

RESEARCH MONOGRAPH . COMPOUND PROFILE

Compound-7p

A synthetic sulfonamidoacetamide identified through phenotypic cell-based screening and structure-activity optimization as a potent inducer of neurite outgrowth in hippocampal, cortical, and retinal primary neurons, with demonstrated in vivo axon regeneration activity in an optic nerve crush injury model.

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ABSTRACT

Compound 7P (CAS 1890208-58-8) is a synthetic sulfonamidoacetamide with the systematic name 2-[(2-methoxyphenyl)[(4-methylphenyl)sulfonyl]amino]-N-(4-methoxy-3-pyridinyl)acetamide and molecular formula C₂₂H₂₃N₃O₅S (molecular weight 441.50 g/mol). The compound was identified at Hanyang University (Republic of Korea) through a phenotypic cell-based screening campaign of chemical libraries followed by iterative structure-activity-guided optimization, culminating in its characterization as the lead compound in a 2016 Journal of Medicinal Chemistry report by Ku, Park, Lee, and colleagues [1]. Compound 7P promotes neurite outgrowth in cultured primary neurons derived from the hippocampus, cerebral cortex, and retina, and in an in vivo rat model of optic nerve crush injury it induces the growth of GAP-43-positive regenerating axons at distances extending beyond 1500 micrometers distal to the crush epicenter, a finding that demonstrates translation of the in vitro neurite outgrowth phenotype into bona fide central nervous system axon regeneration.

The compound was selected as the optimization lead on the basis of three convergent improvements over the parent hit (compound 1): enhanced neurite outgrowth activity in the primary neuron phenotypic assay, improved aqueous solubility attributable to the introduction of a 4-methoxypyridinyl amide pharmacophore replacing a lipophilic aniline, and markedly improved metabolic stability (61.2 percent of parent compound remaining after microsomal incubation, compared to 0.7 percent for the original

hit) driven by reduction of the calculated partition coefficient from 3.76 to 2.43 [1]. These properties rendered compound 7P suitable for the in vivo optic nerve injury study that constitutes the principal translational evidence for the compound class.

The molecular target or targets through which compound 7P stimulates axon regeneration have not been definitively identified. The phenotypic screening approach that generated the compound was target-agnostic, and the published literature does not report a defined receptor, enzyme, or signaling node as the primary binding partner. Contextual evidence from the broader axon regeneration field implicates pathways including the mammalian target of rapamycin (mTOR), the signal transducer and activator of transcription 3 (STAT3), and phosphatase and tensin homolog (PTEN) signaling as determinants of central nervous system axon growth competence, but the specific engagement of these pathways by compound 7P has not been demonstrated in pathway-deconvolution or target-identification studies.

Compound 7P has no clinical development history. No human pharmacokinetic, safety, or efficacy data have been generated. The compound is not approved by any regulatory authority for any indication. It has entered the research-chemical supply chain as a nootropic-marketed powder on the basis of the published preclinical neurite outgrowth and axon regeneration data, but no clinical evidence supports cognitive enhancement, neuroprotection, or any other therapeutic claim in humans. This monograph documents the chemistry, structure-activity optimization, published preclinical pharmacology, the limited mechanistic and pharmacokinetic characterization, sourcing and handling considerations, and a comparative assessment against five alternative axon-regeneration-promoting small molecules on

five competency standards. Investigators should treat compound 7P as an early-stage preclinical research tool with a single peer-reviewed primary publication and no human translational data.

1. Compound Identification

1.1 Names, synonyms, and registry identifiers

Common designation: Compound 7P. The name derives from the internal compound numbering in the Ku et al. (2016) medicinal chemistry optimization campaign, in which series 7 sulfonamidoacetamides bearing a pyridinyl amide were designated with the suffix "p" [1]. The designation has been adopted without modification by the research-chemical supply market.

Chemical Abstracts Service registry number: 1890208-58-8.

PubChem Compound Identifier: CID 127052939.

Reaxys Registry Number: 29568294.

International Union of Pure and Applied Chemistry name: 2-[(2-methoxyphenyl)[(4-methylphenyl)sulfonyl]amino]-N-(4-methoxy-3-pyridinyl)acetamide.

Synonyms in the commercial supply chain: Compound 7P, Compound-7p. No International Nonproprietary Name has been assigned. No development code has been issued by any pharmaceutical sponsor.

1.2 Structural class and chemistry

Compound 7P is a sulfonamidoacetamide. The molecule consists of three principal pharmacophoric elements connected through a central glycinamide backbone: (a) a 4-methylphenylsulfonyl (tosyl) group attached to the backbone nitrogen; (b) a 2-methoxyphenyl substituent on the same backbone nitrogen, rendering it a tertiary sulfonamide nitrogen; and (c) a 4-methoxy-3-pyridinyl group as the amide acceptor. The overall architecture is that of an N,N-disubstituted glycine amide in which the sulfonyl group provides electron-withdrawing character and conformational rigidity, while the two methoxy-bearing aromatic rings provide hydrogen-bond acceptor capacity and moderate lipophilicity.

Molecular formula: C₂₂H₂₃N₃O₅S.

Molecular weight: 441.50 g/mol.

Calculated partition coefficient (cLogP): 2.43 [1]. This value represents a substantial reduction from the parent hit compound 1 (cLogP 3.76) and was a deliberate medicinal chemistry design objective to improve aqueous solubility and metabolic stability.

Predicted density: 1.329 +/- 0.06 g/cm³ at 20 degrees Celsius and 760 Torr [2].

Physical properties: White to off-white crystalline or amorphous powder. Soluble in dimethyl sulfoxide; aqueous solubility improved relative to the parent hit but precise thermodynamic solubility values in water or buffered aqueous media have not been reported in the peer-reviewed literature. The compound is supplied commercially at greater than or equal to 99 percent purity by high-performance liquid chromatography.

Hydrogen bond donors: 1 (amide NH).

Hydrogen bond acceptors: 7 (sulfonyl oxygens, methoxy oxygens, pyridine nitrogen, amide carbonyl oxygen).

Rotatable bonds: Approximately 8, consistent with moderate molecular flexibility.

Stereochemistry: Compound 7P contains no stereocenters. The molecule is achi-
ral and is supplied as a single constitutional isomer.

1.3 Synthesis

The published synthesis of compound 7P proceeds through a convergent route typical of sulfonamidoacetamide medicinal chemistry [1]. The key steps, as described in the Ku et al. (2016) report and inferred from the structural class, are: (a) sulfonylation of 2-methoxyaniline with 4-methylbenzenesulfonyl chloride (tosyl chloride) to generate the N-(2-methoxyphenyl)-4-methylbenzenesulfonamide intermediate; (b) N-alkylation of the sulfonamide nitrogen with ethyl bromoacetate or chloroacetate under basic conditions (typically potassium carbonate in dimethylformamide) to produce the N-alkylated sulfonamidoacetate ester; (c) ester hydrolysis to the corresponding acetic acid; and (d) amide coupling with 3-amino-4-methoxypyridine using a standard peptide coupling reagent (1-ethyl-3-(3-dimethylaminopropyl)carbodiimide with hydroxybenzotriazole, or analogous activation) to generate the final compound 7P. Purification is by silica gel column chromatography and/or recrystallization. The route is amenable to parallel synthesis

for structure-activity relationship exploration, and the Ku et al. campaign evaluated a library of analogues through systematic variation of the sulfonyl arene, the N-aryl substituent, and the amide amine component [1].

Industrial-scale synthesis has not been reported in the peer-reviewed literature. The compound is manufactured by multiple Chinese chemical suppliers (Ningbo Inno Pharmchem, Wuhan Qiami Technology, Suzhou Myland Nutraceuticals, and others) at gram to kilogram scale for the research-chemical market at stated purities of 97 to 99 percent [2, 3].

2. Abstract

Compound 7P (CAS 1890208-58-8; 2-[(2-methoxyphenyl)[(4-methylphenyl)sulfonyl]amino]-N-(4-methoxy-3-pyridinyl)acetamide; C₂₂H₂₃N₃O₅S; molecular weight 441.50 g/mol) is a synthetic sulfonamidoacetamide identified through a phenotypic cell-based screening campaign and iterative medicinal chemistry optimization at Hanyang University, Republic of Korea, as a small-molecule inducer of neurite outgrowth and axon regeneration [1]. The compound represents the terminal optimization product in a series of sulfonamidoacetamide derivatives generated by systematic variation of the sulfonyl, N-aryl, and amide pharmacophores to maximize neurite outgrowth activity in primary hippocampal neurons while simultaneously improving aqueous solubility and metabolic stability relative to the original phenotypic screening hit (compound 1).

The principal published evidence for compound 7P comprises a single peer-reviewed report in the *Journal of Medicinal Chemistry* (Ku et al. 2016) [1]. In that study, compound 7P promoted neurite outgrowth in cultured primary neurons derived from rat hippocampus, cerebral cortex, and retina. The neurite outgrowth activity was confirmed across multiple neuronal subtypes, establishing that the effect was not restricted to a single central nervous system region. In an *in vivo* rat optic nerve crush injury model, intravitreal administration of compound 7P induced statistically significant growth of GAP-43-positive regenerating axons at distances of 500, 1000, and beyond 1500 micrometers distal to the crush epicenter, compared to phosphate-buffered saline controls [1]. GAP-43 (growth-associated protein 43) is a canonical marker of regenerating axons in the mammalian central nervous system, and its presence in fibers extending beyond the lesion site constitutes evidence of bona fide axon regeneration rather than sprouting or local remodeling.

The molecular target of compound 7P has not been identified. The screening campaign employed a phenotypic (target-agnostic) approach, and no target deconvolution, affinity chromatography, photoaffinity labeling, or chemoproteomics study has been published. The precise mechanism of action therefore remains unknown. The broader axon regeneration literature implicates the mTOR, JAK/STAT3, and PTEN/PI3K/Akt signaling axes as principal determinants of neuronal growth

competence in the adult central nervous system, but the engagement of compound 7P with any of these pathways has not been experimentally demonstrated in the published record.

No human data of any kind exist for compound 7P. No pharmacokinetic study in any species has been published beyond the in vitro microsomal stability assay reported in the primary paper (61.2 percent remaining after incubation, compared to 0.7 percent for the parent hit). No toxicology, genotoxicity, reproductive toxicity, or carcinogenicity study has been reported. The compound is not in clinical development and has not been submitted for regulatory review in any jurisdiction. The commercial availability of compound 7P as a "nootropic" research powder is based entirely on the single published preclinical axon regeneration study and on unsubstantiated extrapolation of preclinical neurite outgrowth activity to human cognitive enhancement. Investigators and consumers should interpret the evidence base accordingly.

3. Discovery and Development History

3.1 Origin and screening campaign

Compound 7P was discovered at Hanyang University (Ansan Campus, Republic of Korea) in a research program led by Sung-Hwa Yoon, Jungeun Yang, and Tae-gyu Nam, with the objective of identifying small molecules capable of inducing axon regeneration in injured central nervous system neurons [1]. The research was funded by the Korea Health Technology R&D Project through the Korea Health Industry Development Institute (grant HI12C0003) and the National Research Foundation of Korea (grant NRF-2014R1A2A2A005564).

The screening approach was phenotypic rather than target-based. The investigators conducted a cell-based screen of chemical libraries using primary hippocampal neurons as the biological substrate, with neurite outgrowth (neurite length and branching) as the phenotypic readout. The screen identified a sulfonamidoacetamide hit (designated compound 1 in the published report) with reproducible neurite outgrowth activity but unfavorable drug-like properties: high lipophilicity (cLogP 3.76), poor aqueous solubility, and near-complete metabolic degradation in microsomal stability assays (0.7 percent remaining) [1].

3.2 Structure-activity optimization

The medicinal chemistry optimization campaign systematically modified three regions of the hit molecule: the sulfonyl arene (varied between phenyl, tolyl, and substituted phenyl), the N-aryl substituent (varied between substituted anilines), and the amide amine (varied between anilines, benzylamines, and heteroaromatic amines). The optimization was guided by the primary neurite outgrowth phenotypic assay in hippocampal neurons, with secondary evaluation of aqueous solubility (by nephelometry or kinetic solubility assessment) and metabolic stability (by liver microsomal incubation with quantification of remaining parent compound by liquid chromatography-mass spectrometry) [1].

The critical optimization step was replacement of the lipophilic aniline amide in the parent hit with a 4-methoxy-3-pyridinyl amide. This single modification accomplished three objectives simultaneously: it preserved neurite outgrowth activity

(the pyridinyl amide maintained the hydrogen bonding capacity required for biological activity), it reduced lipophilicity (cLogP decreased from 3.76 to 2.43), and it improved metabolic stability from 0.7 percent to 61.2 percent remaining parent compound after microsomal incubation [1]. The improvement in metabolic stability was attributed to the reduced CYP-mediated oxidative metabolism associated with the lower lipophilicity.

Compound 7P was designated the lead compound from this optimization on the basis of the convergent improvement in the three parameters (neurite outgrowth, solubility, metabolic stability) and was advanced to in vivo evaluation.

3.3 In vivo validation

The in vivo study employed a rat optic nerve crush injury model, a standard paradigm for evaluation of central nervous system axon regeneration [1]. Optic nerve crush produces a defined, reproducible axotomy of retinal ganglion cell axons at the crush site; regeneration is assessed by quantification of GAP-43-immunopositive axons extending distal to the crush epicenter at defined distances. Compound 7P was administered by intravitreal injection (the route of delivery was directly into the vitreous humor of the eye, providing access to retinal ganglion cells without systemic pharmacokinetic confounds). GAP-43-positive axons in the optic nerves of compound-7P-treated animals extended significantly beyond the crush epicenter compared to vehicle-treated controls, with differences evident at 500, 1000, and 1500 micrometers distal to the crush center and reaching statistical significance beyond 1500 micrometers [1].

3.4 Post-publication trajectory

Following publication of the Ku et al. (2016) report, compound 7P entered the research-chemical supply chain. Multiple chemical suppliers, predominantly based in China, began offering the compound as a powder at gram scale with stated purities of 97 to 99 percent [2, 3]. The compound was adopted by the nootropic research-chemical market as a purported cognitive enhancement agent, a positioning based on extrapolation from the neurite outgrowth and axon regeneration data to cognitive function. No peer-reviewed follow-up study, replication study, target identification study, or in vivo pharmacokinetic study has been published as of the monograph revision date. The compound has not entered formal preclinical develop-

ment or investigational new drug-enabling studies under any pharmaceutical sponsor. The evidence base therefore remains limited to a single primary peer-reviewed publication.

4. Mechanism of Action

4.1 Target-agnostic discovery and unknown molecular target

Compound 7P was identified through a phenotypic screening campaign in which the biological readout was neurite outgrowth in primary hippocampal neurons [1]. The screening approach did not presuppose or identify a molecular target. No target deconvolution study (affinity chromatography, photoaffinity labeling, thermal proteome profiling, chemoproteomics, or CRISPR-based genetic screening) has been reported for compound 7P or for any member of the sulfonamidoacetamide series. The molecular target or targets through which the compound promotes neurite extension and axon regeneration are therefore unknown as of the monograph revision date.

4.2 Contextual pathway biology

The broader literature on central nervous system axon regeneration has identified several intracellular signaling pathways that govern the growth competence of injured adult neurons. These pathways provide a contextual framework within which the mechanism of compound 7P might operate, though direct evidence of engagement is lacking.

mTOR signaling: The mammalian target of rapamycin complex 1 (mTORC1) is a principal intracellular integrator of growth signals in neurons. Genetic deletion of PTEN (the principal negative regulator of the PI3K/Akt/mTOR axis) in retinal ganglion cells produces robust optic nerve axon regeneration in mouse models [4]. Pharmacological activation of mTOR by PTEN inhibition or by direct mTOR agonism is a leading strategy in the axon regeneration field. Whether compound 7P activates the mTOR pathway directly or indirectly has not been tested.

JAK/STAT3 signaling: The signal transducer and activator of transcription 3 (STAT3) pathway mediates cytokine-driven axon regeneration in retinal ganglion cells and dorsal root ganglion neurons [5]. Activation of STAT3 through the JAK/STAT axis promotes the expression of regeneration-associated genes (RAGs) including GAP-43, ATF3, SPRR1A, and others. Compound 7P-treated axons express

GAP-43 at the protein level in the optic nerve injury model, consistent with activation of a RAG expression program, but the upstream signaling events have not been characterized.

Epigenetic regulation: Axon regeneration in the central nervous system involves epigenetic reprogramming of the chromatin landscape to permit expression of regeneration-associated gene programs that are silenced in the quiescent adult neuron. Histone acetyltransferases, histone deacetylases, and DNA methyltransferases have all been implicated. Whether compound 7P acts through epigenetic mechanisms has not been investigated.

Cytoskeletal dynamics: Neurite outgrowth requires coordinated regulation of the actin and microtubule cytoskeleton at the growth cone. RhoA/ROCK signaling inhibits growth cone advance; inhibition of this pathway promotes neurite outgrowth and axon regeneration. The sulfonamide functional group in compound 7P is present in several known kinase and enzyme inhibitors, but no evidence links compound 7P to RhoA/ROCK or to other cytoskeletal regulatory targets.

4.3 Phenotypic profile

The empirically characterized phenotypic profile of compound 7P is as follows:

Promotion of neurite outgrowth in primary hippocampal neurons: compound 7P increased both neurite length and neurite branching in cultures of primary rat hippocampal neurons in a concentration-dependent manner [1].

Promotion of neurite outgrowth in cortical neurons: the effect was replicated in primary cultures of rat cortical neurons, establishing that the activity was not hippocampus-specific [1].

Promotion of neurite outgrowth in retinal neurons: the effect was replicated in primary cultures of retinal neurons, the cell type relevant to the optic nerve crush model [1].

Induction of GAP-43-positive axon regeneration in vivo: in the rat optic nerve crush model, compound 7P induced regenerating axons expressing the growth-associated marker GAP-43 at distances extending beyond the lesion epicenter [1].

The phenotypic profile is consistent with activation of an intrinsic neuronal growth program (rather than, for example, removal of an extrinsic inhibitory signal), but this inference has not been validated by gain-of-function or loss-of-function studies on defined targets.

4.4 Selectivity

No selectivity profiling against panels of receptors, ion channels, kinases, or enzymes has been published for compound 7P. The compound's activity profile against off-target pharmacology is unknown.

5. Pharmacokinetics and Routes of Administration

5.1 In vitro metabolic stability

The sole published pharmacokinetic datum for compound 7P is the in vitro microsomal stability assay reported in the Ku et al. (2016) optimization campaign [1]. After incubation with liver microsomes under standard conditions, 61.2 percent of compound 7P remained as intact parent compound. This represents a marked improvement over the parent hit compound 1 (0.7 percent remaining), attributable to the reduction in lipophilicity (cLogP from 3.76 to 2.43) achieved by introduction of the 4-methoxypyridinyl amide. The species of liver microsomes (rat, mouse, or human), the incubation duration, the microsomal protein concentration, and the co-factor conditions were not specified in the accessible abstract and supplementary materials; standard medicinal chemistry practice at the time would suggest rat liver microsomes at 0.5 mg/mL protein with NADPH regenerating system for 30 to 60 minutes.

5.2 Absorption

No oral bioavailability study has been published for compound 7P in any species. The calculated partition coefficient of 2.43, the molecular weight of 441.50 (below the Lipinski Rule of Five threshold of 500), the single hydrogen bond donor, and the seven hydrogen bond acceptors (below the threshold of 10) are consistent with oral drug-likeness by the Lipinski criteria. However, compliance with Lipinski parameters does not guarantee oral absorption, and the actual oral bioavailability remains unknown.

5.3 Distribution

No distribution study has been published. The predicted density is 1.329 g/cm³ [2]. Volume of distribution, plasma protein binding, blood-brain barrier penetration, and tissue partitioning are uncharacterized. The in vivo optic nerve study used intravitreal injection, bypassing systemic distribution entirely.

5.4 Metabolism

Beyond the single microsomal stability data point, no metabolic identification study has been published. The likely metabolic liabilities, inferred from the molecular structure, include: (a) O-demethylation of the 2-methoxyphenyl and/or 4-methoxypyridinyl groups by CYP enzymes (CYP1A2, CYP2C19, CYP3A4 are common mediators of aromatic O-demethylation); (b) oxidation of the tolyl methyl group to a benzylic alcohol or aldehyde; (c) amide bond hydrolysis by carboxylesterases or amidases; and (d) sulfonamide N-dealkylation. None of these metabolic pathways has been experimentally characterized.

5.5 Elimination

No elimination study has been published. Renal and biliary excretion pathways are uncharacterized.

5.6 Routes of administration

In vivo research use (published): Intravitreal injection. The optic nerve crush study used direct injection into the vitreous humor, delivering compound 7P to retinal ganglion cells without systemic exposure [1]. The dose, volume, and vehicle for the intravitreal injection were reported in the primary publication.

In vitro research use: Compound 7P is dissolved in dimethyl sulfoxide for cell culture applications. Typical stock concentrations for phenotypic neurite outgrowth assays are in the millimolar range, with working concentrations in the low-micromolar to mid-micromolar range applied to neuronal cultures. The specific EC₅₀ for neurite outgrowth promotion has not been reported as a discrete value in the accessible literature.

Commercial research-chemical use: The compound is supplied as a powder and is dissolved by the end user in dimethyl sulfoxide or in aqueous vehicles. No standardized reconstitution protocol has been published. No oral, subcutaneous, intraperitoneal, or intravenous dosing study has been reported in the peer-reviewed literature.

5.7 Special populations

No data exist for any special population. No pediatric, geriatric, hepatic impairment, renal impairment, or pregnancy and lactation data have been generated.

6. Preclinical Pharmacology

6.1 In vitro neurite outgrowth

The primary pharmacological characterization of compound 7P was performed in vitro using primary neuronal cultures from postnatal rats [1]. Three neuronal subtypes were evaluated:

Hippocampal neurons: Primary hippocampal neurons were the principal screening substrate throughout the optimization campaign. Compound 7P promoted neurite outgrowth (increased total neurite length and number of branch points per neuron) in a concentration-dependent manner. The hippocampal neurite outgrowth assay was the basis on which the structure-activity relationships were constructed and on which compound 7P was selected as the lead.

Cortical neurons: Primary cortical neurons showed neurite outgrowth promotion qualitatively similar to hippocampal neurons, establishing that the activity was not restricted to a single hippocampal neuronal subtype.

Retinal neurons: Primary retinal neurons, including retinal ganglion cells (the projection neurons whose axons form the optic nerve), showed neurite outgrowth promotion by compound 7P. This finding was the basis for advancing the compound to the optic nerve crush in vivo model, as the relevant neuronal population (retinal ganglion cells) was responsive to the compound in vitro.

The neurite outgrowth assay was performed using standard immunocytochemical methods with antibodies against neuronal markers (beta-III-tubulin or MAP2) and quantified by automated or semi-automated image analysis. Specific EC₅₀ values, maximum efficacy relative to positive controls, and dose-response curve parameters have not been reported as discrete numerical values in the accessible literature.

6.2 In vivo optic nerve crush model

The in vivo study employed the rat optic nerve crush injury model [1]. In this paradigm, the optic nerve is surgically exposed and crushed with calibrated forceps at a defined distance from the globe, producing a complete axotomy of retinal ganglion

cell axons at the crush site. The animals were allowed to survive for a defined post-injury interval (typically 14 to 28 days), during which compound 7P or vehicle (phosphate-buffered saline) was administered by intravitreal injection.

Axon regeneration was quantified by immunohistochemistry for GAP-43 (growth-associated protein 43) in longitudinal sections of the optic nerve. GAP-43 is expressed at high levels in regenerating axons and at low levels in quiescent or degenerating axons; its presence in fibers extending distal to the crush site is accepted as evidence of axon regeneration rather than artifact or sprouting.

The results demonstrated that optic nerves from compound-7P-treated animals contained GAP-43-positive axons extending beyond the crush epicenter at distances of 500, 1000, and greater than 1500 micrometers, while vehicle-treated control animals showed minimal or no GAP-43-positive axons beyond the crush site [1]. The difference was statistically significant at the most distal measurement points. The number of regenerating axons and the maximum regeneration distance were both increased by compound 7P treatment.

This result constitutes the principal translational evidence for the compound class and demonstrates that the *in vitro* neurite outgrowth activity of compound 7P translates to *in vivo* axon regeneration in an established central nervous system injury model.

6.3 Limitations of the preclinical evidence

Several limitations of the preclinical evidence base should be noted:

The *in vivo* study has not been independently replicated. The sole published *in vivo* experiment comes from the original discovery laboratory. Independent replication by other research groups would substantially strengthen the evidence.

The optic nerve crush model, while a standard paradigm, evaluates a specific central nervous system tract (retinal ganglion cell axons) under defined injury conditions. Generalization to other central nervous system regions (spinal cord, corticospinal tract, peripheral nerve) or to other injury types (transection, contusion, chronic degeneration) has not been tested.

Functional recovery was not assessed. The published study evaluated axon regeneration by histological (GAP-43 immunohistochemistry) rather than functional criteria. Whether the regenerating axons form functional synapses, restore visual

function, or produce any behavioral improvement was not reported.

The compound was delivered by intravitreal injection, bypassing systemic pharmacokinetics. The feasibility of systemic (oral or parenteral) administration for the treatment of central nervous system injury or degeneration has not been evaluated.

Dose-response characterization in vivo was limited. A comprehensive dose-response study with multiple doses and time points has not been reported.

7. Clinical Evidence Base

No clinical evidence exists for compound 7P. The compound has not been administered to humans in any context: no Phase 1 safety and pharmacokinetic study, no Phase 2 efficacy study, no compassionate use, and no investigator-initiated trial has been reported or registered. The compound is not listed in any clinical trial registry (ClinicalTrials.gov, EU Clinical Trials Register, WHO International Clinical Trials Registry Platform) as of the monograph revision date.

The absence of clinical evidence is the principal limitation of compound 7P as a translational research tool. The gap between preclinical neurite outgrowth and axon regeneration data and any human therapeutic application (cognitive enhancement, neuroprotection, nerve injury repair) is substantial and encompasses unanswered questions in pharmacokinetics (oral bioavailability, brain penetration, metabolic stability in human hepatocytes), safety (acute and chronic toxicity, genotoxicity, reproductive toxicity, cardiovascular safety), and efficacy (whether promotion of neurite outgrowth in rat primary neurons predicts any measurable benefit in human neurological function).

The commercial positioning of compound 7P as a nootropic research chemical implies cognitive enhancement properties that are not supported by any published evidence. No cognitive or behavioral testing has been reported in any animal model beyond the optic nerve regeneration histological endpoint. The extrapolation from neurite outgrowth promotion to cognitive enhancement is speculative and should be interpreted with corresponding caution.

8. Sourcing and Quality Verification

Compound 7P is not available as a pharmaceutical-grade preparation. No pharmacopoeial monograph exists. The compound is supplied exclusively as a research-grade chemical by commercial chemical suppliers.

Primary suppliers: Multiple Chinese manufacturers and international research-chemical distributors supply compound 7P at gram to kilogram scale. Identified suppliers include Ningbo Inno Pharmchem (NBINNO), Wuhan Qiami Technology, Suzhou Myland Nutraceuticals, Umbrella Labs, PureRawz, BC9, ModernAminos, and others [2, 3]. Pricing at the time of monograph preparation ranges from approximately \$50 to \$150 per gram at the retail level and substantially lower at bulk scale.

Stated purity: Suppliers typically claim greater than or equal to 97 percent or greater than or equal to 99 percent purity by high-performance liquid chromatography. Certificates of analysis are provided by vendors but should be verified independently.

Quality verification: Investigators should confirm compound identity and purity on every research-grade lot through the following analytical methods:

- High-performance liquid chromatography (HPLC) with UV detection at 254 nm or 220 nm, confirming area-percent purity of greater than or equal to 98 percent
- Mass spectrometry confirmation of the protonated molecular ion $[M+H]^+$ at m/z 442.14 (expected for $C_{22}H_{23}N_3O_5S + H$)
- Proton nuclear magnetic resonance spectroscopy (1H NMR) in deuterated dimethyl sulfoxide or deuterated chloroform, confirming the expected resonance pattern: aromatic protons for the 4-methylphenylsulfonyl, 2-methoxyphenyl, and 4-methoxypyridinyl rings; methyl singlets for the tosyl methyl and the two methoxy groups; and the glycine methylene singlet
- Residual solvent analysis by gas chromatography (common synthesis solvents: dimethylformamide, dichloromethane, ethyl acetate, methanol)
- Karl Fischer titration or thermogravimetric analysis for residual water content

Third-party analytical laboratories (Janoshik Analytical, Anabolic Lab, or equivalent) provide independent verification of vendor certificates of analysis. Given that compound 7P is marketed to a nootropic consumer base with no regulatory oversight, independent analytical confirmation is particularly important for research applications where quantitative dose-response precision is required.

9. Reconstitution and Handling

Compound 7P is supplied as a white to off-white powder. The following reconstitution and handling guidelines are based on the general properties of sulfonamidoacetamides and on the limited published data for this specific compound.

Stock solution preparation: For in vitro research applications, compound 7P is dissolved in anhydrous dimethyl sulfoxide (DMSO) at concentrations of 10 to 100 mM. The compound is expected to be freely soluble in DMSO at these concentrations based on its moderate lipophilicity (cLogP 2.43) and absence of ionizable acidic protons at physiological pH. Stock solutions should be aliquoted into single-use volumes to avoid repeated freeze-thaw cycles.

Working solution preparation: Stock solutions in DMSO are diluted into cell culture medium or physiological buffer immediately before use. The final DMSO concentration in the culture medium should not exceed 0.1 to 0.5 percent (v/v) to avoid solvent-mediated cytotoxicity. Vehicle controls containing equivalent DMSO concentrations must be included in all experiments.

Aqueous solubility: The compound was designed with improved aqueous solubility relative to the parent hit, but precise thermodynamic solubility values in water, phosphate-buffered saline, or cell culture medium have not been published. Investigators should assess solubility empirically in the specific vehicle and at the specific concentration required for their application. Turbidity or precipitation at high concentrations may indicate solubility limits.

Storage: The solid compound should be stored at -20 degrees Celsius in a desiccated, light-protected environment. DMSO stock solutions should be stored at -20 degrees Celsius in tightly sealed vials. Aqueous dilutions should be prepared fresh for each experiment and not stored.

Stability: No formal stability study (accelerated or long-term) has been published. The sulfonamide bond and the amide bond are generally stable functional groups under ambient storage conditions. The methoxy groups are stable at neutral pH. Hydrolytic degradation of the amide bond may occur under strongly acidic or strongly basic conditions or at elevated temperatures.

Handling precautions: Compound 7P is classified as a research chemical not intended for human consumption. Standard laboratory personal protective equipment (gloves, laboratory coat, eye protection) should be used. No material safety data sheet with comprehensive toxicological data has been published by any supplier; handling should follow the precautionary approach used for all uncharacterized bioactive small molecules.

10. Stack Interactions and Combinations

No drug-drug interaction, drug-supplement interaction, or combination pharmacology study has been published for compound 7P. The following considerations are inferred from the molecular structure and from the compound class, and should be treated as theoretical rather than empirically validated.

CYP-mediated interactions: Compound 7P contains two methoxy groups that are likely substrates for CYP-mediated O-demethylation (primarily CYP1A2, CYP2C19, CYP3A4). Concurrent administration of strong CYP3A4 inhibitors (ketoconazole, itraconazole, ritonavir, clarithromycin) or strong CYP1A2 inhibitors (fluvoxamine, ciprofloxacin) could theoretically increase compound 7P exposure by reducing metabolic clearance. Conversely, CYP inducers (rifampin, carbamazepine, phenytoin, St. John's wort) could reduce exposure. These interactions have not been characterized experimentally.

Other neurite-outgrowth-promoting agents: Combination with other compounds that promote neurite outgrowth (nerve growth factor, brain-derived neurotrophic factor, cnicin, parthenolide, glycopyrrolate, mexiletine) could theoretically produce additive or synergistic effects on neurite extension. No combination study has been published for compound 7P with any of these agents.

mTOR pathway modulators: If compound 7P acts through or in parallel with the mTOR pathway, concurrent administration with mTOR inhibitors (rapamycin, everolimus, temsirolimus) could attenuate the neurite outgrowth effect, while concurrent PTEN inhibition could produce additive effects. These interactions are speculative.

Cholinergic agents: Compound 7P has no characterized cholinergic activity. The nootropic market context places it alongside acetylcholinesterase inhibitors, racetams, and other cholinergic cognitive enhancers, but no pharmacological interaction data exist.

General considerations: Investigators combining compound 7P with other research compounds in cell culture or in animal models should consider potential cytotoxicity from combined exposure, potential chemical incompatibility in stock solutions, and the absence of any published interaction data. Single-agent characterization should precede combination experiments.

11. Adverse Events and Safety Signal

11.1 In vitro toxicity

No formal cytotoxicity profiling has been published for compound 7P. In the primary neurite outgrowth assay, the compound was used at concentrations that promoted neurite extension without reported neuronal cell death, implying that the active concentration range is below the cytotoxic threshold in primary neurons under the specific culture conditions used [1]. However, a comprehensive cytotoxicity assessment (MTT, LDH release, or live/dead staining across a wide concentration range) has not been reported.

11.2 In vivo toxicity

No formal toxicology study has been published. The optic nerve crush study used intravitreal injection, which limits systemic exposure and provides minimal information about systemic toxicity. No acute toxicity (LD50), subacute toxicity, subchronic toxicity, chronic toxicity, genotoxicity, reproductive toxicity, or carcinogenicity study has been reported for compound 7P.

11.3 Predicted toxicological liabilities

Predictions based on molecular structure and compound class:

Sulfonamide hypersensitivity: Sulfonamide-containing compounds are associated with hypersensitivity reactions in a subset of the population. The 4-methylphenylsulfonyl group in compound 7P is an arylsulfonamide, the structural class most commonly associated with sulfonamide allergy (in contrast to non-arylamine sulfonamides, which have a lower incidence). Whether compound 7P would provoke sulfonamide hypersensitivity in susceptible individuals is unknown.

Hepatotoxicity: Sulfonamides and pyridine-containing compounds have been associated with hepatotoxicity in some cases, though these associations are compound-specific rather than class-wide. No hepatotoxicity data exist for compound 7P.

Cardiac safety: No hERG channel inhibition, QT prolongation, or cardiovascular safety data have been generated.

11.4 Safety in the nootropic market context

Compound 7P is commercially available as a research-chemical powder marketed to nootropic consumers. No human safety data of any kind exist. The marketed product typically carries a disclaimer stating "not for human consumption" or "for laboratory research use only." Individuals who consume the compound do so without the benefit of safety pharmacology, toxicology, or clinical safety data. The risk-benefit assessment for human use cannot be performed because neither the risks nor the benefits have been characterized in humans.

12. Comparative Assessment

This section presents a structured comparative assessment of five alternative small molecules with demonstrated axon-regeneration-promoting or neurite-outgrowth-promoting activity against compound 7P, evaluated on five competency standards: novelty, effect size, promising potential (current development state and trajectory), side-effect profile, and overall validation. The five comparators were selected as representative of the principal mechanistic and developmental approaches in the small-molecule axon regeneration field.

12.1 Parthenolide

Mechanism: Parthenolide is a sesquiterpene lactone derived from the feverfew plant (*Tanacetum parthenium*) that promotes axonal growth through multiple mechanisms, including inhibition of NF-kappaB signaling, activation of the PI3K/Akt pathway, and microtubule stabilization [6]. The compound promotes axonal growth in cultured dorsal root ganglion neurons from adult rats and mice, in primary retinal ganglion cells from rodents and humans, and in vivo in sciatic nerve injury models at a daily intravenous dose of 2 micrograms per kilogram [6].

Novelty: Moderate. Parthenolide is a well-characterized natural product with extensive pharmacological literature spanning anti-inflammatory, anticancer, and now neuroregenerative applications. The axon regeneration application represents a novel extension of known pharmacology.

Effect size: Substantial in preclinical models. Daily intravenous injections of 2 micrograms per kilogram markedly accelerated motor and sensory function recovery following sciatic nerve injury in rats. Notably, functional recovery (behavioral endpoints) was demonstrated, which compound 7P has not shown [6].

Promising potential: Moderate. The compound is a well-characterized natural product with established pharmacology, but its oral bioavailability is low, restricting clinical administration to parenteral routes. This pharmacokinetic limitation constrains translational potential for chronic neurodegenerative indications.

Side-effect profile: Parthenolide contains an alpha-methylene-gamma-butyrolactone and an epoxide, both potentially mutagenic and allergenic functional groups. Gastrointestinal irritation and contact dermatitis are reported adverse effects of feverfew-derived preparations.

Overall validation: Moderate to high. Multiple independent laboratories have characterized parthenolide in axon regeneration models. Functional recovery has been demonstrated. The evidence base is substantially deeper than that of compound 7P.

Comparison to compound 7P: Parthenolide has a larger preclinical evidence base, multiple characterized mechanisms of action, and demonstrated functional recovery, all of which compound 7P lacks. Compound 7P has the advantage of rational medicinal chemistry optimization for drug-like properties (improved metabolic stability and lipophilicity), while parthenolide has intrinsic pharmacokinetic limitations. Neither compound has human clinical data for neuroregenerative indications.

12.2 Cnicin

Mechanism: Cnicin is a sesquiterpene lactone from blessed thistle (*Cnicus benedictus*) that promotes axon regeneration through mechanisms overlapping with but distinct from parthenolide [7]. The compound promotes neurite outgrowth and functional nerve repair in sciatic nerve, facial nerve, and optic nerve injury models.

Novelty: Moderate to high. Cnicin represents a structurally distinct sesquiterpene lactone with improved pharmacokinetic properties relative to parthenolide; its application to nerve regeneration is recent (2022 and subsequent publications).

Effect size: Substantial. Efficacy demonstrated in rats, mice, and rabbits at 2 micrograms per kilogram daily dose. Functional recovery (whisker movement in facial nerve injury, visual function in optic nerve injury, motor function in sciatic nerve injury) has been reported [7].

Promising potential: High among the preclinical comparators. Cnicin exhibits 84.7 percent oral bioavailability in rats and lacks the potentially mutagenic epoxide group present in parthenolide, rendering it a more developable clinical candidate [7]. Intravenous administration at 4 mg/kg (2000-fold higher than the effective oral dose) for 2 weeks did not produce observable toxicity.

Side-effect profile: Favorable in preclinical testing. The absence of the epoxide functional group present in parthenolide reduces the theoretical mutagenicity and allergenicity risk.

Overall validation: Moderate. The evidence base is recent and growing. Multiple species and multiple nerve injury paradigms have been tested. Functional recovery and safety margins have been characterized.

Comparison to compound 7P: Cnicin has a substantially stronger evidence base, including oral bioavailability data, safety margin characterization, multiple species, multiple injury paradigms, and functional recovery endpoints. Compound 7P has the advantage of a defined synthetic route amenable to medicinal chemistry optimization, while cnicin is a natural product with more complex supply chain considerations. Cnicin is the more advanced preclinical candidate overall.

12.3 Glycopyrrolate

Mechanism: Glycopyrrolate is an FDA-approved muscarinic receptor antagonist identified through computational drug repurposing as a promoter of neurite outgrowth and axon regeneration [8]. The mechanism is hypothesized to involve muscarinic receptor antagonism on Schwann cells or direct effects on neuronal growth signaling.

Novelty: High in context. Glycopyrrolate is a well-established anticholinergic drug repurposed for an entirely new indication (nerve regeneration), representing a novel therapeutic application of a known compound.

Effect size: In vivo, the most distal axonal regrowth in mice treated with glycopyrrolate was increased by 61 percent, and the number of regenerating axons was increased by 73 percent, compared to controls [8].

Promising potential: High. The compound is already FDA-approved for other indications (reduction of chronic drooling, treatment of peptic ulcer, reduction of secretions perioperatively), providing an established human safety database, known pharmacokinetics, and a clear regulatory pathway for repurposing.

Side-effect profile: Well characterized from decades of clinical use. Principal adverse events are anticholinergic: dry mouth, constipation, urinary retention, tachycardia, and blurred vision. These are dose-dependent and manageable.

Overall validation: Moderate. The axon regeneration data are from a single research group and have not been independently replicated. However, the compound has extensive human safety data from its primary indications.

Comparison to compound 7P: Glycopyrrolate has the substantial advantage of FDA approval, known human pharmacokinetics and safety, and a clear regulatory pathway for clinical testing in nerve regeneration. Compound 7P has no human data of any kind. The axon regeneration effect sizes are in a comparable range in preclinical models.

12.4 Mexiletine

Mechanism: Mexiletine is an FDA-approved sodium channel blocker (class IB antiarrhythmic) identified alongside glycopyrrolate through the same computational drug repurposing screen as a promoter of neurite outgrowth [8]. The mechanism in the context of nerve regeneration may involve sodium channel modulation affecting growth cone dynamics or neuronal excitability-growth coupling.

Novelty: High in context. Like glycopyrrolate, mexiletine represents a drug repurposing approach.

Effect size: The most distal axonal regrowth in mice treated with mexiletine was increased by 59 percent, and the number of regenerating axons was increased by 67 percent, compared to controls [8].

Promising potential: High. FDA-approved with established human safety data. Used clinically for ventricular arrhythmias and for neuropathic pain (particularly in erythromelalgia and sodium channelopathies).

Side-effect profile: Well characterized. Principal adverse events are gastrointestinal (nausea, vomiting), neurological (tremor, dizziness, paresthesia), and cardiac (proarrhythmic risk at high doses).

Overall validation: Moderate. Single-group preclinical data for the nerve regeneration application, but extensive clinical experience for other indications.

Comparison to compound 7P: Similar to glycopyrrolate, mexiletine has the substantial advantage of FDA approval and known human safety. Compound 7P has neither, and the preclinical nerve regeneration effect sizes are comparable.

12.5 RLA-47

Mechanism: RLA-47 is a small-molecule inhibitor of the RhoA/ROCK signaling pathway, which is a principal intracellular brake on axon growth cone advance in the inhibitory environment of the injured central nervous system [9]. By inhibiting RhoA/ROCK signaling, the compound releases the cytoskeletal brake and permits growth cone extension through normally non-permissive substrates such as myelin-associated inhibitors and chondroitin sulfate proteoglycans.

Novelty: Moderate. RhoA/ROCK inhibition is a well-established strategy in the axon regeneration field. Multiple RhoA/ROCK inhibitors (fasudil, Y-27632, ibuprofen at high doses) have been characterized in preclinical models. RLA-47 represents a next-generation compound with improved potency and selectivity within this mechanism.

Effect size: RLA-47 promoted substantial axon regeneration and functional crossing in a spinal cord injury model, extending the mechanism beyond the optic nerve injury paradigm [9].

Promising potential: Moderate. The compound is in preclinical development. The RhoA/ROCK mechanism is well validated in the field and has attracted pharmaceutical interest (the Rho-kinase inhibitor fasudil is approved in Japan for cerebral vasospasm).

Side-effect profile: Limited preclinical data. RhoA/ROCK inhibitors as a class produce vasodilation, hypotension, and smooth muscle relaxation as pharmacological effects.

Overall validation: Moderate. The mechanism is well validated by multiple independent groups. RLA-47 itself has a smaller evidence base.

Comparison to compound 7P: RLA-47 has a defined molecular target (RhoA/ROCK), which compound 7P does not have. The defined target enables mechanism-based optimization, combination design, and biomarker development. Compound 7P has no characterized target. Both compounds are in early preclinical stages.

12.6 Comparator summary

The five comparator compounds span three principal approaches to small-molecule promotion of axon regeneration: natural-product-derived growth promoters (parthenolide, cnicin), repurposed FDA-approved drugs identified by computational screening (glycopyrrolate, mexiletine), and mechanism-directed synthetic compounds (RLA-47).

Compound 7P occupies a distinct position as a synthetic compound identified by phenotypic (target-agnostic) screening with rational medicinal chemistry optimization for drug-like properties. Its principal strength is the demonstrated translation from in vitro neurite outgrowth to in vivo optic nerve axon regeneration, established in a single, well-executed preclinical study. Its principal weaknesses are the unknown molecular target, the absence of functional recovery data, the absence of any in vivo pharmacokinetic characterization, the absence of any safety or toxicology data, and the reliance on a single published peer-reviewed study.

Among the comparators, **glycopyrrolate** and **mexiletine** hold the strongest translational positions owing to their FDA approval, established human safety databases, and known pharmacokinetics. **Cnicin** holds the strongest preclinical position among the non-approved compounds owing to its oral bioavailability, multi-species multi-paradigm efficacy data, functional recovery endpoints, and characterized safety margins. **Parthenolide** has a deeper mechanistic and preclinical literature but pharmacokinetic limitations. **RLA-47** has the advantage of a defined molecular target within a validated pathway.

Compound 7P's position in this landscape is that of an early-stage preclinical research tool. The compound may have value for investigators seeking a synthetic, drug-like small molecule with demonstrated neurite outgrowth and optic nerve regeneration activity in a well-defined chemical series amenable to further optimization. It does not, at present, have the evidence base to support clinical translation or human use claims.

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This monograph is provided for research use only. Not for medical, diagnostic, or therapeutic purposes. Not for human consumption. Compound 7P is not approved by any regulatory authority for any indication. The compound has a single peer-reviewed primary publication and no human safety, pharmacokinetic, or efficacy data. The evidence base does not support therapeutic claims of any kind.

Investigators should obtain independent analytical confirmation of identity and purity on every lot. The nootropic market positioning of this compound is not supported by clinical evidence.