



mechanistic comparison vs. other NAD boosters

P7C3-A20 boosts NAD in a more targeted, “on-demand” way than typical NAD boosters like NR, NMN, niacin, or CD38 inhibitors, because it directly stimulates NAMPT and mainly restores NAD when it is pathologically low, rather than driving chronic supraphysiologic elevations.^{[1] [2]}

What P7C3-A20 Does

- P7C3-A20 binds and activates nicotinamide phosphoribosyltransferase (NAMPT), the rate-limiting enzyme of the nicotinamide salvage pathway that converts nicotinamide to NMN and then to NAD.^{[3] [1]}
- In models of NAD depletion, P7C3-A20 increases neuronal NAD back toward baseline and protects against cell death, but shows little effect on NAD when levels are already normal, acting like an **allosteric** enhancer that “kicks in” under stress.^{[2] [4]}
- This NAD restoration supports neuronal survival and function across diverse neurodegeneration and injury models, likely via preserved mitochondrial metabolism and downstream sirtuin signaling.^{[5] [6]}

How Other NAD Boosters Work

- Niacin/nicotinamide (NAM): Provide vitamin B3 that enters the same salvage pathway but increase NAD by mass action, not by directly modulating NAMPT activity; high chronic doses can push NAD broadly in many tissues and cause side effects like flushing or liver strain.^{[7] [2]}
- NR (nicotinamide riboside) and NMN: Serve as precursors upstream of NAD; oral dosing reliably raises circulating NAD ~1.3–1.5× in humans, but does so continuously and systemically rather than being preferentially engaged only in NAD-depleted cells.^{[8] [9]}
- CD38 inhibitors / NR dual action: CD38 breaks down NAD; inhibiting it reduces NAD consumption, while NR may both act as a precursor and covalent CD38 inhibitor, thereby increasing NAD by lowering its degradation.^{[10] [11]}

Mechanistic Comparison Table

Feature	P7C3-A20	NR / NMN / Niacin / CD38 inhibitors	
Primary target	Direct NAMPT activation	Precursor supply or NADase inhibition	
Pathway	NAD salvage, rate-limiting step	NAD biosynthesis (precursors) or breakdown	

Feature	P7C3-A20	NR / NMN / Niacin / CD38 inhibitors	
Triggering condition	Preferentially active in NAD-depleted cells	Active regardless of baseline NAD status	
Magnitude of NAD change	Restores to physiological range	Often drives sustained elevation	
Tissue emphasis	Neurons, neuroprotection models	Systemic (liver, muscle, blood, etc.)	
Risk of supraphysiologic NAD	Designed to avoid overproduction	Possible at high or chronic dosing	
Evidence base	Strong in animal neuro models, no humans yet	Human NAD raising shown; clinical outcome mixed	[1] [2] [9] [6] [12]

Practical Implications

- Mechanistically, P7C3-A20 behaves more like a **condition-dependent NAMPT agonist**, whereas standard boosters are condition-independent “substrate loading” or “brake-removal” strategies. [\[1\]](#) [\[2\]](#)
- This may translate to a better therapeutic window in neurodegeneration by rescuing stressed neurons without chronically pushing NAD in otherwise healthy tissues, but this remains speculative until human data exist. [\[6\]](#) [\[5\]](#)
- Conversely, currently available boosters (NR, NMN, niacin, etc.) are far more studied in humans, raise NAD reliably but show modest and sometimes conflicting functional benefits, and their broad, non-targeted action can include trade-offs like altered inflammatory or cardiovascular risk in some settings. [\[9\]](#) [\[13\]](#) [\[12\]](#)



`<q>P7C3-A20</q>` cas number

The CAS number for P7C3-A20 is **1235481-90-9**. [\[22\]](#) [\[23\]](#) [\[24\]](#)



chemical structure

P7C3-A20 is an aminopropyl carbazole with a dibrominated carbazole core, a fluorinated propyl linker at the 9-position, and a 3-methoxyaniline moiety on the terminal amine. [\[38\]](#) [\[39\]](#)

Text description

- Core ring system: A **carbazole** (tricyclic indole-like system) bearing bromine atoms at the 3 and 6 positions, giving a 3,6-dibromo-9H-carbazole scaffold. [\[39\]](#) [\[38\]](#)
- Linker: The nitrogen at the 9-position of the carbazole is substituted with a three-carbon propyl chain that carries a fluorine on the β -carbon (2-position), making a β -fluoropropyl side

chain.^[40] ^[38]

- Terminal aryl amine: The distal end of that propyl chain is attached to a 3-methoxyaniline ring (meta-methoxy-substituted phenyl bearing an anilide nitrogen), yielding an N-(3-methoxyphenyl)-aminopropyl connection.^[39] ^[40]

Key identifiers

- IUPAC / formal name examples:
 - N-(3-(3,6-dibromo-9H-carbazol-9-yl)-2-fluoropropyl)-3-methoxyaniline.^[41] ^[40]
 - 3,6-dibromo-β-fluoro-N-(3-methoxyphenyl)-9H-carbazole-9-propanamine.^[42] ^[39]
- Molecular formula: C₂₂H₁₉Br₂FN₂O; molecular weight ≈ 506.21 g/mol.^[43] ^[44] ^[39]

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sources

P7C3-A20 is sold only as a laboratory research chemical, not for human use, by several specialty suppliers.^[55] ^[56]

Major research suppliers

- Focus Biomolecules: Markets P7C3-A20 as a NAMPT activator/proneurogenic agent (5–25 mg sizes), explicitly labeled “for laboratory research use only; not for human or veterinary applications.”^[57] ^[55]
- Aobious: Offers high-purity (≈98%) P7C3-A20 under CAS 1235481-90-9 in multiple mg quantities with typical storage at 0 to –20 °C and DMSO solubility.^[56]
- Other catalog vendors: MedChemExpress, Selleck, MedKoo, LKT Labs, Abbexa, TargetMol, and similar companies list P7C3-A20 as a neuroprotective NAMPT activator for in vitro/in vivo research, again strictly labeled **research-only** and not for diagnostic or therapeutic use.^[58] ^[59] ^[60] ^[61] ^[62] ^[63]

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How was this administered in mouse studies

In mouse studies, P7C3-A20 has been given mainly by intraperitoneal (IP) injection and oral gavage, at doses around 10–20 mg/kg once or twice daily.^[75] ^[76] ^[77]

Intraperitoneal injection

- Traumatic brain injury and stroke models: P7C3-A20 is commonly dissolved in a DMSO/Kolliphor/D5W vehicle and administered IP at 10–20 mg/kg, starting 30 minutes after injury and continued once or twice daily for 7 days to 1 month.^[78] ^[76] ^[77]
- ICH and BBB studies: Mice received 10 mg/kg IP, either as a single dose shortly after insult or as daily injections, which reduced lesion volume, BBB damage, and neurodegeneration.^[75] ^[78]

Oral (gavage) dosing

- Metabolic / liver and gut microbiota models: P7C3-A20 has been formulated for oral gavage and given at 20 mg/kg/day, often split b.i.d. (twice daily) in mice. [\[79\]](#)
- Pharmacokinetic work: Earlier P7C3-class studies show that oral administration achieves sustained plasma exposure, and oral formulations were optimized in rodents before primate studies. [\[80\]](#) [\[81\]](#)

General patterns and context

- Typical mouse dose range: 10–20 mg/kg per day, with treatment windows from 7 days (acute injury) up to several weeks depending on the model. [\[82\]](#) [\[76\]](#) [\[83\]](#)
- Formulation: Usually dissolved in DMSO plus a surfactant (e.g., Kolliphor/cremophor) and diluted in aqueous dextrose or similar vehicle for IP, or in oil/syrup-type vehicles for oral dosing, always as a **research** intervention, not a clinical regimen. [\[80\]](#) [\[78\]](#)

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How long was daily dosing continued in mouse experiments

Across mouse experiments, daily P7C3-A20 dosing ranges from very brief (about 1 week) up to about 1 month, depending on the disease model. [\[95\]](#) [\[96\]](#)

Short courses (about 1–2 weeks)

- Ischemic stroke and acute injury: In stroke models, P7C3-A20 was typically given for about 7 days (often twice daily) after the ischemic event to test acute neuroprotection and early behavioral effects. [\[97\]](#) [\[98\]](#)
- Stress/depression paradigms: In neurogenesis/antidepressant studies with related P7C3 compounds, daily (or twice-daily) injections were continued for around 10 days during the stress protocol. [\[99\]](#)

Intermediate courses (about 2 weeks)

- Metabolic/NAFLD model: In a fatty-liver and gut-microbiota study, high-fat-fed mice received P7C3-A20 by oral gavage at 20 mg/kg/day for **2 weeks**. [\[100\]](#)
- Some injury/inflammation models: Certain intracerebral hemorrhage or inflammatory brain injury experiments used daily IP P7C3-A20 for roughly 10–14 days to assess lesion size, BBB integrity, and microglial activation. [\[101\]](#) [\[102\]](#)

Longer courses (about 1 month)

- Chronic TBI model (key paper): In the “1 year after TBI” mouse study, animals received **10 mg/kg IP once daily for 4 weeks (30 days)**, starting 12 months post-injury, with benefits persisting at least 4 months after stopping treatment. [\[103\]](#) [\[104\]](#) [\[95\]](#)
- Safety and tolerability: A broader overview notes that P7C3-A20 has been dosed in mice at up to 40 mg/kg/day for **30 days** without obvious toxicity in behavior, weight, or appearance. [\[96\]](#)

Overall, most mouse work clusters around **7–30 days of daily dosing**, with chronic-injury and safety studies using the upper end (≈30 days) and acute models using 1–2 weeks. [\[105\]](#) [\[95\]](#) [\[97\]](#)

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“In neurogenesis/antidepressant studies with related P7C3 compounds” **what are the other related compounds**

The main “related” P7C3 compounds used in neurogenesis/antidepressant mouse studies are the parent P7C3 molecule, the more active analog P7C3-A20, and the inactive analog P7C3-S184; later work also highlights P7C3-S243 as an improved analog. [\[114\]](#) [\[115\]](#) [\[116\]](#)

Core P7C3-class molecules

- **P7C3**: The original aminopropyl carbazole, proneurogenic and neuroprotective; shows antidepressant-like effects in chronic social defeat stress (CSDS) by preserving hippocampal neurogenesis. [\[116\]](#) [\[117\]](#) [\[114\]](#)
- **P7C3-A20**: A fluorinated, methoxy-substituted analog with greater neuroprotective and antidepressant efficacy than P7C3; strongly enhances hippocampal neurogenesis and is the lead compound in many later studies. [\[118\]](#) [\[119\]](#) [\[114\]](#)
- **P7C3-S184**: An “inactive” analog used as a negative control; lacks neuroprotective and antidepressant effects in the same CSDS and neurogenesis paradigms, helping confirm that behavioral effects depend on neuroprotection. [\[120\]](#) [\[114\]](#)

Optimized later analog

- **P7C3-S243**: A more drug-like aminopropyl carbazole analog with increased polarity and an aminopyridine in place of aniline, reported to retain or improve neuroprotective activity while offering better pharmacokinetic properties. [\[115\]](#) [\[116\]](#)

Broader P7C3 analog set

- Structure–activity work describes “dozens” of P7C3 analogs; at least eight additional analogs were tested in hippocampal neurogenesis assays, where proneurogenic potency tracked with neuroprotection in Parkinson’s and other models. [\[121\]](#) [\[119\]](#) [\[118\]](#)
- Patents and reviews refer to the entire **P7C3 class of aminopropyl carbazoles**, but in published antidepressant/neurogenesis work the named comparators are mainly P7C3, P7C3-A20, P7C3-S184, and later P7C3-S243. [\[122\]](#) [\[114\]](#) [\[116\]](#)

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<q>P7C3-S243</q> cas number

The CAS number for **P7C3-S243** (—P7C3-S243) is **1597443-57-6**. [\[133\]](#) [\[134\]](#)

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chemical structure

P7C3-S243 is an aminopropyl carbazole with a dibrominated carbazole core, a β -fluoropropyl linker, and a 6-methoxypyridin-2-amine head group, making it more polar and drug-like than P7C3-A20. [\[153\]](#) [\[154\]](#)

Text description

- Core scaffold: A **3,6-dibromo-9H-carbazole** ring system (same carbazole core as P7C3-A20) substituted at the 9-position. [\[155\]](#) [\[154\]](#)
- Linker: A three-carbon propyl chain bearing a fluorine on the 2-position (β -fluoro) connects the carbazole nitrogen (N-9) to the terminal heterocycle. [\[155\]](#) [\[153\]](#)
- Terminal heterocycle: Instead of an aniline, P7C3-S243 carries a **6-methoxypyridin-2-amine** moiety, increasing polarity and removing the aniline group that limited drug-likeness in earlier analogs. [\[156\]](#) [\[153\]](#)

Key identifiers

- Representative full name (IUPAC-style): (*S*)-N-(3-(3,6-dibromo-9H-carbazol-9-yl)-2-fluoropropyl)-6-methoxypyridin-2-amine. [\[157\]](#) [\[154\]](#)
- Class: Single-enantiomer, neuroprotective **aminopropyl** carbazole optimized for oral bioavailability, BBB penetration, and reduced toxicity versus earlier P7C3 analogs. [\[158\]](#) [\[153\]](#)

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Compare structures of P7C3 S243 and P7C3 A20

Both P7C3-A20 and P7C3-S243 share the same dibrominated carbazole core and β -fluoropropyl linker, but they differ in the "head group": P7C3-A20 carries a 3-methoxyaniline, whereas P7C3-S243 carries a 6-methoxypyridin-2-amine, making S243 more polar and drug-like. [\[171\]](#) [\[172\]](#)

Shared structural features

- **Core scaffold:** Both are 3,6-dibromo-9H-carbazole derivatives substituted at the 9-nitrogen. [\[173\]](#) [\[174\]](#)
- **Linker:** Both use a three-carbon propyl chain with a fluorine on the 2-position (β -fluoro), giving an N-(3-(3,6-dibromo-9H-carbazol-9-yl)-2-fluoropropyl) backbone. [\[175\]](#) [\[176\]](#)

Key differences

- P7C3-A20 "head group": Terminal **aniline** ring (3-methoxyaniline), i.e., N-(3-methoxyphenyl) at the end of the linker. [\[177\]](#) [\[174\]](#)
- P7C3-S243 "head group": Terminal **aminopyridine** ring (6-methoxypyridin-2-amine), replacing the aniline with a pyridine nitrogen and shifting the methoxy to the 6-position. [\[172\]](#) [\[176\]](#)

Consequences of the substitution

- Polarity and drug-likeness: Replacing the aniline of A20 with an aminopyridine in S243 increases polarity and removes the aniline moiety that was considered less desirable for drug-like properties. [\[171\]](#) [\[173\]](#)
- Enantiomeric form: P7C3-S243 is developed as the single *S* enantiomer (—P7C3-S243), whereas A20 is typically used as a single active fluorinated analog without the same emphasis on chirality optimization. [\[176\]](#) [\[172\]](#)

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are there any human studies on any P7C3- family of compounds

There are **no published human clinical trials** of any P7C3-family compound as of late 2025; all in vivo work is in rodents and non-human primates. [\[192\]](#) [\[193\]](#)

What has been done so far

- Rodents: P7C3, P7C3-A20, and P7C3-S243 have extensive mouse and rat data in models of TBI, stroke, Parkinson's, Alzheimer's, depression, fatty liver disease, and others, all preclinical. [\[194\]](#) [\[195\]](#) [\[196\]](#) [\[192\]](#)
- Non-human primates: Daily oral P7C3-A20 for 38 weeks in monkeys increased hippocampal neurogenesis without detectable toxicity, supporting translational potential but still not

constituting human testing. [\[193\]](#) [\[197\]](#)

Explicit statements about human data

- A 2018–2023 series of reviews on P7C3 compounds describes them as “preclinical” agents and highlights primate safety/efficacy as a step **toward** first-in-human studies, with no human trial results reported. [\[192\]](#) [\[193\]](#)
- No entries for P7C3, P7C3-A20, or P7C3-S243 appear in these reviews as having Phase I or later trials; any discussion of human use is framed as future, hypothetical translation. [\[198\]](#) [\[192\]](#)

Takeaway for practical use

- At present, P7C3-family compounds should be regarded strictly as **experimental research tools**, with safety and efficacy established only in animals and cell systems, not in people. [\[193\]](#) [\[192\]](#)
- Even where news releases mention P7C3-A20 as a potential treatment for TBI or Alzheimer's, they refer to animal models and non-human primate work, not to completed or ongoing human trials. [\[199\]](#) [\[200\]](#) [\[201\]](#)

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what would the HED for P7C3-A20 based on primate studies

Using standard body-surface-area scaling, the human-equivalent dose (HED) corresponding to the 10 mg/kg/day oral dose used in rhesus monkeys is about **3.2 mg/kg/day** in humans (≈ 220 mg/day for a 70-kg adult). [\[212\]](#) [\[213\]](#)

Step 1 – Primate dose used

- Adult male rhesus monkeys received **10 mg/kg/day oral P7C3-A20** for 38 weeks, with no detected central or peripheral toxicity and sustained plasma exposure. [\[214\]](#) [\[212\]](#)
- This 10 mg/kg dose is the relevant starting point for HED estimation. [\[212\]](#)

Step 2 – HED calculation

- Standard BSA conversion:
 - K_m (monkey, rhesus) ≈ 12 ; K_m (human, adult) ≈ 37 . [\[213\]](#)
 - $\text{HED (mg/kg)} = \text{Animal dose} \times (K_m_{\text{animal}} / K_m_{\text{human}})$.
- Plugging in:
 - $\text{HED} \approx 10 \text{ mg/kg} \times (12/37) \approx 3.2 \text{ mg/kg}$. [\[213\]](#)
- For a 70-kg human, that is **~ 225 mg/day**, often rounded to **~ 200 – 250 mg/day** as a theoretical equivalent to the primate neurogenesis study. [\[212\]](#) [\[213\]](#)

Critical caveats

- P7C3-A20 has **no human safety data**, and PK/PD in humans are unknown; this HED is a regulatory toxicology convention, **not** a recommended or permissible dose. [\[215\]](#) [\[216\]](#)
- Route, formulation, and metabolism can differ substantially between monkeys and humans; any real first-in-human study would start far below this HED and escalate cautiously. [\[217\]](#) [\[213\]](#)



Estimate starting clinical dose after applying safety factor to HED

Using standard regulatory practice (FDA MRSD approach), a conservative starting clinical dose from the primate HED of ~3.2 mg/kg/day would be about **0.3 mg/kg/day** (≈20 mg/day for a 70-kg adult). [\[233\]](#) [\[234\]](#)

How that estimate is derived

- From primate data, the HED for P7C3-A20 was ~3.2 mg/kg/day based on 10 mg/kg/day in rhesus monkeys. [\[235\]](#) [\[236\]](#)
- FDA guidance: After converting an animal NOAEL to an HED, a **default safety factor of 10** is typically applied to get the maximum recommended starting dose (MRSD) for healthy volunteers. [\[237\]](#) [\[233\]](#)
- Applying that factor:
 - $\text{MRSD} \approx 3.2 \text{ mg/kg} \div 10 \approx \mathbf{0.32 \text{ mg/kg}}$. [\[238\]](#) [\[233\]](#)
 - For 70 kg, that is $\approx 22 \text{ mg/day}$, usually rounded to **~20 mg/day** as a theoretical first-in-human starting dose. [\[239\]](#) [\[233\]](#)

Important cautions

- This calculation assumes the **monkey dose is at or below the NOAEL**, which has not been formally established for P7C3-A20 in GLP tox; without a defined NOAEL, a real program might use an even larger safety factor (e.g., 10–30+). [\[234\]](#) [\[233\]](#)
- Modern practice often also considers a MABEL (minimal anticipated biological effect level) approach, which can yield **lower** starting doses than NOAEL/HED-based MRSD. [\[240\]](#) [\[234\]](#)
- P7C3-A20 remains a purely preclinical compound; these numbers are only illustrative of regulatory math, **not** a dosing recommendation for human use. [\[241\]](#) [\[242\]](#)



is there an established LD50 dose

There is **no published LD₅₀** (median lethal dose) for P7C3-A20 in any species. [\[256\]](#) [\[257\]](#)

What is known about toxicity instead

- Mouse/rat studies describe P7C3 and P7C3-A20 as “non-toxic at doses several-fold higher” than those needed for neuroprotection, but do **not** report an LD₅₀ value. [\[258\]](#) [\[259\]](#)
- Non-human primate work reports daily oral 10 mg/kg P7C3-A20 for 38 weeks with no detectable central or peripheral toxicity, again without defining an LD₅₀. [\[260\]](#) [\[261\]](#)

Implications

- Without formal acute toxicity studies designed to determine LD₅₀, only a general statement of “well tolerated” at tested doses is possible; any precise LD₅₀ number you might see online is not supported by the primary literature. [\[262\]](#) [\[257\]](#)
- P7C3-A20 should therefore be considered an experimental compound with incomplete toxicology, not a drug with a characterized lethal-dose profile. [\[257\]](#) [\[263\]](#)

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what is the half life

For P7C3-class compounds, including P7C3-A20, reported in vivo half-lives are on the order of **>6 hours**, but precise species- and route-specific $t_{1/2}$ values for P7C3-A20 are not consistently published. [\[276\]](#) [\[277\]](#)

What is reported

- A review of P7C3 chemistry states that both P7C3 and P7C3-A20 have “**long half-lives... in vivo ($t_{1/2} > 6$ h)**”, based on hepatocyte and animal PK studies, without giving exact numbers by route or species. [\[276\]](#)
- For the parent P7C3, one pharmacology overview notes **oral bioavailability ~32% and a terminal plasma half-life of about 6.7 h after IP dosing** in rodents, used as a benchmark for the series’ PK behavior. [\[277\]](#)

What is *not* available

- No open-source paper or datasheet gives a definitive numeric half-life curve (e.g., “ $t_{1/2} = 7.2$ h IV, 9.1 h PO in mouse”) specifically for P7C3-A20. [\[278\]](#) [\[279\]](#)
- Human half-life is completely unknown, as there are no clinical PK studies with any P7C3-family compound. [\[280\]](#) [\[278\]](#)

Practical interpretation

- P7C3-A20 is generally characterized as having a **“long half-life” sufficient for once-daily dosing** in rodent and primate experiments, but current literature only supports saying **t_{1/2} is greater than roughly 6 hours in vivo**, not a precise value. [\[281\]](#) [\[276\]](#)

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<q>P7C3-S243</q> are there any in vivo studies on this

Yes. P7C3-S243 has been tested extensively in vivo in multiple rodent models, but not in humans. [\[297\]](#) [\[298\]](#)

Parkinson's disease models

- Mouse/rat 6-OHDA and MPTP models: P7C3-S243 protects dopaminergic neurons in the substantia nigra and improves motor behavior when given systemically (e.g., 5–10 mg/kg/day), showing robust neuroprotection in vivo. [\[299\]](#) [\[297\]](#)
- Mechanistic work shows preserved nigral neuron survival and striatal innervation, with S243 outperforming earlier P7C3 analogs in potency and tolerability. [\[300\]](#) [\[297\]](#)

Alzheimer's disease models

- TgF344-AD rat model: (—P7C3-S243) chronically treated aging wild-type and transgenic rats, protecting them from cognitive decline and depressive-like behavior without changing amyloid deposition. [\[301\]](#) [\[298\]](#)
- Treated AD-model rats retained neuron numbers and synaptic markers despite persistent plaques and tangles, indicating direct neuroprotection rather than disease-modifying effects on amyloid. [\[302\]](#) [\[303\]](#)

Other in vivo contexts

- Blast/TBI and axonal injury: Reviews and mechanistic papers note P7C3-S243 efficacy in mouse blast-TBI and axonal degeneration paradigms, similar to P7C3-A20. [\[304\]](#) [\[300\]](#)
- General summary: A 2014–2023 body of work classifies P7C3-S243 as an “optimized” aminopropyl carbazole with improved drug-like properties and proven in vivo neuroprotection across multiple rodent models of neurodegenerative disease. [\[305\]](#) [\[297\]](#)

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Summarize in vivo models and species used with P7C3-S243

Yes. P7C3-S243 has been used in multiple in vivo rodent models, mainly rats and mice, across neurodegenerative and injury paradigms. [\[317\]](#) [\[318\]](#)

Species used

- **Rats**
 - 6-OHDA Parkinson's disease models (unilateral nigrostriatal lesion). [\[319\]](#) [\[317\]](#)
 - TgF344-AD transgenic rats (Alzheimer's disease model, both sexes, long-term dosing). [\[320\]](#) [\[318\]](#)
- **Mice**
 - Blast-mediated traumatic brain injury (TBI) and axonal degeneration models. [\[321\]](#)
 - Additional mouse neurodegeneration and injury paradigms referenced in reviews (e.g., ALS and age-related decline) for the P7C3 series including S243. [\[322\]](#) [\[323\]](#)

Parkinson's disease models (rat)

- 6-OHDA lesion: Systemic P7C3-S243 (often 5–10 mg/kg/day) before and/or after toxin exposure protected dopaminergic neurons in substantia nigra, preserved striatal dopamine and metabolites, and improved motor behavior. [\[317\]](#) [\[319\]](#)
- These studies showed efficacy even when S243 was started **after** lesioning, modeling delayed treatment. [\[317\]](#)

Alzheimer's disease model (rat)

- TgF344-AD rats: Long-term daily P7C3-S243 from 6 months of age for 9–18 months prevented cognitive deficits and depressive-like behavior and blocked neurodegeneration, without altering amyloid or neuroinflammation markers. [\[318\]](#) [\[320\]](#)

Traumatic brain injury / axonal injury (mouse)

- Blast-TBI: In mice exposed to blast, orally administered P7C3-S243 (e.g., 30 mg/kg/day) reduced axonal degeneration in hippocampal circuits and preserved synaptic and electrophysiologic function. [\[321\]](#)
- These experiments established that S243 crosses the intact blood–brain barrier and blocks early axonal damage preceding neuron loss. [\[324\]](#) [\[321\]](#)

High-level summary

- Across models, P7C3-S243 has **in vivo** evidence in:
 - Rat: Parkinson's (6-OHDA), Alzheimer's (TgF344-AD), neuropsychiatric/behavioral endpoints. [\[318\]](#) [\[317\]](#)
 - Mouse: Blast/TBI and axonal degeneration, with additional support from series-wide reviews. [\[323\]](#) [\[321\]](#)
- All studies are **preclinical**; there are no human in vivo data for P7C3-S243. [\[322\]](#) [\[323\]](#)



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