



DOGWOOD THERAPEUTICS

**Deep Commitment to Cancer
Patients Suffering from Neuropathy**

Q4 2025 Update

NASDAQ: DWTX

Forward-Looking Statements and Disclaimers



Forward-Looking Statements

Statements in this presentation contain “forward-looking statements,” within the meaning of the U.S. Private Securities Litigation Reform Act of 1995, that are subject to substantial risks and uncertainties. All statements, other than statements of historical fact, contained in this presentation are forward-looking statements. Forward-looking statements contained in this presentation may be identified by the use of words such as “anticipate,” “believe,” “contemplate,” “could,” “estimate,” “expect,” “intend,” “seek,” “may,” “might,” “plan,” “potential,” “predict,” “project,” “suggest,” “target,” “aim,” “should,” “will,” “would,” or the negative of these words or other similar expressions, although not all forward-looking statements contain these words. Forward-looking statements are based on the current expectations of Dogwood Therapeutics, Inc. (“Dogwood”) and are subject to inherent uncertainties, risks and assumptions that are difficult to predict, including risks related to the completion, timing and results of current and future clinical studies relating to Dogwood’s product candidates. Further, certain forward-looking statements are based on assumptions as to future events that may not prove to be accurate. These and other risks and uncertainties are described more fully in the section titled “Risk Factors” in the Annual Report on Form 10-K for the year ended December 31, 2024, filed with the Securities and Exchange Commission. Forward-looking statements contained in this presentation are made as of this date, and Dogwood undertakes no duty to update such information except as required under applicable law.

Important Additional Information and Where to Find It

Dogwood, its directors and certain of its executive officers are deemed to be participants in the solicitation of proxies from Dogwood stockholders in connection with Dogwood’s expected special meeting seeking stockholder approval of conversion of Dogwood’s preferred stock (“Preferred Stock”) and other matters related to the business combination with Wex Pharmaceuticals, Inc. (the “Combination”). Information regarding the names of Dogwood’s directors and executive officers and their respective interests in Dogwood by security holdings or otherwise can be found in Virios Therapeutics, Inc.’s proxy statement for its 2025 Annual Meeting of Stockholders, filed with the SEC on April 30, 2025. To the extent holdings of Dogwood’s securities have changed since the amounts set forth in Virios Therapeutics Inc.’s proxy statement for the 2025 Annual Meeting of Stockholders, such changes have been or will be reflected on Initial Statements of Beneficial Ownership on Form 3 or Statements of Change in Ownership on Form 4 filed with the SEC. These documents are available free of charge at the SEC’s website at www.sec.gov. Dogwood intends to file a proxy statement and accompanying proxy card with the SEC in connection with the solicitation of proxies from Dogwood stockholders in connection with Dogwood’s expected special meeting seeking stockholder approval of conversion of the Preferred Stock and other matters related to the Combination. Additional information regarding the identity of participants, and their direct or indirect interests, by security holdings or otherwise, will be set forth in Dogwood’s proxy statement for such special meeting, including the schedules and appendices thereto. INVESTORS AND STOCKHOLDERS ARE STRONGLY ENCOURAGED TO READ ANY SUCH PROXY STATEMENT AND THE ACCOMPANYING PROXY CARD AND ANY AMENDMENTS AND SUPPLEMENTS THERETO AS WELL AS ANY OTHER DOCUMENTS FILED BY DOGWOOD WITH THE SEC CAREFULLY AND IN THEIR ENTIRETY WHEN THEY BECOME AVAILABLE AS THEY WILL CONTAIN IMPORTANT INFORMATION. Stockholders will be able to obtain copies of the proxy statement, any amendments or supplements to the proxy statement, the accompanying proxy card, and other documents filed by Dogwood with the SEC for no charge at the SEC’s website at www.sec.gov. Copies will also be available at no charge at the Investor Relations section of Dogwood’s corporate website at <https://ir.DWTX.com/> or by contacting Dogwood’s Investor Relations at Dogwood Therapeutics, Inc., 44 Milton Avenue, Alpharetta, GA 30009 or by emailing

Dogwood’s Investor Relations at IR@dwtx.com or (866) 620-8655.

Dogwood is Led by an Executive Team with Extensive Drug Development and Commercialization Experience



DWTX Executive Team



Greg Duncan
Chairman & CEO



R. Michael Gendreau
MD, PhD CMO



Angela Walsh
CFO



Ralph Grosswald
SVP of Operations



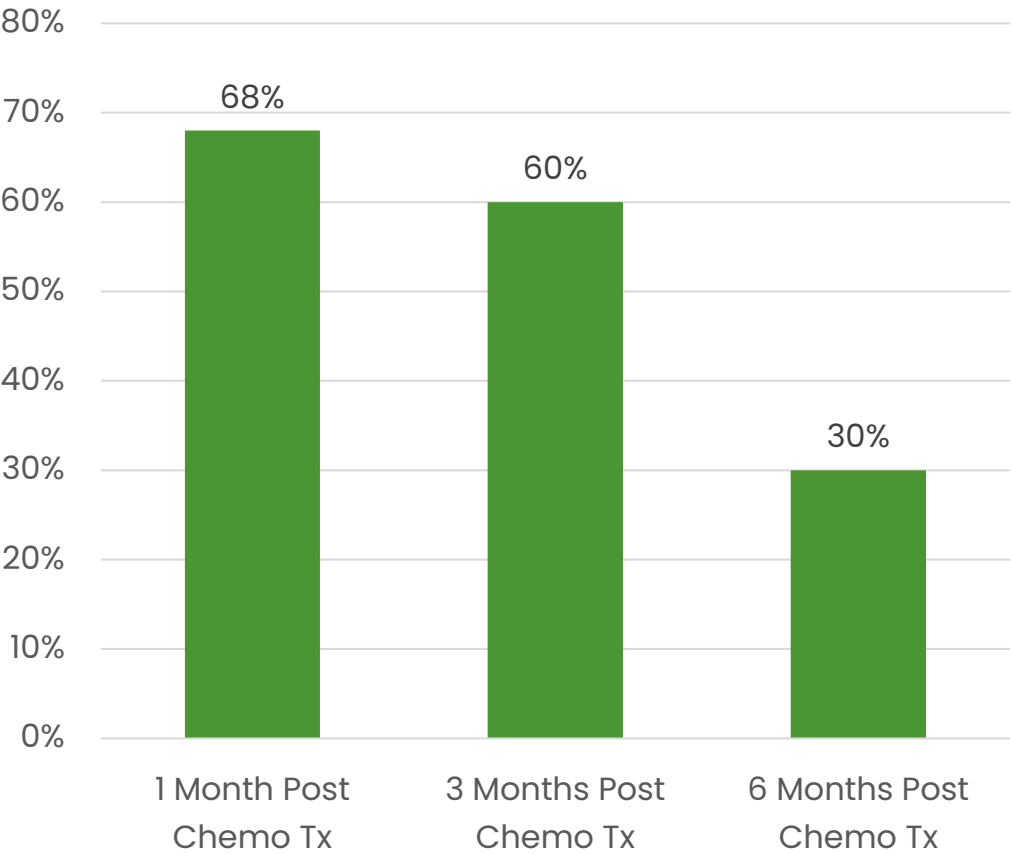
Management's Brand Development & Commercialization Experience Includes:



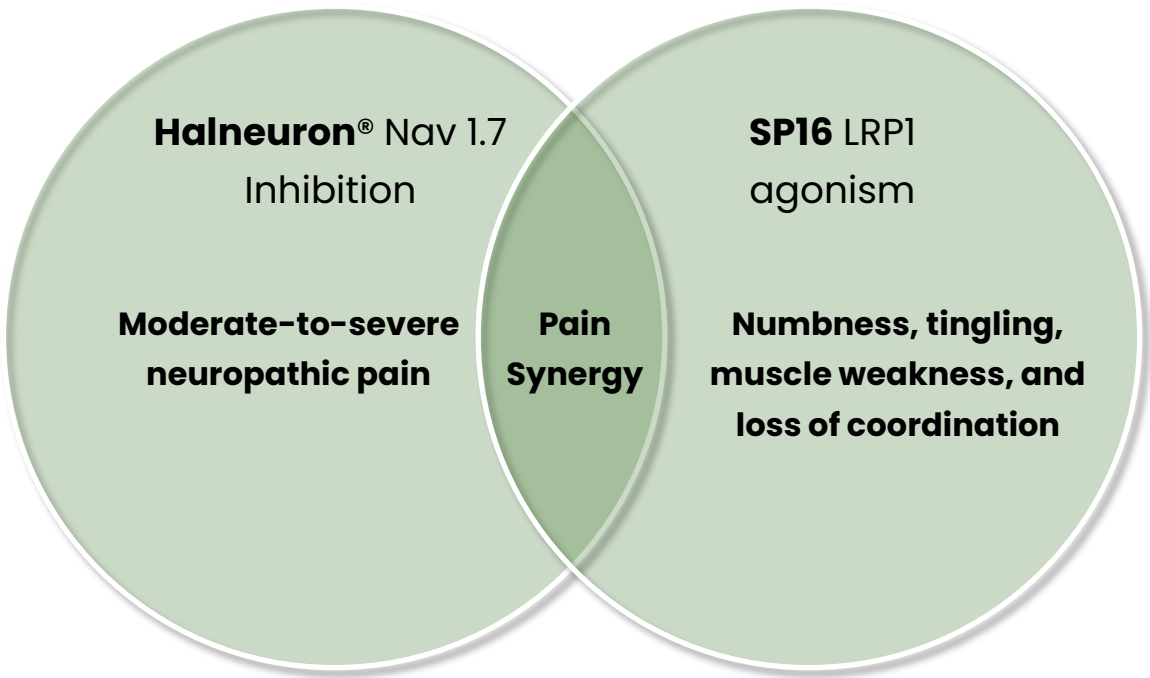
Deep Commitment to Cancer Patients Suffering from Neuropathy, with the Goal to Expand to General Cancer Pain and Acute Surgical Pain



Neuropathy Prevalence Post Chemotherapy Treatment



Two Potential Solutions to Address Significant Unmet Neuropathic Medical Need



Complementary and Synergistic Research Pipeline

- Halneuron® (TTX) = Nav1.7 channel blocker, analgesic → best for treating established chemo induced pain
- SP16 = LRP1 (Low density lipoprotein receptor related protein-1) -agonist, anti-inflammatory, neuroprotection → best for attenuation of neuropathy during chemo;
 - SP16 may preserve full chemo regimen
 - SP16 potential to synergistically complement Halneuron® in treating pain post chemotherapy
- Common commercial call points (oncology and pain centers) and potential partners
 - Bundled protocols/formularies
 - Deeper penetration into the global CIPN treatment opportunity ~\$1.5B market, expand to ~\$5B cancer pain market

Target Indication	Candidate/Target	Preclinical	Phase 1	Phase 2	Phase 3
Phase 2b CIPN	Halneuron® Na_v1.7 Injection	FDA Fast Track Designation: Ongoing Phase 2b			
General Cancer Pain	Halneuron® Na_v1.7	Phase 2a Complete			
Acute Surgical Pain	Halneuron® Na_v1.7				
Phase 1b CIPN	SP16 Intravenous Administration	NCI Funded			

DWTX Projected Research Program Milestones and Catalysts

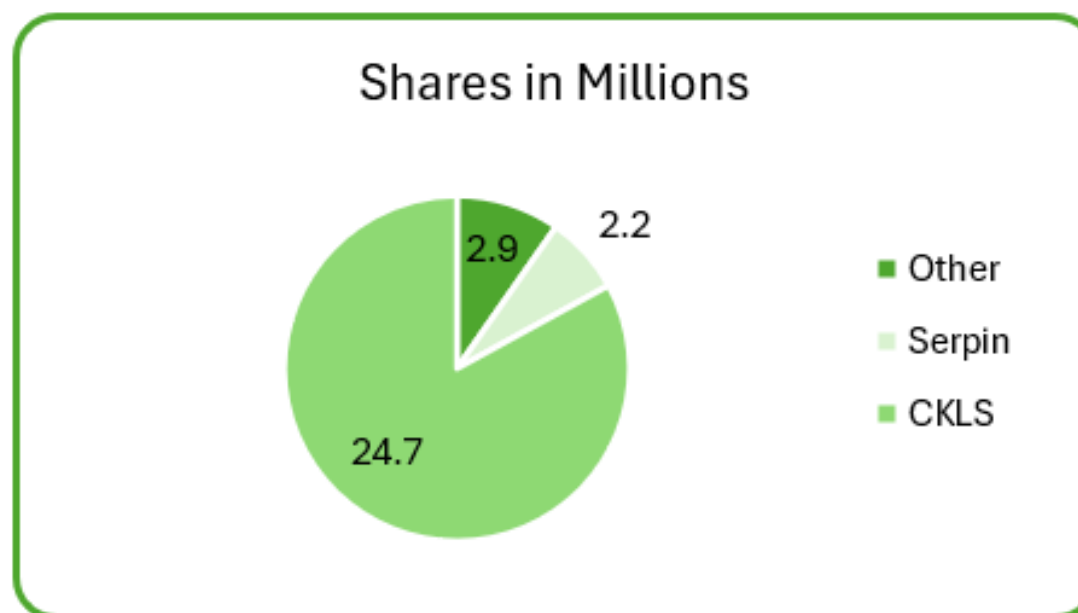


Candidate/Target	Target Indication	Next Key Milestone
Halneuron® Na_v1.7	FDA Fast Track Designation for Treatment of Chemotherapy Induced Neuropathic Pain (CINP)	Q4: Recruitment of 100 (50% of Current Target) Patients in Phase 2b Q4: New Synthetic Halneuron® IP Filed Q4 '25: Phase 2b Interim Data Readout Q2/Q3 '26: Final data for 200 Patient Phase 2b CINP
SP16 LRP1 agonist	Novel New Treatment for Chemotherapy Induced Peripheral Neuropathy (CIPN)	Q4: Filed SP16 IND to Advance to Phase 1b Safety Study 1H '26: Patient Enrollment in Phase 1b CIPN Study*

*Subject to be review with FDA

DWTX Ownership on a Fully Diluted Basis, Presuming Positive Shareholder Vote

- Upon conversion of the existing Preferred Stock following shareholder approval, there will be ~ 30 million shares of common stock on a fully diluted basis
 - CK Life-Sciences (CKLS) owns ~ 2,108 shares of Series A and ~ 284 shares of Series A-1 Preferred Stock
 - Serpin Pharma owns ~ 179 preferred (A-2)
 - Each Preferred share converts into 10,000 common shares, subject to approval by DWTX shareholders
- Post conversion, two large, stable shareholders CKLS (~ 83%) and Serpin Pharma (~7%) will own ~ 90% of DWTX stock



Halneuron[®] Research Program Overview

Halneuron® – Logical Approach Fulfills Many Requirements Of An Ideal Analgesic



Halneuron® Therapeutic Profile Demonstrated in Clinical Research to-date



Reduced pain in both Cancer Related Pain and CINP clinical trial



Long-lasting relief, with responders exhibiting almost of 2 months of pain relief



No evidence of addiction, euphoria or tolerance



Demonstrated acceptable safety profile from tests in over 700 patients



IP and exclusivity protected via manufacturing know-how and trade secrets

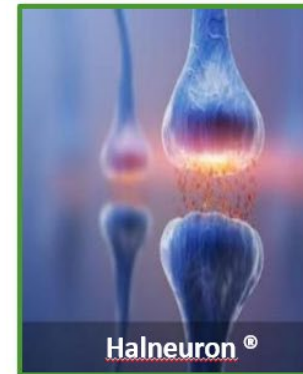


There are no FDA approved CINP medicines, highlighting a large market opportunity

Halneuron's® Na_v1.7 Inhibition Mechanism Supported by Real World Patient Experience



Loss of Na_v1.7 Function Leads to Congenital Insensitivity to Pain Syndrome



Halneuron® inhibits sodium channels, including Na_v1.7, reducing pain signal transmission



Erythromelalgia: Sodium channels remain open increasing pain signals

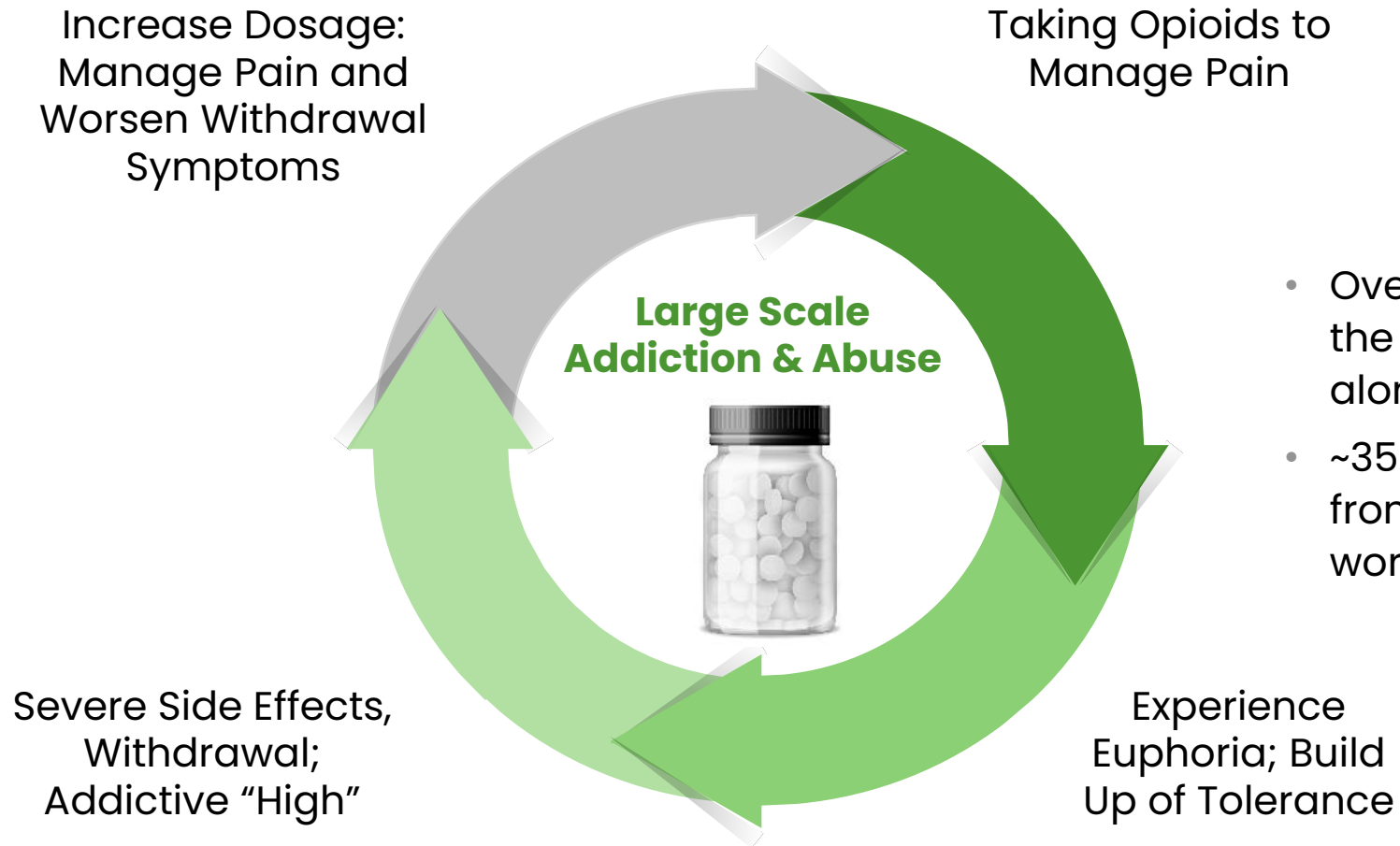
CIPN Represents a Major Unmet Medical Need

- Approximately 20M new patients were diagnosed with cancer worldwide in 2022
 - ~2M new cancer cases in the US in 2025
 - ~40% of cancer patients live with chronic pain
- Over 50% of cancer patients are treated annually with chemotherapy
- CIPN is nerve damage caused by certain chemotherapy drugs, leading to a range of neuropathic symptoms, including pain, numbness, and tingling, often in the hands and feet
 - CIPN severity characterized as mild (25%), moderate (50%), or severe (25%) across most markets
- Estimates suggest almost 70% of patients treated with chemotherapy experience CIPN
 - 30% of chemotherapy treated patients continue to experience CIPN six months post treatment
- Chemotherapy utilization is expected to increase by 54% by 2040
- A 2024 review states that up to 97% of patients with painful CIPN use opioid analgesics, but due to side effects, addiction risks, and potential for reduced effectiveness over time
 - Opioids are now relegated to third-line therapy for neuropathic pain



Notes: American Cancer Society; 2024, WHO, 2024; Lancet Oncology, 2019; DelveInsight, 2018; de Stoutz ND, Bruera E, Suarez-Almazor M, J Pain Symptom Manage 1995; Trescot AM, Boswell MV, Atluri SL, et al., Pain Physician 2006

Vicious Cycle With Opioid Pain Therapies



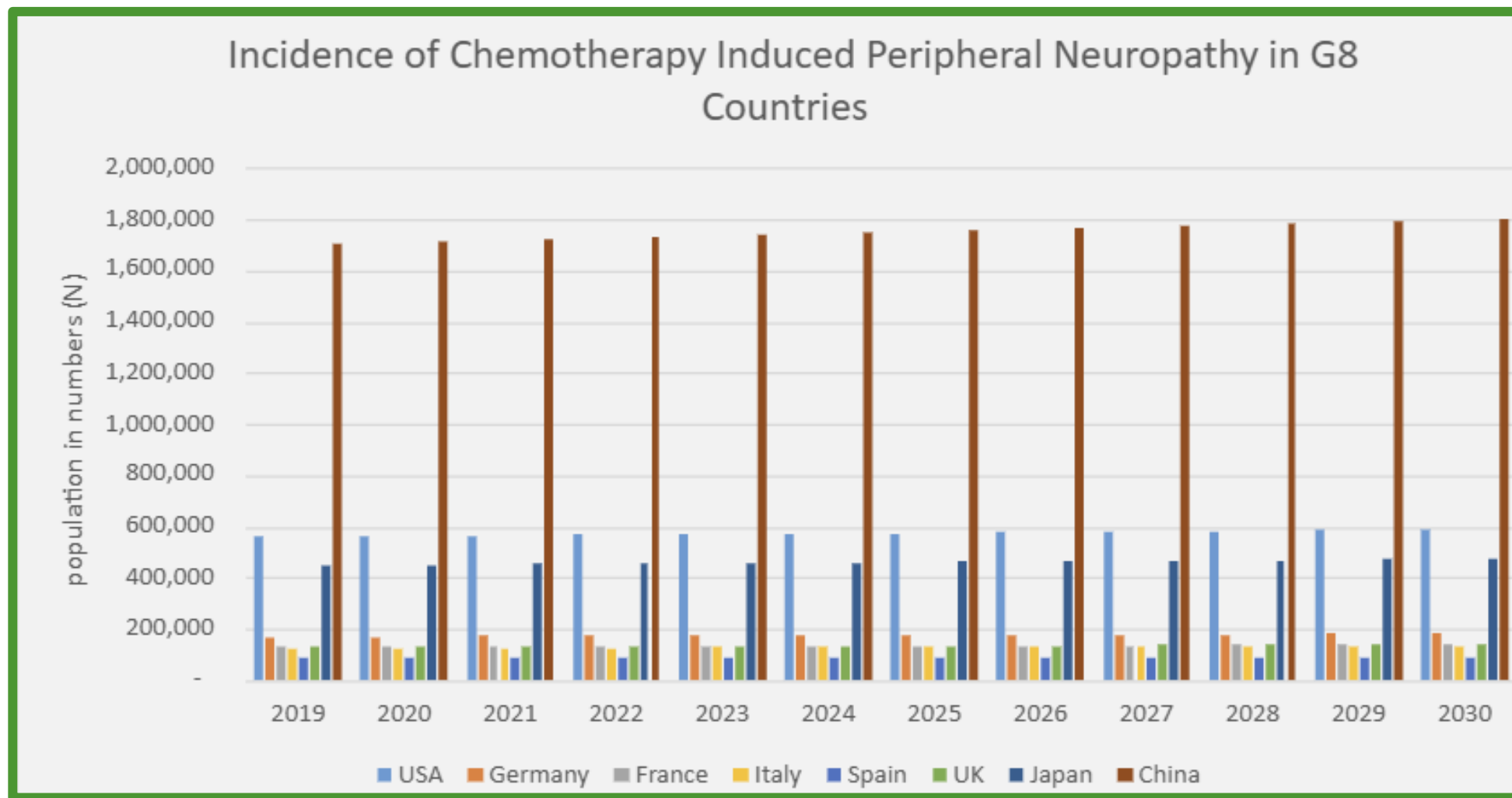
- Over 53,000 opioid deaths in the past 12 months in the US alone¹
- ~35.6 million people suffered from drug use disorders worldwide in 2018²

Notes:

1. CDC - <https://www.cdc.gov/nchs/nvss/vsrr/drug-overdose-data.htm>

2. WHO - <https://www.who.int/news-room/fact-sheets/detail/opioid-overdose>

China CINP Patient Population = 7 Major Markets Combined, Opioid Therapy is the Leading Treatment



Notes: Thelansis, 2020

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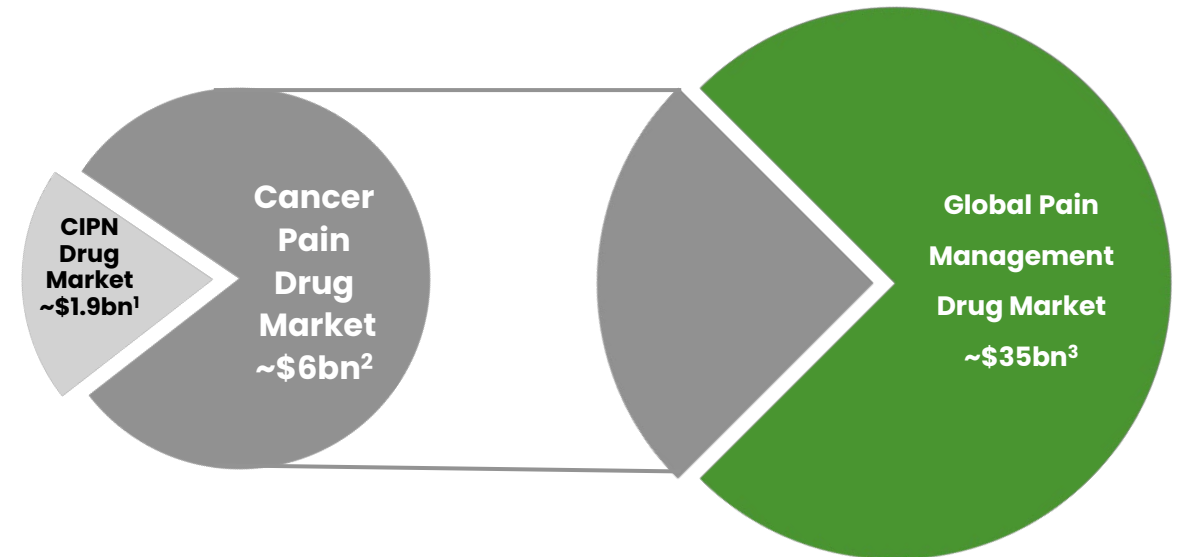
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Ability to Effectively Treat CIPN Opens a Large Market Opportunity

- Medications like duloxetine, gabapentin, pregabalin, or tricyclic antidepressants and opioids are used to help manage neuropathic pain
 - Opioids account for 38%–58% of CIPN treatment, depending on the market
- Halneuron[®] CRP and acute surgical pain life-cycle plan target represents an even larger opportunity than CIPN population

Pain Management Drug Markets

~57% and ~45% of the Global Cancer Pain and Global Pain Management drug markets are opioids respectively ^{2,3}

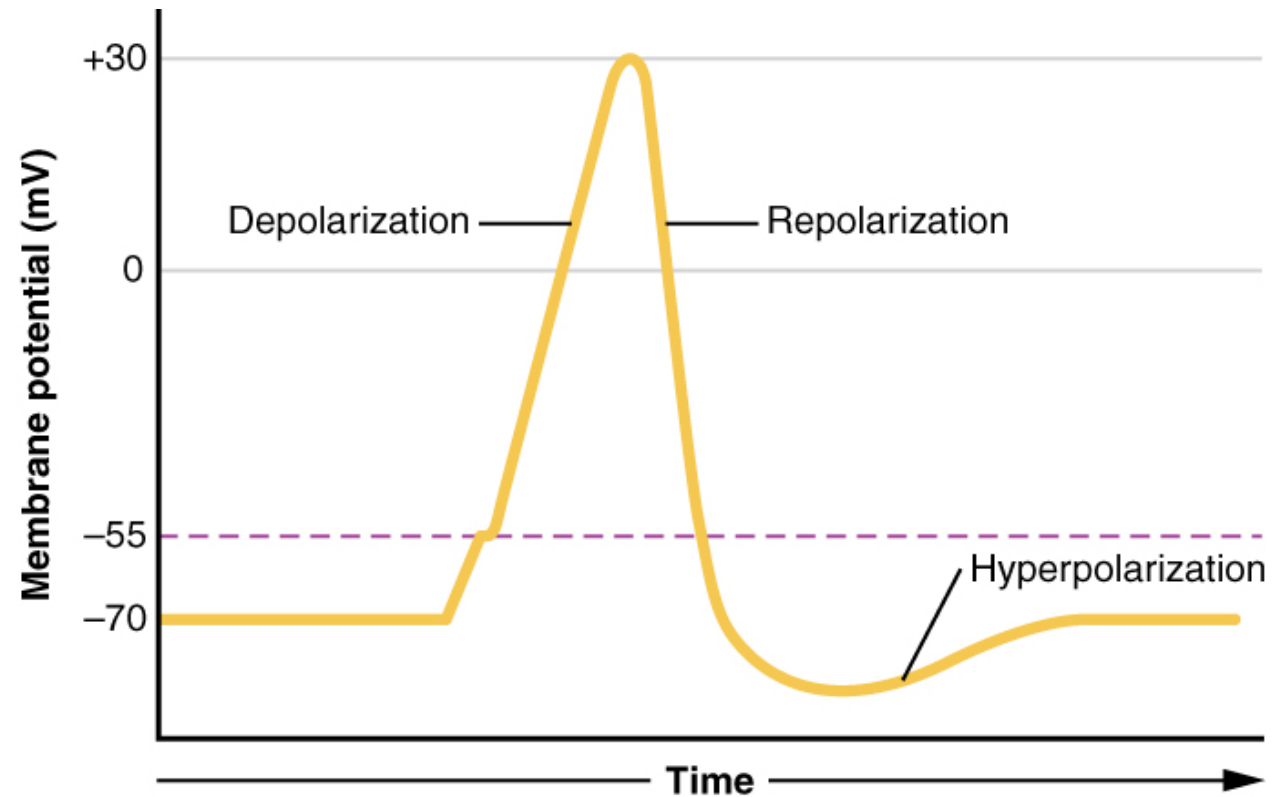


Notes:

1. Delveinsight December 2018, Chemotherapy-Induced Peripheral Neuropathy, Market insights, Epidemiology and Market Forecast 2018–2027
2. Allied Market Research December 2018, Global Cancer Pain Market, Opportunity Analysis and Industry Forecast 2018–2025
3. LP Information December 2019, Global Pain Management Drugs Market growth 2019 –2024
4. Windbank, Annals of Neurol, Neurol, 2017
5. Cancer facts & Figures 2021, CA: A Cancer Journal for Clinicians

Pain and Pain Signaling Related to Chemotherapy

- Inflow of sodium ions causes the membrane to depolarize, thus transmitting an electrical pulse that travels to the spinal cord and ultimately the brain
- This electrical pulse is known as the action potential
- In neuropathic pain, underlying nerve damage makes the peripheral neurons involved in pain more excitable
- Chemotherapy increases the expression of NaV channels in nociceptors, resulting in increased action potential, neuronal hyperexcitability, increasing pain sensitivity and neuropathic pain



Source: Li, Y., et al., *DRG Voltage-Gated Sodium Channel 1.7 Is Upregulated in Paclitaxel-Induced Neuropathy in Rats and in Humans with Neuropathic Pain*. J Neurosci, 2018; Zhang, H. and P.M. Dougherty, *Enhanced excitability of primary sensory neurons and altered gene expression of neuronal ion channels in dorsal root ganglion in paclitaxel-induced peripheral neuropathy*. Anesthesiology, 2014

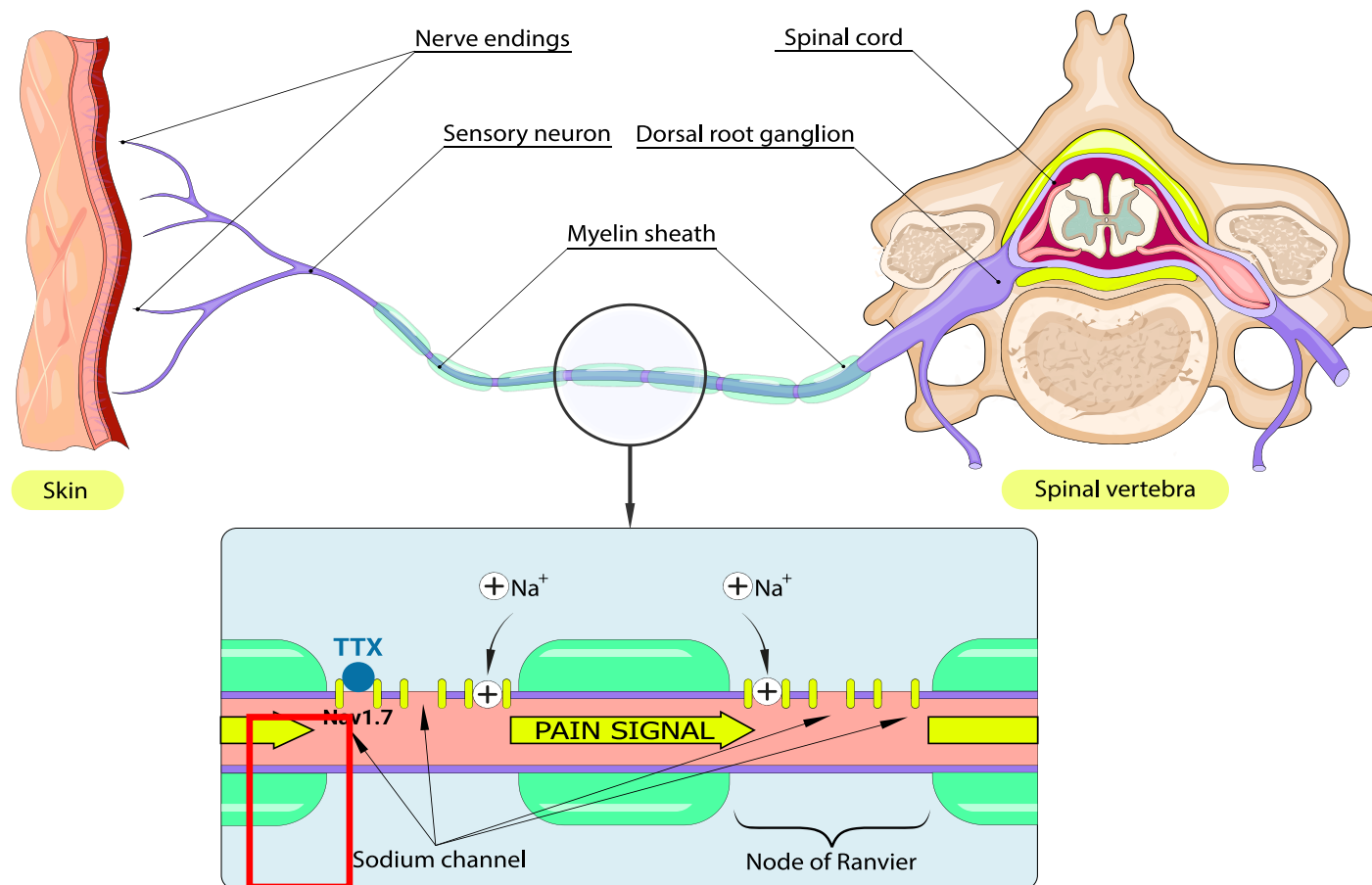
Our Approach – What is Halneuron®?

- Halneuron® is Tetrodotoxin (TTX), a sodium channel blocker and potent small molecule found in puffer fish and several other marine animals (not a peptide or protein)
- Halneuron® is administered as a sub-Q injection

How Does Halneuron® Work?

Pain signals are nerve impulses that travel along a nerve as electrical signals generated by the movement of sodium ions through ion channels on the surface of nerve cells.

Halneuron® works as an analgesic by binding the $Na_v1.7$ channel, a sodium channel responsible for pain signal transmission and associated with certain neuropathies.



Sodium Channels in Mammals

- There are 9 primary sodium channels known in mammals
- The 1.7, 1.8 and 1.9 channels are directly related to pain transmission in the peripheral nervous system
- Halneuron is specific for the 1.7 channel

Table 1. Mammalian sodium channel α subunits

Type	Gene symbol	Chromosomal location	Primary tissues
Na _v 1.1	SCN1A	Mouse 2 Human 2q24	CNS neurons
Na _v 1.2	SCN2A	Mouse 2 Human 2q23–24	CNS neurons
Na _v 1.3	SCN3A	Mouse 2 Human 2q24	CNS neurons
Na _v 1.4	SCN4A	Mouse 11 Human 17q23–25	SkM
Na _v 1.5	SCN5A	Mouse 9 Human 3p21	Uninnervated SkM, heart
Na _v 1.6	SCN8A	Mouse 15 Human 12q13	CNS neurons
Na _v 1.7	SCN9A	Mouse 2 Human 2q24	PNS neurons
Na _v 1.8	SCN10A	Mouse 9 Human 3p22–24	DRG neurons
Na _v 1.9	SCN11A	Mouse 9 Human 3p21–24	DRG neurons
Na _x	SCN7A SCN6A	Mouse 2 Human 2q21–23	uterus, astrocytes, hypothalamus

Halneuron[®] is Selective for the Na_v1.7 Channel in Peripheral Tissues

Channel	TTX Sensitivity	Predominant Distribution
Na_v1.7	EC₅₀ = 24.5 nM	Peripheral nervous system (PNS)
Na _v 1.8	EC ₅₀ = 60,000 nM	PNS & dorsal root ganglion (DRG)
Na _v 1.9	EC ₅₀ = 40,000 nM	PNS & DRG
Na _v 1.5	EC ₅₀ = 5,700 nM	Cardiac

- Na_v1.7 through 1.9 are found in the peripheral nervous system (PNS) and are involved in regulating pain signaling
- Halneuron's low affinity for cardiac sodium channels (Na_v1.5) provides 200X safety margin, avoiding limitations of previous approaches to modulating Na_v1.7
- Halneuron does not cross the blood-brain barrier, minimizing central nervous system adverse events

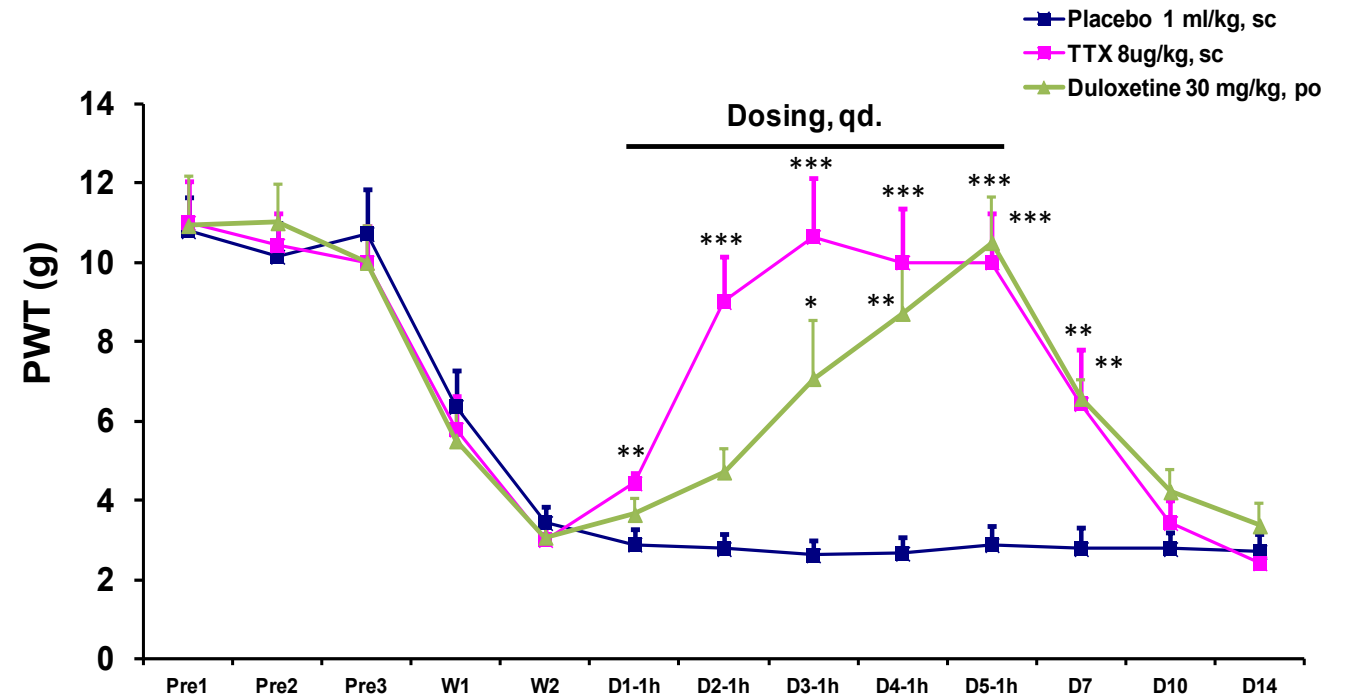
Source: Catterall WA, Goldin AL, Waxman SG. International Union of Pharmacology. XLVII. Nomenclature and structure-function relationships of voltage-gated sodium channels. Pharmacol Rev. 2005 Dec;57(4):397-409J; Channels 2(6): 407-412, 2008. Lee et al.; Osteen et al, Pain Ther, 2025

Preclinical Efficacy of Halneuron® in Rat Oxaliplatin-Induced Neuropathic Pain Study



- Adult male Sprague-Dawley rats.
- Oxaliplatin 4 mg/kg, injected intravenously, twice a week, repeated up to 9 times to induce mechanical allodynia.
- Paw withdrawal threshold (PWT) used as an indicator of neuropathy
 - PWT $\leq$ 4g were used as an indicator.
- The rats showing significant mechanical allodynia
 - TTX 8ug/kg or vehicle injected subcutaneously, q.d. for 5 days
- Duloxetine given orally, at 30 mg/kg, q.d.as active control (3,750 X the TTX dose)

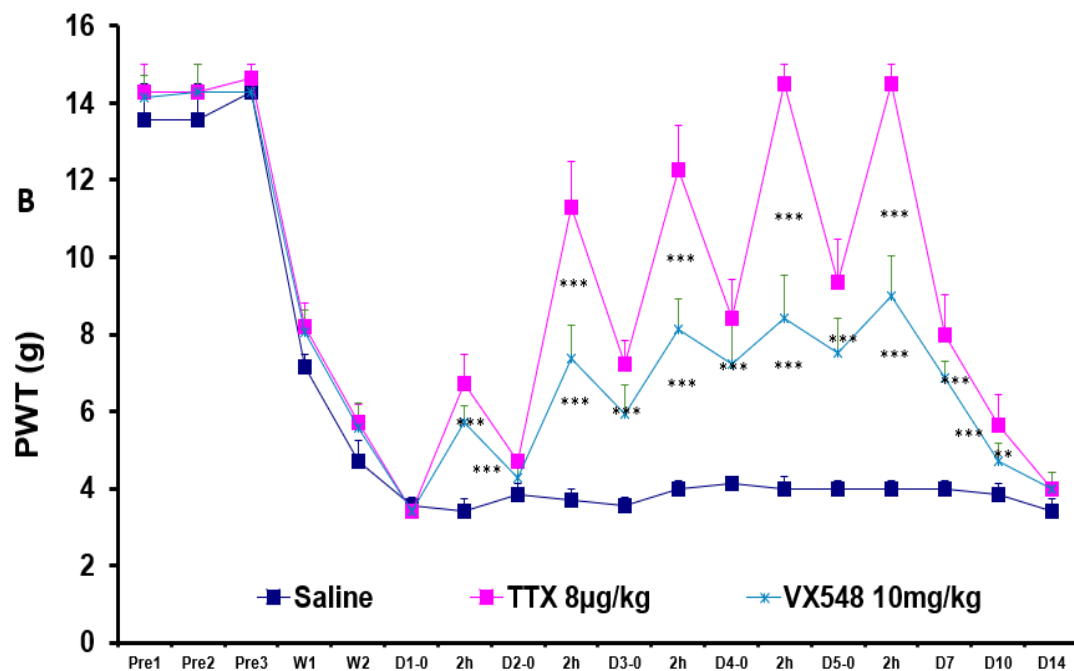
Halneuron Effect on PWT in Oxaliplatin Induced Rat Pain Model



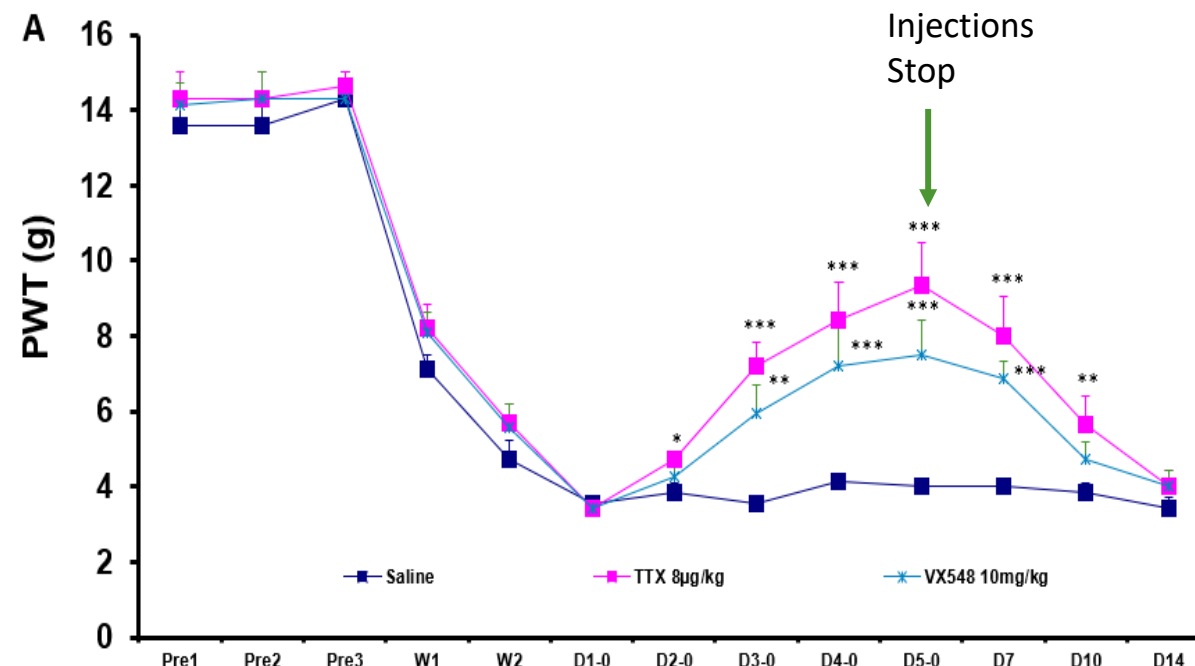
*, **, ***: p<0.05, 0.01, 0.001, respectively, compared to placebo group, one-way ANOVA, n=7

Halneuron® Pain Reduction in Preclinical Paw Withdrawal Model Compared to VX548 (TTX 1000 X lower dose)

Halneuron® Efficacy Compares Favorably with Recently Approved Suzetrigine at all Tested Doses



Halneuron® Efficacy Builds as Evidenced by Higher Pre-Dose Threshold Prior to Future Doses



Cancer Related Pain Phase 2 Study (n=165)

- Tested for efficacy and safety of Halneuron® for moderate to severe inadequately controlled pain post cancer therapy
 - Included neuropathic and non-neuropathic pain patients
- Randomized, double-blind, placebo-controlled, parallel-design, multicenter trial
- Statistically significant efficacy achieved based on a pain reduction endpoint
- On average, Halneuron® responders demonstrated pain relief for 57.7 days post injection
- Halneuron® showed an acceptable safety profile in cancer patients

Phase 2 CRP Study – Halneuron® Demonstrated Statistically Significant Pain Reduction



Cancer Related Pain – 8 Injections over 4 days – long term follow-up every 15 days after primary endpoint

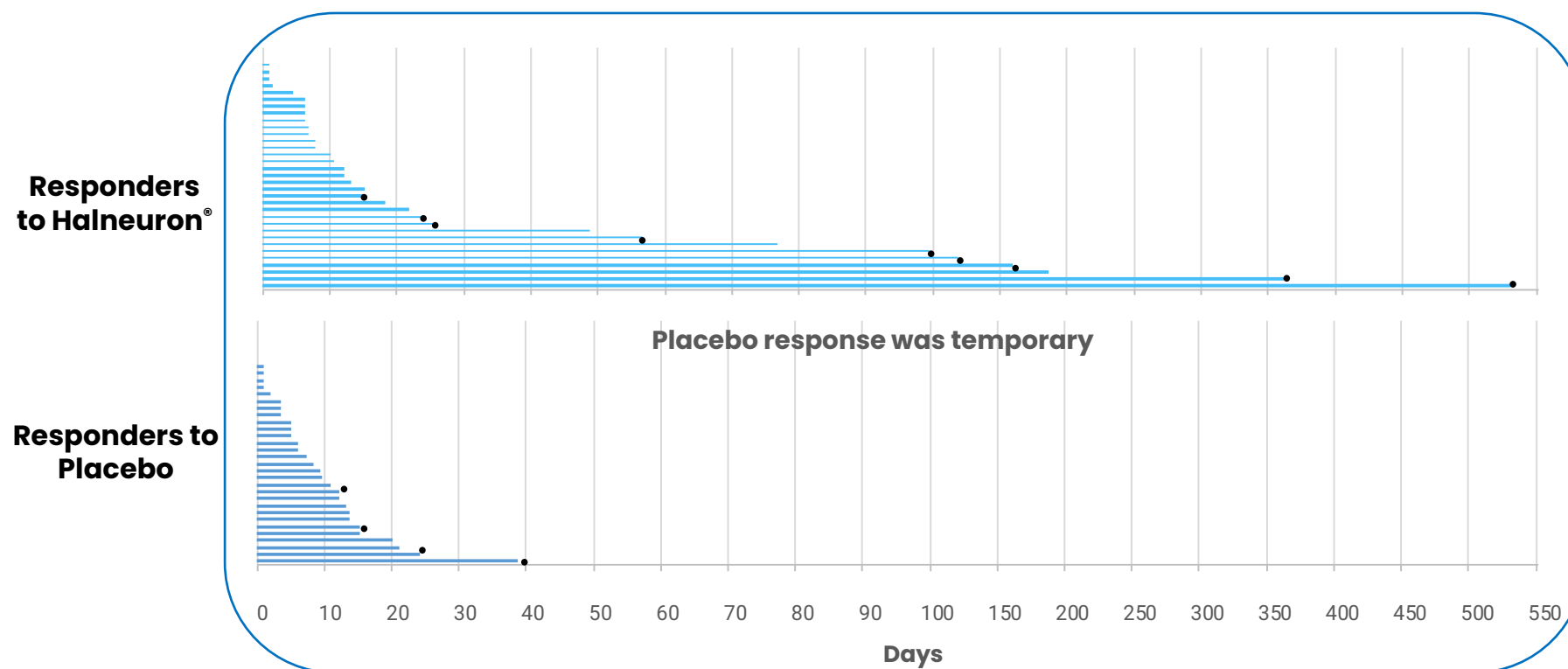
51% of patients on Halneuron® experienced a $\geq 30\%$ reduction in pain vs. 35% on Placebo

	TTX ¹		Placebo ²		Difference
Responder ³	33	51%	29	35%	16%
Non-Responder	32	49%	55	65%	
Total	65		84		
95% C. I.	0.4 - 32.1				
p-value	0.046				

A “Responder” was defined as a patient who had a mean reduction in pain intensity of $\geq 30\%$; or $\geq 50\%$ reduction in opioid use

Phase 2 CRP Study: Long Duration of Pain Relief for Initial Responders

Mean Pain Response For Halneuron® Responders Was 57.7 Days Vs 10.5 Days For Placebo Responders



- A “Responder” is defined as a patient who had a mean reduction in pain intensity of $\geq 30\%$ or a decrease of at least 50% of opioid use at endpoint
- One-in-four (27%) Halneuron® responders had pain relief for >30 days after 4 days of treatment

Previous Phase 2a Signal-Seeking Study to Treat Chemotherapy Induced Neuropathic Pain



Phase 2a Signal-Seeking Study (n=125)

- Randomized, double-blind, dose-finding, placebo-controlled, multicenter study evaluating efficacy and safety of Halneuron® in CINP patients
 - Tested 7.5 ug, 15 ug and 30 ug sub-Q injections in patients with neuropathic pain
 - Compared 30 ug BID x 4 days to 30 ug QD x 4 days
- Results:
 - 30 ug results superior to lower doses and placebo
 - 30 ug BID vs QD showed comparable efficacy (with half the total amount of drug delivered)
 - 30 ug QD demonstrated a superior adverse event profile to BID dosing
 - Halneuron® showed an acceptable safety profile in CINP patients, similar to that seen in CRP
- Conclusion: 30 ug dosed 1x day selected to advance to Phase 2b studies in CINP
 - Determined treatment 'effect size' used to power the Phase 2b study (i.e 0.4 units)

Halneuron® Adverse Event Profile

Most Frequent Adverse Events During Phase 2a CINP Study

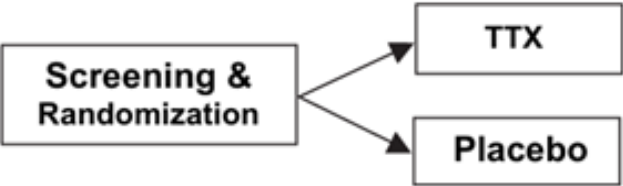
Adverse Event	TTX 30 µg QD	TTX 30 µg BID	Placebo
	x 4 days	x 4 days	x 4 days
	N=25 N (%)	N=26 N (%)	N=25 N (%)
Paraesthesia oral (tingling or prickling sensation in oral region)	10 (40.0%)	11 (42.3%)	3 (12.0%)
Hypoaesthesia oral (numbness or decreased sensation in oral region)	6 (24.0%)	10 (38.5%)	3 (12.0%)
Headache	1 (4.0%)	9 (34.6%)	5 (20.0%)
Dizziness	3 (12.0%)	8 (30.8%)	5 (20.0%)
Paraesthesia (tingling or prickling sensation in extremities)	5 (20.0%)	7 (26.9%)	6 (24.0%)
Nausea	1 (4.0%)	6 (23.1%)	6 (24.0%)
Fatigue	5 (20.0%)	3 (11.5%)	4 (16.0%)
Pain in extremity	4 (16.0%)	3 (11.5%)	2 (8.0%)
Dysgeusia (taste distortion)	2 (8.0%)	3 (11.5%)	0
Back pain	1 (4.0%)	3 (11.5%)	3 (12.0%)
Burning sensation	1 (4.0%)	2 (7.7%)	2 (8.0%)



**Selected Dose
for Phase 2b**

- Majority of AEs were mild to moderate in severity
- Most common AEs were expected and resolved naturally within a few hours after each injection
- No clinically significant impact on laboratory tests, vital signs, or ECGs was noted

*Ongoing Halneuron® 4-Week P2b CINP Study



Baseline	Week 1	Week 2	Week 3	Week 4
Run-in Period Avg. of Days -7 to -1	8 Halneuron® treatment injections spaced over 2 weeks			Primary Endpoint End of Study

- **Primary Objective of the 4-Week Phase 2b study**
 - To explore the safety and efficacy of Halneuron® in the treatment of patients with moderate-to-severe CINP
- **Primary Efficacy Endpoint**
 - Change from baseline at Week 4 in the weekly average of daily 24-hour recall pain intensity scores, comparing Halneuron® to placebo
 - Based on entries in e-diary implemented on personal smartphone
- **Secondary Efficacy Endpoints**
 - Patient Global Impression of Change (PGIC), PROMIS Fatigue, PROMIS Sleep, PROMIS-29, Pain Interference, Hospital Anxiety and Depression Scale (HADS), Neuropathic Pain Symptom Inventory (NPSI)
- **Target enrollment of 200 patients, subject to modification post Phase 2b interim analysis (projected in Q4 2025)**

New Synthetic API for Phase 3 and Commercialization

- Target CoM Filing Q4 2025
- Potential Extension to 2045+

Manufacturing Process and Trade Secrets

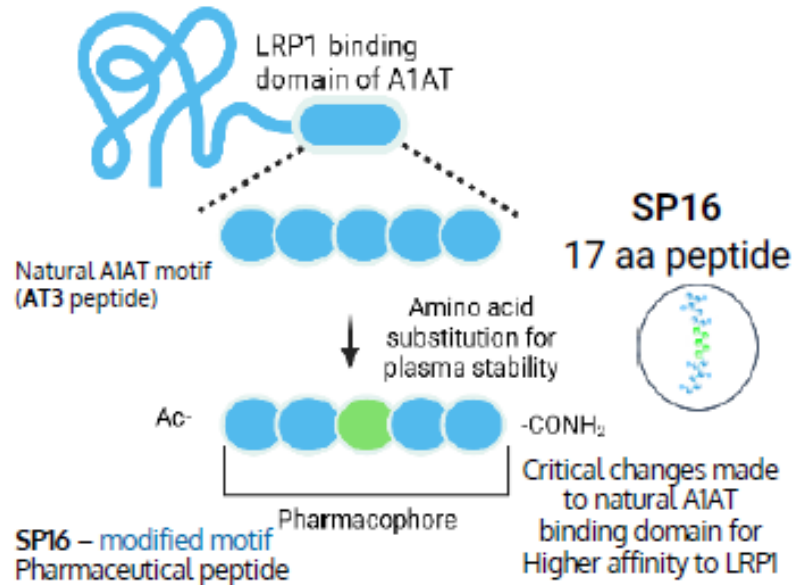
Method of Use

New Chemical Entity

SP16 IV CIPN Program Overview

SP16 Target Background

SP16 is a safe and natural solution to inflammatory disease

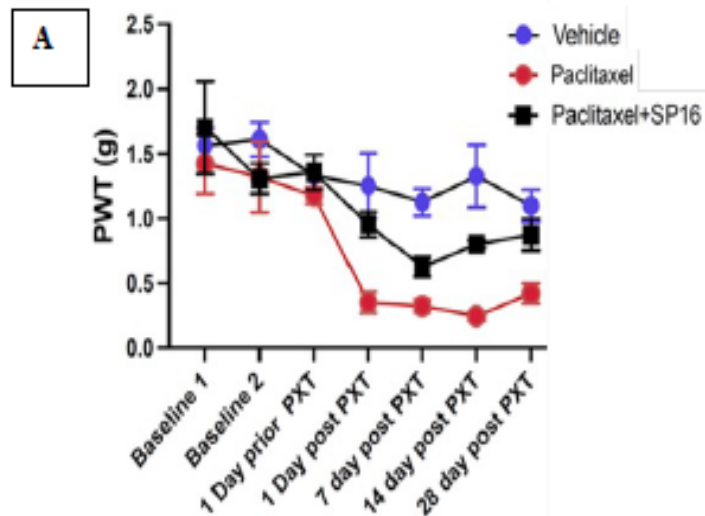


- Alpha 1 antitrypsin (A1AT) is a member of the serpin (serine protease inhibitor family) that plays a critical role in protecting the body from the damaging effects of powerful enzyme proteases
 - A1AT acts as an "off switch" or inhibitor for proteases including neutrophil elastase, preventing them from damaging healthy tissue
- Serpin the company has discovered the active portion of A1AT responsible for this activity
 - SP16 is a 17 amino acid peptide containing the active portion of A1AT activating LRP1 (low density lipoprotein receptor related protein-1)
- SP16 administered via IV formulation with two hypothesized actions:
 - Anti-inflammatory (analgesic) action via reduction of IL-6, IL-8, IL-1 β and TNF-alpha
 - Repairs tissue via increases in pAKT and pERK that regulate fundamental processes like growth, proliferation, and survival
- Human PoC is the next stage of SP16 development

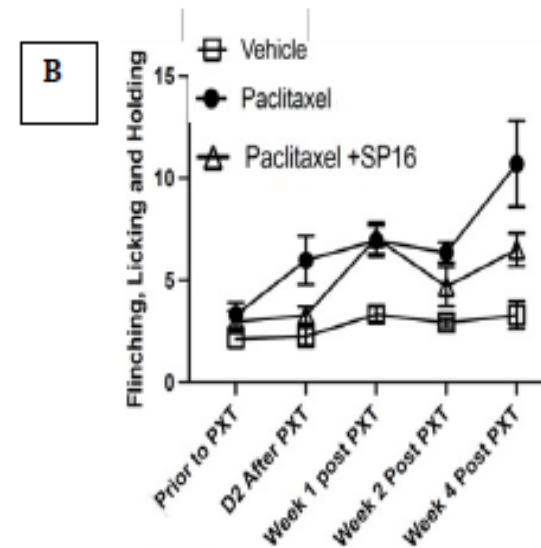
Preclinical Research Demonstrates SP16 Analgesic Effects

SP16 Reduced Both Mechanical and Cold Sensitivity in a Murine Model of Paclitaxel Induced Neuropathy

Von Frey Mechanical Hypersensitivity

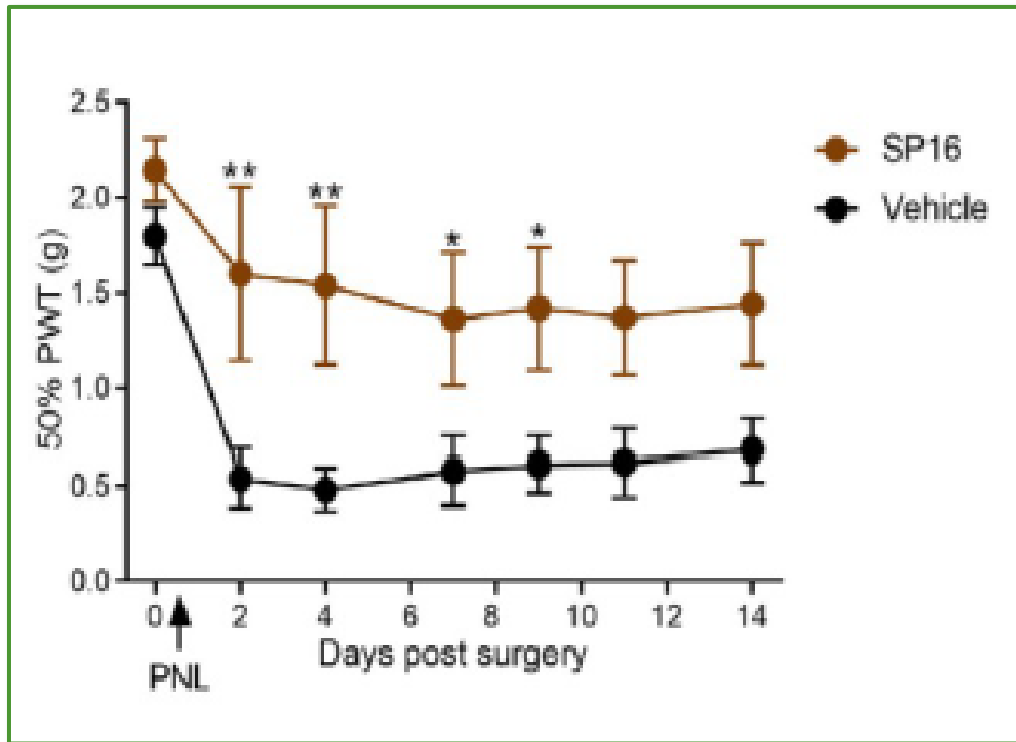


Thermal Allodynia



SP16 reduces sensory hypersensitivity in taxane induced model. C57BL/6 mice were administered PXT (4 mg/kg, IP) every other day for 8 days. Mice were treated with SP16 (2mg/kg, SC) or vehicle control at the start of PXT treatment and 3x/week for 3 weeks, dropping to 1x/week at the start of the 4th week. A) mechanical hypersensitivity (von Frey) and B) Cold allodynia (acetone test) was evaluated every week for 4 weeks. n=6 mice

SP16 Anti-inflammatory Effects Blocks the Development of Pain in Peripheral Nerve Injury Model



Study Highlights

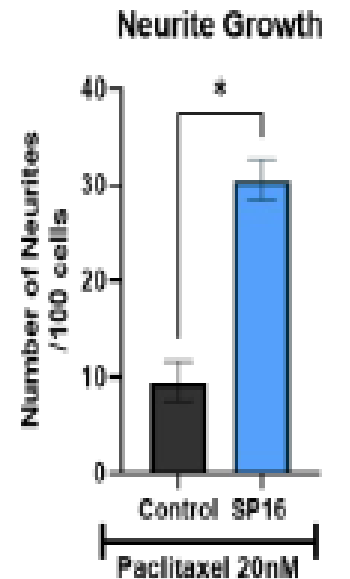
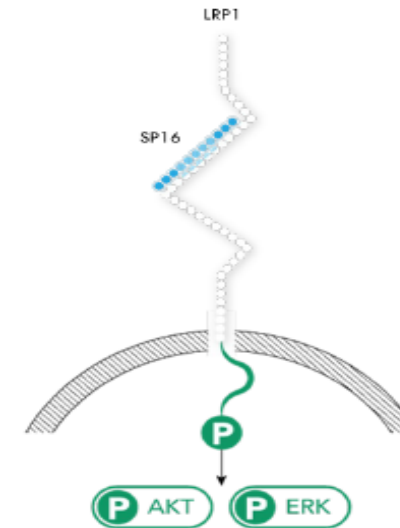
- Tactile allodynia develops after peripheral nerve ligation and are sustained for 14 days
- SP16(2μg/g) delivered daily (S.C.) significantly prevented the development of tactile allodynia (**p<0.01)

SP16 Exhibits Potential to Prevent and/or Repair Nerve Damage Associated Chemotherapy

- In collaboration with Dr. Wendy Campana at the University of California San Diego, SP16 was tested for its regenerative effects on neurons
- Neurotrophic effects of SP16 and associated increase in regenerative genes in neurons [Wang, 2022]
- SP16 was neuroprotective, activating neurite survival and growth, pro-regenerative genes and proteins, and protective signaling pathways
- SP16 significantly increased neurite growth in the presence of paclitaxel

Reparative Function of SP16

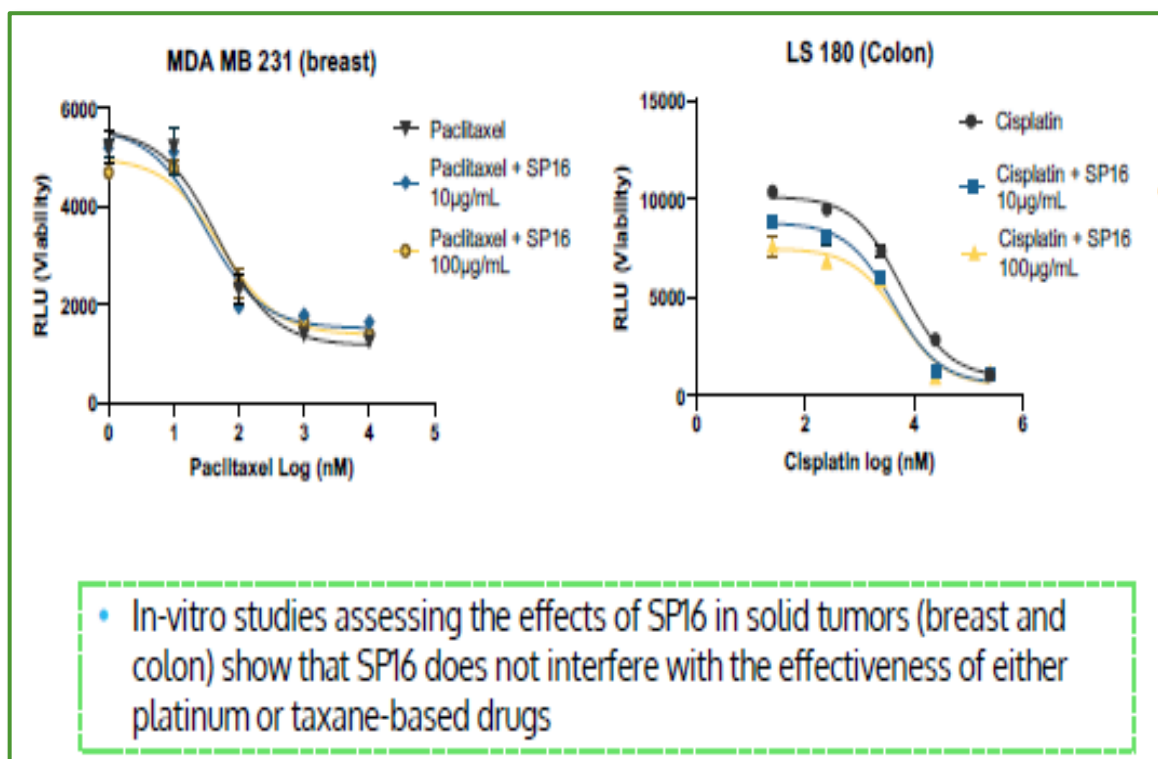
By activation of LRP1 dependent pathways (pAkt and pERK), SP16 treatment induces neurite survival and growth



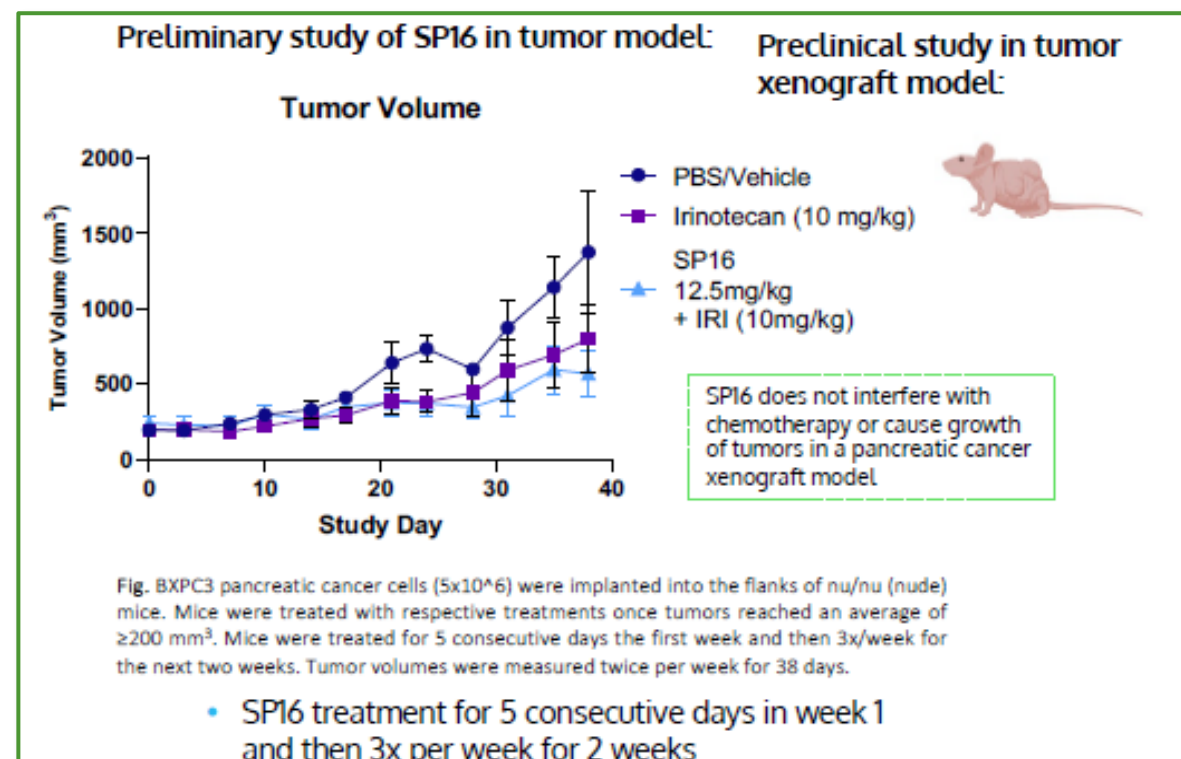
Source: Wang et al., 2021 *FASEB J*

In-vitro Assays in Several Cancer Types Shows SP16 Does Not Interfere with Common Chemotherapy Regimes

In-vitro assays in breast and colon cancer shows SP16 does not interfere with the effectiveness of either platinum or taxane drugs



In-vitro assays in pancreatic cancer cells shows SP16 does not interfere with the effectiveness of a topoisomerase 1 inhibitor

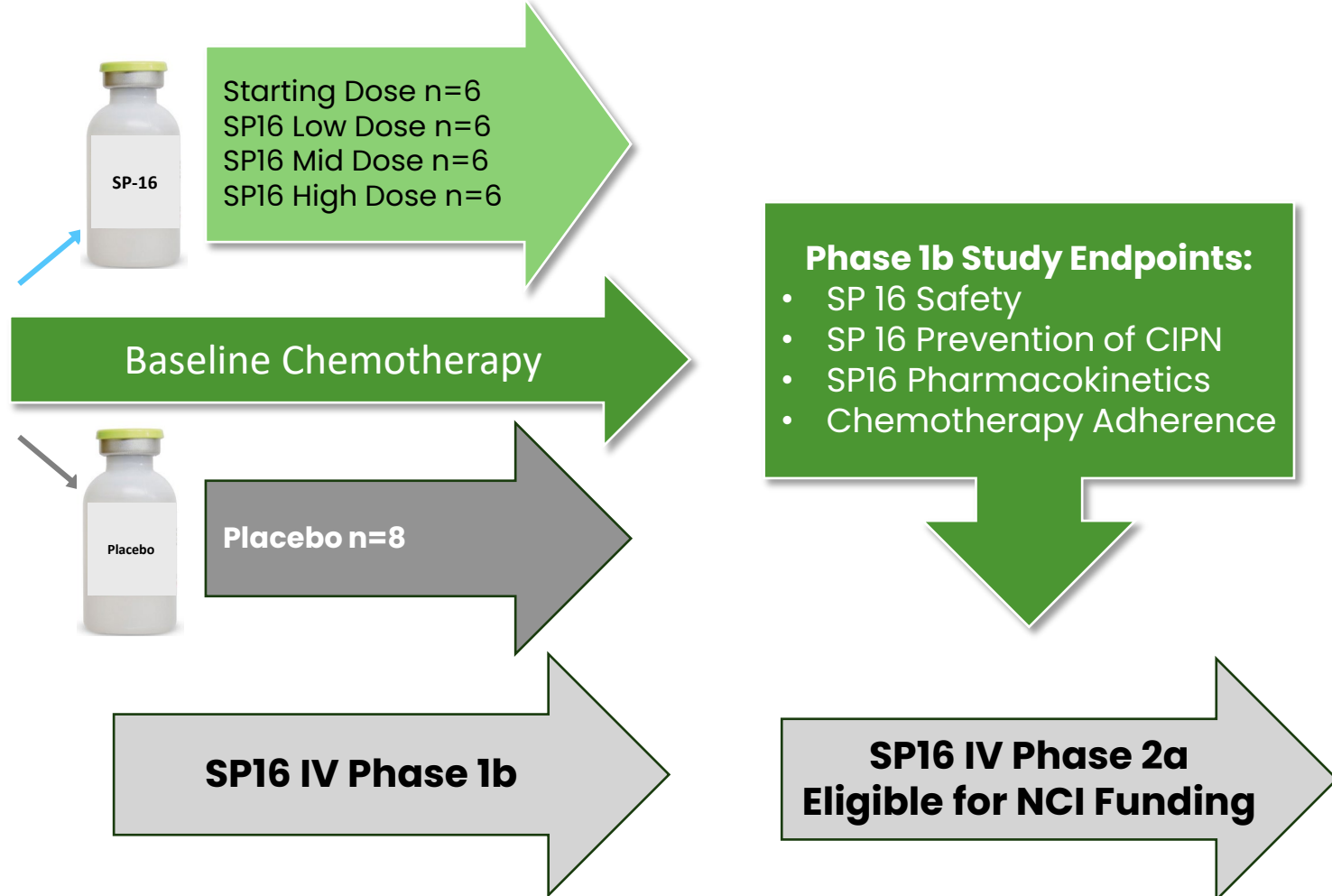


NCI Funded SP16 Research Plan to be Finalized with FDA and Executed at University of VA

NCI Funded Trial in collaboration with UVA

Patient Population

Up to 32 Metastatic Cancer
Patients Experiencing
Neuropathy from their
Concurrent Chemotherapy



- Halneuron® (TTX) = Nav1.7 channel blocker, analgesic → best for treating established CIPN pain
 - Halneuron® is in Phase 2b development for chemotherapy induced neuropathic pain
 - Opportunity to expand
 - Nav1.7 channel blocker should have utility on cancer related pain, as well as post surgical pain
- SP16 = LRP1-agonist, anti-inflammatory, neuroprotection → best for attenuation of CIPN during chemo;
 - SP16 is in early clinical stage development and may enable neuroprotection, may preserve full chemo regimen and potential to synergistically complement Halneuron® in treating pain post chemotherapy
 - SP16 Phase 1b development for CIPN
 - Program is fully funded by NCI through next clinical milestone
- Common commercial call points (oncology and pain centers) and potential partners
 - Bundled protocols/formularies with major cancer centers/providers
 - Co-promotion targeting infusion suites and pain clinics
 - Together deeper penetration into the global CIPN treatment opportunity ~\$1.5B market
 - Ability to expand into larger ~\$5B cancer pain market



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