DOGWOOD THERAPEUTICS

Advancing First-in-Class, Non-opioid Medicines to Treat Pain and Neuropathy

August 2026

NASDAQ: DWTX

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, 2025, 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.

Corporate Snapshot

Headquarters	Atlanta, GA
Nasdaq	DWTX
Shares Outstanding	33,992,553
Cash	$13.2 million March 31, 2026 Pro forma
Analyst Coverage	H.C. Wainwright Maxim Group
Pipeline History	Cancer and Chemotherapy Related Pain Represents A Major Unmet Medical Need Lead Asset Halneuron has Demonstrated Statistical and Clinically Meaningful Reduction in Cancer Pain Nav 1.7 Target Further Validated Genetic Defect Responsible for Congenital Insensitivity to Pain Syndrome Dogwood Therapeutics Executive Team has Developed or Commercialized Blockbuster Pain Medicines, Including Celebrex, Lyrica, Savella and Cimzia Halneuron Phase 2b Data Readout Fall 2026

Team Introductions

Greg Duncan
Chairman & CEO

R. Michael Gendreau MD, PhD
Chief Medical Officer

Angela Walsh
Chief Financial Officer

Ralph Grosswald
Senior VP, Operations

Scott Millard
VP Research

Jerry Evarts
VP Mfg

Proven Record of Drug Development & Commercial Success

Therapeutic Pain and Neuropathy Pipeline

Candidate	Indication	Preclinical	Phase 1	Phase 2	Phase 3	2026 Milestones
Halneuron® Injection	**Chemotherapy Induced Neuropathic Pain (CINP)** Fast Track Designation					Fall ’26 P2b Data Readout Q4’26 EOP2 FDA Submission
	General Cancer Pain					Completed P2 Study Demonstrated Statistically Significant Pain Reduction
	Acute Surgical Pain					
SP16 IV	**CIPPN** NCI Funded					IND Accepted by FDA Q3‘26 P1b Commences Enrollment

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Chemotherapy induced neuropathic pain (CINP), Chemotherapy Induced Neuropathic Peripheral Neuropathy (CIPPN)

The Problem: CIPN Patient Journey

Chemotherapy Induced Neuropathy Symptom Onset

Patients Experience

- Pain
- Numbness
- Tingling
- Sensitivity to cold
- Muscle weakness
- Balance disturbance, and others

Oncologist Cancer Treatment Considerations

- **Mild** – continue chemo + monitor
- **Moderate** – dose reduction/delay
- **Severe** – discontinue chemo

Pain Specialists Initial Commercial CINP Call Point

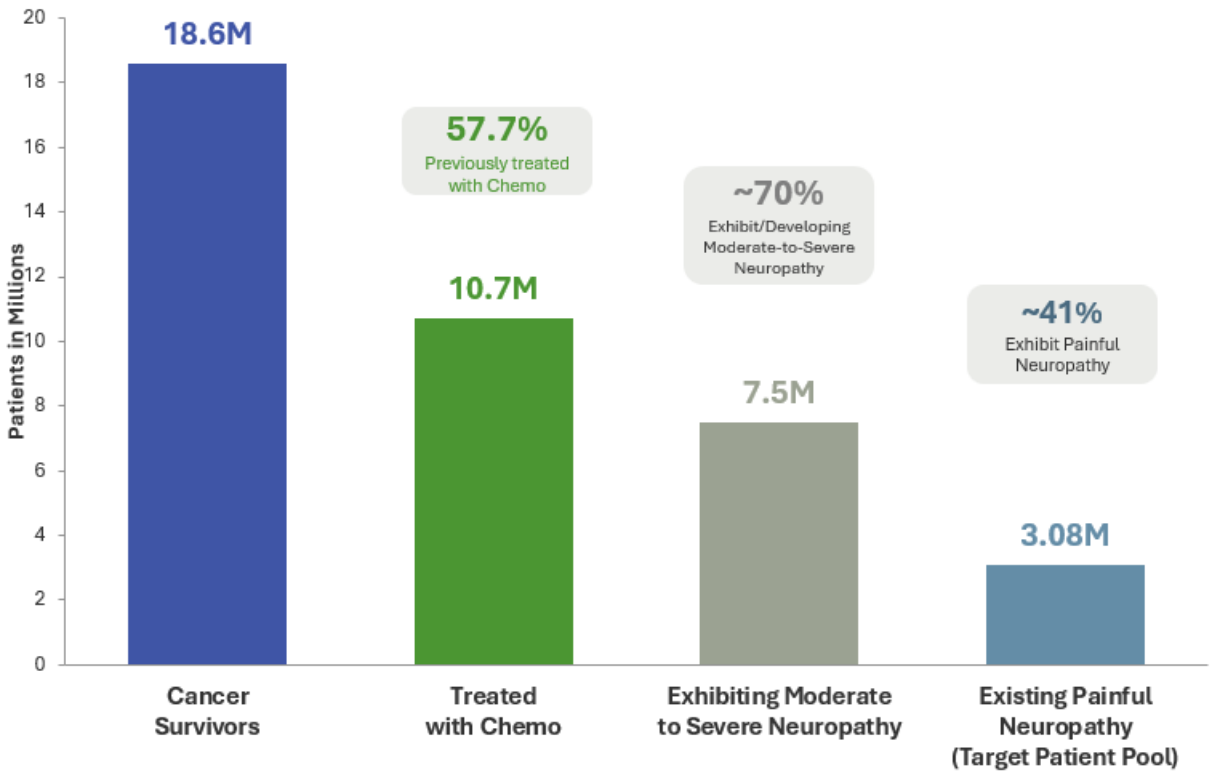
- Post chemo chronic pain patients referred to pain centers
- Pain specialist (i.e. Neurologist, anesthesiologists, etc.) treat “off-label” chronic pain meds (e.g. duloxetine, pregabalin, opioids)

Oncologists Represent Market Development Opportunity

- Oncologist treatment of pain represents a growth option
- Buy and bill model aligns well for injectable treatments
- LCM expansion to general cancer related pain

Moderate-to-Severe CINP Represents a Major Unmet Medical Need

There are > 3 Million Existing US CINP Patients



Annual Estimated Burden of New Cancer Type

Cancer Type	New Cases
Breast	313,310
Prostate	299,010
Lung	234,580
Colorectal	152,810
Skin	100,640
Bladder	83,190
Kidney	81,610
Non-Hod Lymphoma	80,620
Uterine	67,880
Pancreatic	66,440
Thyroid	44,020
Liver	41,630
Myeloma	35,780
Total New	**2,003,140**

2,003,140

57.7%
Treated of Cancer Patients Treated with Chemo

1,154,658

~70%
Exhibit/Developing Moderate-to-Severe Neuropathy

808,260

~41%
Exhibit Painful Neuropathy

331,387

New Moderate/Severe CINP Patients/Annum

Worldwide incidence of cancer is projected to grow by 53% by 2040

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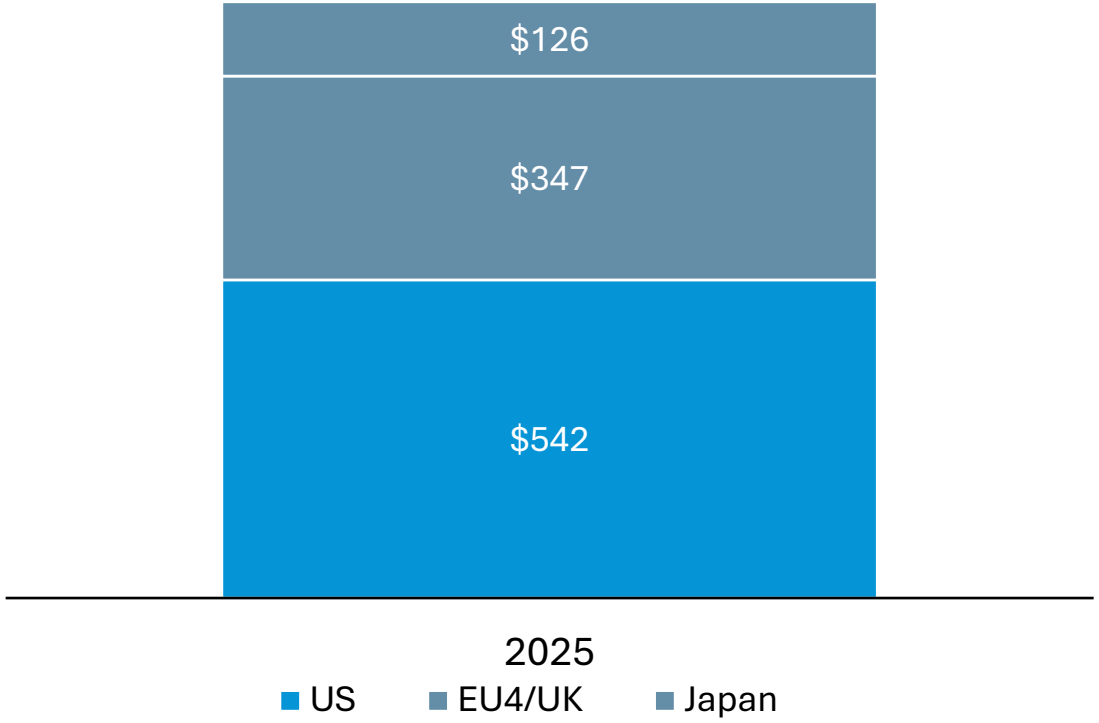
Sources: ACS Cancer Facts & Figures 2025; Sreeram 2023; D'Souza 2025;

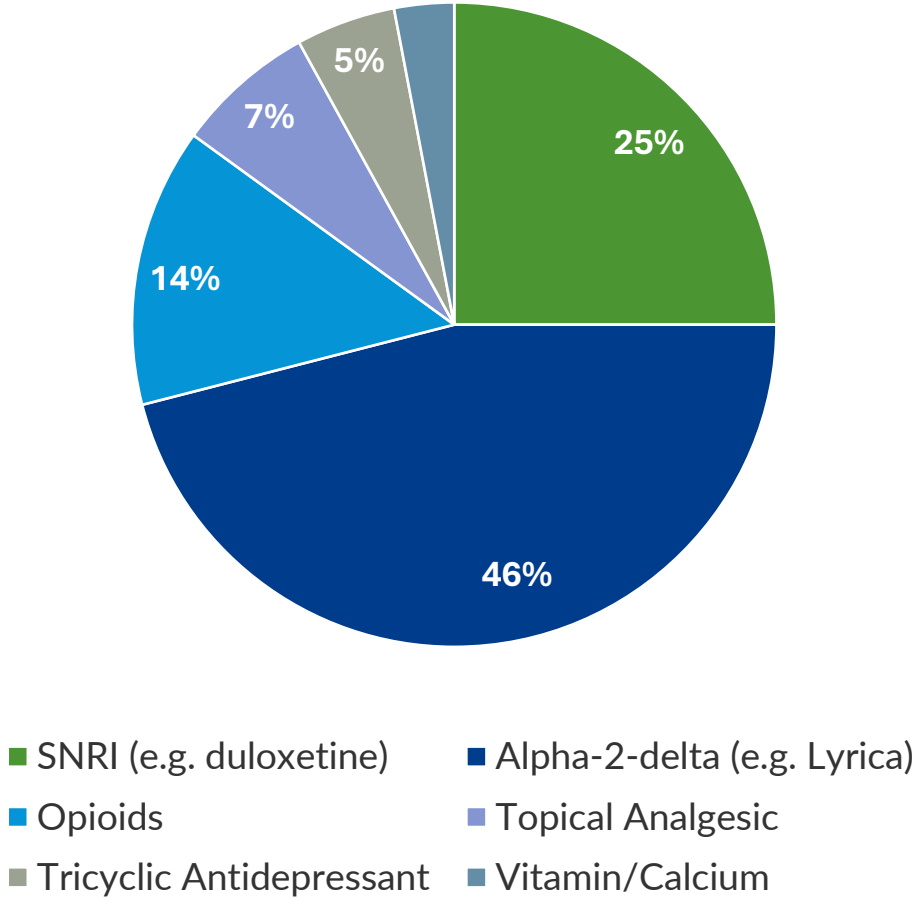
Significant CINP Treatment Market Across 7 Major Markets (7MM), Despite No FDA Approved Treatments

Total Market Sales ($M)

$1,015B



Market Share (%) by Therapy in the 7MM, 2026



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Sources: Delve Insight , 2025

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Halneuron® CINP Program Overview

Our Solution - Halneuron®

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

- Prevents or relieves pain by interrupting nerve conduction
- Modulates pain transmission in the peripheral nerves, no CNS effects
- Administered as a sub-Q injection
- Halneuron® treatment demonstrated statistically significant, durable pain reduction, with acceptable safety profile, in cancer and chemotherapy related clinical pain studies to date

How Does Halneuron® Work?

Halneuron® works as an analgesic by binding to the $Na_v1.7$, channel, a sodium channel responsible for pain signal transmission

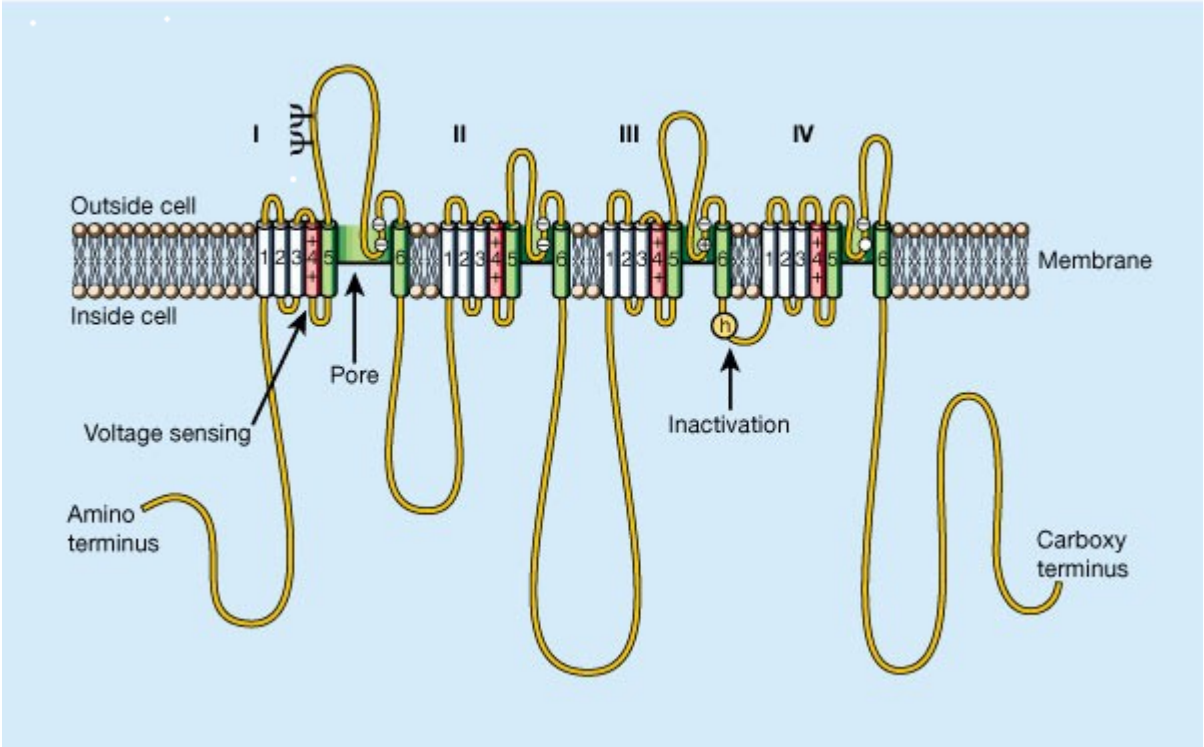
What are NaV channels

- Voltage gated sodium channels (NaV) comprises a family of integrated membrane proteins that conduct sodium ions from the extracellular space through a cell's membrane

- Structurally, they consist of alpha subunits that form a pore in the plasma membrane through which Na ions flow and beta subunits that exert a modulatory function on their activity

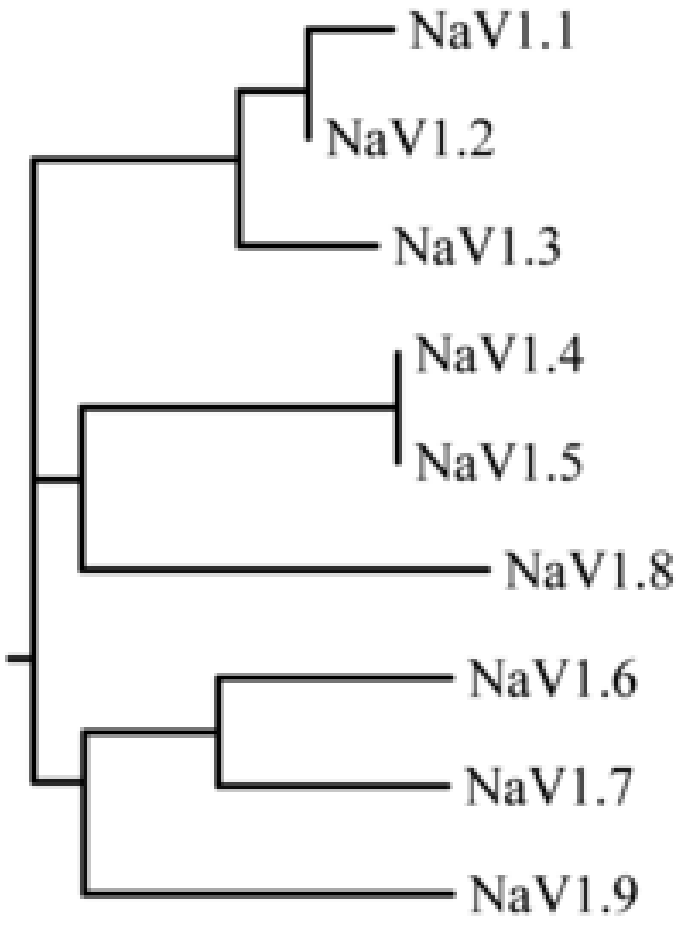
- The alpha subunit consist of 4 repeated domains (I-IV) each of which contains six (S1-S6) membrane-spanning segments; S4 serves as the voltage sensor and controls the opening of the channel in response to a change in the transmembrane voltage potential

- In excitable cells the opening of NaVs enable the rising phase of action potentials and their propagation



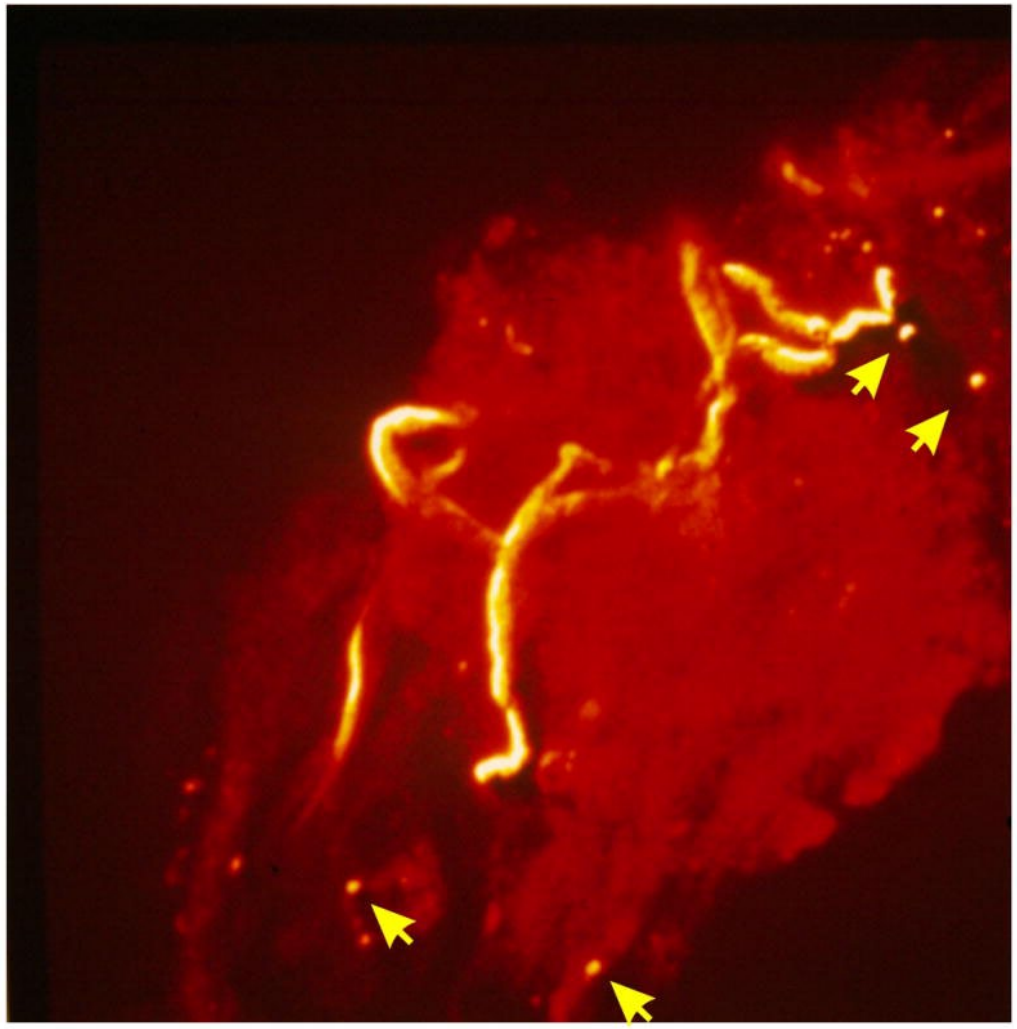
NaV isoforms are Differentially Expressed

- There are 9 NaV family members that differ in their structure, regulation and tissue distribution
- The NaV 1.x family are all expressed in the brain, whereas a few isotypes are also expressed in the periphery
  - NaV 1.5 in cardiac tissue
  - NaV 1.4 in skeletal muscle
  - NaV 1.7-1.9 dorsal root ganglia
- Dorsal root ganglia expression is a target tissue for peripheral pain modulation



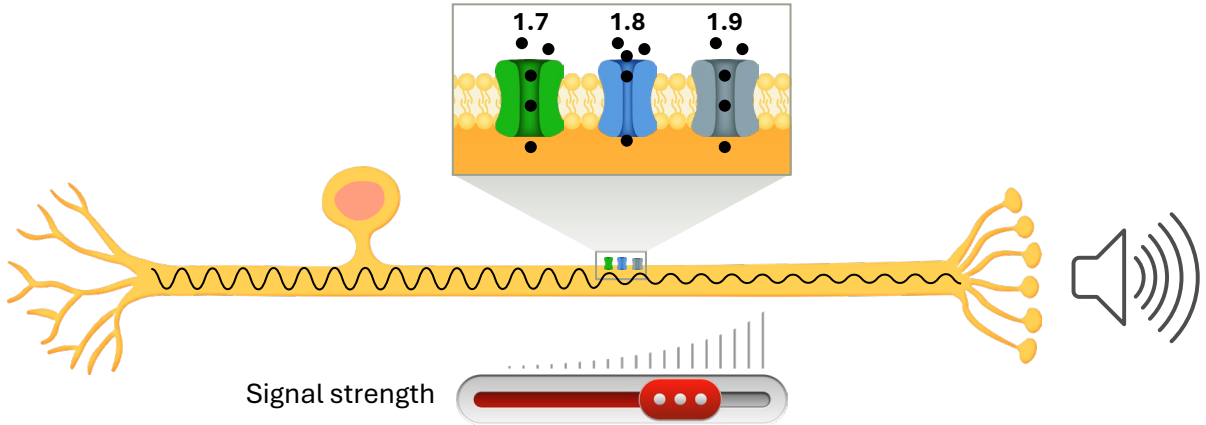
Role of NaV in pain

- Voltage gated sodium channels (NaV1.7-1.9) are important in the peripheral signaling of pain
- NaV1.7 is of particular interest because individuals with NaV1.7 loss of function mutations are congenitally insensitive to the perception of acute and chronic pain
- Non-central components to pain perception that are related to NaV1.7 activation include transduction of pain stimuli at peripheral nerve terminals and axonal transmission of action potentials to the CNS
- Chronic pain conditions- including inflammatory pain- are associated with an increase in expression levels of NaV1.7, pointing to its critical role in hyperalgesia and allodynia



Nav Sodium Channel Signaling

Nerve Damage from Chemotherapy Triggers Pain Signaling



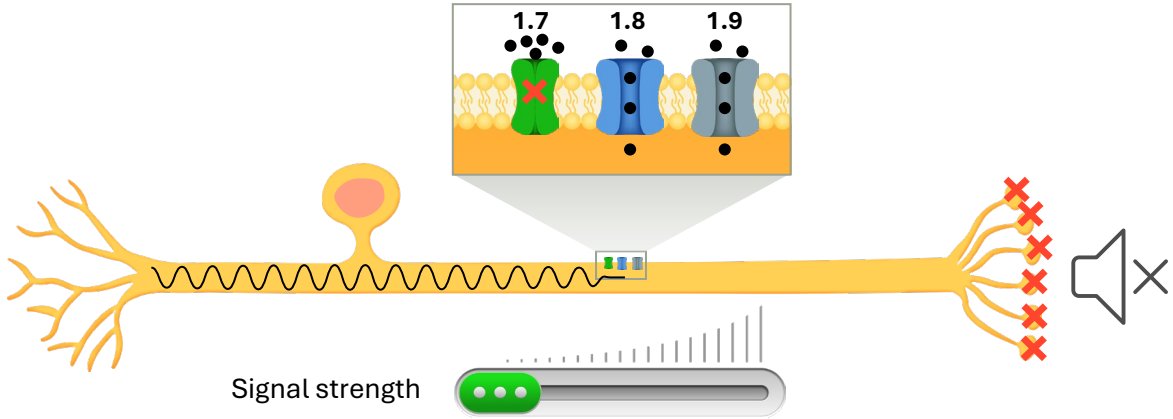
The Role of Nav Sodium Channels

Nav 1.7
Critical to setting the depolarization threshold, and therefore the probability of triggering an electrical signal required to transmit pain signals (“on/off switch“)

Nav 1.8
Acts as a "dimmer or amplifier “ thought to increase pain signaling duration or intensity

Nav 1.9
Thought to be involved in cold pain sensing and small fiber neuropathy.

Halnueron Nav1.7 Inhibition



What makes Halneuron different?

- Selectively inhibits Nav 1.7 sodium channel signaling rather than Nav 1.8
- Preclinical data demonstrates:
  - Pain reduction > Nav 1.8 inhibitor in preclinical rat model
  - No additional pain reduction effect when combining a Nav 1.8 inhibitor with Halneuron, highlighting importance of the Nav 1.7 target

Mammalian Sodium Channel Isoforms

Channel	TTX Sensitivity	Predominant Distribution
$Na_v1.7$	**EC_{50} = 24.5 nM**	**PNS (DRG)**
$Na_v1.8$	EC_{50} = 60,000 nM	PNS (DRG)
$Na_v1.9$	EC_{50} = 40,000 nM	PNS (DRG)
$Na_v1.4$	EC_{50} = 25 nM	Skeletal Muscle
$Na_v1.5$	EC_{50} = 5,700 nM	Heart
$Na_v1.1$	EC_{50} = 6 nM	CNS
$Na_v1.2$	EC_{50} = 18 nM	CNS
$Na_v1.3$	EC_{50} = 4 nM	CNS
$Na_v1.6$	EC_{50} = 6 nM	CNS

- TTX preferentially acts to block pain signals through $Na_v1.7$
- TTX unable to cross the blood-brain barrier
- Relief of chronic pain without the common CNS side effects

Loss of NA_V 1.7 Function Leads to Congenital Insensitivity to Pain Syndrome

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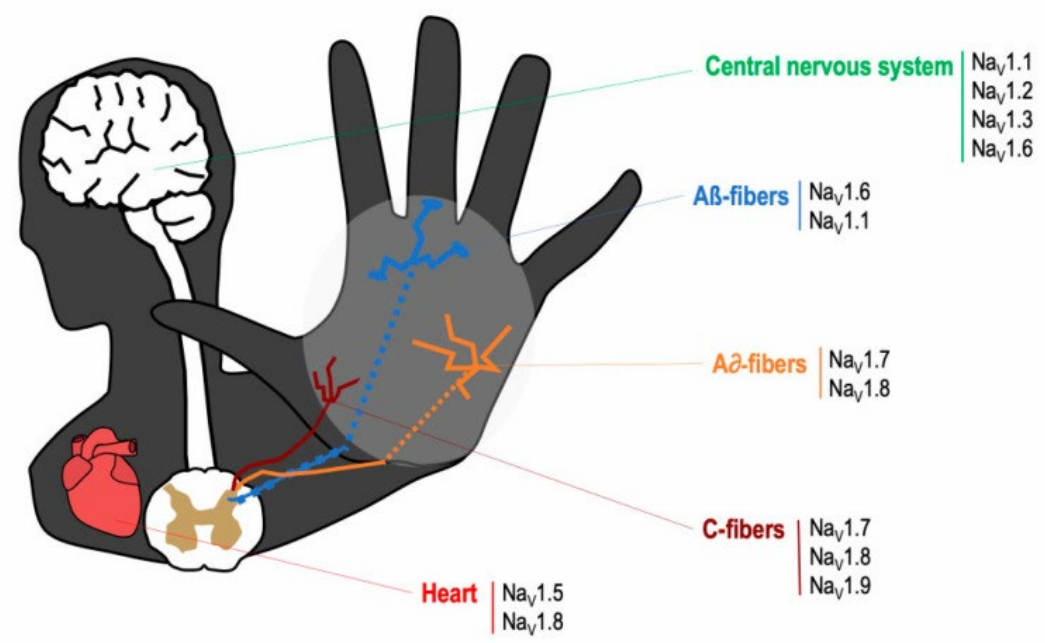
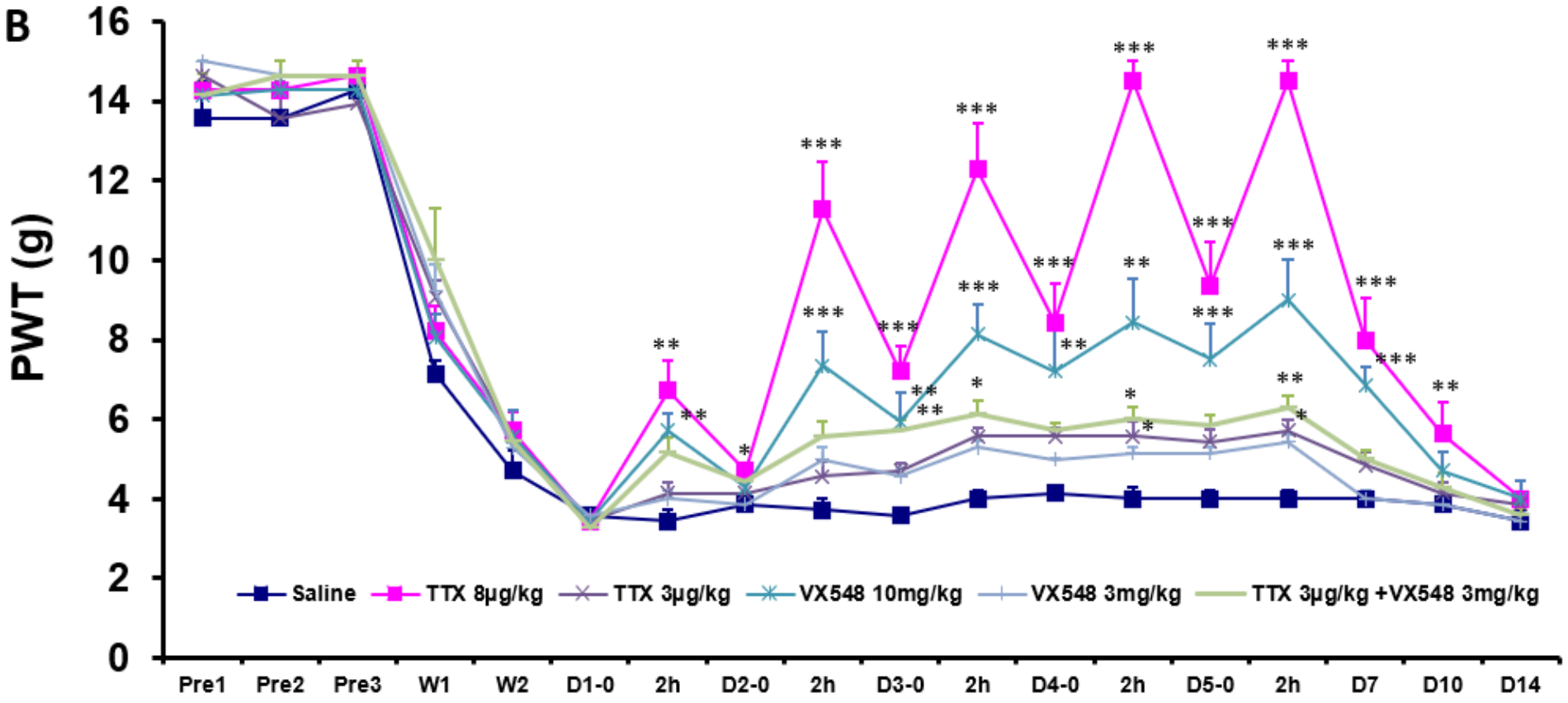
Channels 2(6): 407-412, 2008. Lee et al.

Validation of TTX as a potential Best in Class Analgesic

- Preclinical studies involving local as well as sub-chronic administration demonstrated analgesic effects in a variety of animal models
- Multiple clinical studies have been performed with TTX in cancer patients as well as in patients with chemotherapy-induced neuropathic pain
- All studies reported durable efficacy in subjects who demonstrate an initial response
- TTX was generally well tolerated with oral hypoesthesia and paresthesia (numbness or tingling in the oral region) as the principal reported AEs



Halneuron® Pain Reduction in Preclinical Paw Withdrawal Model > VX548 at all High, Medium and Low Doses



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

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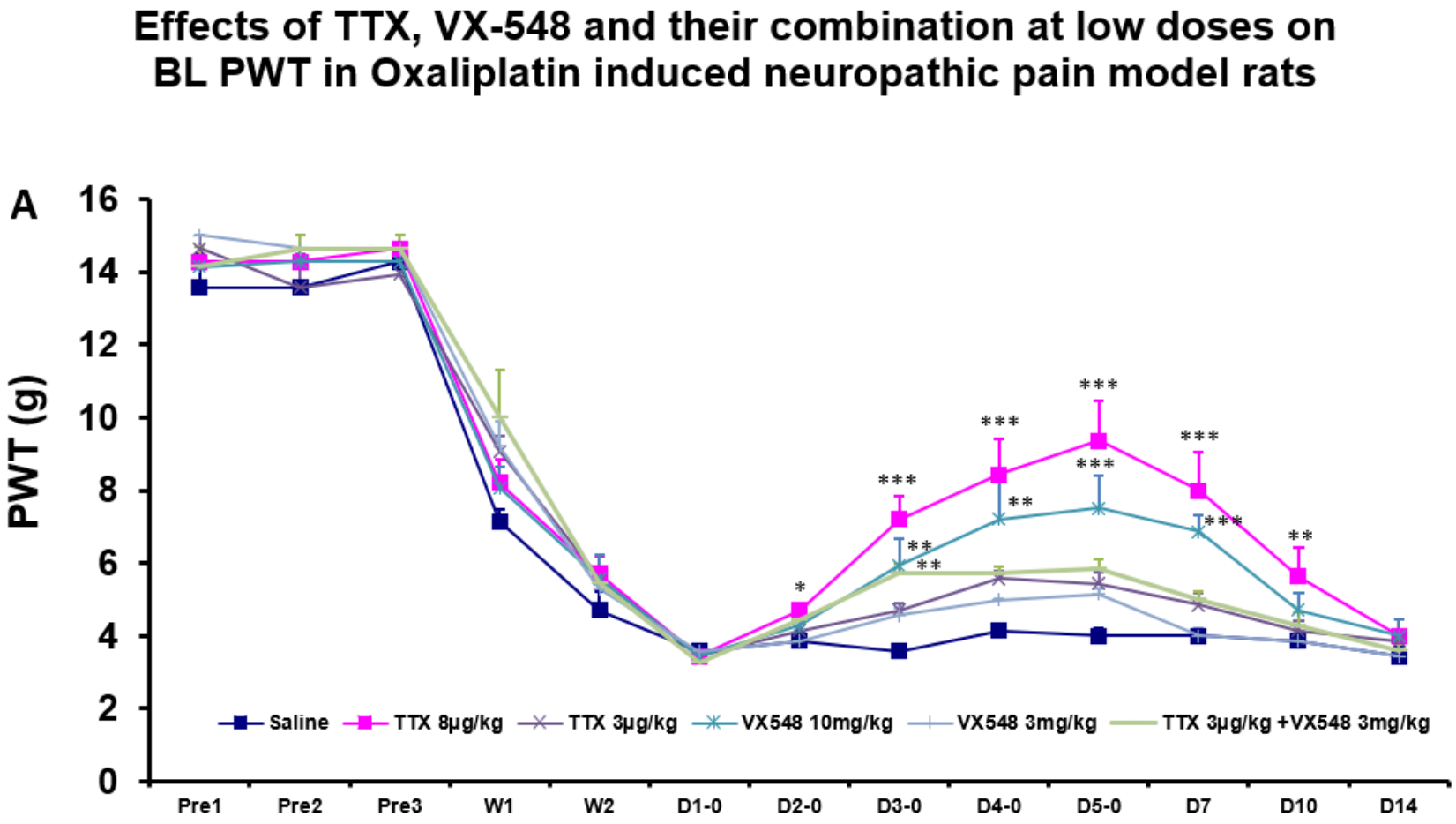
Halneuron® Efficacy Builds as Evidenced by Higher Pre-Dose Threshold Prior to Future Doses (i.e. D2-0, D3-0, D4-0 and D5-0)



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

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Halneuron® Demonstrated Statistically Significant Pain Reduction in Phase 2 Study

Compared

30 µg BID x 4 days

Placebo BID x 4 days

A "Responder" was defined as a patient who had a mean reduction in pain intensity of ≥ 30%; or ≥ 50% reduction in opioid use

Phase 2 Cancer Related Pain (n=165) – 8 Injections over 4 days – long term follow-up every 15 days after primary endpoint

	TTX		Placebo		Δ
Responder	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**				

Conclusions

Halneuron® treatment demonstrated statistically significant reduction in cancer related pain

Halneuron® treatment reduced both neuropathic and non-neuropathic pain

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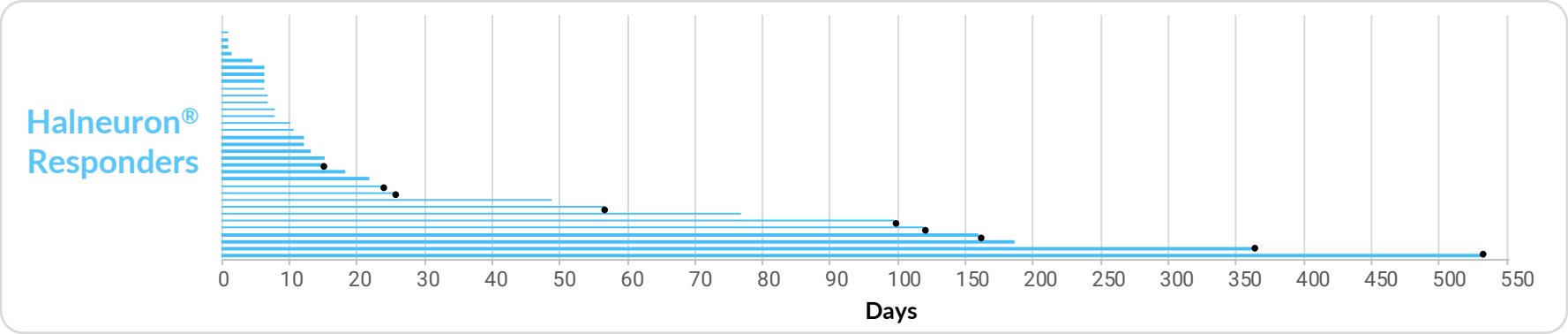
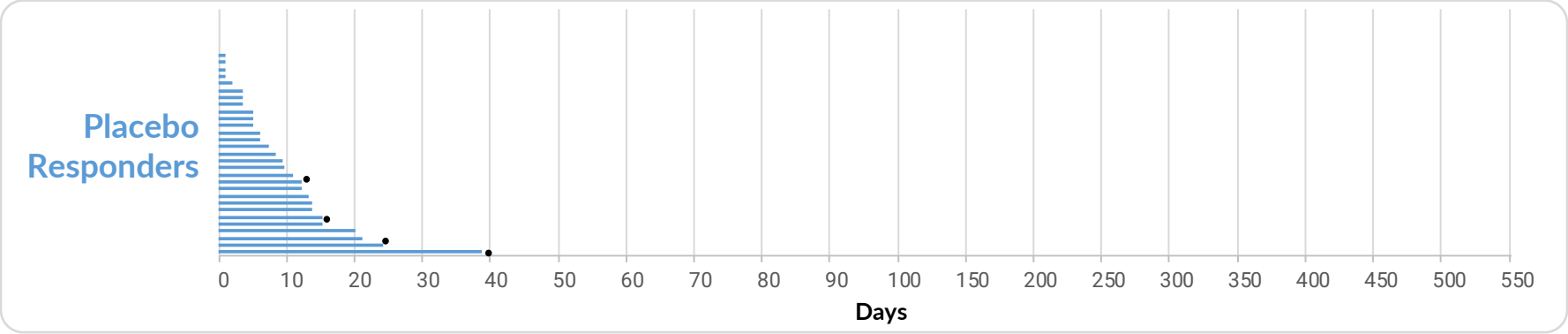
Phase 2 CRP Study Demonstrated Durable Pain Relief in Initial Responders

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



One-in-four (27%) Halneuron® responders had pain relief for >30 days after 4 days of treatment



Placebo response was temporary

A "Responder" was defined as a patient who had a mean reduction in pain intensity of ≥ 30%; or ≥ 50% reduction in opioid use

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Phase 2a Achieved Efficacy and Identified Optimal Dose for Phase 2b

Randomized, double-blind, dose-finding, placebo-controlled, multicenter study evaluating efficacy and safety of Halneuron® in CINP patients (n=125)

Doses Tested

sub-Q injections in patients with neuropathic pain

- 7.5 µg
- 15 µg
- 30 µg

Compared

- 30 µg BID x 4 days
- 30 µg QD x 4 days
- 15 µg BID x 4 days
- 7.5 µg BID x 4 days
- Placebo x 4 days

Results

- 30 µg results superior to lower doses and placebo
- 30 µg BID vs QD showed comparable efficacy (with half the total amount of drug delivered)
- 30 µg 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

Conclusions

30 µg 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) through interim assessment

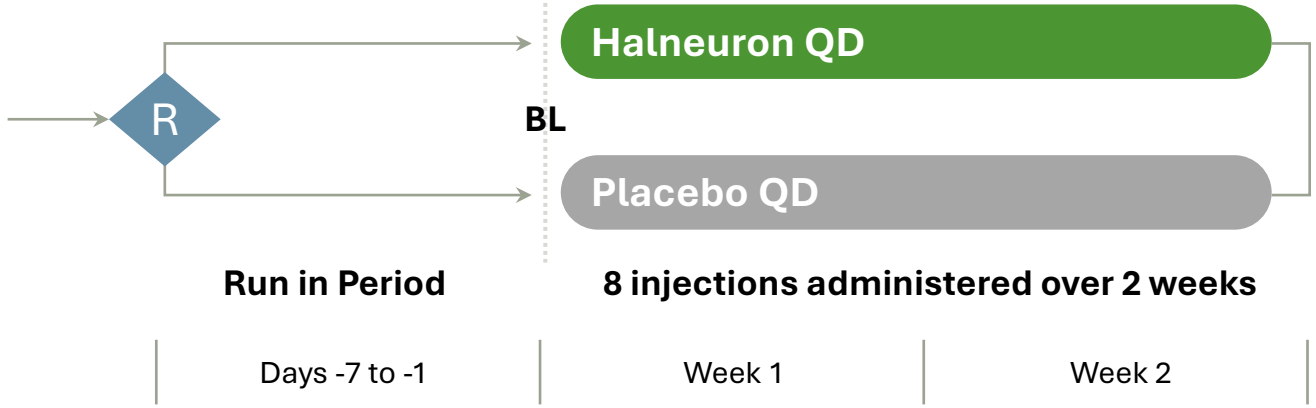
Phase 2b CINP Study De-Risked with Interim Analysis, Robust Statistical Power

4 Week Phase 2b Study

Primary Objective to explore the safety and efficacy of Halneuron® in the treatment of patients with moderate-to-severe CINP

Screening & Randomization

Treatment Phase



Endpoints (Week 4)

- Change from Baseline in the weekly average of daily 24-hour pain
- Incidence of SAEs, AEs, and AESIs

EOS

Final Phase 2b Study (HALT-CINP-203) Data Projected Fall 2026

Interim Results

- Completed Phase 2b interim analysis for sample size and to choose the analytical method for the primary analysis for the statistical analysis plan
- Committee consists of two senior statisticians not otherwise involved with our study
- Interim analysis based on change in pain from baseline compared to placebo:
  - Statistical methodology based on >50% responder analysis
  - Sample size required to maintain 80-85% statistical power ranges from 210 to 240 patients

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Phase 2b Interim Analysis Also Demonstrates Efficacy Signal, Durable Response in Refractory CINP

Key Highlights (n=97)

- **Halneuron demonstrating treatment effect versus placebo**
  - Responders exhibit durable treatment pattern
  - Halneuron treatment effect +/- concomitant pain meds also > placebo
  - Halneuron drop out rate (~4.4%) far below other compounds: duloxetine (20+%), pregabalin (30-40%)
- **Halneuron treatment effect noteworthy given:**
  - 5-year (mean) duration of moderate-to-severe neuropathic pain for Ph2b enrollees
  - Includes refractory CINP patients treated with other chronic pain medicines (67%)
- **First randomized control clinical trial to demonstrate pain reduction effect in CINP patients conducted under FDA chronic pain guidance**
  - Second cancer related pain trial exhibiting pain reduction via responder analysis
- **Final Data Projected Fall 2026**

Halneuron® Drop-out rate is <5% with Manageable AE Rates Across CINP Studies

Adverse Event (AE)	CINP 201 TTX 30 µg QD x 4 days N=25 N (%)	CINP-201 Placebo x 4 days N=25 N (%)	CINP-203 Currently Blinded Summary: TTX 30µg QD x 8 days Combined Drug + Placebo N=125 (%)
Paresthesia oral (tingling or sensation in oral region)	10 (40.0%)	3 (12.0%)	**51 (41%)**
Hypoesthesia oral (numbness or decreased sensation in oral region)	6 (24.0%)	3 (12.0%)	**24 (19%)**
Paresthesia (tingling or sensation in extremities)	5 (20.0%)	6 (24.0%)	**23 (18%)**
Headache	1 (4.0%)	5 (20.0%)	**17 (14%)**
Nausea	1 (4.0%)	6 (24.0%)	**16 (13%)**
Fatigue	5 (20.0%)	4 (16.0%)	**9 (7%)**
Dizziness	3 (12.0%)	5 (20.0%)	**6 (5%)**
Dysgeusia (taste distortion)	2 (8.0%)	0 (0%)	**4 (3%)**
Pain in extremity	4 (16.0%)	2 (8.0%)	**2 (1.6%)**
Burning sensation	1 (4.0%)	2 (8.0%)	**2 (1.6%)**
Back pain	1 (4.0%)	3 (12.0%)	**0 (0%)**

Halneuron has appeared to be safe and well tolerated across multiple clinical trials to date, with oral paresthesia being the most common, yet transient adverse effect

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Advantages of New Synthetic Manufacture of Halneuron® 30 ug for Injection

- Scalability - synthetic process not limited by raw material availability. Starting material is a common sugar.
- Faster and greater control over the manufacturing process.
- Absence of bioburden, largest impurity of natural TTX.
- Toxicity enters in last step of >30 chemical transformations. High potency only for last step and purification.
- Purity is unrelated to environment.
- Utilizes robust purification technique (IP) developed for naturally sourced tetrodotoxin.
- New IP developed for purification of crude TTX and concentration of purified fractions filed in 2025.
    - If granted, may give Composition of Matter and Process protection until 2045
    - Process for the Synthesis of Tetrodotoxin and Intermediates Thereof
    - Intermediates and Process for the Synthesis of Tetrodotoxin

Halneuron® Intellectual Property

Patent Name	Type	App/Patent #	Date Filed	Anticipated Expiry
Stable Pharmaceutical Composition of Freeze-Dried Tetrodotoxin Powder	Composition of Matter	8,124,608	7/14/2004	8/4/2028
Use of Sodium Channel Blockers for the Treatment of Neuropathic Pain Developing as a Consequence of Chemotherapy	Method of Use	9,018,222	3/26/2007	3/18/2030
Sodium Channel Blocking Compounds Tetrodotoxin Galactopyranosides	Composition/Method of Use	8,486,901	3/26/2010	4/7/2030
Tetrodotoxin Liquid Formulations	Composition of Matter	18/556,683	4/22/2022	2042
Process For the Extraction and Purification of Tetrodotoxin	Process	18/880,674	4/26/2023	2043
Process for the Synthesis of Tetrodotoxin and Intermediates Therefor	Synthetic Mfg Process	63/672,146	11/28/2025	2046
Intermediates and Process for the Synthesis of Tetrodotoxin	Composition of Matter/Process		11/28/2025	2046

Halneuron Offers Potential Relief for Millions Pain Sufferers in the US Alone

Mild CINP
~142,000 New Patients/Annum[1]

Diabetic Peripheral Neuropathy
~716,000 New Patients/Annum[2]

Halneuron Initial Approval
for **~4 Million** Moderate-to-Severe CINP Patients in 2030

Acute Pain Associated with Ambulatory Surgery
~6 Million Patients/Annum[3,4]

Chemo Pain Prevention
~2 Million New Patients/Annum[5]

1. Sreeram 2023; 2. CDC 2026, Current Diabetes 2026; 3. JAMA Surgery 2023, Canadian Jrnl Anesthesiology, '04; 4. Cancer Journal Anesthesiology 2024; 5. American Cancer Society 2026

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SP16 IV Therapy

Chemotherapy Induced Pain & Peripheral Neuropathy (CIPPN)

Program Overview

SP16 Mechanism Allows for Healing During Inflammation

A1AT Target

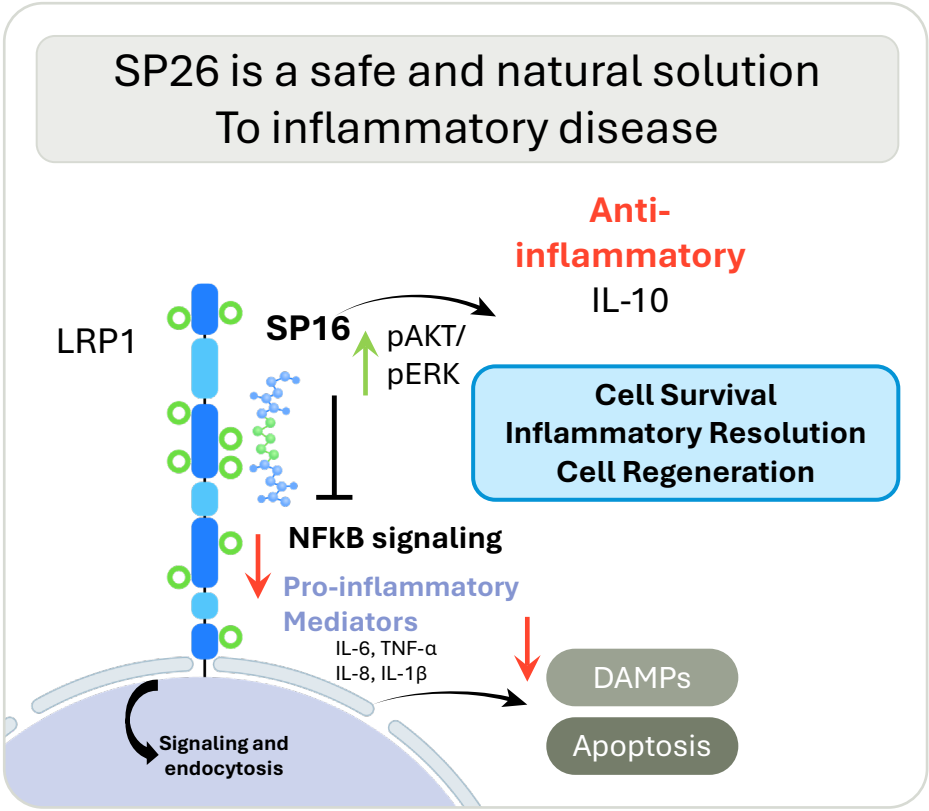
- A1AT (serine protease inhibitor family) regulates proteases to protect healthy tissue
- Dysregulated protease activity drives inflammation and tissue damage

SP16 Design

- 17–amino acid peptide derived from active region of A1AT
- Selectively activates LRP1 (LDL-receptor related protein 1) to drive anti-inflammatory and reparative signaling to restore immune balance

Hypothesized Outcomes:

- Anti-inflammatory: reduces IL-6, IL-8, IL-1β, TNF-α to decrease inflammation and pain
- Tissue repair: increases pAKT / pERK signaling to support cell growth



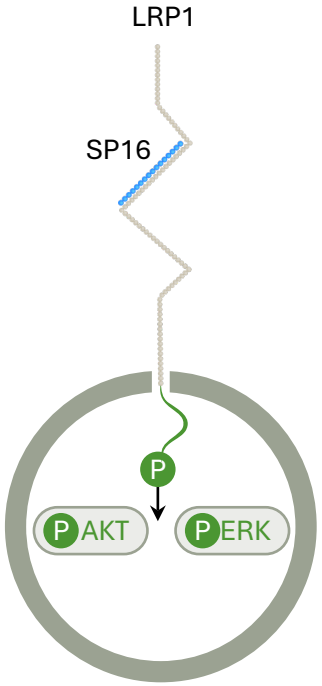
Dual mechanism anticipated to reduce inflammation while promoting tissue repair

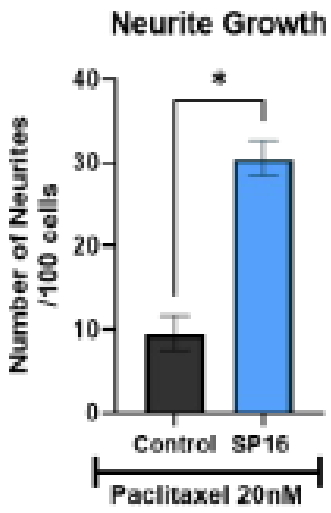
SP16 LRP1 Agonism Exhibits Potential to Prevent and/or Repair Nerve Damage Associated Chemotherapy

- 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

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

Patient Population

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

University of Virginia

NIH NATIONAL CANCER INSTITUTE

SP16 IV Phase 1b

SP16 Low Dose n=8

SP16 Mid Dose n=8

SP16 High Dose n=8

Placebo n=8

Baseline Chemotherapy

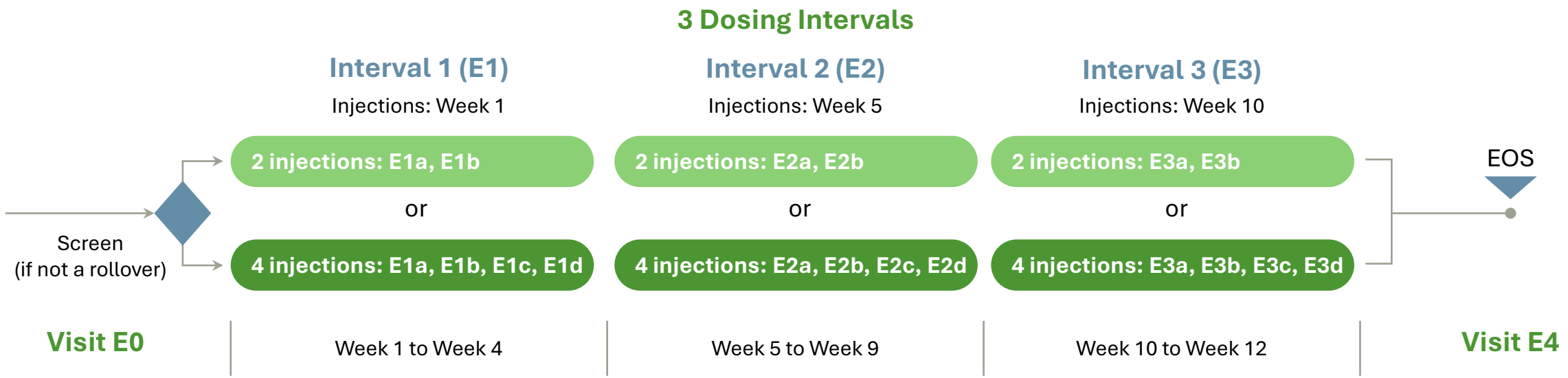
Phase 1b Study Endpoints

- SP 16 Safety
- SP 16 Prevention of CIPN
- SP16 Pharmacokinetics
- Chemotherapy Adherence

SP16 IV Phase 2a
Eligible for NCI Funding

Open Label Extension Study Design

12 Week Open Label Study



- Patients will continue daily pain diaries entry scores
- IRT will assign either 2 or 4 injections for the interval, based on 7-day average pain scores
- Halneuron® dosing as follows:
  - NRS Pain Change > 4 gets 4 injections at treatment interval, or
  - NRS Pain Change <3 gets 2 injections at treatment interval

Commenced Enrollment May 2026

A First-in-Class Approach to Pain and Neuropathy

The Problem of Neuropathic Pain

- Neuropathy includes chronic pain, numbness, tingling, and poor coordination, with significant impact on QoL
- ~7.5 million patients suffer from moderate–to-severe chemotherapy induced peripheral neuropathy (CIPN)
- ~3 million patients suffer from painful chemotherapy induced neuropathic pain (CINP)

Lead Assets Establish Beachhead

- Lead asset Halneuron® inhibits the Na_V 1.7 sodium channel critical for pain signaling
- SP16 activates LDL-receptor related protein 1 (LRP1) to drive pain reduction (anti-inflammatory) and reparative signaling to restore immune balance

Clinically Validated

- Halneuron demonstrated statistically significant and durable pain reduction, with acceptable safety profile, in cancer and chemotherapy related clinical pain studies to date

2026 Catalysts

- Halneuron in Phase 2b CINP development, data anticipated 3Q26
- SP16 Chemotherapy Induced Peripheral Neuropathy (CIPN) Phase 1b enrollment anticipated mid-2026

Significant Opportunity

- CINP treatment market $1B in US/UK/EU4/Japan, projected to increase to ~$3B by 2036[1]
- Expand into larger ~$7.5B Cancer Related Pain (CRP) market[2]

DOGWOOD THERAPEUTICS

[1]Delve Insight [2]Allied Market Research

2026 Pipeline Milestones

Key 2026 Milestone	Estimated Timing
FDA Acceptance of Filing of SP16 IND to Advance to Phase 1b CIPN Study	✓ April 2026
Global Combination Antiviral License	✓ May 2026
Halneuron Maintenance Study Commences (Part II of Phase 2b)	✓ May 2026
SP16 Phase 1b Enrollment Begins	• 2H 2026
Halneuron® Phase 2b Fully Recruited	• August 2026
Projected Final Data Patient Halneuron® Phase 2b CINP	• Fall 2026
Halneuron End of Phase 2 FDA Clinical Submission	• December 2026