DOGWOOD THERAPEUTICS

Developing a Non-opioid, $Na_v1.7$ Specific Sodium Channel Inhibitor to Treat Pain

Q3 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

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DWTX Executive Team

Greg Duncan
Chairman & CEO

Pfizer · UCB · Celtaxsys

R. Michael Gendreau
MD, PhD CMO

Cypress · INTEC PHARMA

Angela Walsh
CFO

ALTEA THERAPEUTICS · Celtaxsys

Ralph Grosswald
SVP of Operations

EMORY HEALTHCARE · Celtaxsys

Management's Brand Development & Commercialization Experience Includes:

- Zoloft (sertraline HCl)
- VIAGRA (sildenafil citrate) tablets
- LIPITOR atorvastatin calcium tablets
- CELEBREX
- Aricept
- LYRICA PREGABALIN
- cimzia (certolizumab pegol)
- Savella milnacipran HCl

Novel, Non-Opioid $Na_v1.7$ Research Pipeline Targeting Chronic and Acute Pain

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Target Indication	Candidate/Target	Preclinical	Phase 1	Phase 2	Phase 3
Chemotherapy-Induced Neuropathic Pain (CINP)	Halneuron® $Na_v1.7$				
Cancer Pain (CRP)	Halneuron® $Na_v1.7$				
Acute pain	Halneuron® $Na_v1.7$				

Halneuron® - Fulfills Many Requirements Of An Ideal Analgesic

- 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

$Na_v1.7$ Inhibition Represents a Logical Target to Reduce Pain

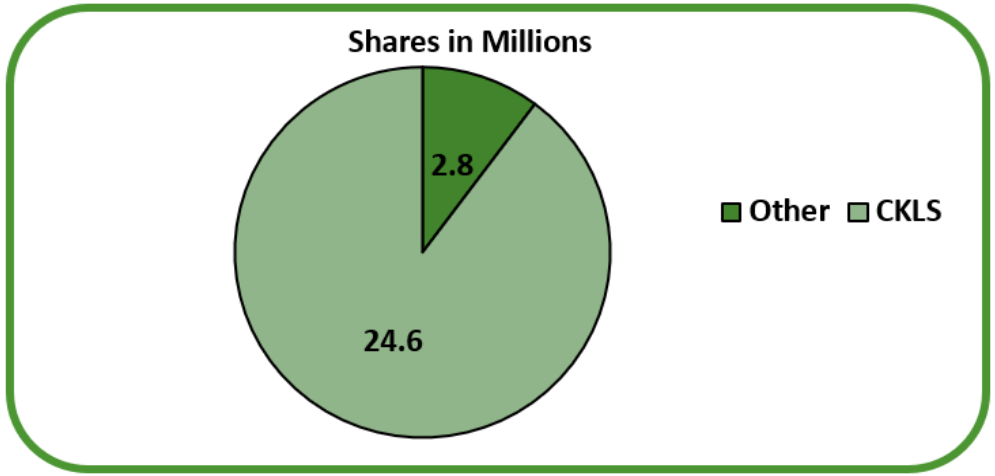
Loss of $Na_v1.7$ Function Leads to Congenital Insensitivity to Pain Syndrome

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

Erythromelalgia: Sodium channels remain open increasing pain signals

Strategic Transaction to Acquire Novel, Non-opioid Development Candidate Halneuron®

- Business combination with Pharmagesic Holdings (i.e. Wex Pharmaceuticals) and VIRI completed in Q4 2024
- CKLS, a publicly listed company on the Hong Kong exchange, was issued **211,383 shares of Common Stock** and **2,108.3854 shares of Series A Preferred Stock** in exchange for Pharmagesic, including its novel new pain development candidate Halneuron®
  - Each Preferred share converts into **10,000** shares of common stock, subject to approval by DWTX shareholders
  - Included a $20M strategic financing (loan) from an affiliate of CK Life Sciences International, (Holdings) Inc. ("CKLS"), which was since converted into **284.2638 shares of Series A-1 Preferred Stock in Q1 2025**
- Upon conversion of the Preferred Stock following shareholder approval, CKLS and its affiliates will hold 24.6 million shares of common stock, or approximately 90% of the Company's common stock on a fully diluted basis.



CINP 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
- CINP 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
  - CINP severity characterized as mild (25%), moderate (50%), or severe (25%) across most markets
- Estimates suggest almost 70% of patients treated with chemotherapy experience CINP
  - 30% of chemotherapy treated patients continue to experience CINP six months post treatment
- Chemotherapy utilization is expected to increase by 54% by 2040
- No therapy for CINP to date, including opioids, has proven to be generally effective
- One-in-three to six in ten CINP patients are likely to be treated with opioids in 2025
  - Cancer patients using opioids develop clinically significant adverse effects (e.g. cognitive impairment and hallucinations)
  - Most patients develop constipation or nausea and vomiting

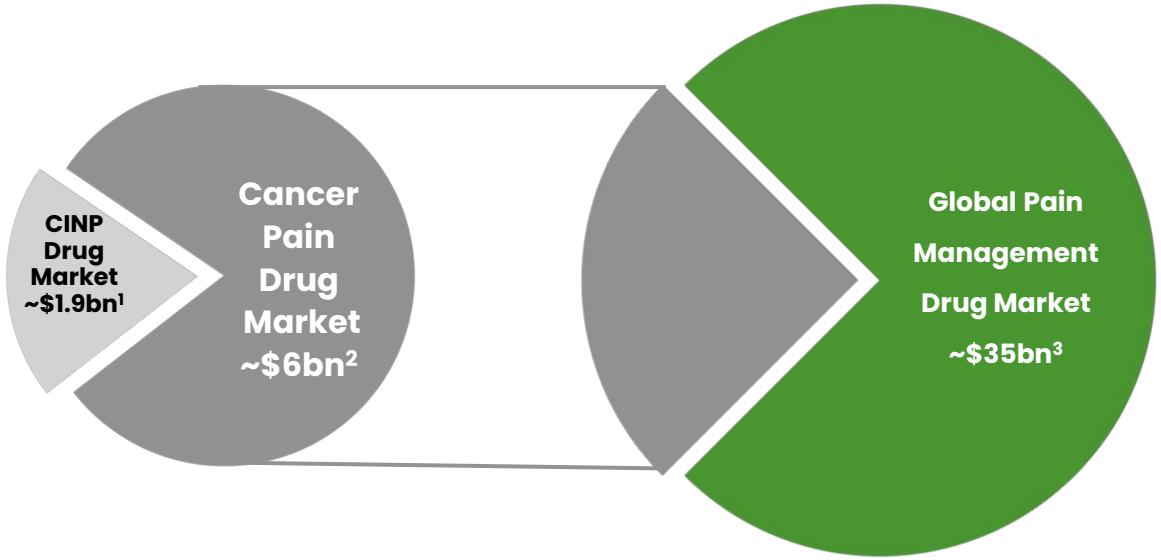
Notes: *American Cancer Society;* 2024, *WHO*, 2024; *Lancet Oncology*, 2019; *DelveInsight,* 2018; de Stoutz ND, Bruera E, Suarez-Almazor M, *J Pain Symptom Manage* 1995; Trescot AM, Boswell MV, Atluri SL, et al., *Pain Physician* 2006

Ability to Effectively Treat CINP Opens a Large Market Opportunity

- No approved treatments for CINP
- Medications like duloxetine, gabapentin, pregabalin, or tricyclic antidepressants and opioids are used to help manage neuropathic pain
  - Opioids account for 38%-58% of CINP treatment, depending on the market
- Halneuron® CRP and acute surgical pain life-cycle plan target represents an even larger opportunity than CINP population

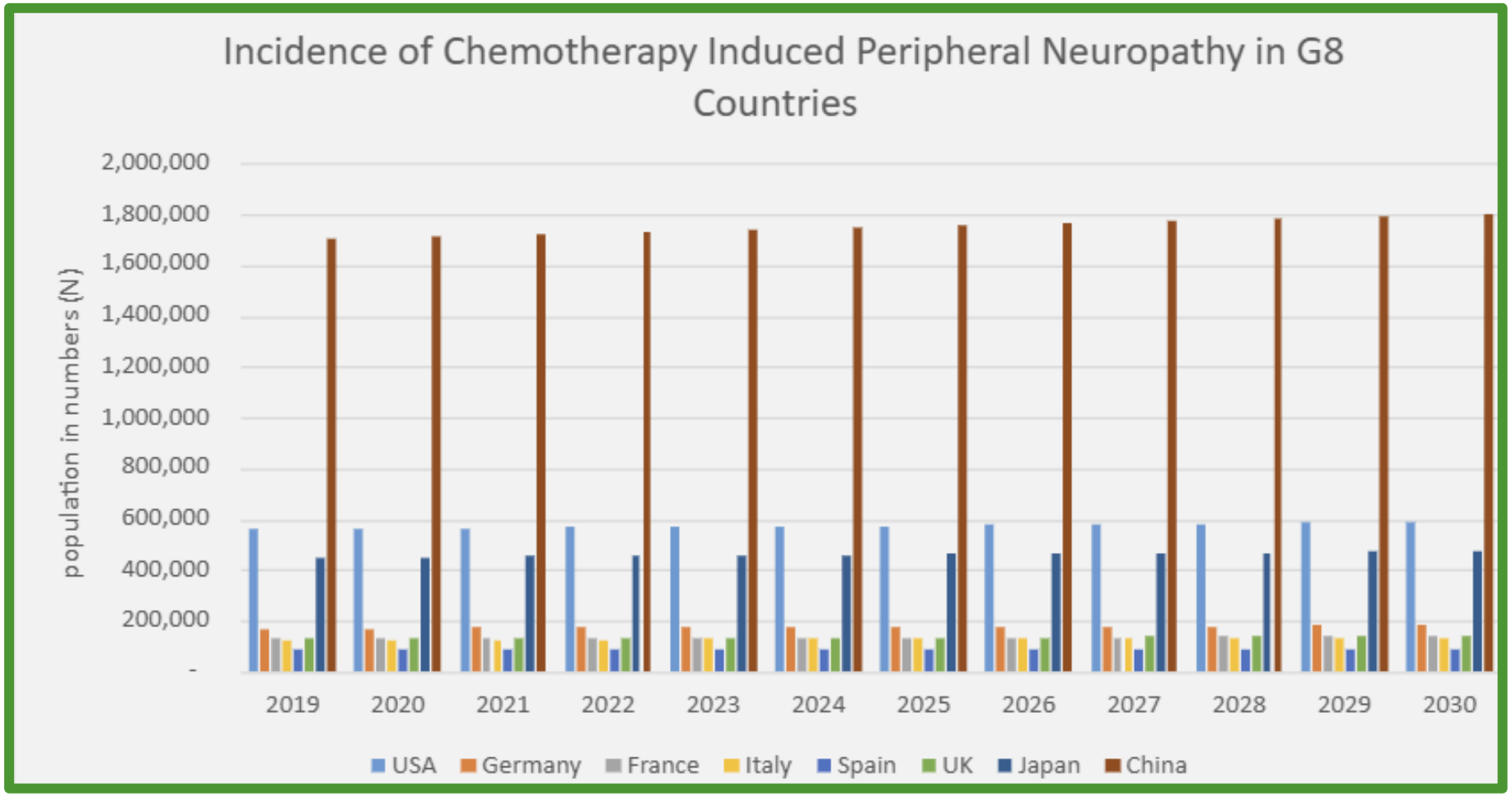
Pain Management Drug Markets

~57% and ~45% of the Global Cancer Pain and Global Pain Management drug markets are opioids respectively [2,3]



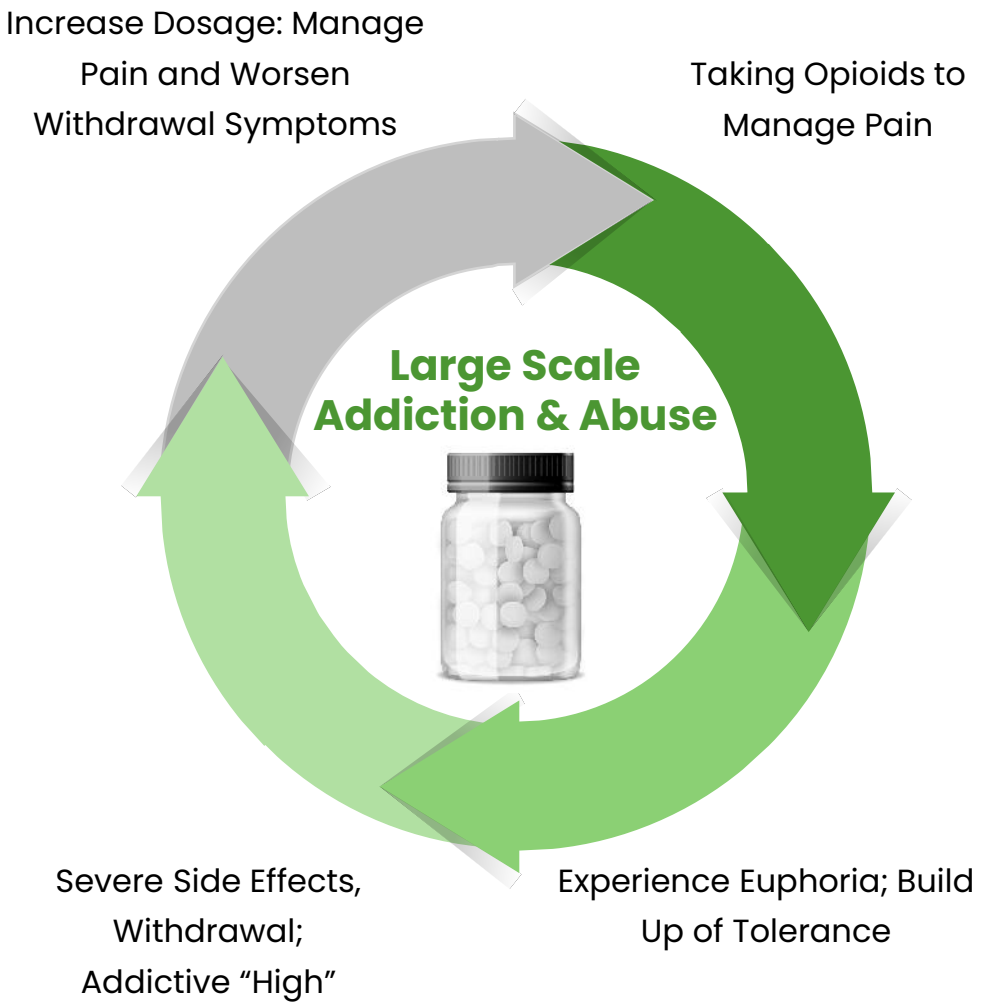
Notes:
1. Delveinsight December 2018, Chemotherapy-Induced Peripheral Neuropathy, Market insights, Epidemiology and Market Forecast 2018-2027
2. Allied Market Research December 2018, Global Cancer Pain Market, Opportunity Analysis and Industry Forecast 2018-2025
3. LP Information December 2019, Global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

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



Notes: Thelansis, 2020

Vicious Cycle With Opioid Pain Therapies



Current Problem:

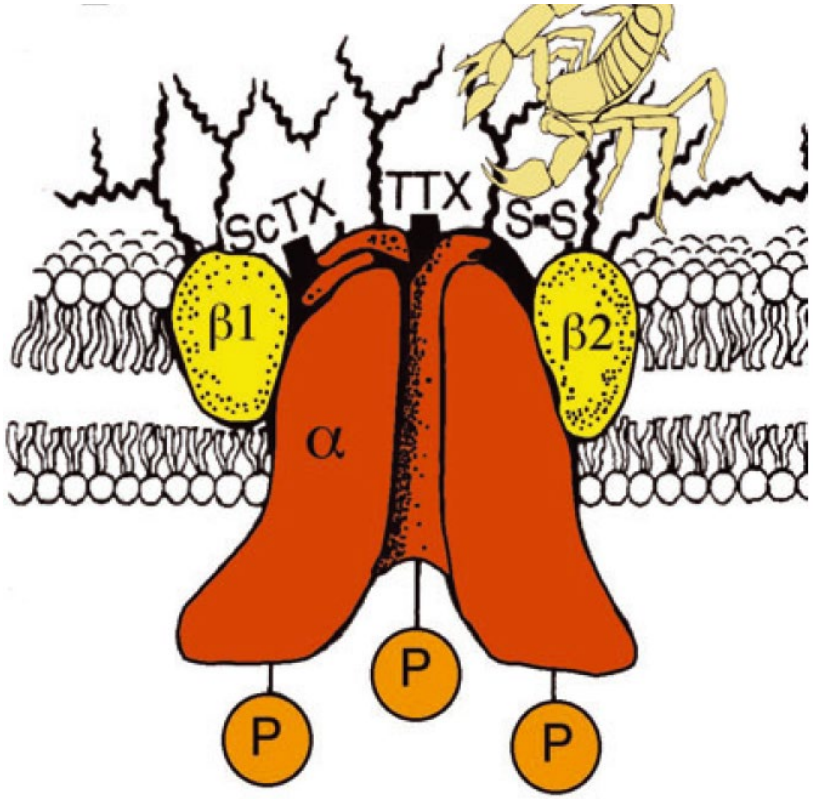
While effective at managing pain, opioids have numerous concerning issues:

- Highly addictive, a recognized public health crisis
- Severe side effects
- Withdrawal symptoms prevent reducing dosage
- Addiction, withdrawal symptoms, and side effects worsens as potency increases to manage long-term and chronic pain conditions
- Over 53,000 opioid deaths in the past 12 months in the US alone[1]
- ~35.6 million people suffered from drug use disorders worldwide in 2018[2]

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

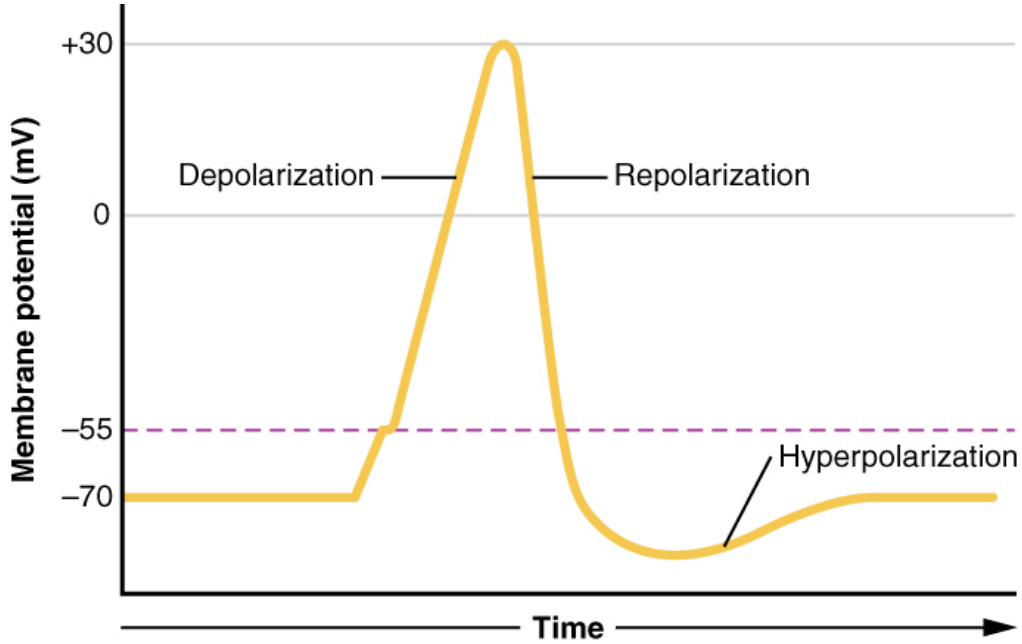
Voltage-Gated Sodium Channels and Pain

- In the 1950's, it was first shown that the electrical signals (action potential) that occurs in nerves and muscles is triggered by an inflow of sodium ions (Na+) into the cell body
  - This became known as the sodium current
- The sodium channel in the nerve cell membrane is composed of multiple protein subunits that protrude through the cell membrane and regulate the flow of ions into the cell
- When sodium ions (Na+) flow into the cell, the membrane can depolarize and send a signal
- There are multiple distinct sodium channels, and there are several classes of molecules that bind specific sodium channels



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 such as CINP, 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

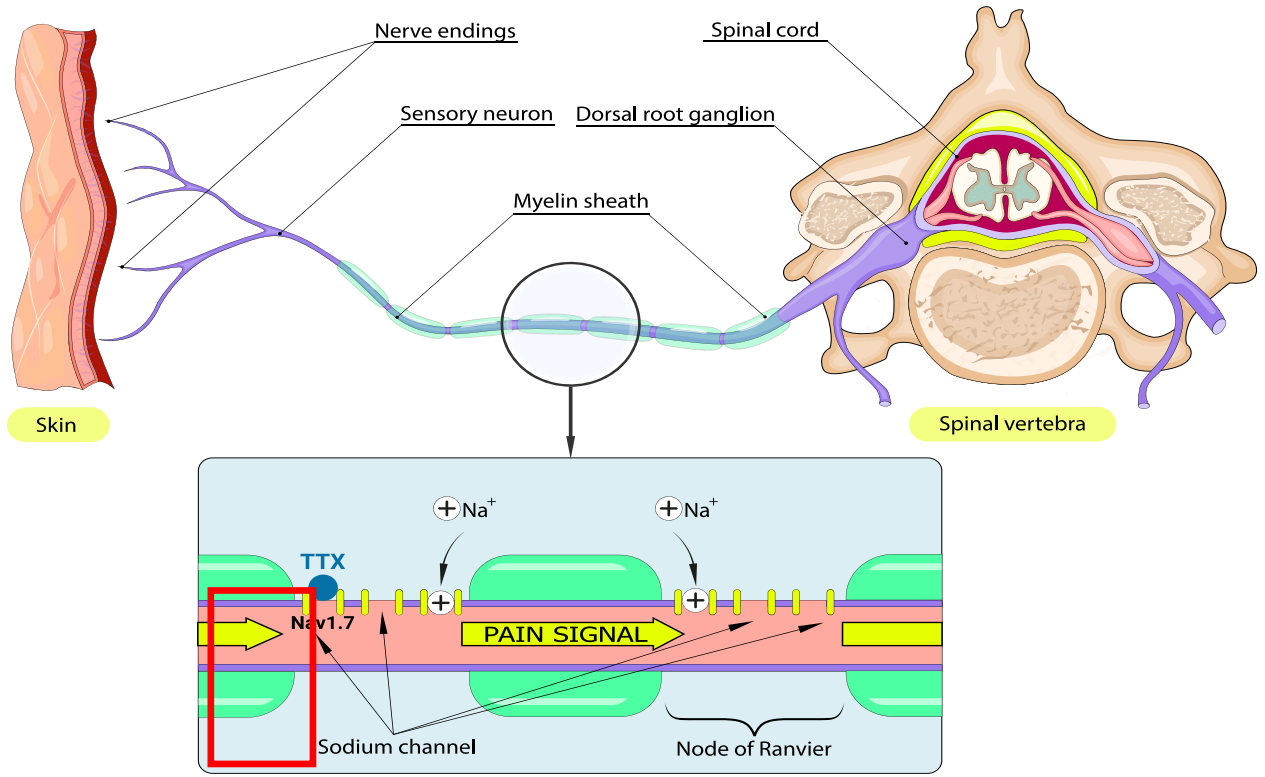
Our Approach – What is Halneuron®?

- 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
$Na_v1.1$	*SCN1A*	Mouse 2 Human 2q24	CNS neurons
$Na_v1.2$	*SCN2A*	Mouse 2 Human 2q23–24	CNS neurons
$Na_v1.3$	*SCN3A*	Mouse 2 Human 2q24	CNS neurons
$Na_v1.4$	*SCN4A*	Mouse 11 Human 17q23–25	SkM
$Na_v1.5$	*SCN5A*	Mouse 9 Human 3p21	Uninnervated SkM, heart
$Na_v1.6$	*SCN8A*	Mouse 15 Human 12q13	CNS neurons
$Na_v1.7$	*SCN9A*	Mouse 2 Human 2q24	PNS neurons
$Na_v1.8$	*SCN10A*	Mouse 9 Human 3p22–24	DRG neurons
$Na_v1.9$	*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

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Halneuron® $Na_v1.7$ Selectivity

Channel	TTX EC_{50}	Predominant Distribution
$Na_v1.7$	**EC_{50} = 24.5 nM**	Peripheral nervous system (PNS)
$Na_v1.8$	EC_{50} = 60,000 nM	PNS & dorsal root ganglion (DRG)
$Na_v1.9$	EC_{50} = 40,000 nM	PNS & DRG
$Na_v1.5$	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® 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

Sodium Channels Involvement in Pain Transmission

- The flow of small ions- sodium, potassium and calcium have been associated with electrical activity in cells throughout evolution
  - It is believed that some 800 million years ago, prior to the development of modern neurons, there were simple calcium channels that were not very specific or efficient for regulating electrical signals
  - As evolution and neuronal development proceeded, sodium and potassium ion channels evolved and differentiated so that now in mammals, there are more than 9 known variants, each with their own gene, distinguished by changes in the large alpha subunit protein that defines the pore on the cell membrane

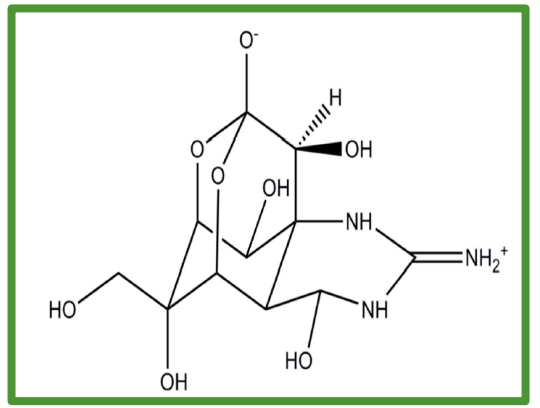
Table 1. Peripheral Sodium Channels.* Pain Related

Channel	Gene	Role in Neuronal Function	Distribution
$Na_V1.7$	*SCN9A*	Boosts subthreshold depolarization; sets the gain on the neuron	Peripheral sensory neurons (e.g., DRG and trigeminal ganglion neurons); sympathetic ganglion neurons
$Na_V1.8$	*SCN10A*	Resistant to depolarization (remains operable even when other sodium channels are inactivated); produces the action potential upstroke and drives repetitive firing	Peripheral sensory neurons (e.g., DRG and trigeminal ganglion neurons)
$Na_V1.9$	*SCN11A*	Modulates resting potential; amplifies response to depolarization	Peripheral sensory neurons (e.g., DRG and trigeminal ganglion neurons)

* DRG denotes dorsal-root ganglion.

Halneuron ® (TTX)

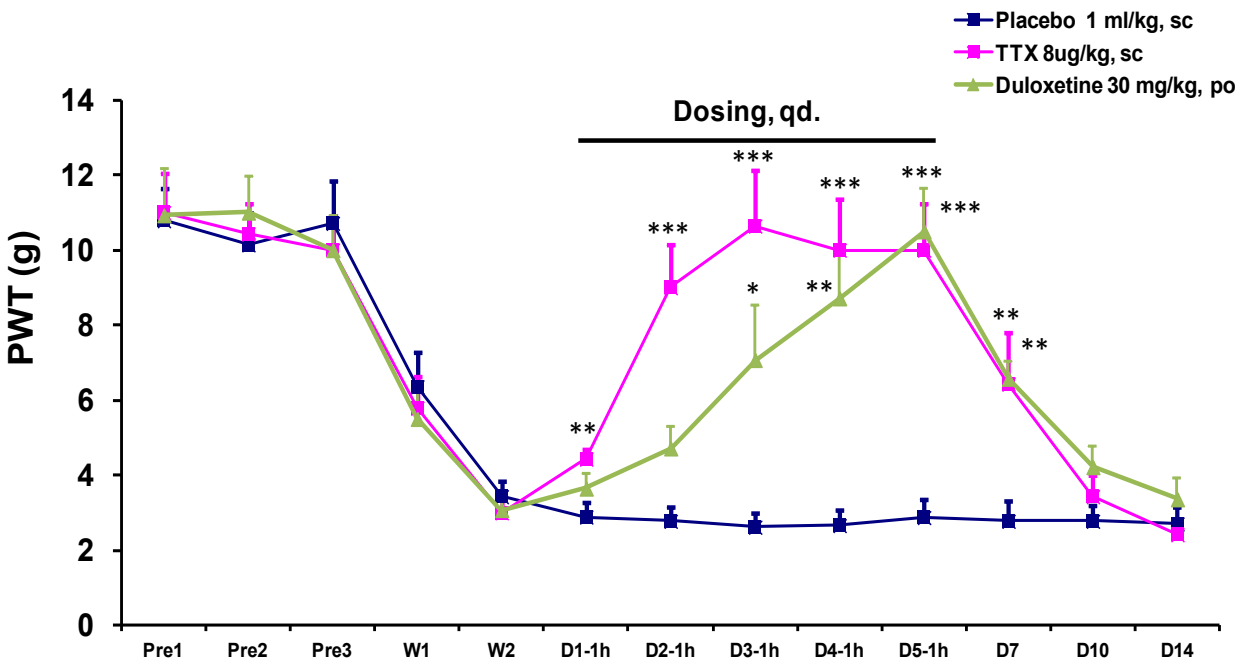
- **Structure**:
- The structure of tetrodotoxin is a complicated highly oxygenated small molecule that binds the pore of the sodium channel with high affinity, blocking the influx of ions and subsequent membrane depolarization
- While it currently is extracted from puffer fish, synthetic approaches to production are being developed for Phase 3 studies
- **TTX for Injection:**
- Dosage Form : Sterile Lyophilized Powder
- Stabel at room temperature for 60 months
- In-use Stability: After reconstitution, stable for 96 hours under refrigeration (2 to 8°C)
  - Stable for 24 hours at ambient room temperature (20 to 25°C)



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

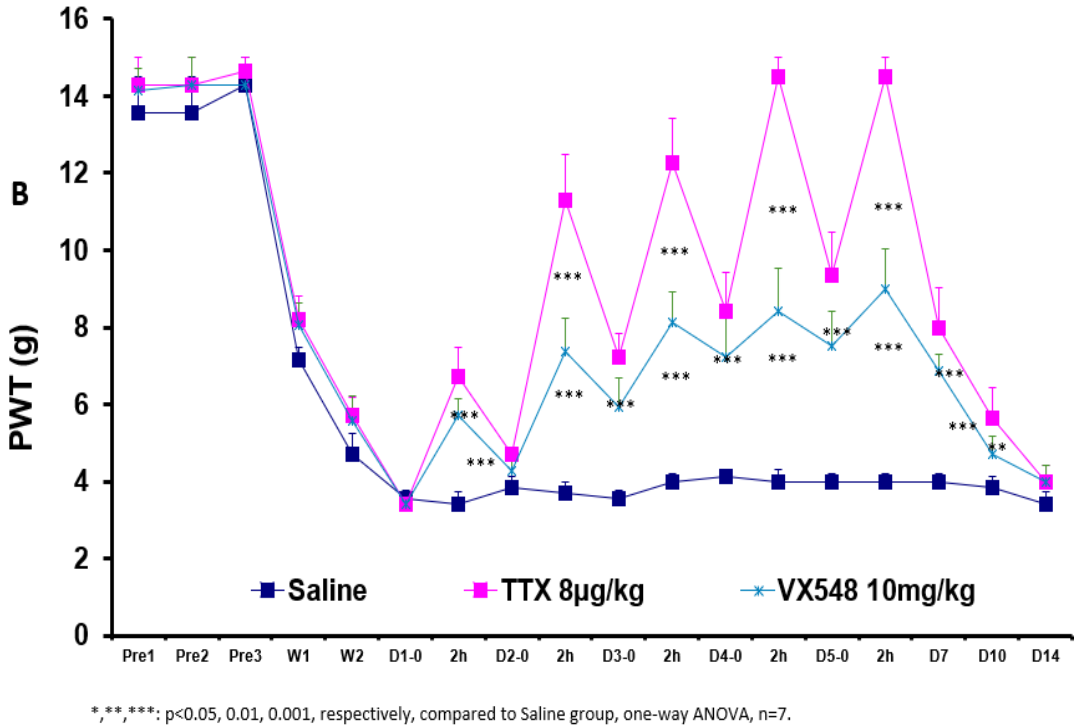
- Adult male Sprague-Dawley rats.
- Oxaliplatin 4 mg/kg, injected intravenously, twice a week, repeated up to 9 times to induce mechanical allodynia.
- Paw withdrawal threshold (PWT) used as an indicator of neuropathy
- The rats showing significant mechanical allodynia
  - PWT ≤ 4g were used as an indicator.
- 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

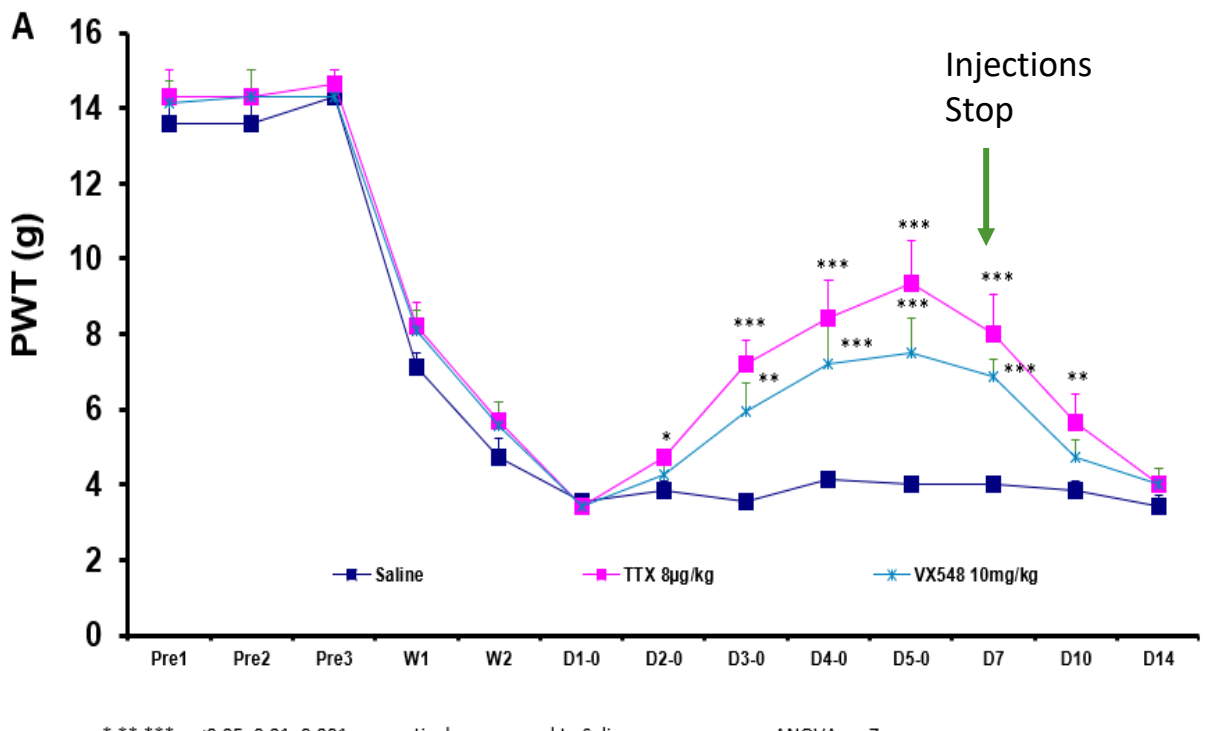


*,**,***: p<0.05, 0.01, 0.001, respectively, compared to placebo group, one-way ANOVA, n=7

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



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



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

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

Previous Phase 2 Human Studies- Cancer Related Pain (CRP)

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 ≥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**				

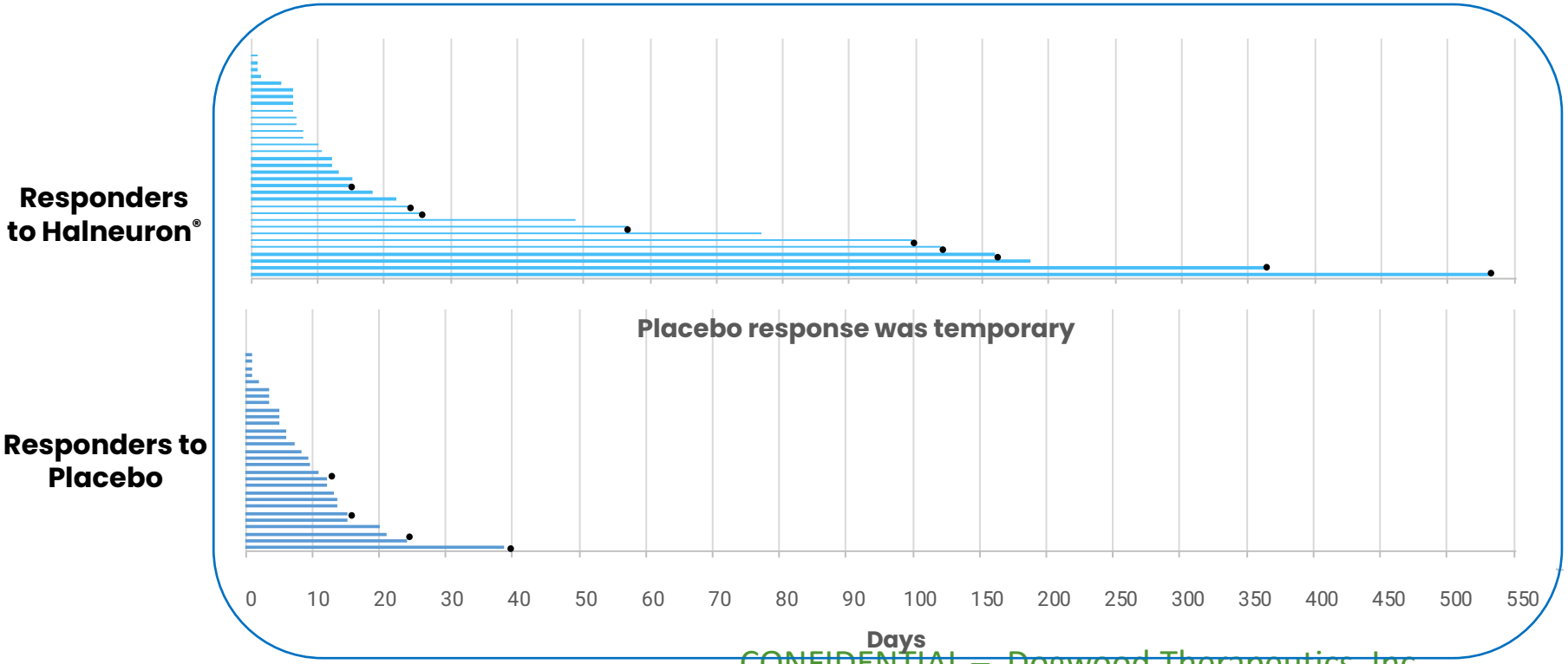
A "Responder" was defined as a patient who had a mean reduction in pain intensity of ≥ 30%; or ≥ 50% reduction in opioid use

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

Duration of response assessed for those with 30% or greater reduction in pain at the primary endpoint (initial responders)

- After a single cycle of 4 days of treatment, Halneuron® initial responders (50.8% of treated) showed a greater duration of pain reduction as compared to placebo responders (34.5% of treated)

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 ≥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 in Chemotherapy Induced Neuropathic Pain (CINP)

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CINP 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® CINP Adverse Event Profile

Most Frequent Adverse Events During Phase 2a CINP Study

Adverse Event	TTX 30 µg QD x 4 days N=25 N (%)	TTX 30 µg BID x 4 days N=26 N (%)	Placebo x 4 days 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

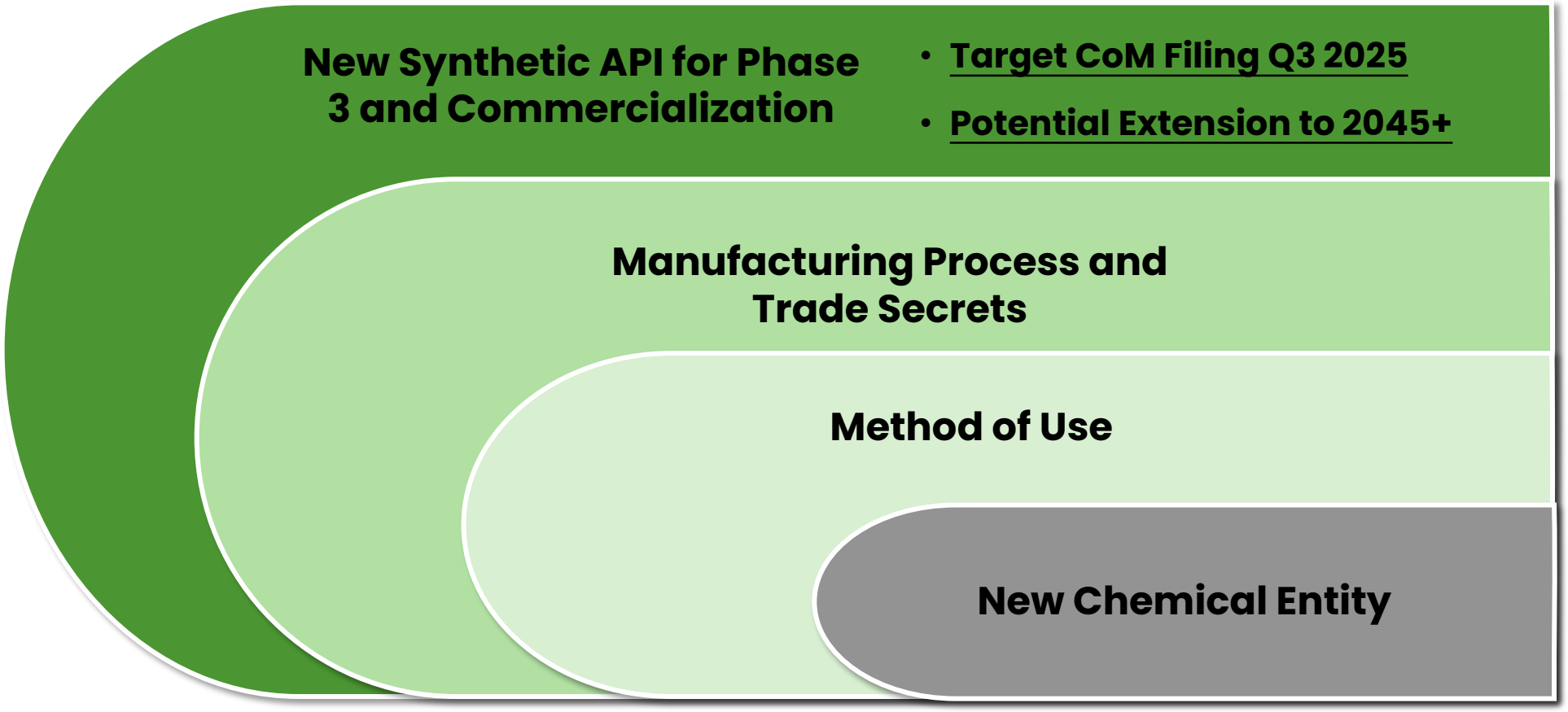
- Majority of AEs were mild to moderate in severity
- Most common AEs were expected and resolved naturally within a few hours after each injection
- No clinically significant impact on laboratory tests, vital signs, or ECGs was noted

Current Halneuron® 4-Week Phase 2b 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)

Intellectual Property & Drug Manufacturing



Halneuron® 2025 Milestones and Catalysts

Candidate/Target	Target Indication	Next Key Milestone(s)
Halneuron® $Na_v1.7$	CINP	August: Phase 2b Trial 50 Patients Enrolled September: New Synthetic IP Filed December: Phase 2b Interim Analysis June/July: Phase2b Final Data

Novel Combination Antiviral Program Targets Two Areas of Unmet Medical Need

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Two novel, late-stage clinical stage development assets:

- **IMC-1 (famciclovir + celecoxib) ready for Phase 3 development as treatment for fibromyalgia:**
  - FDA agreement to enter Phase 3 post End-of-Phase 2 meeting;
    - Pharmacokinetic/Food Effect Study
    - Study 1: Head-to-Head 12-Week Study of IMC-1 vs Placebo
    - Study 2: Multifactorial, 12-Week Study of IMC-1 vs Placebo vs Famciclovir vs Celecoxib
    - Study 3: Long-term safety extension study
  - Exploring Phase 3 partnership and extended-release dosage formulation to extend IP
- **IMC-2 (valacyclovir + celecoxib) Phase 2 Long-COVID study ongoing:**
  - Proof-of-concept study completed in 2023, new IP filed with protection potential to 2044
  - We have clarity from FDA on the development requirements associated with advancing IMC-2 into Phase 2 development as a treatment for Long-COVID symptoms
  - Exploring Phase 2b funding/partnership options with new IMC-2 formulation