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Wave Life Sciences

PRECISION-HD clinical trial results and business update

March 29, 2021

Forward-looking statements

This document contains forward-looking statements. All statements other than statements of historical facts contained in this document, including statements regarding possible or assumed future results of operations, preclinical and clinical studies, business strategies, research and development plans, collaborations and partnerships, regulatory activities and timing thereof, competitive position, potential growth opportunities, use of proceeds and the effects of competition are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause the actual results, performance or achievements of Wave Life Sciences Ltd. (the "Company") to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "expect," "plan," "aim," "anticipate," "could," "intend," "target," "project," "contemplate," "believe," "estimate," "predict," "potential" or "continue" or the negative of these terms or other similar expressions. The forward-looking statements in this presentation are only predictions. The Company has based these forward-looking statements largely on its current expectations and projections about future events and financial trends that it believes may affect the Company's business, financial condition and results of operations. These forward-looking statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, including those listed under Risk Factors in the Company's Form 10-K and other filings with the SEC, some of which cannot be predicted or quantified and some of which are beyond the Company's control. The events and circumstances reflected in the Company's forward-looking statements may not be achieved or occur, and actual results could differ materially from those projected in the forward-looking statements. Moreover, the Company operates in a dynamic industry and economy. New risk factors and uncertainties may emerge from time to time, and it is not possible for management to predict all risk factors and uncertainties that the Company may face. Except as required by applicable law, the Company does not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

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Agenda

Topic	Speaker
Opening remarks	**Paul Bolno, MD, MBA** President & CEO
PRECISION-HD and OLE trial results	**Michael Panzara, MD, MPH** Chief Medical Officer, Head of Therapeutics Discovery and Development
Evolution of Wave	**Paul Bolno, MD, MBA** President & CEO
WVE-003 (SNP3) and clinical pipeline	**Michael Panzara, MD, MPH** Chief Medical Officer, Head of Therapeutics Discovery and Development

Q&A

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Wave's commitment to innovation in HD

Scientific vision around biology of Huntington's disease including the role of wild-type HTT

Committed to novel approach to selectively reduce mutant HTT while sparing wild-type HTT

Innovative approaches – SNP phasing, wild-type HTT assay

Collaborations with academia, clinicians, industry and the community

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WVE-003: Highly differentiated program supported by Wave's next generation chemistry

First generation chemistry

WVE-120101 SNP1

WVE-120102 SNP2

WVE-003
SNP3

Differentiated wild-type sparing approach
Only allele-selective clinical program

First HD clinical candidate containing PN chemistry
PN chemistry has demonstrated enhanced potency, exposure, durability in CNS

Clinical starting dose informed by preclinical *in vivo* model
Insight into PK / PD relationships

Clinical trial efficiencies
Adaptive trial design may enable rapid POC

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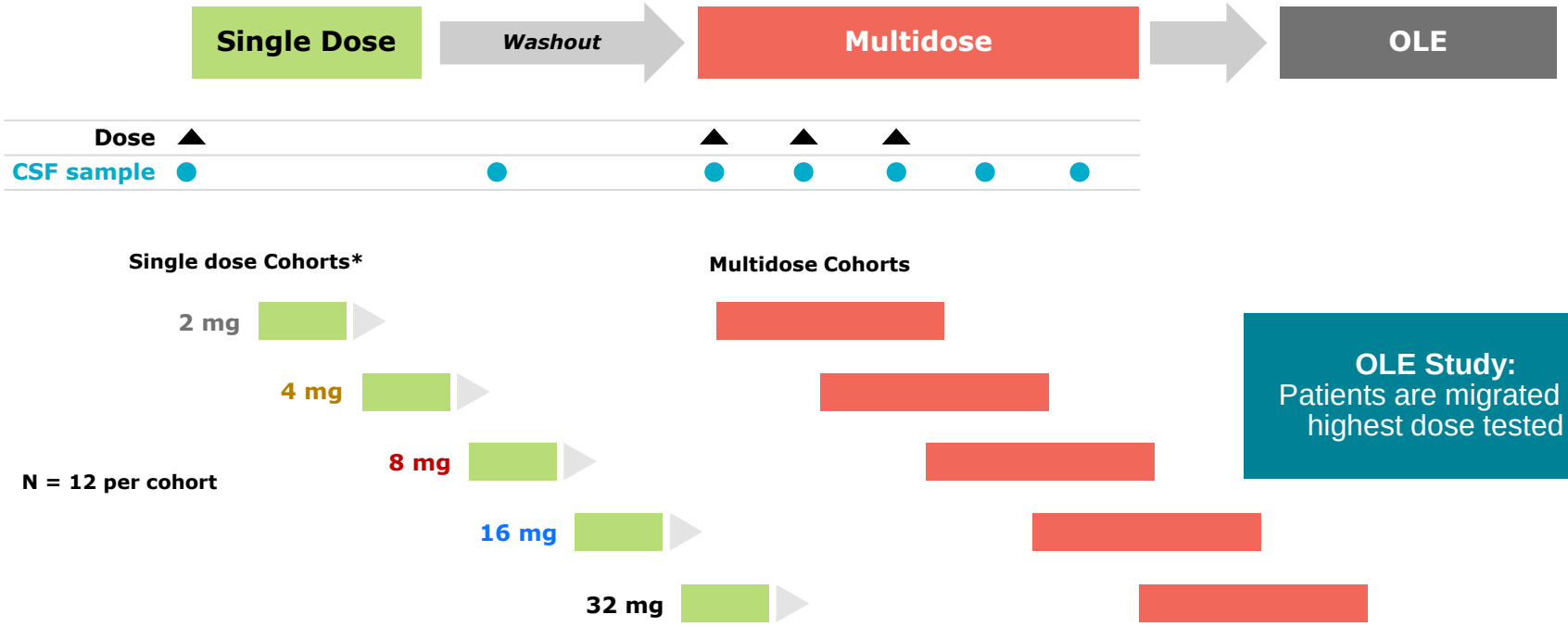
Mike Panzara, MD, MPH
Chief Medical Officer,
Head of Therapeutics
Discovery and Development

mHTT results from PRECISION-HD trials do not support further development of WVE-120102 and WVE-120101

- **PRECISION-HD2: Core Study**
  - No statistically significant reductions of mHTT after single or multiple doses of WVE-120102 (doses 2-32mg)
  - No dose response
- **PRECISION-HD2: Open Label Extension (OLE)**
  - Modest and inconsistent reductions in mHTT over course of study
  - Additional dose escalation unlikely to achieve drug concentrations needed for robust mHTT knockdown
  - No correlation between >20% mHTT reduction and wtHTT change, suggesting allele selectivity
- **PRECISION-HD1: Core and OLE**
  - Results consistent with PRECISION-HD2 up to 16 mg; 32 mg core and OLE results pending
- **Additional observations from all studies**
  - No changes in neurofilament light-chain (NfL) over time
  - No worsening of disease progression in treated participants versus expected based on natural history
  - Biomarker assays (mHTT, wtHTT, and NfL) performed reliably
- **Next-Generation Compound, WVE-003, in Phase 1b/2a**
  - New PN backbone modifications have potential to address limitations of first-generation chemistry
  - WVE-003, incorporating PN backbone chemistry, demonstrates improved preclinical *in vivo* pharmacology
  - Sites being activated, dosing expected in 2021

PRECISION-HD study design



OLE: Open label extension; CSF: cerebrospinal fluid; mHTT: mutant huntingtin; *12mg single-dose cohort in PRECISION-HD2, US only

PRECISION-HD2: Patient Disposition

	Pooled Placebo	WVE-120102						
		2 mg	4 mg	8 mg	12 mg	16 mg	32 mg	Total
Randomized patients	22	9	12	15	8	9	13	88
Number of doses per patient								
1	6	3	4	7	8	0	6	28
2	0	0	0	0	0	0	0	0
3	3	0	3	6	0	0	3	12
4	13	6	5	2	0	9	4	26
Mean	3.0	3.0	2.8	2.2	1.0	4.0	2.4	2.5
Patients who prematurely discontinued treatment	0	2 (22.2)	1 (8.3)	0	0	0	7 (53.8)	10 (11.4)
Adverse Event	0	0	0	0	0	0	6 (46.2)	6 (6.8)

Note: Numbers in parentheses are percentages.

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PRECISION-HD2: Patient Demographics and HD Disease History

	Pooled	WVE-120102						Active
	Placebo (N=22)	2 mg (N=9)	4 mg (N=12)	8 mg (N=15)	12 mg (N=8)	16 mg (N=9)	32 mg (N=13)	Total (N=66)
Time since initial diagnosis (years)								
Mean	6.1	9.9	3.8	5.6	3.9	3.2	5.6	5.3
Age at HD onset (years)								
Mean	40.18	42.00	41.75	43.33	42.50	49.00	48.08	44.47
Diagnosis stage								
1	9 (40.9)	5 (55.6)	6 (50.0)	8 (53.3)	1 (12.5)	5 (55.6)	11 (84.6)	36 (54.5)
2	13 (59.1)	4 (44.4)	6 (50.0)	7 (46.7)	7 (87.5)	4 (44.4)	2 (15.4)	30 (45.5)
CAG repeat length								
Mean	45.00	44.22	44.00	43.73	45.13	43.44	42.62	43.76
CAP score								
Mean	502.36	533.72	459.89	475.51	519.65	504.84	473.92	489.64

Note: Numbers in parentheses are percentages.

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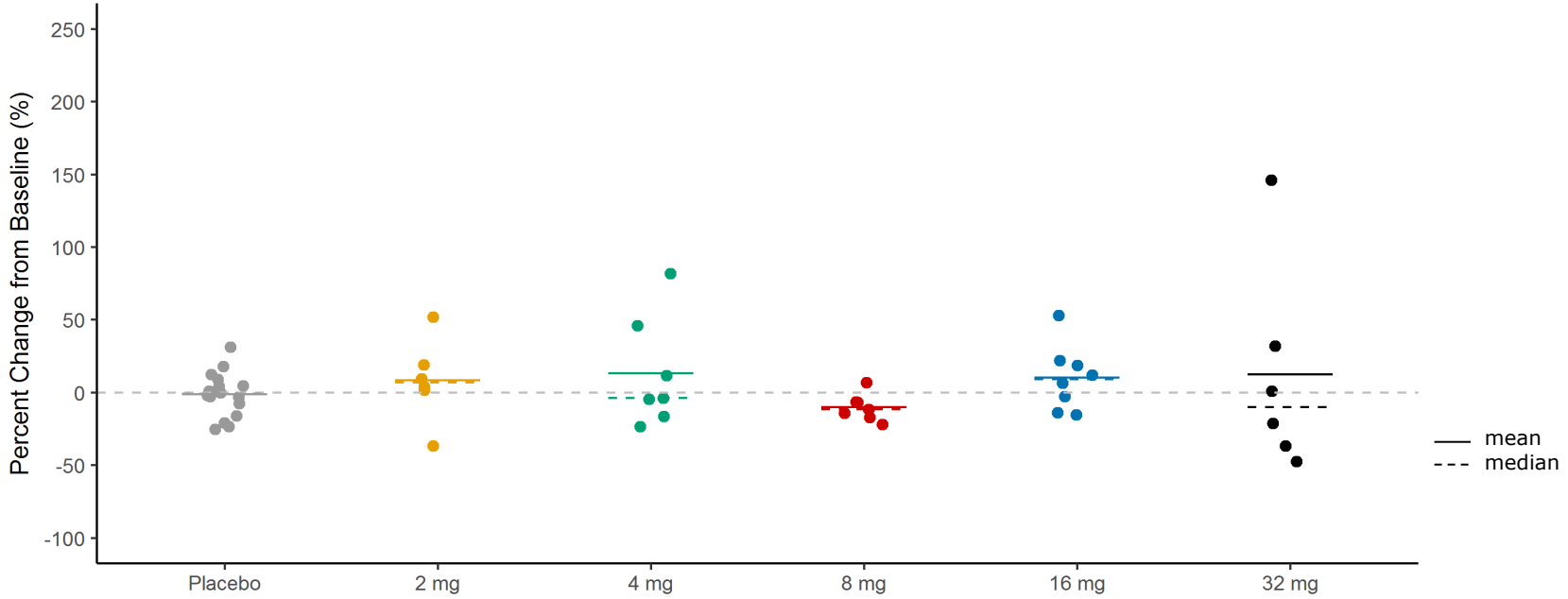
PRECISION-HD2 core: No statistically significant reduction of mHTT detected after 3 or 4 doses of WVE-120102



- Loss of small, statistically significant effect seen at December 2019 interim analysis
  - Incomplete follow-up after 3rd and 4th dose visits previously
  - Additional patients added to analysis
  - Placebo results changed with additional analyses and new patients

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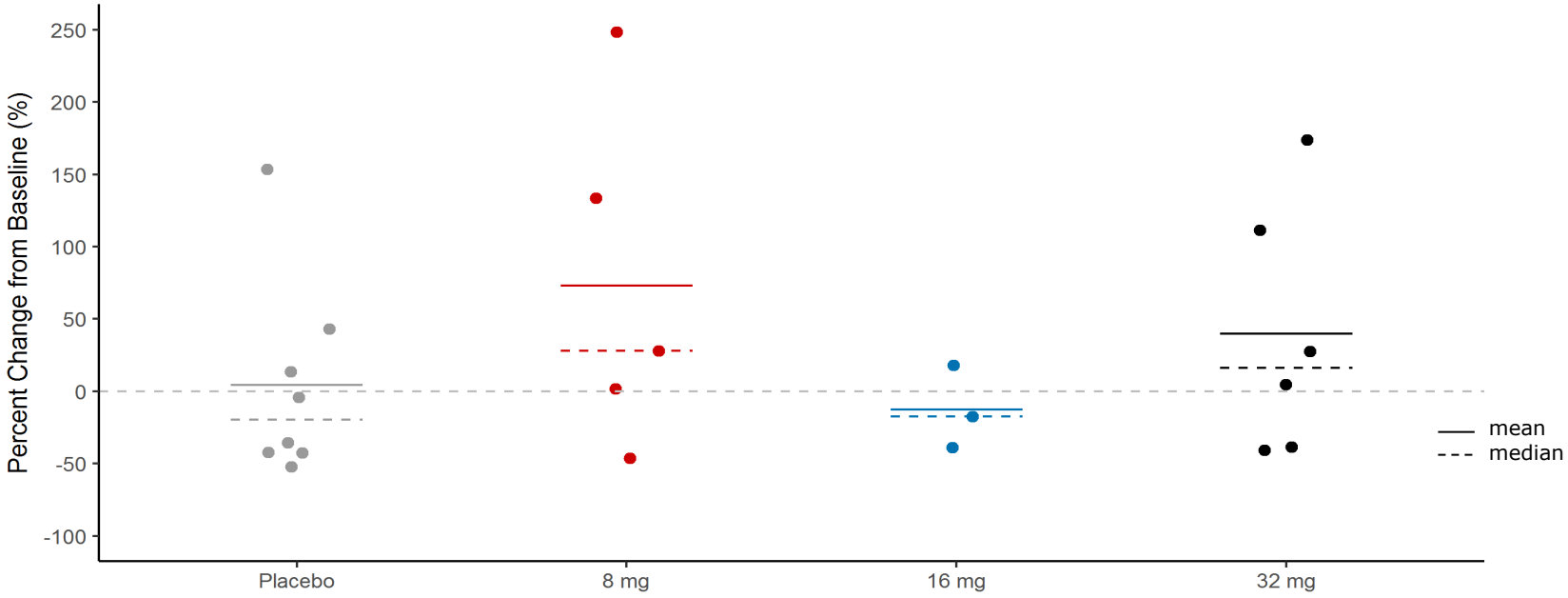
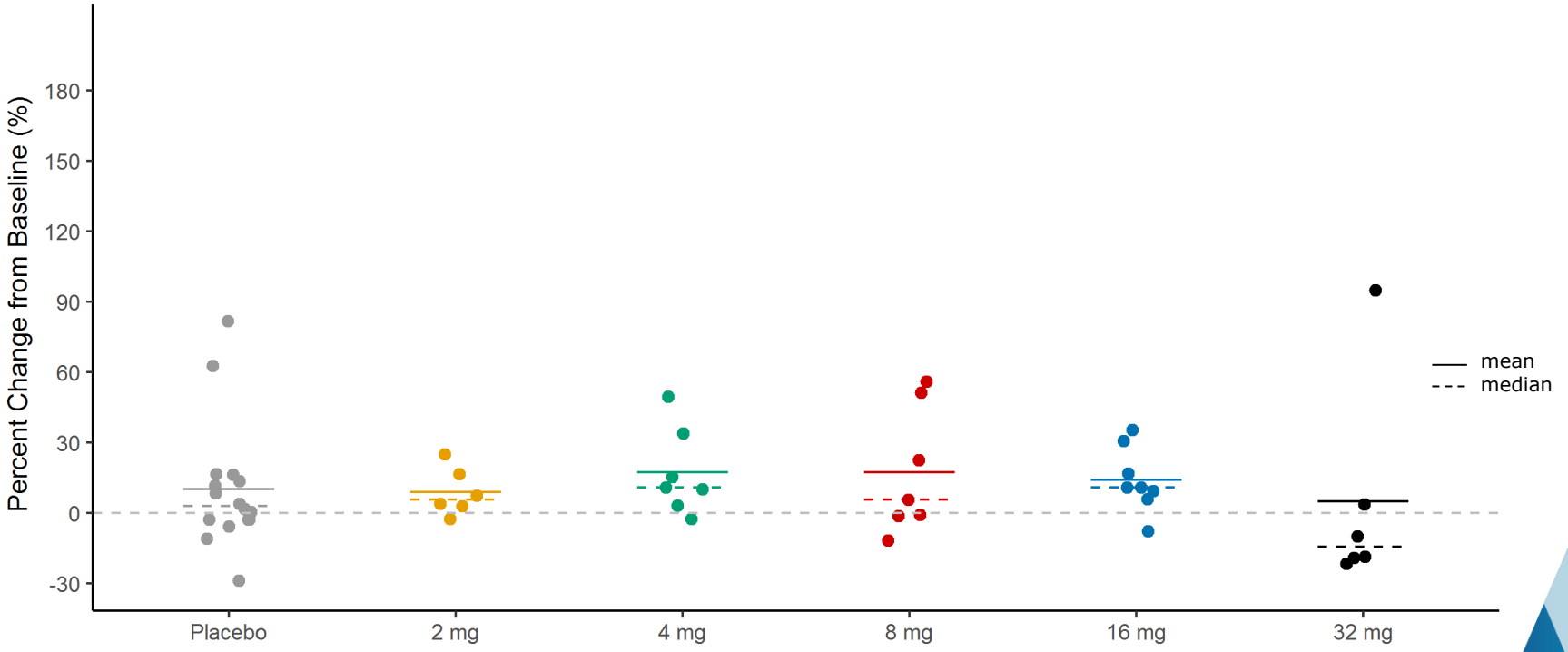
PRECISION-HD2 core: No statistically significant changes in wtHTT detected after 3 or 4 doses of WVE-120102



One patient (16mg) with percent change from baseline 1125.4% not shown
Fewer wtHTT assessments were performed in core study as baseline sample quantity was limited and prioritized for use in higher doses and OLE

PRECISION-HD2 core: No statistically significant changes in NfL detected after 3 or 4 doses of WVE-120102



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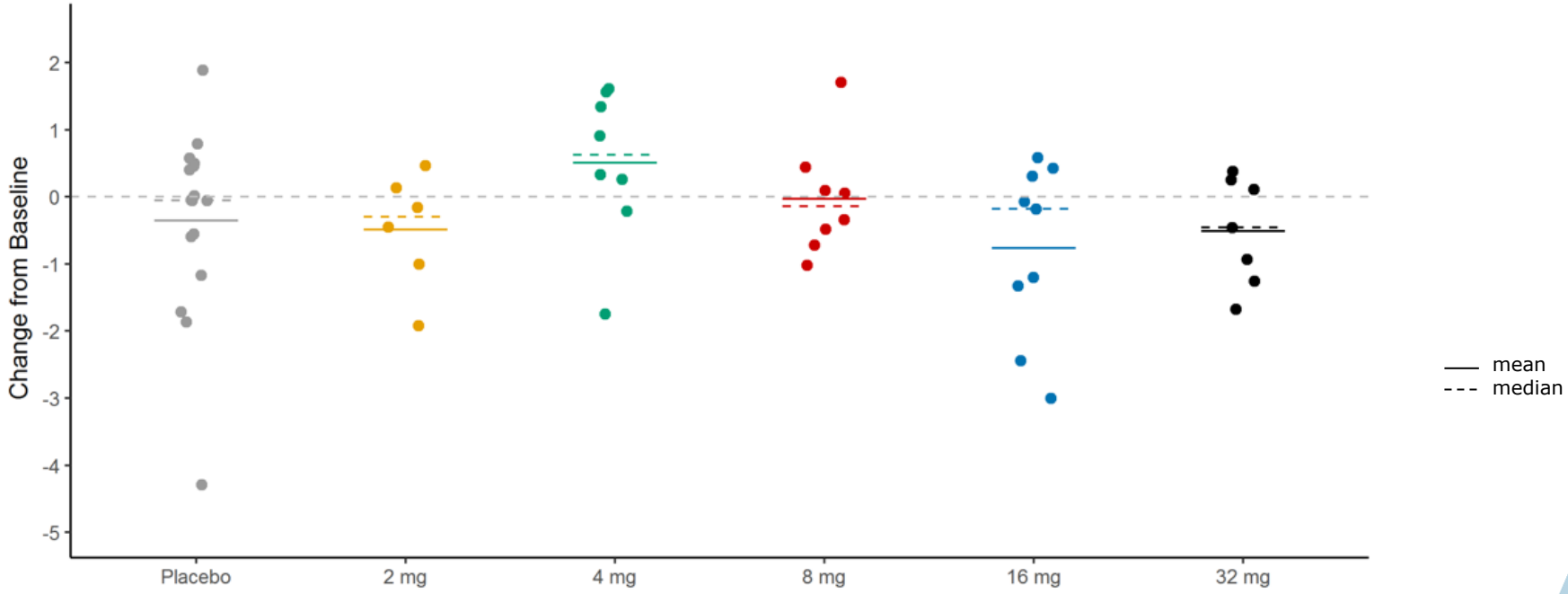
PRECISION-HD2 core: No change in UHDRS based clinical outcomes after 3 or 4 doses of WVE-120102



Similar results observed for the TMS and TFC

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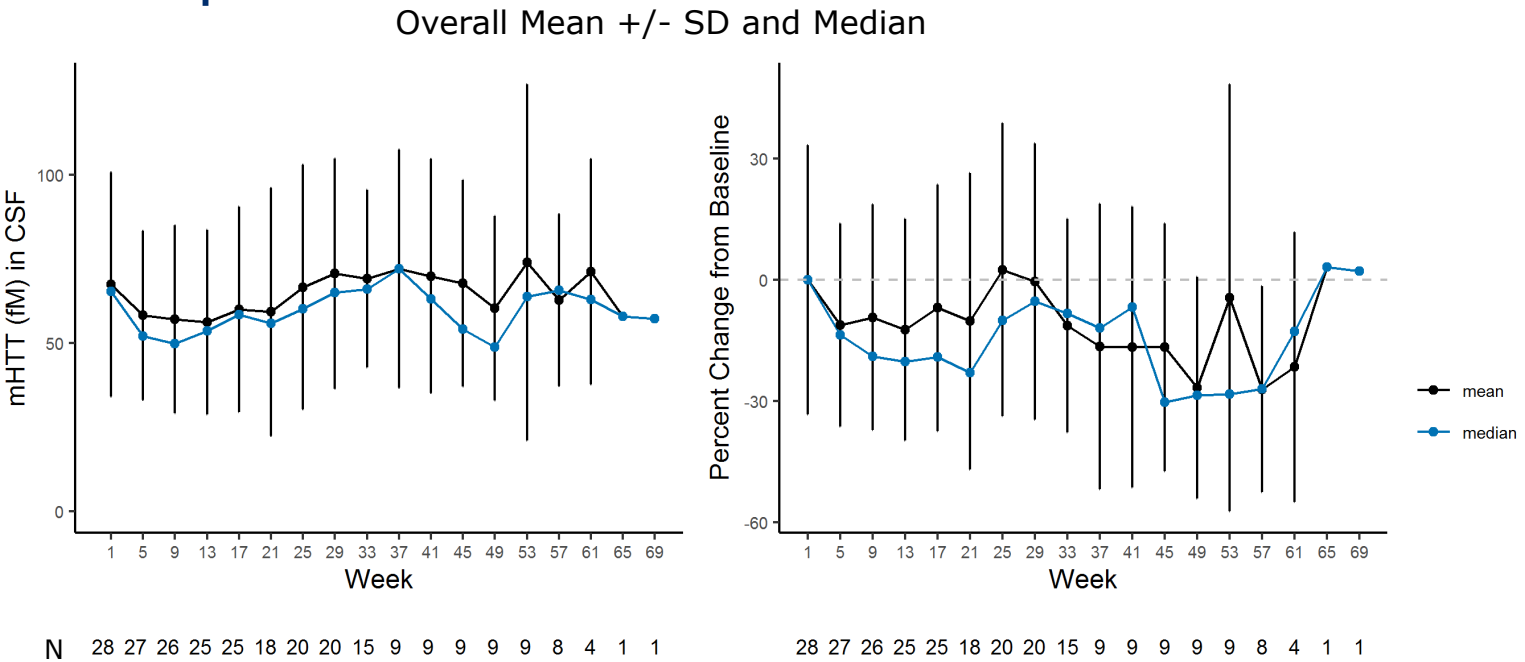
PRECISION-HD2 OLE: Modest reductions of mHTT at some timepoints



- 36 patients were receiving active treatment in the PRECISION-HD2 OLE and were included in the safety assessment, mean = 9.3 monthly doses, range = (1,19)
- 28 patients were included in the biomarker analysis, mean = 8.1 monthly doses, range = (1, 17)
- Multiple pre-specified and ad hoc sensitivity analyses confirmed no consistent effects

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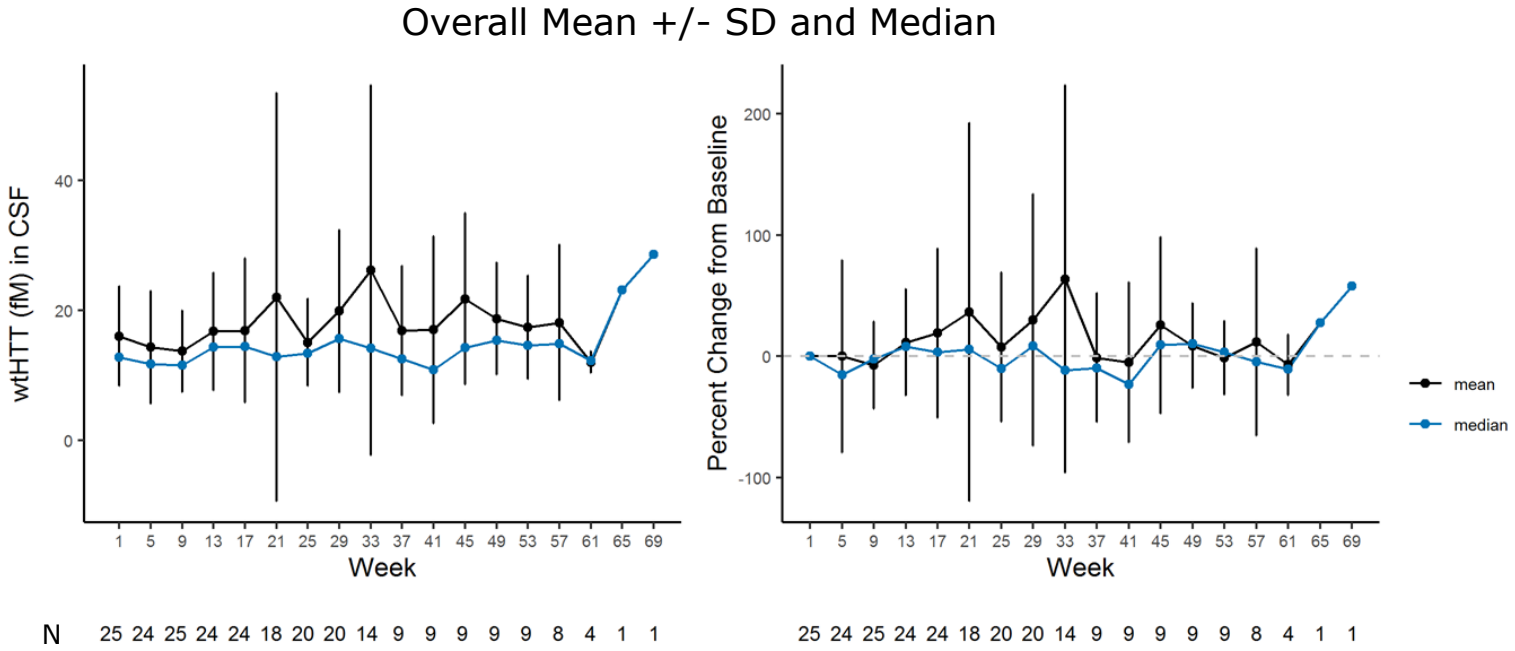
PRECISION-HD2 OLE: No reductions in wtHTT over time



- No correlation between greater than 20% reduction in mHTT and wtHTT change (Correlation coefficient = 0.04, 95% CI: -0.22, 0.19)
- Suggests allele-selectivity; Lack of robust reduction in mHTT limits ability to be definitive

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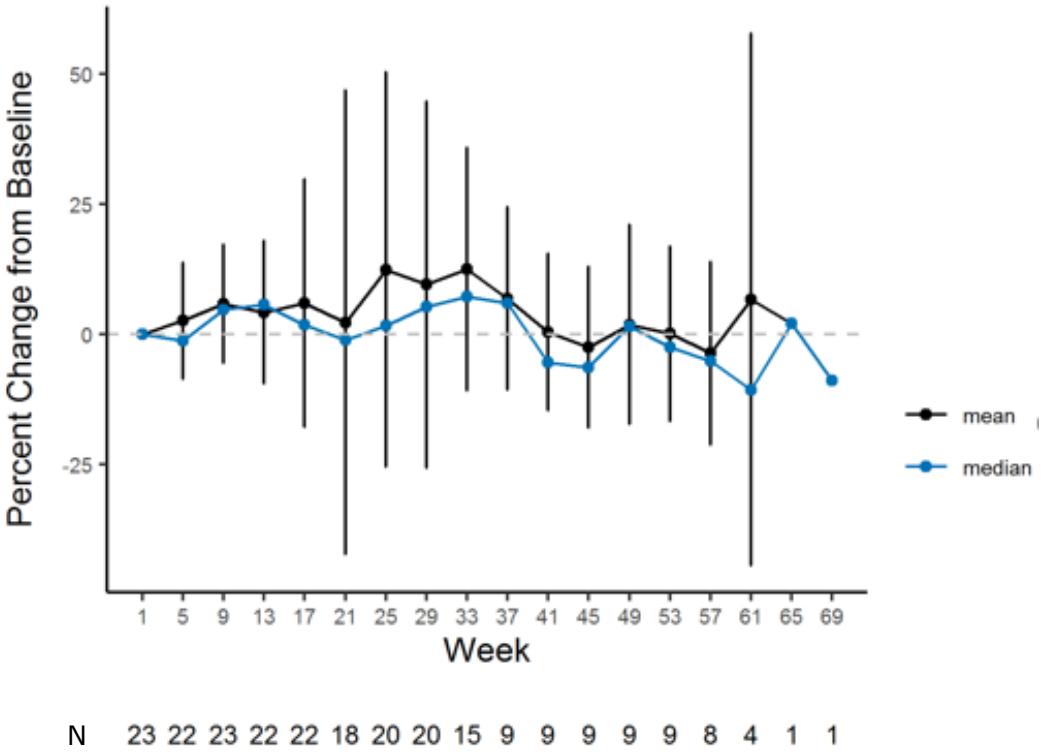
PRECISION-HD2 OLE: No changes in NfL over time

Overall mean +/- SD and median



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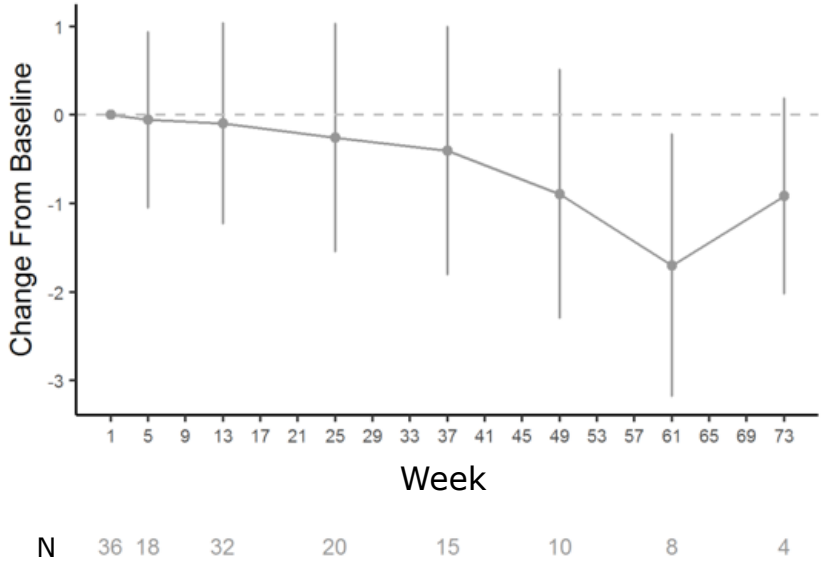
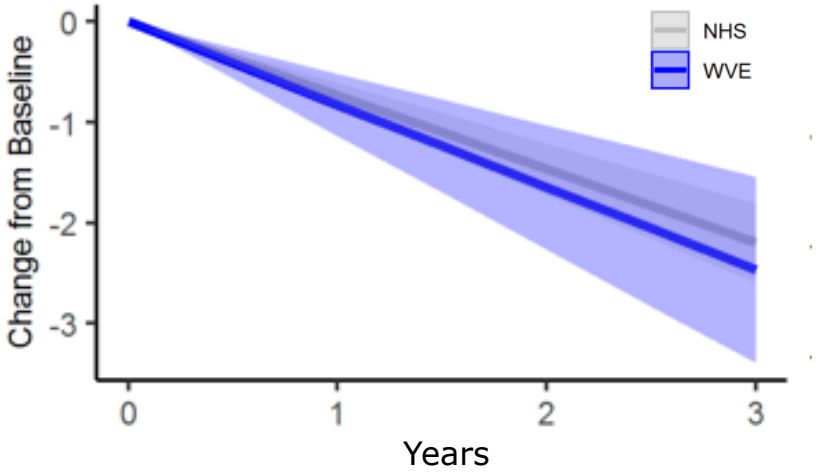
Progression on UHDRS-based endpoints matched that expected from natural history studies (NHS)

PRECISION-HD2 OLE: cUHDRS



PRECISION-HD2 Core and OLE: cUHDRS vs. NHS

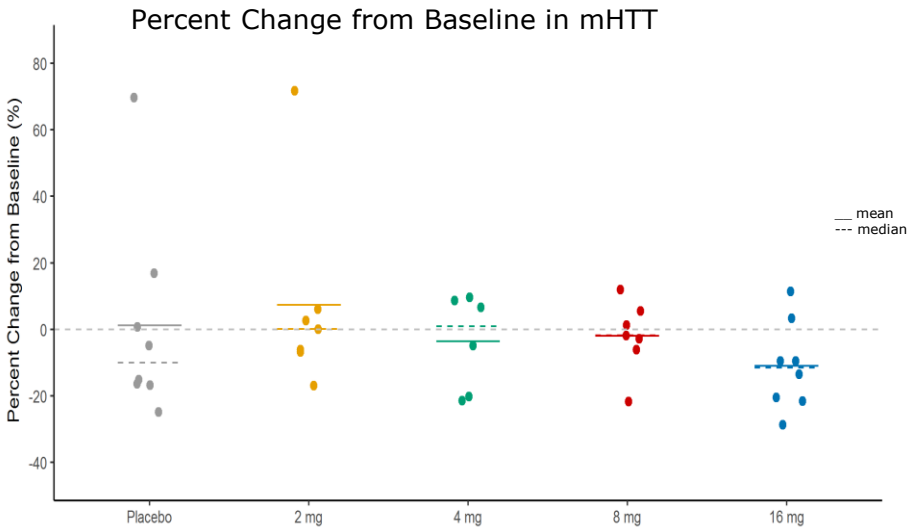


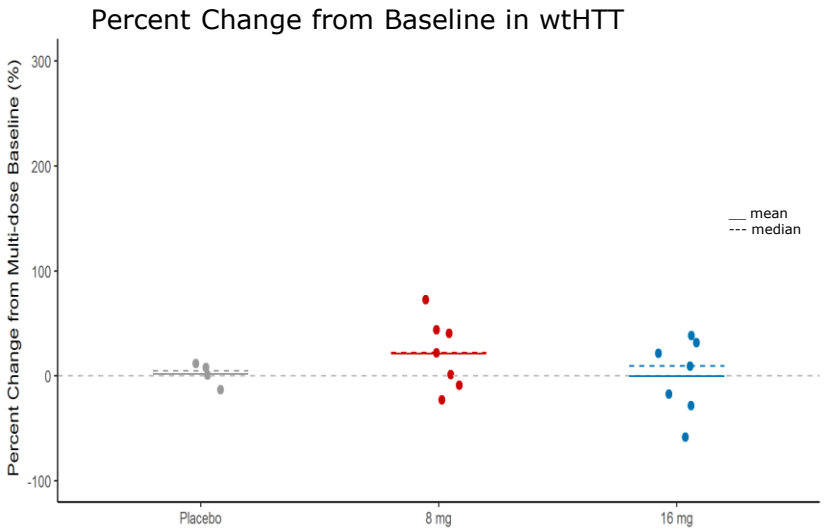
CHDI provided Track-HD, Predict-HD, Enroll-HD

PRECISION-HD2 Core and OLE Safety

- Adverse events reported in 83% of WVE-120102-treated participants in the PRECISION-HD2 core study versus 90% on placebo, most mild to moderate in intensity
  - Most common (reported in ≥10% receiving WVE-120102): Headache, procedural pain, back pain, falls, viral upper respiratory tract infection, dizziness, and post-lumbar puncture syndrome
  - Serious adverse events (SAEs) increased in the 32 mg group as compared to lower doses
    - 7 of 13 patients were reported with an SAE related to treatment
    - 6 discontinued treatment due to AE
    - SAEs were transient and included disorientation, delirium, ataxia, slurred speech, amnesia, meningitis, fever and vertigo
- Adverse events reported in the PRECISION-HD2 OLE were similar
  - 36 patients reported with an event over 327 person/months of exposure
  - Incidence of SAEs related to treatment with 32 mg WVE-120102 was lower than in the core study
    - 3 patients discontinued treatment due to AEs (2 receiving 16 mg, 1 receiving 32 mg).
- No clinically meaningful trends in clinical laboratory values including no CSF white blood cell and protein elevations in either study

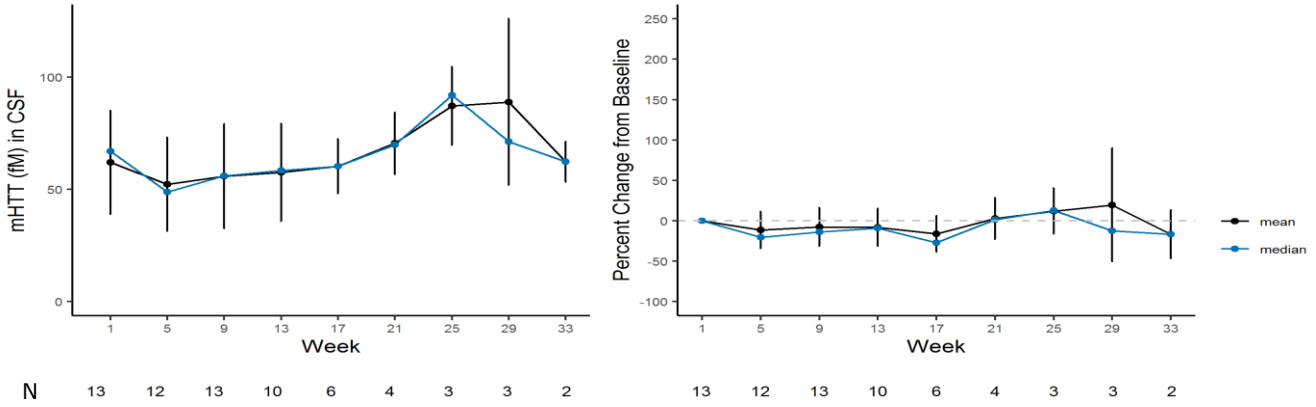
PRECISION-HD1 Core: No statistically significant reductions in mHTT and no change in wtHTT



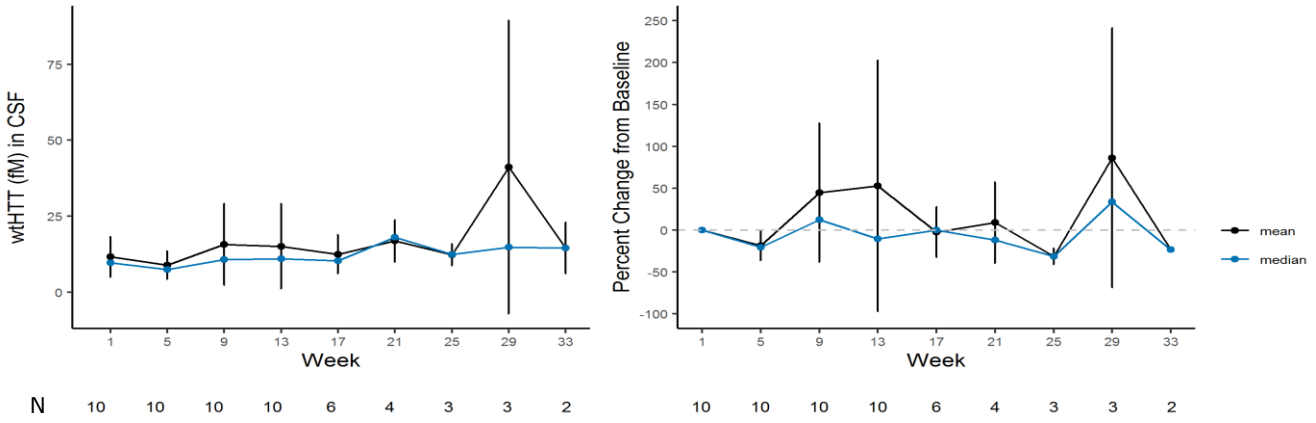


PRECISION-HD1 OLE: No statistically significant reductions in mHTT and no change in wtHTT

mHTT



wtHTT



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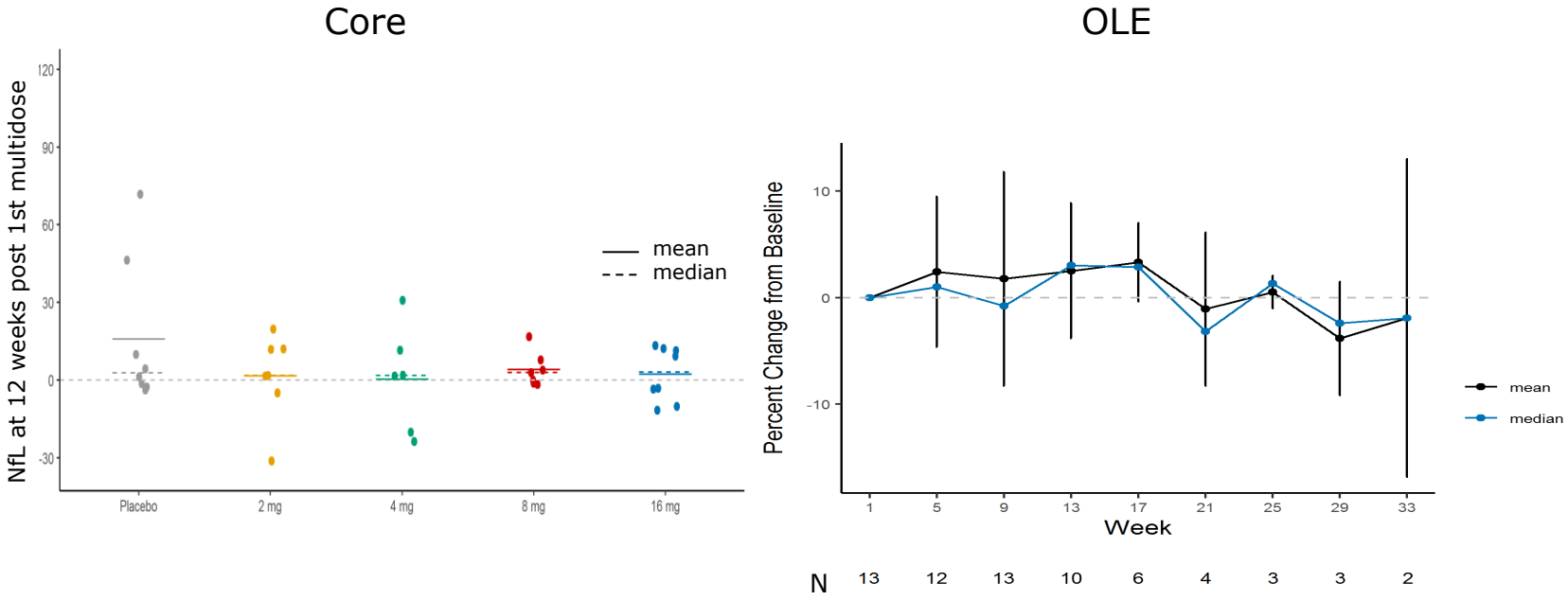
PRECISION-HD1 Core and OLE: No statistically significant changes in NfL over time



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PRECISION-HD1: AE incidence balanced between groups up to 16mg

- Adverse events reported in 91% of WVE-120101-treated patients who received up to 16 mg in the core study versus 75% who received placebo, most of which were mild to moderate in intensity
  - Most common (reported in ≥10% receiving WVE-120101): Headache, procedural pain, dizziness, back pain, falls and viral upper respiratory infection
  - No patients were reported with SAEs related to WVE-120101 up through 16 mg, 2 patients discontinued treatment due to AEs, one patient each in the 2 mg and 4 mg groups.
- Adverse events reported in the PRECISION-HD1 OLE were similar
  - 25 patients reported with an event over 95 person/months of exposure with no discontinuations due to AE
  - 1 patient reported an SAE of gait disturbance related to treatment
- No clinically meaningful trends in clinical laboratory values including CSF white blood cell and protein elevations in either study

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  - No changes in neurofilament light-chain (NfL) over time
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  - Biomarker assays (mHTT, wtHTT, and NfL) performed reliably
- **Next-Generation Compound, WVE-003, in Phase 1b/2a**
  - New PN backbone modifications have potential to address limitations of first-generation chemistry
  - WVE-003, incorporating PN backbone chemistry, demonstrates improved preclinical *in vivo* pharmacology
  - Sites being activated, dosing expected in 2021

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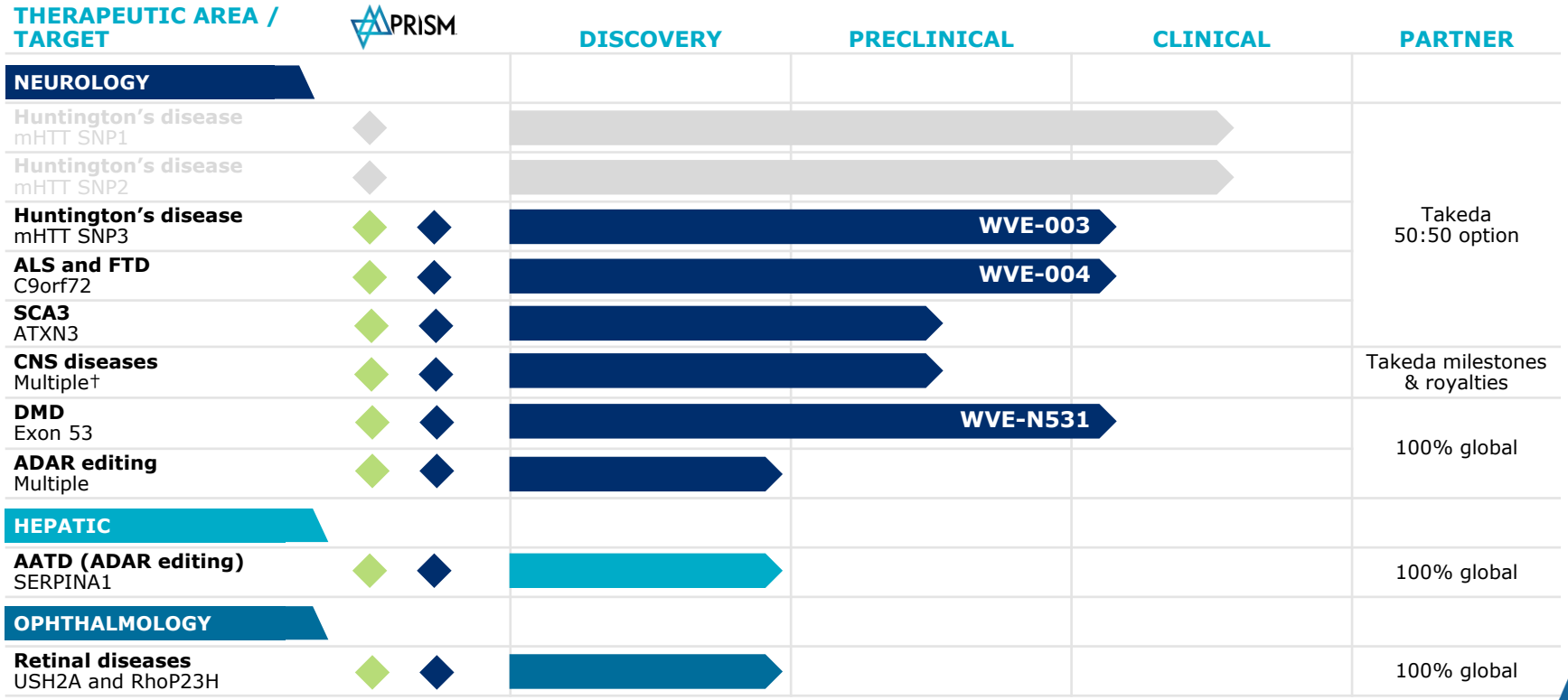
Paul Bolno, MD, MBA

President and CEO

Pipeline reflects company's evolution



†During a four-year term, Wave and Takeda may collaborate on up to six preclinical targets at any one time.
ALS: Amyotrophic lateral sclerosis; FTD: Frontotemporal dementia; SCA3: Spinocerebellar ataxia 3; CNS: Central nervous system; DMD: Duchenne muscular dystrophy; AATD: Alpha-1 antitrypsin deficiency

Foundation for next generation programs

- **Oligonucleotide innovation and optimization**
  - PN backbone chemistry modifications
  - Interactions between sequence, chemistry and stereochemistry
- ***In vivo* models**
  - Insight into PK / PD relationships
  - Novel model generation
- **Leverage learnings of first-generation programs**
  - Translational pharmacology
  - Adaptive clinical trial design

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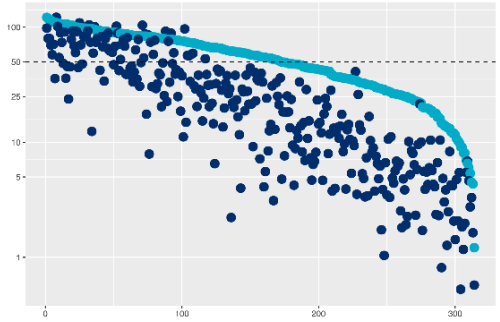
PK: Pharmacokinetics PD: Pharmacodynamics

PN chemistry increases potency in silencing, splicing, and editing preclinical studies

Silencing

Target knockdown (% remaining)



Improved knockdown

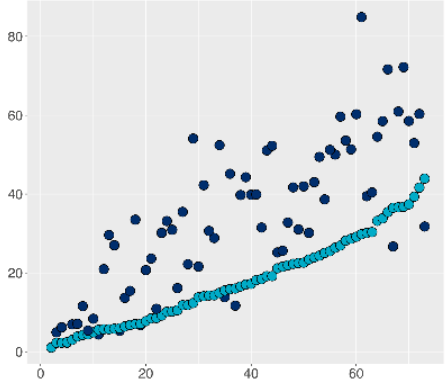
Ranked by potency of reference PS/PO compound

PS/PO reference compound

PS/PN modified compound

Splicing

% Skipping

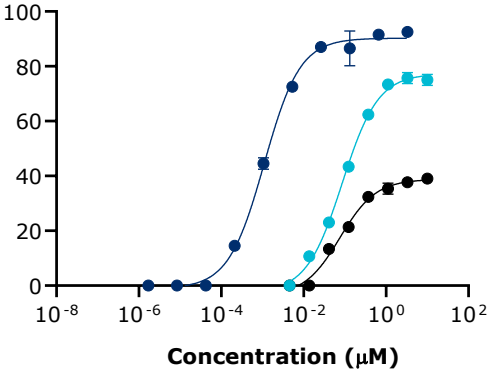


Improved skipping

Ranked by potency of reference PS/PO compound

Editing

% Editing



Improved editing

PS/PO/PN

PS/PO (Stereopure)

PS/PO (Stereorandom)

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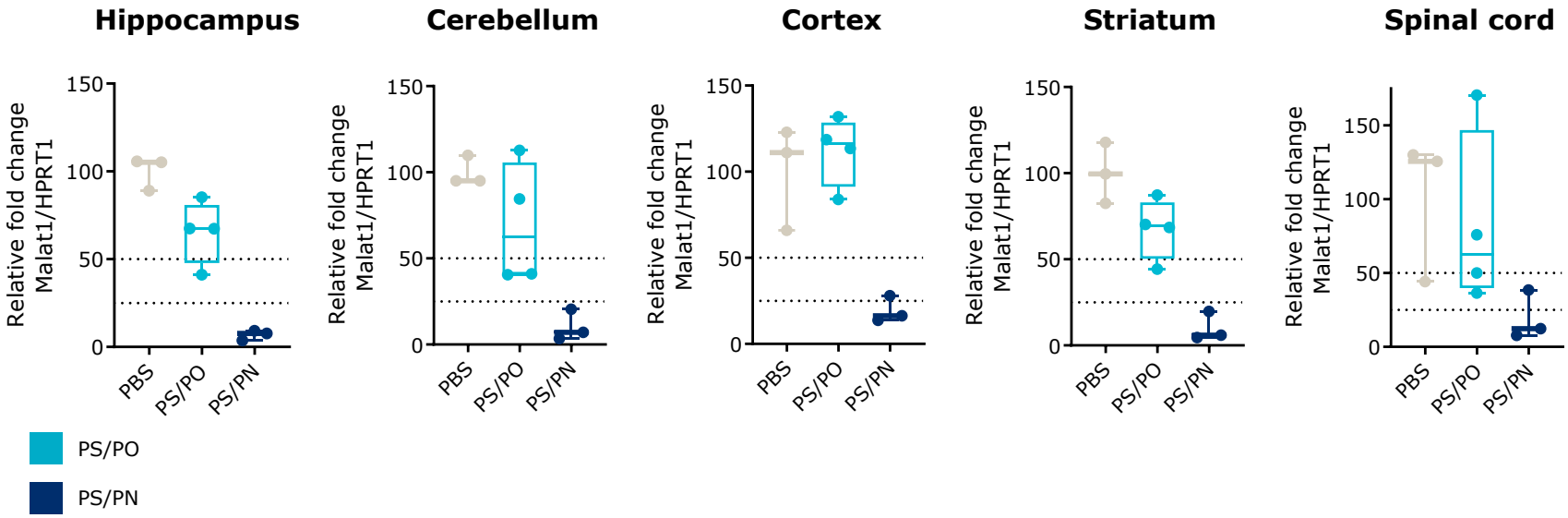
Presented at Analyst & Investor Research Webcast on August 25, 2020; Right: Data from independent experiments; PS phosphorothioate; PO phosphodiester; PN Nitrogen-containing backbone

PRISM

PN chemistry increases durability across key CNS tissues *in vivo*

Malat1 knockdown at 10 weeks in CNS (100 µg)



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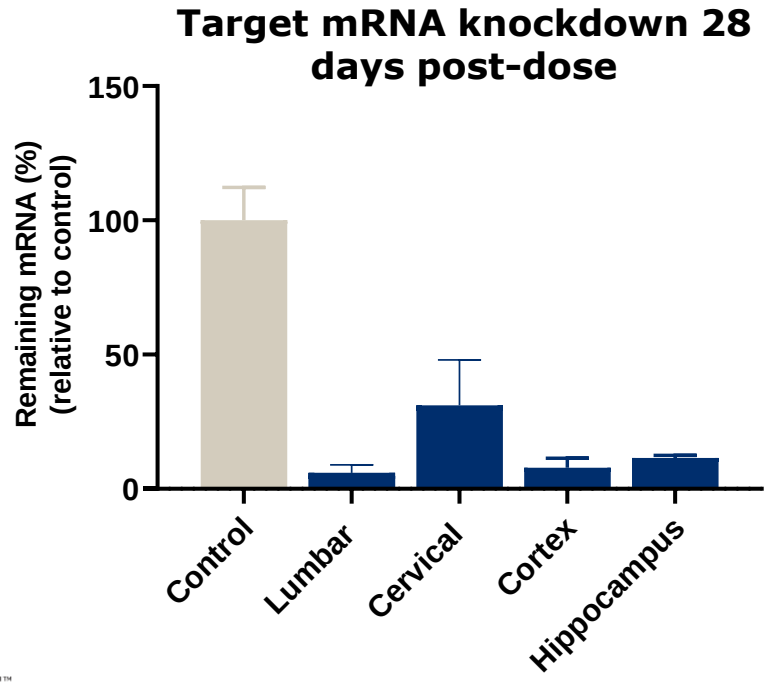
Mice received a single 100 ug ICV injection (n=3 per group). Relative fold-change in Malat1 expression is shown for the indicated tissues 10-weeks post-dose. Malat1 expression normalized to Hprt1. PBS, phosphate buffered saline; PS phosphorothioate; PO phosphodiester; PN Nitrogen-containing backbone; Hprt1, hypoxanthine-guanine phosphoribosyl transferase

PRISM

Potential of PN chemistry demonstrated in NHP CNS

Substantial and widespread target mRNA reduction following single intrathecal dose in NHPs

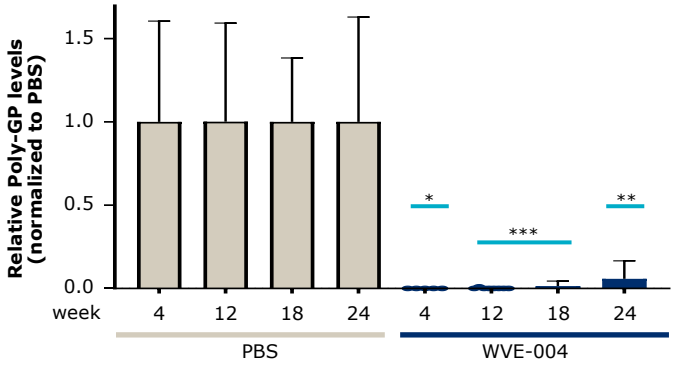


- Single IT dose of 12 mg (n=3)
- Therapeutic candidate widely distributed across brain and spinal cord
- ~90% mRNA knockdown one-month following single dose

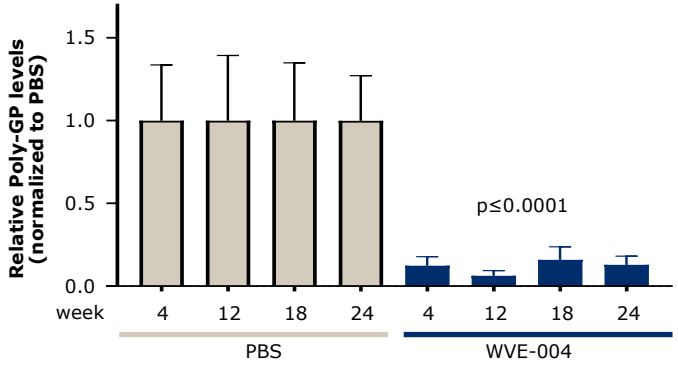
NHPs: Non-human primates; IT: intrathecal
NHPs were administered 12 mg on day 1 via IT bolus injection; tissue samples were collected from 3 NHPs at 28 days post-dose.

WVE-004 (C9orf72) demonstrates durable reduction of DPRs *in vivo* after 6 months in spinal cord and cortex

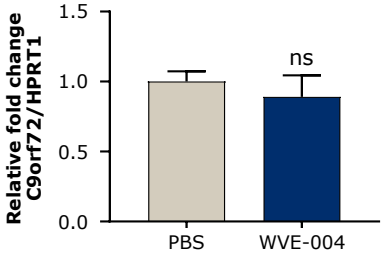
Spinal cord

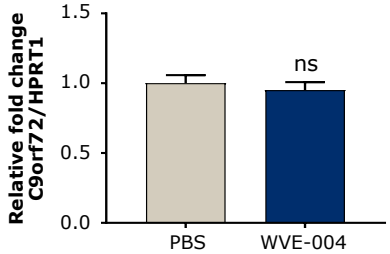


Cortex



Healthy C9orf72 protein relatively unchanged ~6 months after WVE-004 administration





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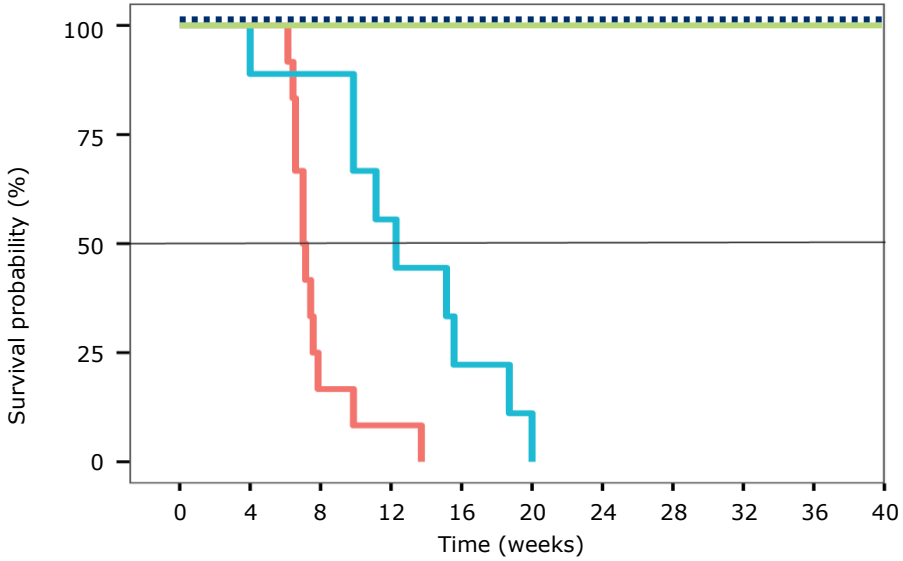
Full results presented at the 31st International Symposium on ALS/ MND (December 2020)
2 x 50 ug (days 0 & 7) dosed ICV to C9BAC transgenic mice; Top: DPRs measured by Poly-GP MSD assay. *: *P*≤ 0.05 **: *P*≤ 0.01, ***: *P*≤ 0.001. Bottom: relative expression of C9orf72 protein. ns: not significant; PBS: phosphate-buffered saline; ICV: intracerebroventricular; DPR: Dipeptide repeat protein; Poly GP: poly glycine-proline; MSD meso scale discovery

PN chemistry led to overall survival benefit in dKO model

PN-containing molecules led to 100% dKO survival at time of study termination



Next generation compounds

- PS/PO/PN, 150 mg/kg weekly
- PS/PO/PN, 75 mg/kg bi-weekly
- PS/PO, 150 mg/kg weekly
- PBS

Note: Untreated, age-matched mdx mice had 100% survival at study termination [not shown]

dKO; double knockout mice lack dystrophin and utrophin protein. mdx mice lack dystrophin. Mice with severe disease were euthanized. dKO: PS/PO/PN 150 mg/kg n=8 (p=0.0018); PS/PO/PN 75 mg/kg n=9 (p=0.00005); PS/PO n=9 (p=0.0024), PBS n=12 Stats: Chi square analysis with pairwise comparisons to PBS using log-rank test

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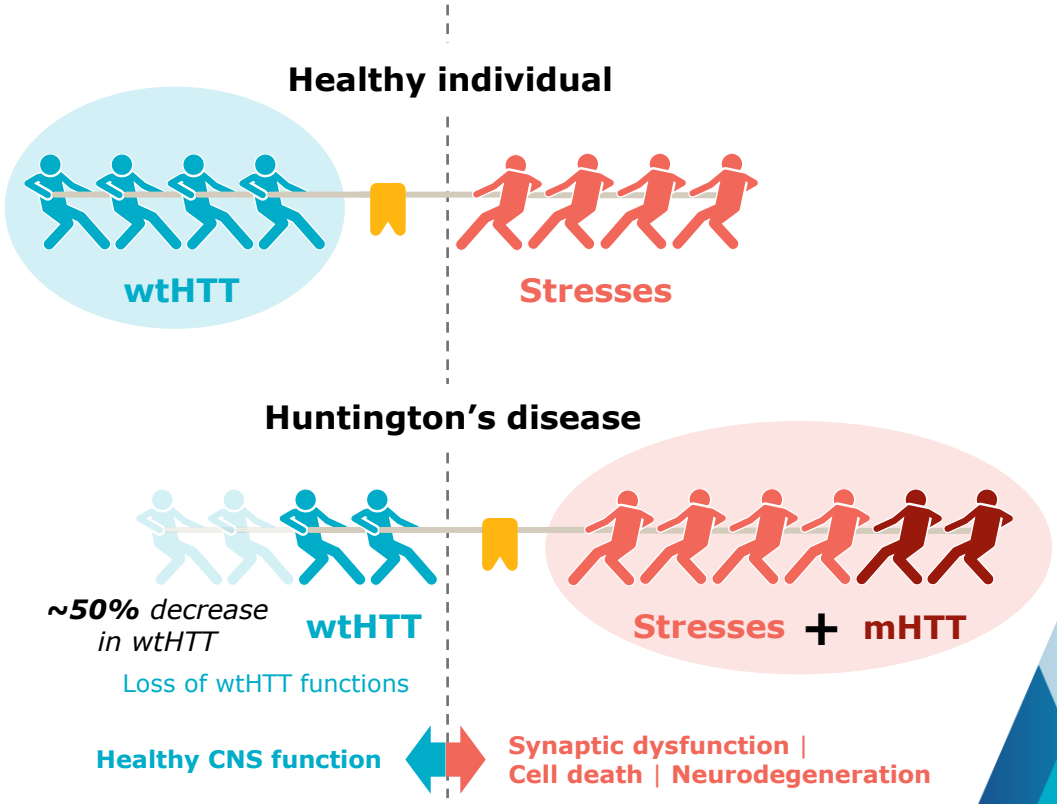
Mike Panzara, MD, MPH
Chief Medical Officer,
Head of Therapeutics
Discovery and Development

HD: mHTT toxic effects lead to neurodegeneration, loss of wtHTT functions may also contribute to HD

- Wild-type HTT is critical for normal neuronal function
- Expanded CAG triplet repeat in HTT gene results in production of mutant huntingtin protein
- Huntington's disease affects entire brain
- Monogenic autosomal dominant genetic disease; fully penetrant
- Characterized by cognitive decline, psychiatric illness, and chorea; fatal disease



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Wild-type HTT is a critical protein for important functions in the central nervous system

NEURON

Promotes neuronal survival by protecting against stress

SYNAPSE

Plays essential role in transport of synaptic proteins to their correct location at synapses

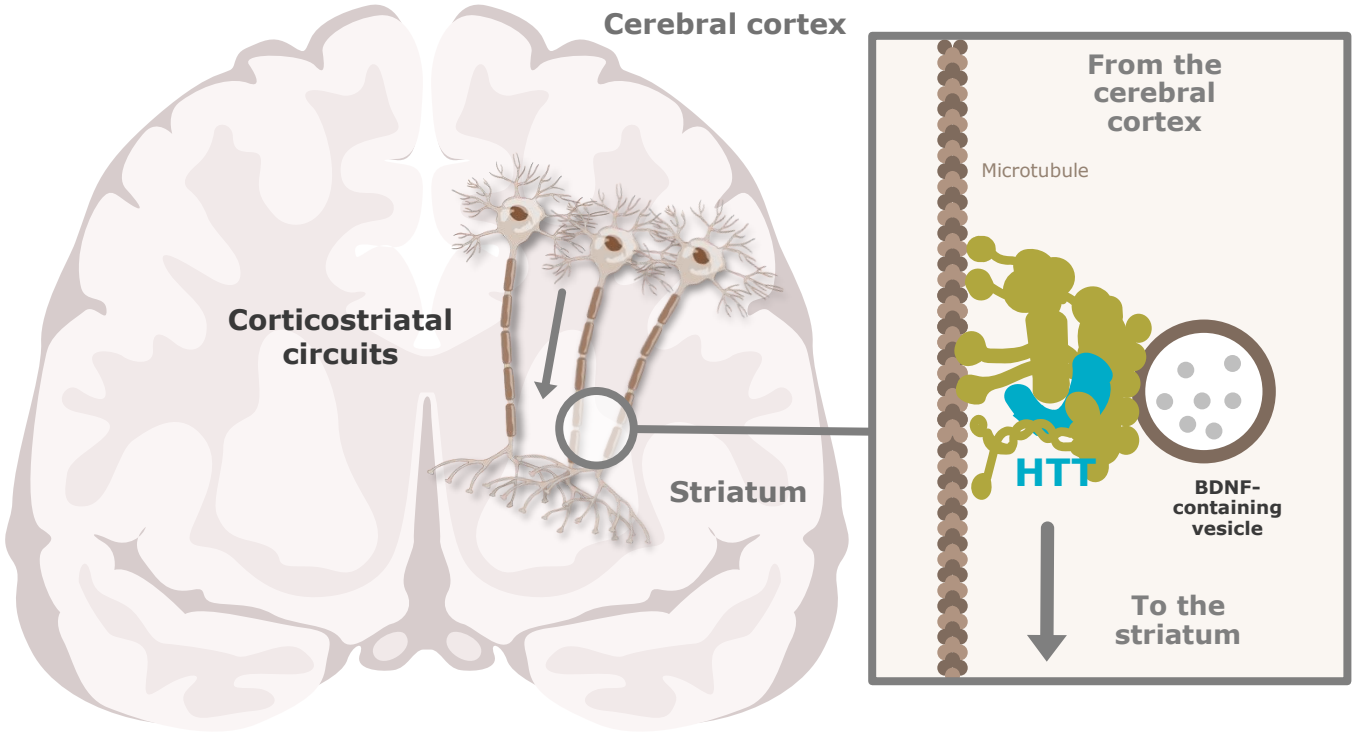
BRAIN CIRCUITS

Supplies BDNF to striatum to ensure neuronal survival and regulates synaptic plasticity, which underlies learning and memory

CSF CIRCULATION

Plays critical role in formation / function of cilia, which are needed to clear catabolites and maintain homeostasis

HTT provides BDNF, a growth factor critical for the survival of striatal neurons



Striatal neurons do not produce BDNF, but they need it to survive[1]

HTT promotes the production of BDNF and transports BDNF from the **cortex** to the striatum[2,3]

In HD, decreased levels of BDNF contribute to degeneration of corticostriatal circuits[2,4,5]

Reduction of wtHTT may decrease the availability of BDNF and accelerate corticostriatal degeneration[6]

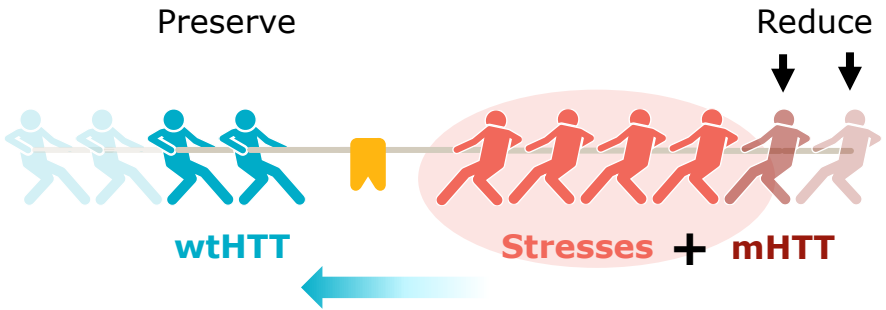
BDNF, brain-derived neurotrophic factor; HD, Huntington's disease; HTT, huntingtin protein.

1. Altar CA, Cai N, Bliven T, et al. *Nature*. 1997;389(6653):856-860. 2. Zuccato C, Ciammola A, Rigamonti D, et al. *Science*. 2001;293(5529):493-498. 3. Gauthier LR, Charrin BC, Borrell-Pagès M, et al. *Cell*. 2004;118(1):127-138. 4. Ferrer I, Goutan E, Marín C, et al. *Brain Res*. 2000;866(1-2):257-261. 5. Baquet ZC, Gorski JA, Jones KR. *J Neurosci*. 2004;24(17):4250-4258. 6. Cattaneo E, et al. *Nat Rev Neurosci*. 2005;6(12):919-930.

Allele-selective approach to treating HD

Wave has only allele-selective clinical program in Huntington's disease

- ✓ Target mutant mRNA HTT transcript to reduce mutant HTT protein
- ✓ Preserve wild-type HTT protein reservoir in brain



Only an allele-selective approach is designed to address <u>both</u> toxic gain of function and toxic loss of function drivers of HD

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Highly differentiated program supported by Wave's next generation chemistry

WVE-003
SNP3

Differentiated wild-type sparing approach
Only allele-selective clinical program

First HD clinical candidate containing PN chemistry
PN chemistry has demonstrated enhanced potency, exposure, durability in CNS

Clinical starting dose informed by preclinical *in vivo* model
Insight into PK / PD relationships

Clinical trial efficiencies
Adaptive trial design may enable rapid POC

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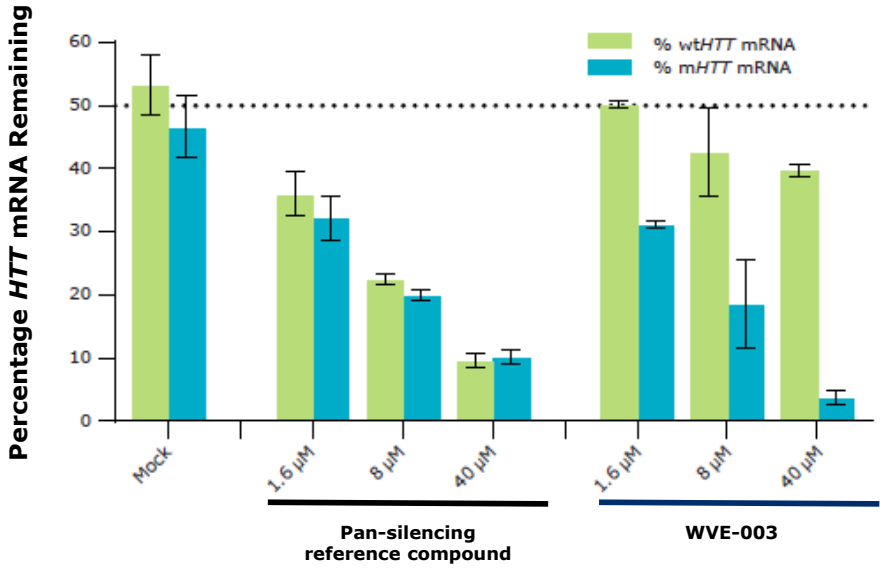
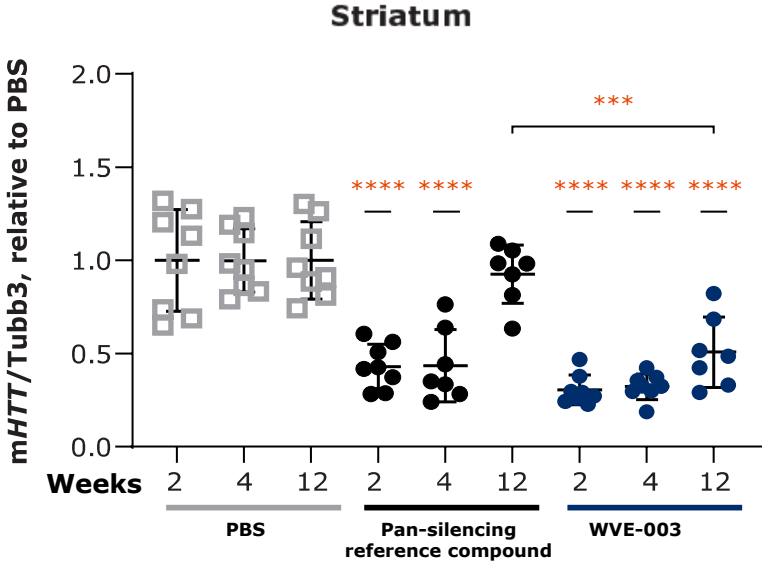
WVE-003 demonstrates selective, potent, and durable reduction of mHTT in preclinical models

Incorporates PN backbone chemistry modifications

Selectively reduces mHTT mRNA in HD iPSC neurons in vitro



Durable striatal mHTT knockdown for 12 weeks in BACHD mouse model



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Results from ND50036 iPSC-derived medium spiny neurons. Total *HTT* knockdown quantified by qPCR and normalized to HPRT1
Oligonucleotide or PBS [100 µg ICV injections on days 1, 3, and 5] delivered to BACHD transgenic. Mean ± SD (n=8, **P*<0.0332, ****P*<0.0002, *****P*<0.0001 versus PBS unless otherwise noted).
HPRT1, hypoxanthine-guanine phosphoribosyl transferase; iPSC, induced pluripotent stem cell; ICV, intracerebroventricular; PBS, phosphate-buffered saline

PK-PD modeling to guide dosing in clinical trial

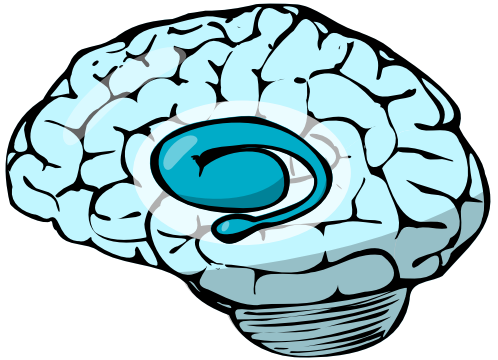
BACHD

Ascending dose studies
- PK & mHTT knockdown data
- IC_{50} determination

NHP

Concentrations in **cortex** and **striatum** sufficient for target engagement

Human
(cortex, striatum)



Anticipated mHTT knockdown in **cortex** and **striatum**

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PK: pharmacokinetic PD: pharmacodynamic IC_{50}: the concentration of observed half of the maximal effect mHTT: mutant huntingtin protein

Clinical trial incorporates adaptive design

Adaptive SAD/MAD design

- Patients with confirmed manifest HD diagnosis with SNP3 mutation (up to 40 patients planned); Participants from PRECISION-HD trial to be offered screening for SNP3 trial
- Dose escalation and dosing interval guided by independent safety committee
- Safety and tolerability
- Biomarkers
  - mHTT
  - NfL
  - wtHTT
- Clinical trial site activation ongoing

Dosing in Phase 1b/2a trial expected to initiate in 2021

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SAD: Single ascending dose MAD: Multiple ascending dose mHTT: mutant huntingtin protein NfL: neurofilament light chain wtHTT: wild-type huntingtin protein

WVE-004 (C9orf72) and WVE-N531 (Exon 53) clinical programs also supported by next generation chemistry

Oligonucleotide innovation and optimization
- PN backbone chemistry modifications
- Interactions between sequence, chemistry and stereochemistry

***In vivo* models**
- Insight into PK / PD relationships
- Novel model generation

Leverage learnings of first generation programs
- Translational pharmacology
- Adaptive clinical trial design

SNP3

WVE-003

Allele-selective silencing candidate in HD

C9orf72

WVE-004

Variant-selective silencing candidate in ALS and FTD

Exon 53

WVE-N531

Exon skipping candidate in DMD

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HD: Huntington's disease ALS: amyotrophic lateral sclerosis FTD: frontotemporal dementia DMD: Duchenne muscular dystrophy

Expected upcoming milestones

THERAPEUTIC AREA / TARGET	PRISM	Milestone
NEUROLOGY		
Huntington's disease mHTT SNP3	◆ ◆	**2021:** Dosing of first patient in clinical trial of WVE-003
ALS and FTD C9orf72	◆ ◆	**2021:** Dosing of first patient in clinical trial of WVE-004
DMD Exon 53	◆ ◆	**2021:** Dosing of first patient in clinical trial of WVE-N531
ADAR editing Multiple	◆ ◆	**1H 2021**: Humanized mouse model validation
HEPATIC		
AATD (ADAR editing) SERPINA1	◆ ◆	**1H 2021:** *in vivo* AATD data

PRISM ◆ Stereopure ◆ PN chemistry

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ALS: Amyotrophic lateral sclerosis FTD: Frontotemporal dementia AATD: Alpha-1 antitrypsin deficiency

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Q&A

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Realizing a brighter future for people affected by genetic diseases

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