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Company presentation

Carnegie Nordic Health Care Seminar

Stockholm, 25 March 2009

Forward looking statements

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This presentation contains certain “forward-looking Statements”, relating to NeuroSearch’s activities and business, which can be identified by the use of forward-looking terminology such as “estimates”, “believes”, “expects”, “may”, “are expected to”, “will”, “should” or similar expressions, or by discussions of strategy, plans or intentions. Such statements include descriptions of NeuroSearch’s research and development programmes and anticipated revenues and earnings in connection therewith. Such statements reflect the current views of the Company with respect to future events and are subject to certain risks, uncertainties and assumptions. Many factors could cause the actual results, performance or achievements of NeuroSearch to be materially different from any future results, performances or achievements that may be expressed or implied by such forward-looking statements. Such factors include, among others, risks associated with product discovery and development, uncertainties related to the performance and outcome of clinical trials, unforeseen product safety issues, manufacturing issues, and the lack of market approval or acceptance of our products as well as competitive risk and the lack of patents and proprietary rights’ protection. Should one or more of these risks or uncertainties materialize, or should underlying assumptions prove incorrect, actual results may vary materially from the forward-looking statements described in this presentation.

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  - Huntington's – ACR16; **Attractive orphan product in Phase III**
  - Obesity – Tesofensine; **Best in class**
  - Parkinson's dyskinesias – ACR325; **New speciality drug candidate**
- Expectations for 2009

Company profile

- Key competencies: CNS diseases - Ion channels
- Pipeline: 13 programmes
  - Phase III orphan drug for Huntington's disease
  - Tesofensine ready for Phase III in obesity
- Partners: Lilly, GSK and Abbott
- Cash position (4 March '09): $m +100
- Organisation: ~240 employees in DK and SE
- Spin-offs: Ownership in 6 biotech companies

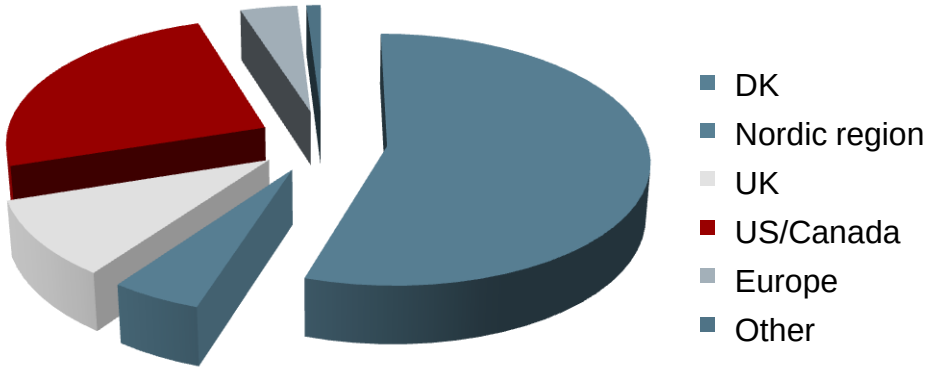
Equity and shareholder structure

- Shares outstanding (DKK 20 nom.) 16.3 million
- Closing price (23 March 2009) DKK 72
- Market Cap $m 206 / DKKm 1,133

Largest share holder groups

ATP	>10%
Oppenheimer Funds, USA	> 5%
GlaxoSmithKline Ltd	> 5%
Eli Lilly and Company	> 3%
Other institutional investors	~ 50%
Retail investors	~ 20%

Geographic shareholder distribution



2008 – 2009; Major highlights

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- Start of major in-house Phase III programme with lead specialist drug; ACR16 for Huntington's
- Tesofensine for obesity fully prepared for Phase III
- Phase II POC with ABT-894 in adult ADHD (Abbott collaboration)
- Novel specialist drug ready for first patient study in Parkinson's dyskinesias; ACR325
- New alliance agreements with Lilly and GSK
- Successful completion of Phase I with novel drug for schizophrenia; ACR343
- Additional pipeline strengthening; Novel PC and clinical drug candidates

Pipeline

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Indication	Programme	Mechanism	Partner	PC dev.	Phase I	Phase II	Phase III	NDA / Reg.
Huntington's disease	ACR16	Dopaminergic stabil.						
Obesity/Type 2 diabetes	Tesofensine	MRI						
ADHD	ABT-894	NNR modulator	Abbott					
Dyskinesias (PD)	ACR325	Dopaminergic stabil.						
Schizophrenia	ACR343	Dopaminergic stabil.						
Cognitive dysfunctions	ABT-560	NNR modulator	Abbott					
Alzheimer's /schizophr.	ABT-107	NNR modulator	Abbott					
Anxiety	NSD-788	MRI						
Pain	NSD-721	GABA modulator	GSK					
Autoimmune diseases	NSD-726	Ion channel mod.	GSK					
Schizophrenia	NSD-761	Ion channel mod.	GSK					
Psychosis	NSD-847	Dopaminergic stabil.	GSK					
ADHD	NSD-867	Cortical enhancer	GSK					

Strategy and business model

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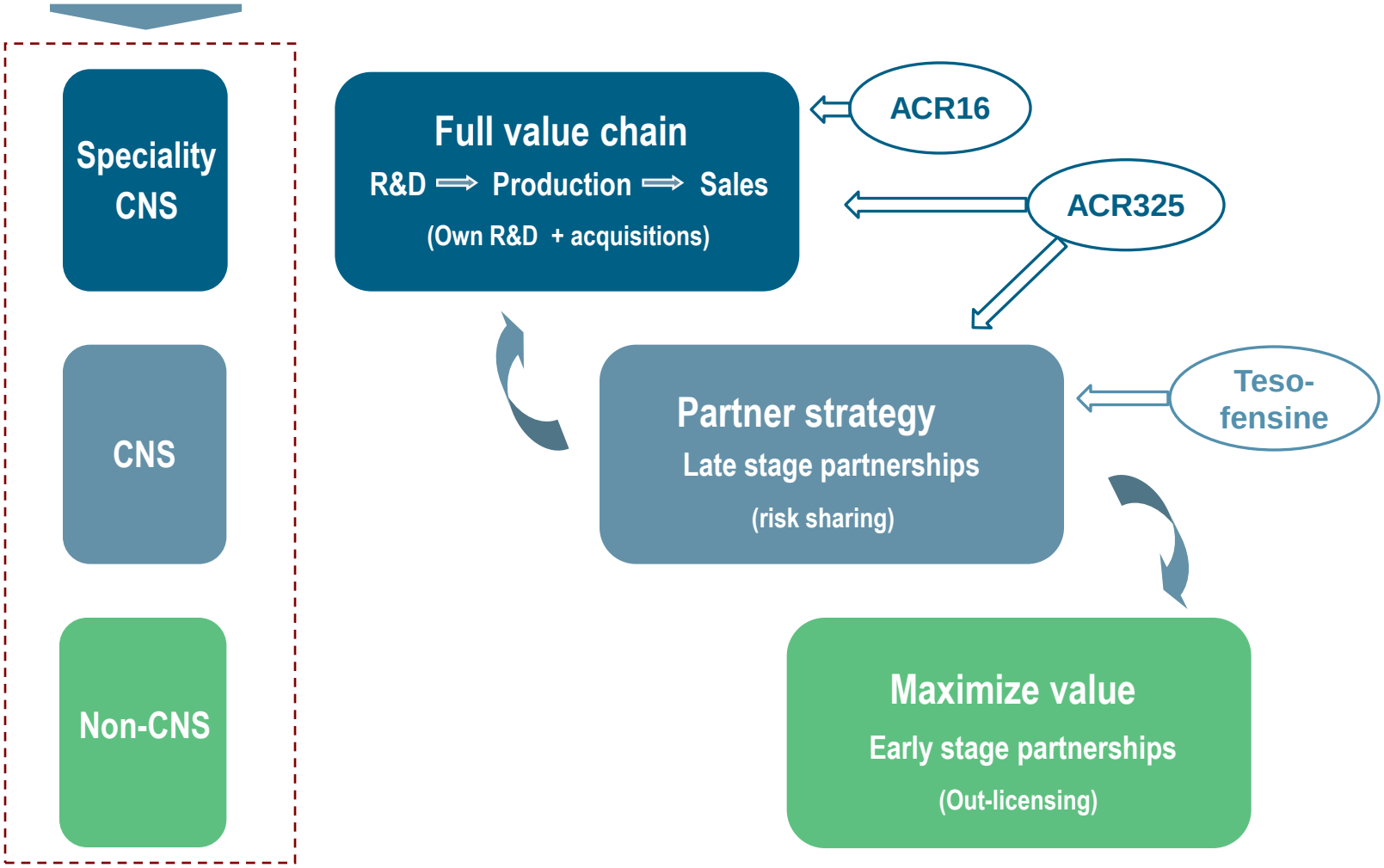
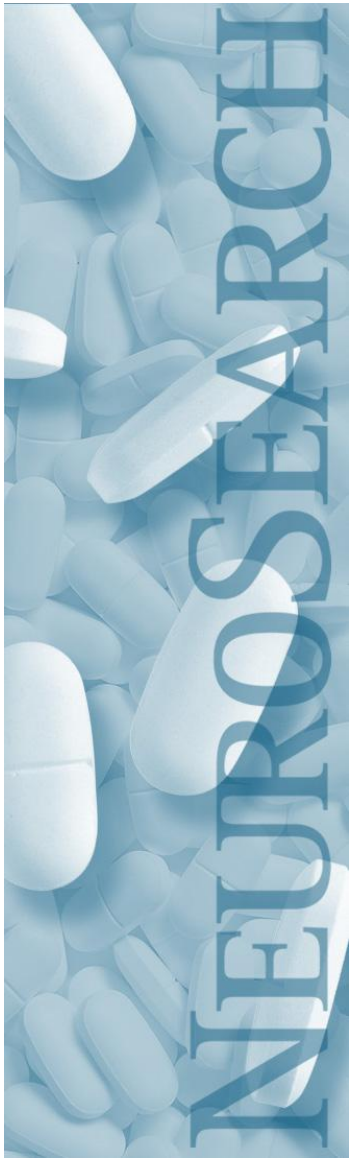
Current business areas



Business objectives

- Build speciality business;
  - Pivotal ACR16 (Huntington's) Phase III results end 2009
  - ACR16 launch planned for 2011
- Tesofensine (obesity); Aim for pre-Phase III partnering
- Ensure financing through to ACR16 Phase III data and launch
  - Funding through structured drug discovery alliances
  - Diligent cost control relative to income from partners
- Continuous pipeline growth



Alliance with Eli Lilly

Drug discovery and drug development collaboration

- Defined ion channel targets
- 3 years' duration – option for 1-2 years extension
- NeuroSearch to commit a number of FTE's p.a.
- Collaborative structure
  - NeuroSearch is responsible for discovery and PC phase
  - Lilly has different options to take over full responsibility and development
- Financing
  - Upfront; 22 m$ (17 m$ as equity investment at 187 DKK/share)
  - Upfront and research funding 2009-2011; 30 m$
  - Substantial upside in milestones and royalties
  - Developmental milestones will cover NeuroSearch's costs

Development portfolio under GSK alliance

Indication	Programme	Mechanism	PC dev.	Phase I	Phase II	Phase III	NDA / Reg.
Pain	NSD-721	GABA modulator					
Autoimmune diseases	NSD-726	Ion channel mod.					
Schizophrenia	NSD-761	Ion channel mod.					
Psychosis	NSD-847	Dopa. stabiliser					
ADHD	NSD-867	Cortical enhancer					

Additional number of compounds to enter PC dev in 2009

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gsk GlaxoSmithKline

Financials

- Up to EURm 109 in premarketing milestone payments on each compound
- Milestones from Phase I start – and earlier for the added compounds
- 20 m€ in GSK share put options relating to Phase I start with 4 drugs before end 2010
- Cost + milestone structure to secure more than full NeuroSearch funding

ACR16 - Lead specialist product

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Huntington's disease; Major commercial opportunity

- Market potential (orphan indication)
  - ~100,000 Huntington's patients worldwide
- High unmet need – and fast penetration
  - No effective treatment, poor symptoms relief
  - Only very few new drugs in development
- Attractive value increment in orphan pricing
- Production secured; very low cost price
- Limited in-house sales force: ~30-40 reps
- Global rights and IP protection until 2020 + 2-5 yrs ext.

Huntington’s disease (HD)

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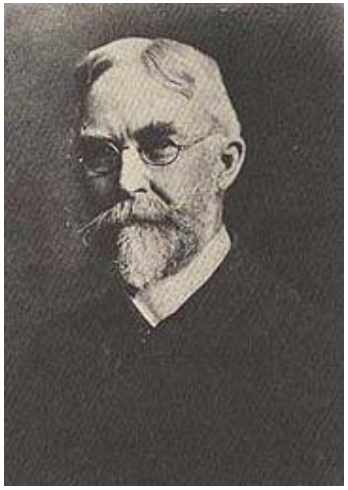
- Fatal, hereditary neurodegenerative disease
- Several disruptions in the brain
- Onset of symptoms at 35-45 yrs of age
  - ’Positive’ and ’negative’ motor dysfunctions
  - Cognitive impairment
  - Psychiatric/behavioural changes
- Progresses without remission and 10-15 yrs life expectancy after symptoms onset
- The majority of Huntington’s pts end in 24h care



DR. GEORGE HUNTINGTON (1850-1916)

Dopaminergic stabilisers – Unique MoA

- Dopaminergic stabilisers are able to
  - ***enhance*** or ***inhibit*** dopaminergic functions, depending on the initial level of activity
  - stabilise impaired **psychomotor functions** through effect at D2 receptors

- Effect in Huntington's disease
  - Improve voluntary motor functions *and* stabilize psychomotor function

- Effect in other psychomotor disorders
  - Reduce dyskinesia and dystonia *and* stabilize psychomotor function

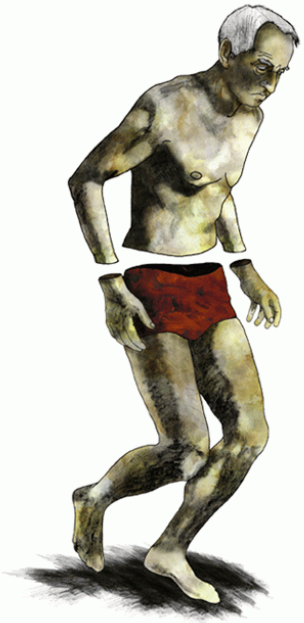


ILLUSTRATION FROM THE BOOK *SYMPTOM*. A COLLECTION OF SYMBOLIC PORTRAITS AND INTERVIEWS WITH PEOPLE DIAGNOSED WITH A SURTAIN DISEASE - **PARKINSONS**

Huntington's disease – Clinical features

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- Impairment of voluntary movements (i.e. 'negative' motor symptoms) most closely linked to functional decline

  (Mahant et al, Neurology, 2003)

- Chorea and dystonia (i.e. 'positive' motor symptoms) are not major determinants of functional disability in HD

- Cognitive decline correlates with loss of voluntary movement ability

Key target for HD treatment:
Alleviation of voluntary motor impairment

ACR16 (Huntington's) - Results from Phase II

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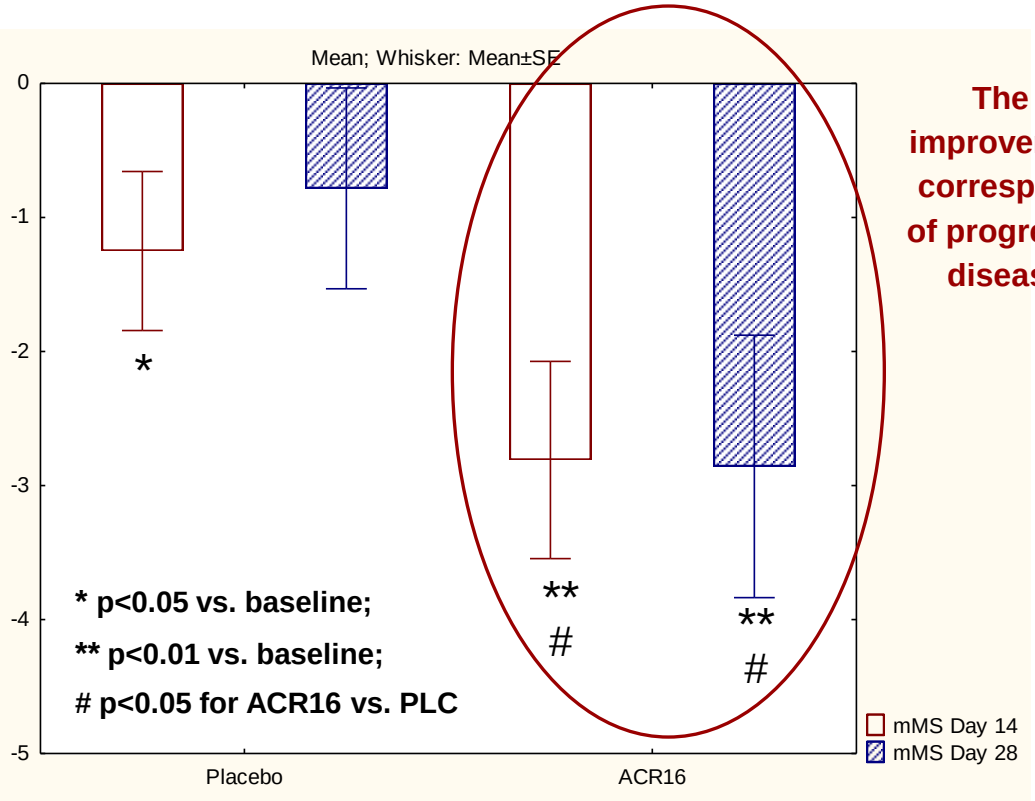
Significant effect of ACR16 on voluntary movements (mMS);



The observed improvement on mMS corresponds to ~1 yr of progression on this disease measure

Phase II design

-60 Huntington's patients

- 2 arm placebo controlled double-blinded study

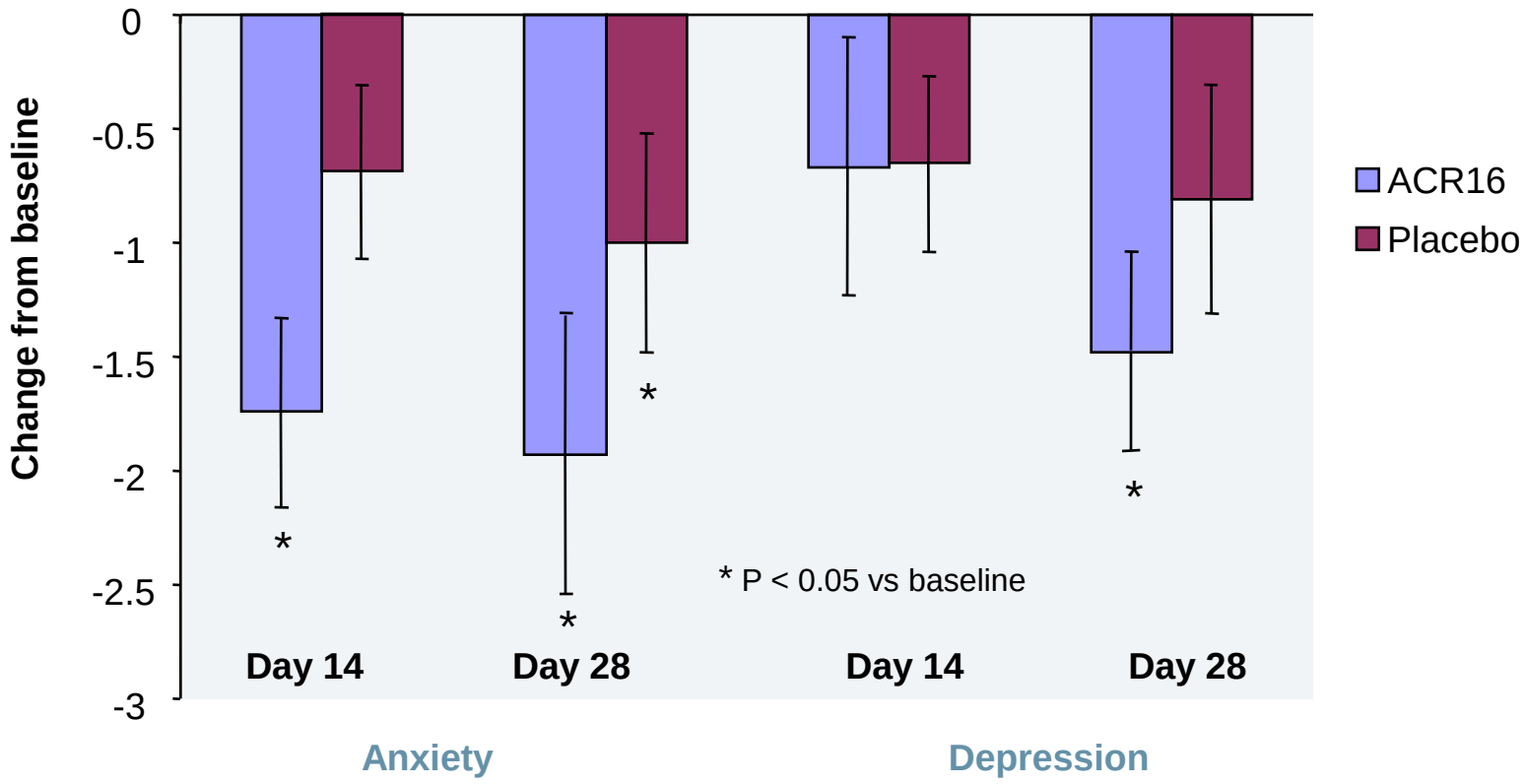
- 45 mg ACR16 QD for 28 days

Modified motor score (mMS) change vs. baseline after 14/28 days of treatment.

Mean SEM, subjects displaying mMS > 10 at baseline.

ACR16 (Huntington's) - Results from Phase II

Positive trends in ACR16's effect on psychiatric symptoms (HADS)



ACR16 (Huntington's) – Pivotal programme

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MermaiHD; Pivotal European Phase III study

- 420 patients, ~ 30 centres in 8 EU countries
- 6 months treatment; 45 mg QD and 45 mg BID + 6 months FU
- Enrolment soon to complete – first patients to complete 12 mths treatment
- Initiated Apr '08 - Results expected end 2009

HART; US Phase IIb confirmatory study

- 220 patients, ~ 30 centres in US/Canada
- 3 months treatment; 10 mg, 22.5 mg, 45 mg – all BID
- Initiated Oct '08 - Results expected in the beginning of 2010

PE; Voluntary motor functions

- Measured on the modified Motor Score (mMS) – accepted by FDA and EMEA

Obesity - Tesofensine

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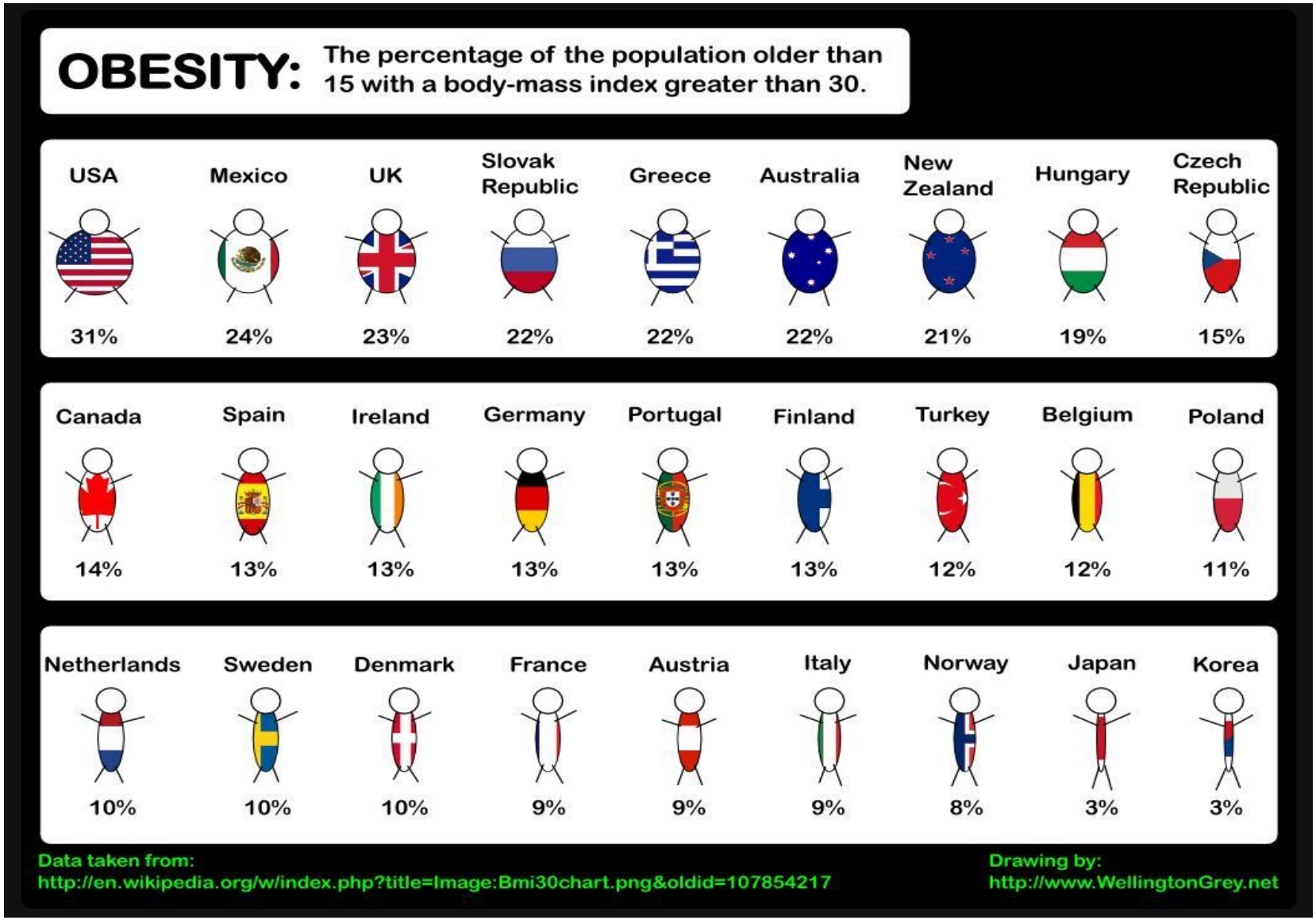

Tesofensine (obesity) - New mechanism

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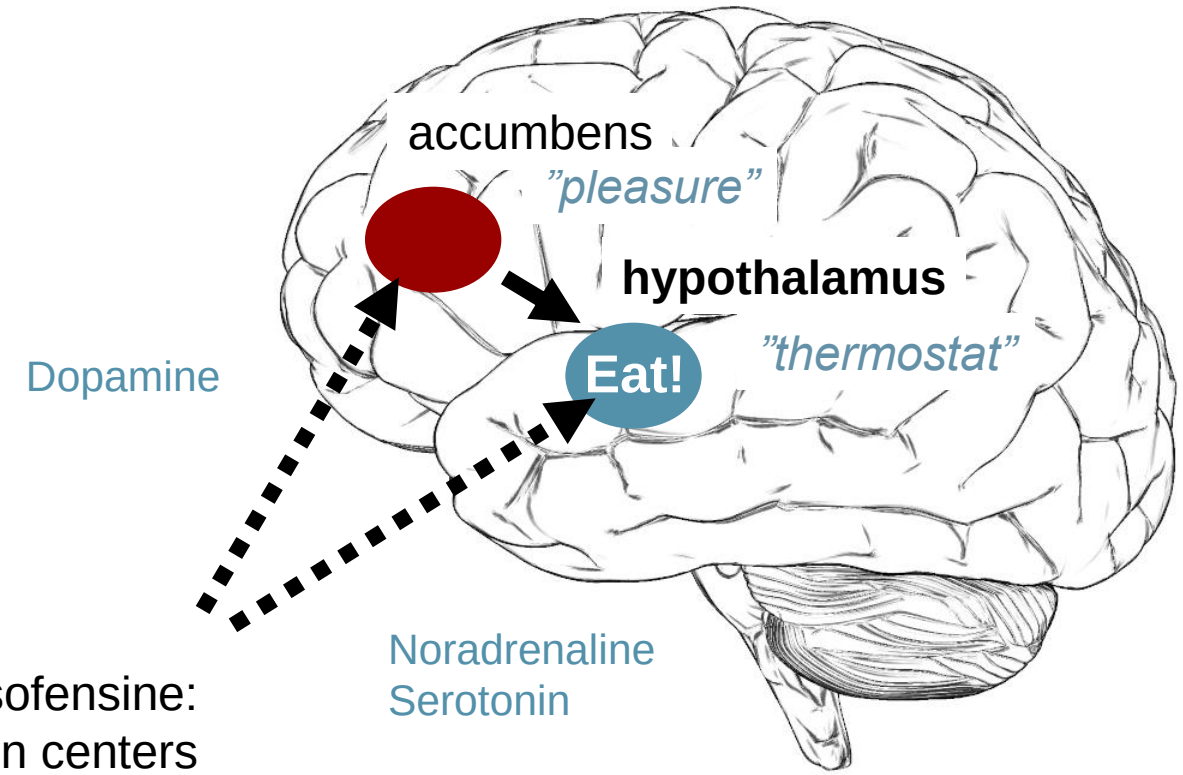
Dual acting mechanism for weight loss:

Pre-synaptic inhibition of DA, NA and 5-HT reuptake



Tesofensine:
Acts on two brain centers

Tesofensine (obesity) – POC results

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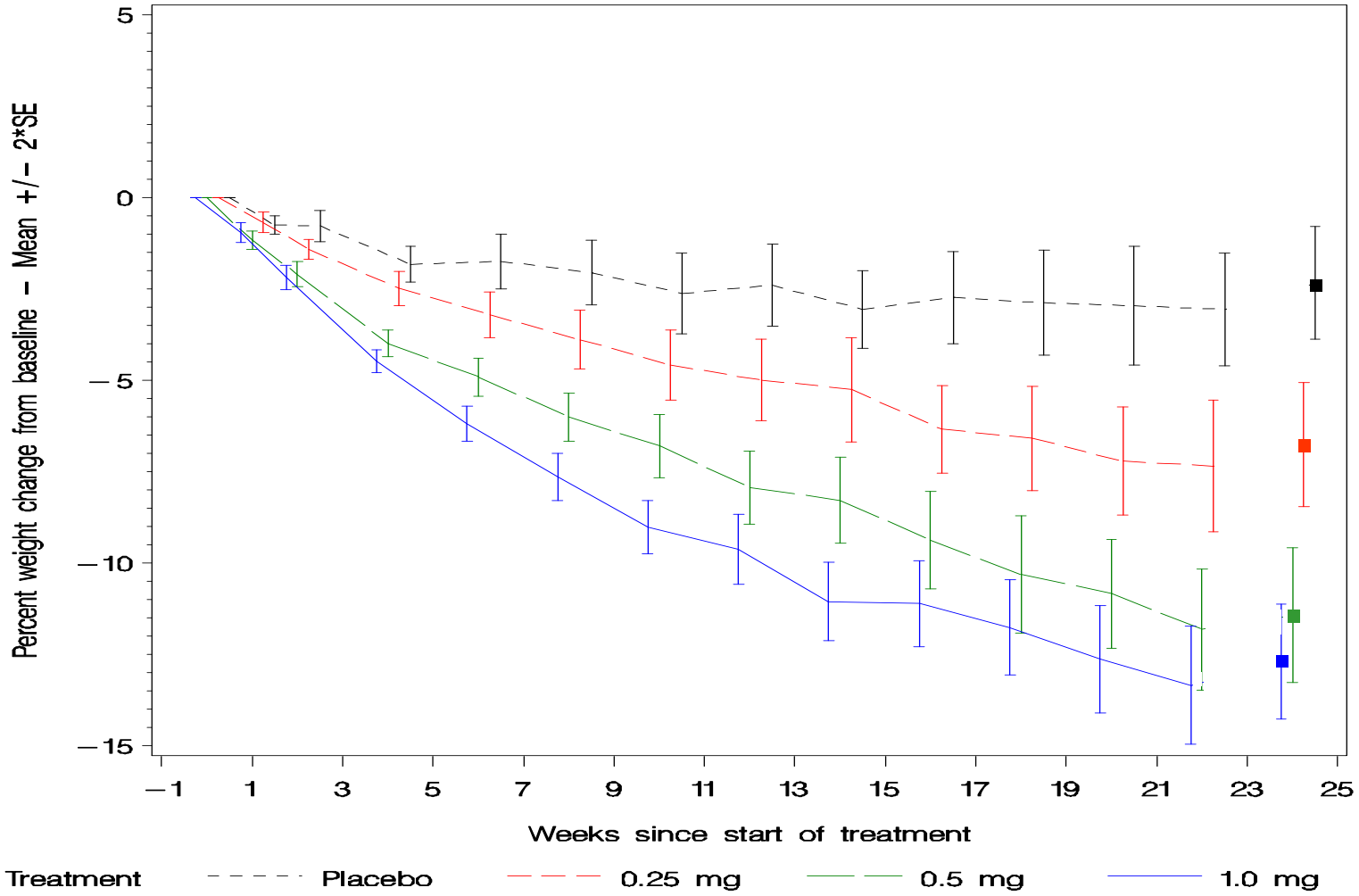
TIPO-1 results: Relative changes (%) in body weight



Tesofensine (obesity) – Phase IIb results

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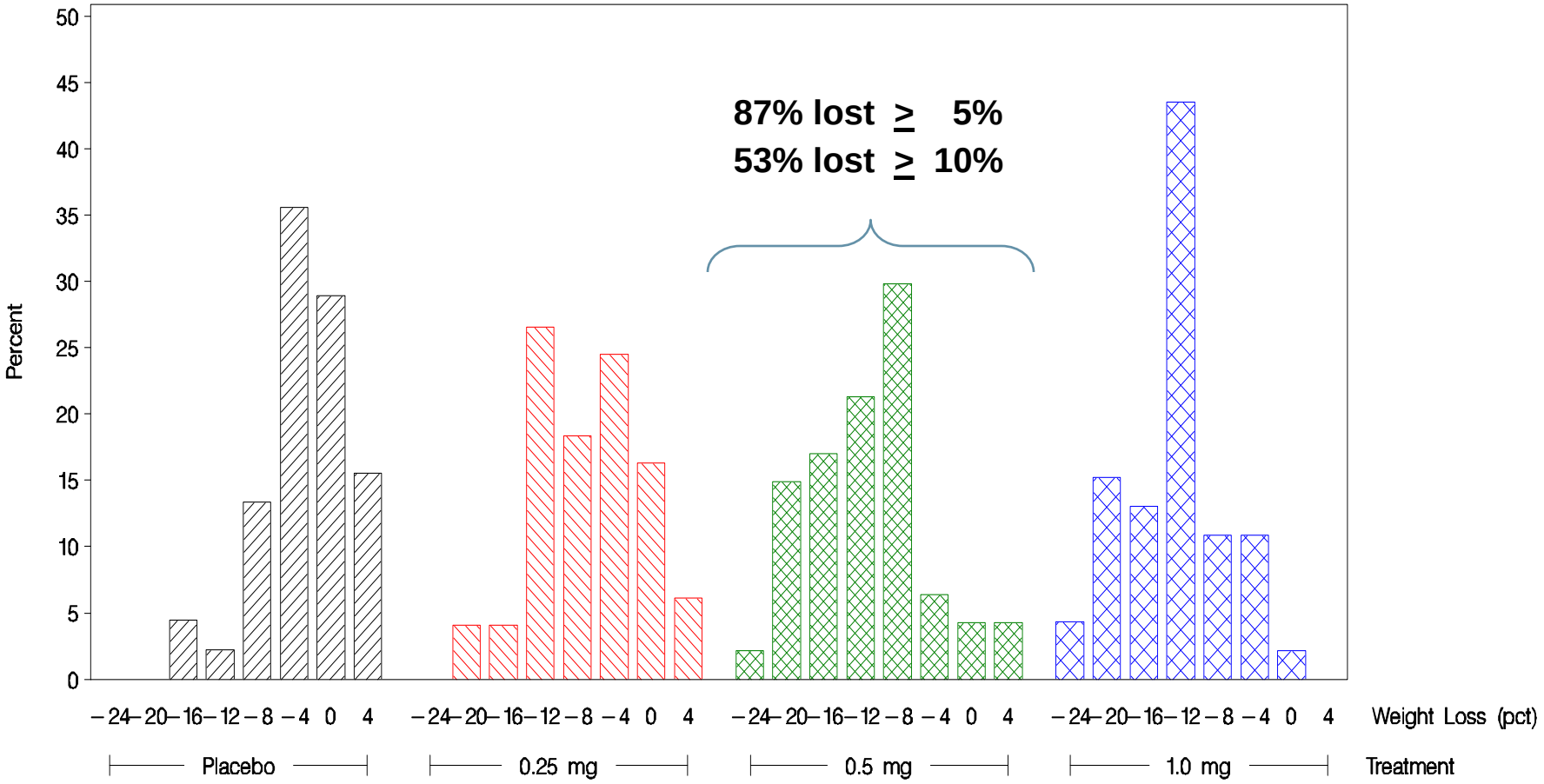
Relative (%) weight change from baseline (ITT = 203)



Tesofensine – Unique weight loss efficacy

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Phase II PoC (TIPO-1) results published in the Lancet, Oct 2008;

Effect of tesofensine on bodyweight loss, body composition, and quality of life in obese patients: a randomised, double-blind, placebo-controlled trial

Arne Astrup, Sten Madsbad, Leif Breum, Thomas J Jensen, Jens Peter Kroustrup, Thomas Meinert Larsen

Summary

Background Weight-loss drugs produce an additional mean weight loss of only 3–5 kg above that of diet and placebo over 6 months, and more effective pharmacotherapy of obesity is needed. We assessed the efficacy and safety of tesofensine—an inhibitor of the presynaptic uptake of noradrenaline, dopamine, and serotonin—in patients with obesity.

Methods We undertook a phase II, randomised, double-blind, placebo-controlled trial in five Danish obesity management centres. After a 2 week run-in phase, 203 obese patients (body-mass index 30–≤40 kg/m²) were prescribed an energy restricted diet and randomly assigned with a list of randomisation numbers to treatment with tesofensine 0·25 mg (n=52), 0·5 mg (n=50), or 1·0 mg (n=49), or placebo (n=52) once daily for 24 weeks. The [illegible] outcome was percentage change in bodyweight. Analysis was by modified intention to [illegible] with measurement after at least one dose of study drug or placebo). The [illegible] number NCT00394667.

Published Online October 23, 2008 DOI:10.1016/S0140-6736(08)61525-1

Department of Human Nutrition, Faculty of Life Sciences (Prof A Astrup MD, T M Larsen PhD) and Department of Endocrinology (Prof S Madsbad MD)

Conclusion:

Tesofensine provides over 24 weeks at least twice the efficacy as compared to available drugs for weight management

Source: www.thelancet.com Published online October 23, 2008 DOI:10.1016/S0140-6736(08)61525-1

Tesofensine (obesity) - Long term results

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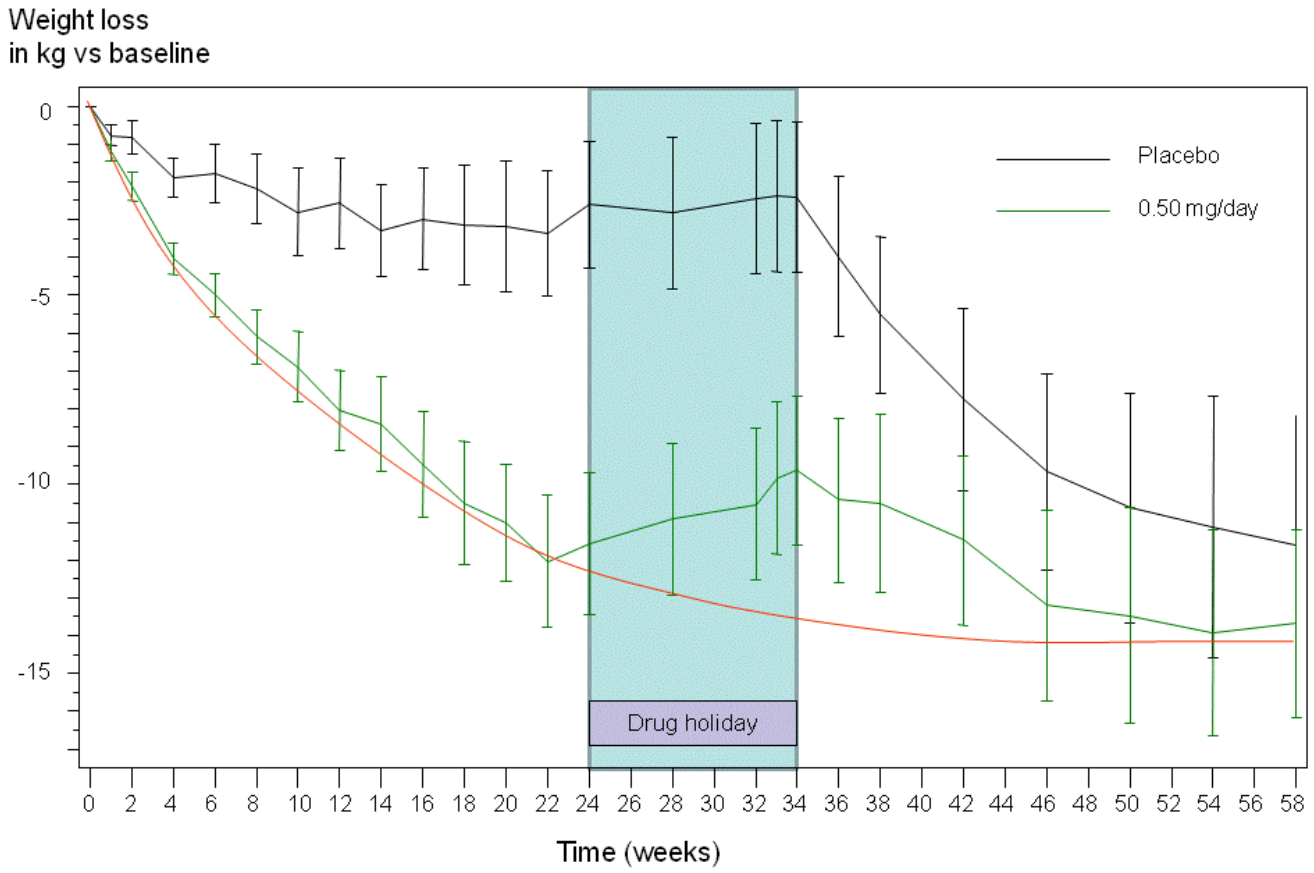
Combined weight loss from TIPO-1 + TIPO-4

- Combined 48 wks weight loss of ~13 kg (sustained after 72 wks)
- Confirmed 24 wks placebo-adjusted weight loss of > 9 kg



Tesofensine (obesity) – Best in class

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TIPO-1 (24 wk POC study)

- Efficacy second to none; 2-3x the effect of existing drugs
- Low drop-out and only mild and transient side-effects

TIPO-4 (48 wk, 0.5 mg open-label extension study)

- Additional weight loss; ~13 kg at interim, sustained over 48 weeks
- Safety results in line with TIPO-1

Abuse liability study with highly supportive results

CV assessment study (supra-therapeutic dosing)

- Results showed no BP or pulse increases during CV challenges

Tesofensine (obesity) – Ready for Phase III

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Ongoing activities

- Discuss data package with FDA and EMEA in 1H'09
- Final Phase III strategy based on outcome of regulatory interactions

Outline of Phase III programme

- Full compliance with FDA guidelines (approx. 6,000 pts)
- 12 months + 12 months FU
- Include studies in pts with co-morbidities (T2D, hypertension etc)

Partnering process runs in parallel

ACR325 – Selected as new specialist drug

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Highly favourable results from PC and Phase I studies

- Very good tolerability and safety profile up to supra-therapeutic levels
- Linear and predictable PK profile
- Novel and unique brain activity (supported by PET data)
- Highly relevant mechanism for the treatment of dyskinesias

Dyskinesias in Parkinson's disease – Specialist indication

- Approximately 4,000,000 Parkinson's patients
- L-dopa is the standard treatment
- Dyskinesias occur in approximately 30% of patients after 5 years of L-dopa treatment and in 60-80% of patients after 10 years of L-dopa treatment

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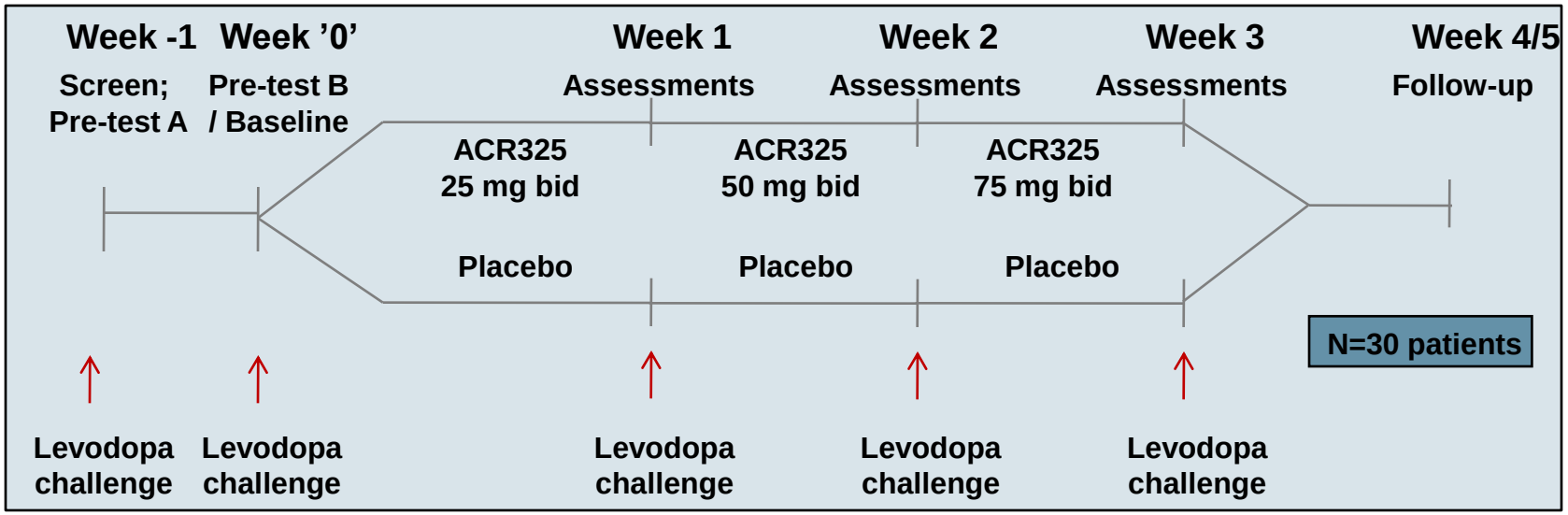
Initiation of Phase Ib study in PD dyskinesias expected in H1 2009

– In Parkinson patients with moderate to severe L-dopa induced dyskinesias



Study endpoints:

– Effect on levodopa-induced dyskinesias (induced L-Dopa challenge)

– Safety, tolerability and PK/PD

Expectations for 2009

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- **Huntington's disease - ACR16**
  - Results from pivotal Phase III programme
- **Obesity - Tesofensine**
  - Outcome of regulatory interactions
  - Continue partner discussions
- **Dyskinesias in Parkinson's disease - ACR325**
  - Initiation of Phase Ib study in Parkinson's patients with dyskinesias
- **Schizophrenia - ACR343**
  - Initiation of Phase II studies
- **Further pipeline advances; Abbott, GSK and own programmes**
- **Additional drug discovery-based collaborations**

For more information, please visit www.neurosearch.com
or write to investor@neurosearch.dk