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Interim Report H1 2015
Reporting period January – June 2015

Halle (Saale), 27 August 2015

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Content

1. **Corporate introduction**
2. Pipeline Update
3. Results January – June 2015
4. Outlook
5. Q & A

Key Highlights January to June 2015

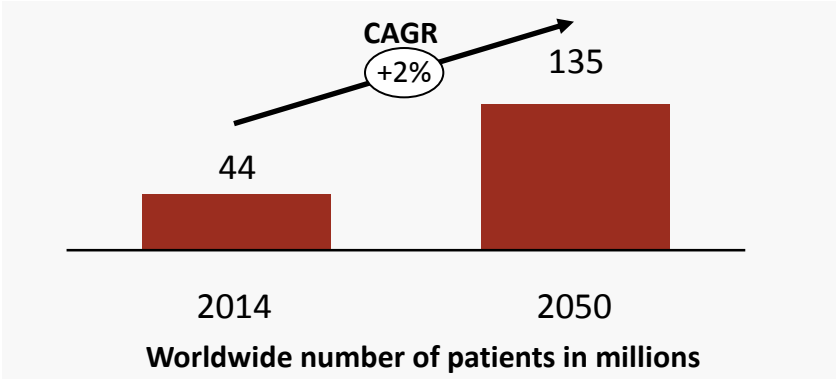
- Phase 2a study of novel treatment for Alzheimer's disease, the SAPHIR trial, initiated
- Additional data on Glutaminyl Cyclases (QCs) in Alzheimer's disease published in *Acta Neuropathologica*
- Data on Probiodrug's Anti-pGlu-3 Abeta monoclonal antibody presented at the 12th AD/PD™ 2015, Nice
- Key patents on Glutaminyl Cyclase (QC) inhibition for the treatment of AD granted in Japan
- Winner of the European Mediscience Award 2015 for Best Technology
- Funding of Alzheimer research at Brigham and Women's Hospital, affiliated with Harvard Medical School
- Cash and cash equivalents of EUR 14.8 million as of 30 June, 2015
- Net loss of EUR 6.2 million for the first six-months period compared with EUR 3.4 million in 2014 - in line with company expectations
- Annual General Meeting held in June, all resolutions proposed by Management and Supervisory Board approved
- New members of the Supervisory Board with distinguished industry expertise appointed

Alzheimer's disease: growing burden, no cure

Alzheimer's disease introduction*

- Leading cause of dementia, ultimately leading to death
- Large burden on families
- Growing cost for society
- Available treatments marginally effective and focus on symptoms only
- Current symptomatic treatments generate ~$4bn p.a.***
- No disease modifying beneficial treatments available
- No new drugs approved since 2007****

Worldwide patient population will triple in the next 30 years**



* Company estimates, ** WHO Dementia Report 2014, ***Datamonitor, **** FDA, Source picture: Alzheimers.org

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Investment highlights

1	**Alzheimer's disease: major burden, no cure**
2	**Attractive industry landscape**
3	**Clearly differentiated approach**
4	**Focused proprietary pipeline**
5	**Strong IP protection**
6	**Experienced management team and renowned investor base**

- Alzheimer's disease ("AD") is a devastating neurological disease affecting over 44 million people world-wide*
- No cure or long-term beneficial treatment available
- No new drugs approved since 2007**

- After years of "drought" rising interest in AD/ neurodegeneration
- Only few major pharma players in the field with clinical programs
- Limited number of innovative approaches available on the biotech side

- Developing a differentiated approach aimed to treat AD
- Building on proprietary know-how of AD biology, taking into account the latest insights in AD drug development

- A novel target in AD: pGlu-Abeta
  - PQ912: small molecule, first of its kind in clinical trials – phase 2
  - PBD-C06: antibody, complementary mode of action – pre-clinical

- Extensive ownership of IP:
  - Granted composition of matter patents
  - Granted medical use patents

- Established drug development and CNS expertise
- Track record of monetizing cutting-edge science (diabetes/DP4-inhibitor drugs)
- Committed support from leading financial and strategic investors

* World Alzheimer Report 2014 ** FDA

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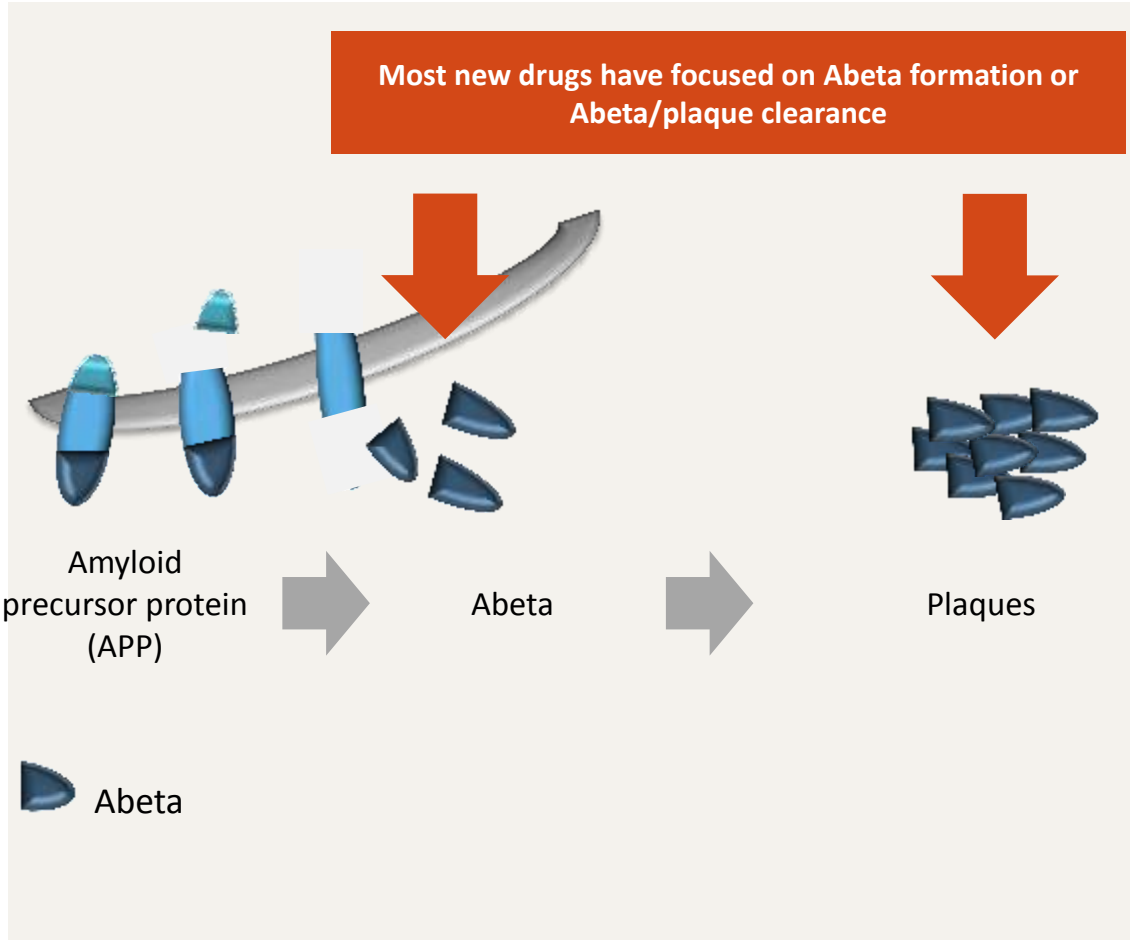
Experienced management team

Management team		Biography
Konrad Glund, PhD, CEO Co-founder Chairman of the management board		▪ Co-founder of Probiodrug, CEO since 2006 ▪ Led development of DP 4 inhibitors, transactions with Merck, Novartis, OSI and Ferring ▪ COO & VP business development OSI (Prosidion) in 2004-2006 ▪ > 10 deals at OSI, including phase 1 deal with pharma
Hendrik Liebers, PhD CFO Member of the management board		▪ Longstanding track record in venture and private capital, CFH and IBG ▪ Numerous board seats in biotech companies ▪ > 20 financing rounds, M&A transactions, trade sales
Inge Lues, PhD CDO Member of the management board		▪ Advisor to biotech companies and public research institutions ▪ Family office E. Merck KG ▪ EVP member of the Pharma Board, Merck KGaA ▪ Head Global Drug Discovery and Non-Clinical Development; Head, Business Area Team, CNS Pharma, Merck KGaA
Frank Weber MD, CMO		▪ Global Clinical Advisor of InterMune ▪ Chief Medical Officer at Merck KGaA ▪ Several medical affairs and clinical development management positions at American Cyanamid/Lederle, Synthelabo, Merck KGaA

Original Abeta approach

Probiodrug targets toxic structures in Alzheimer's disease



Considerations

- Most new drug treatments have targeted Abeta or plaques
- Therapies have focused on:
  1. Reduction of Abeta formation
  2. Clearance of existing Abeta or plaque
- To date, several drug development attempts based on this original Abeta approach have failed – except one Abeta antibody in an early trial - others are ongoing and have yet to show benefit

Source: Company analysis, Mullard, Nature Review Drug Discovery, September 2012

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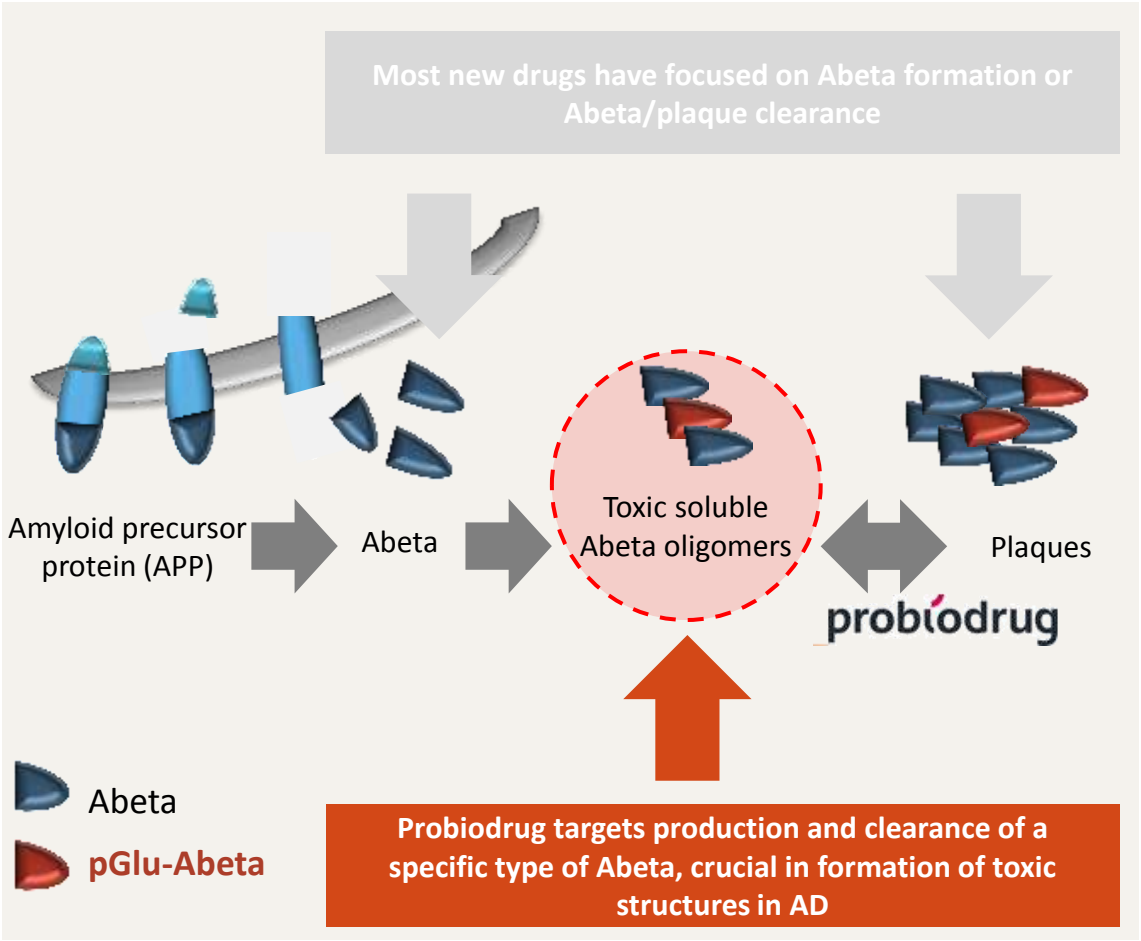
Probiodrug's **differentiated** approach

Probiodrug targets toxic structures in Alzheimer's disease



Considerations

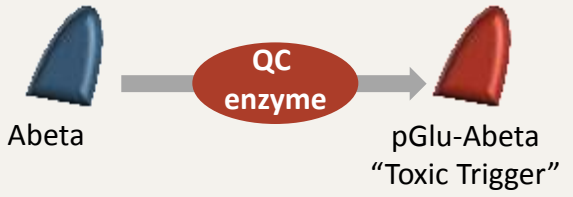
- Probiodrug and others have progressed insights on Abeta and its role in AD
- Abeta has a physiological function
- Plaques are not the primary toxic culprit
- In fact, a "pre-plaque"* structure is most toxic and relevant from a clinical perspective
- Probiodrug targets a specific type of Abeta, **pGlu-Abeta**, which is crucial in the formation of these toxic "pre-plaques"

Source: Company analysis, Mullard, Nature Review Drug Discovery, September 2012

“Prevent formation” concept

Mechanism:

QC (Glutaminyl Cyclase) is crucial for pGlu-Abeta formation

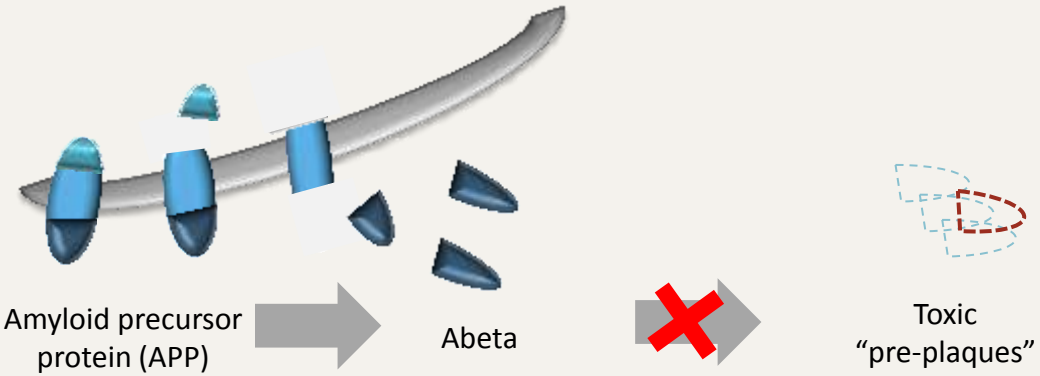


QC enzyme is crucial for pGlu-Abeta formation

Inhibiting QC, no pGlu-Abeta

Therapeutic effect:

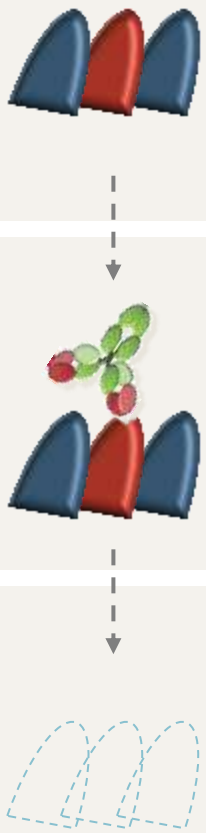
Aim to prevent formation of toxic “pre-plaques”



Probiodrug was first to discover the role of QC and has full ownership of broad target IP

“Capture and Clear” concept

1	Toxic “pre-plaques” and plaques containing pGlu-Abeta
2	Antibody to capture and clear existing pGlu-Abeta
3	Reduced number of toxic oligomers of Abeta



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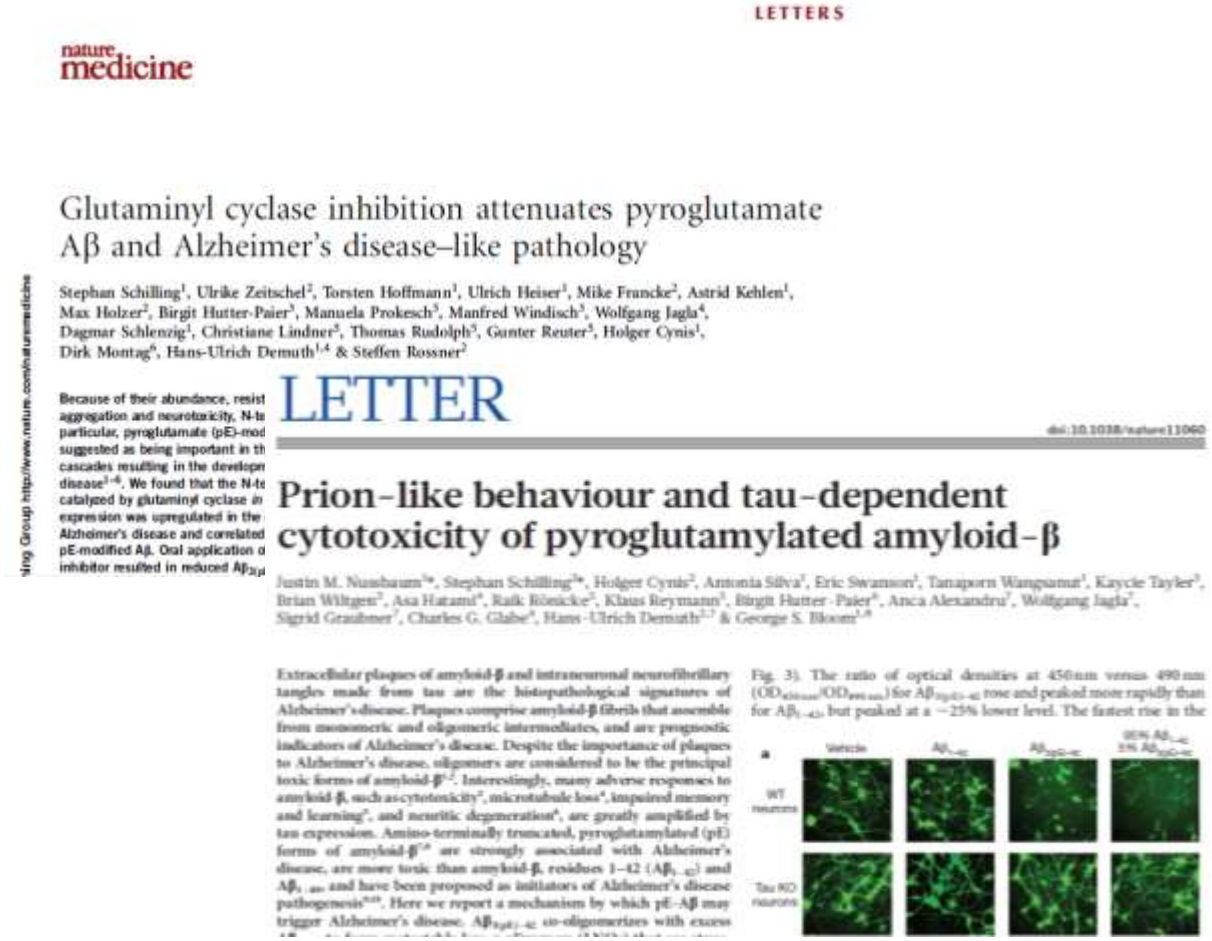
Extensive evidence for differentiated approach

Publications *(selection)*



LETTERS

nature medicine

Glutaminyl cyclase inhibition attenuates pyroglutamate Aβ and Alzheimer's disease–like pathology

Stephan Schilling[1], Ulrike Zeitschel[2], Torsten Hoffmann[1], Ulrich Heiser[1], Mike Francke[2], Astrid Kehlen[1], Max Holzer[2], Birgit Hutter-Paier[3], Manuela Prokesch[3], Manfred Windisch[3], Wolfgang Jagla[4], Dagmar Schlenzig[1], Christiane Lindner[5], Thomas Rudolph[5], Gunter Reuter[5], Holger Cynis[1], Dirk Montag[6], Hans-Ulrich Demuth[1,4] & Steffen Rossner[2]

LETTER

doi:10.1038/nature11060

Prion-like behaviour and tau-dependent cytotoxicity of pyroglutamylated amyloid-β

Justin M. Nussbaum[1]*, Stephan Schilling[2]*, Holger Cynis[2], Antonia Silva[1], Eric Swanson[1], Tanaporn Wangsanut[1], Kaycie Tayler[1], Brian Wiltgen[1], Asa Hatami[1], Ralf Rönicke[3], Klaus Reymann[3], Birgit Hutter-Paier[4], Anca Alexandru[5], Wolfgang Jagla[5], Sigrid Graubner[5], Charles G. Glabe[1], Hans-Ulrich Demuth[2,5] & George S. Bloom[1,6]

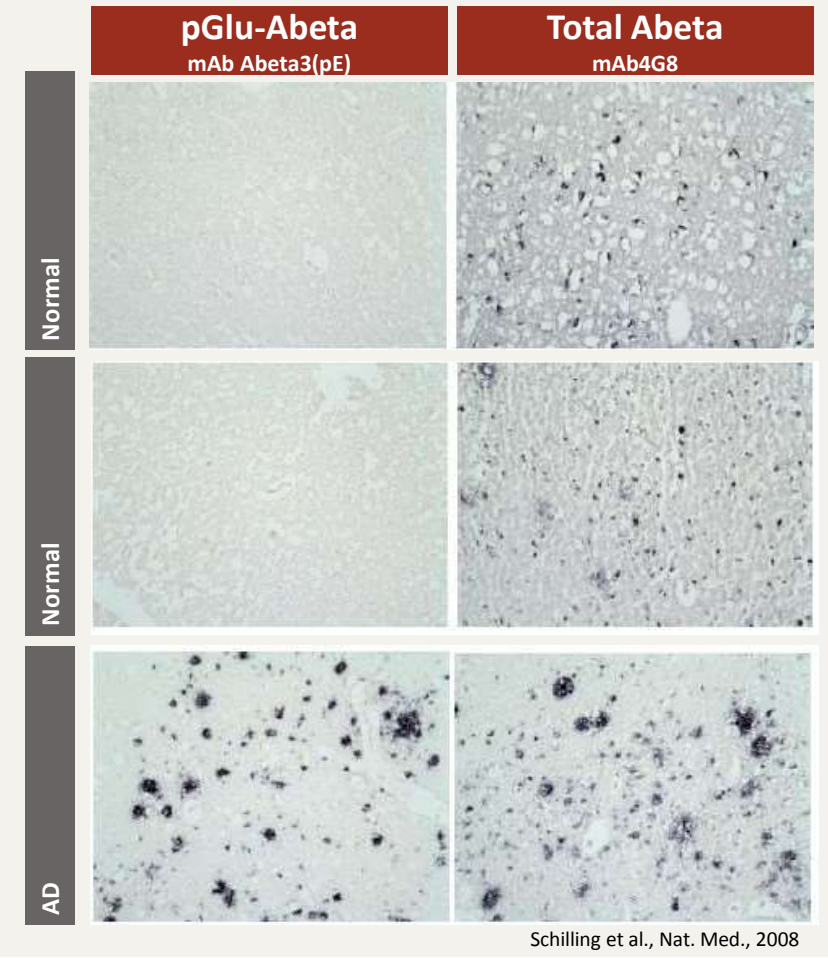
Core data

- pGlu-Abeta is specific for AD
- pGlu-Abeta and oligomers correlate with disease progression
- QC is crucial for pGlu-Abeta production
- QC overexpression drives pGlu-Abeta and AD pathology

Source: Schilling et al. Glutaminyl cyclase inhibition attenuates pyroglutamate Aβ and Alzheimer's disease–like pathology, Nature Medicine 14 – 2008, Nussbaum et al. Prion-like behaviour and tau-dependent cytotoxicity of pyroglutamylated amyloid-β – Nature 485, 2012

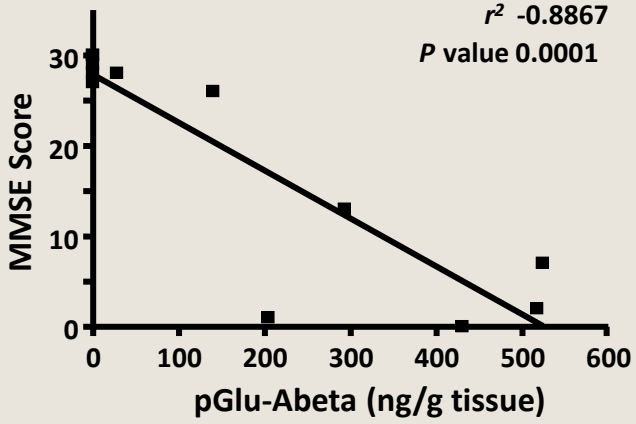
pGlu-Abeta appears in brains of AD patients only

Appearance of pGlu-Abeta in AD brains



Schilling et al., Nat. Med., 2008

pGlu-Abeta level versus MMSE in brain



MORAWSKI M, Schilling S et al; J Alzheimers Dis. 2014, 39, 385-400

Considerations

- Abeta plaques also seen in cognitively normal individuals and in AD
- **pGlu-Abeta (3-42) is characteristic for AD - not found in healthy individuals**
- In brains of AD patients pGlu-Abeta correlates with progression of Alzheimer's disease pathology (based on the Mini-Mental-State Examination "MMSE")

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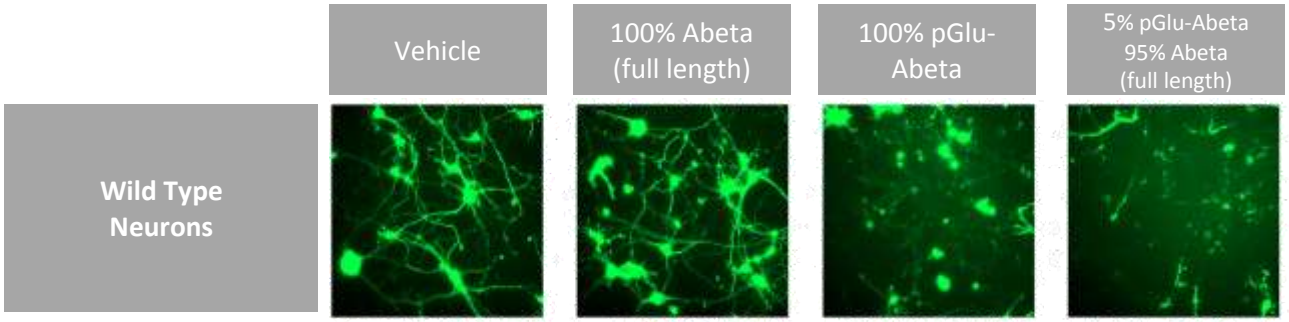
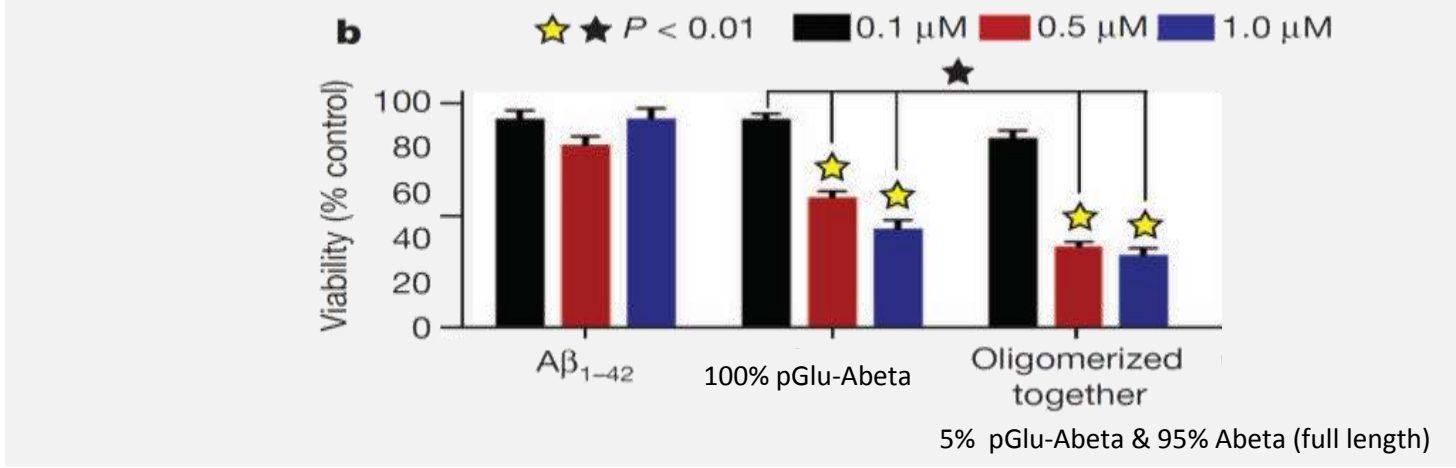
Probiodrug demonstrated toxic role of Abeta oligomers in AD

Toxic role of pGlu-Abeta and oligomers



Cell viability analysis (XTT plate reader assay) on Primary Mouse Neurons after various peptide treatments



- pGlu-Abeta(3-42) exerts strong cell-toxicity
- pGlu-Abeta and Abeta (1-42) form mixed oligomers with enhanced toxicity compared to pure pGlu-Abeta
- Toxic properties may be transferred to other Abeta species based on structural rearrangements in a mechanism of „molecular priming“

Source: Nussbaum et. al , Nature, 2012

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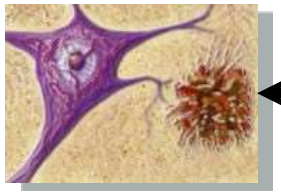
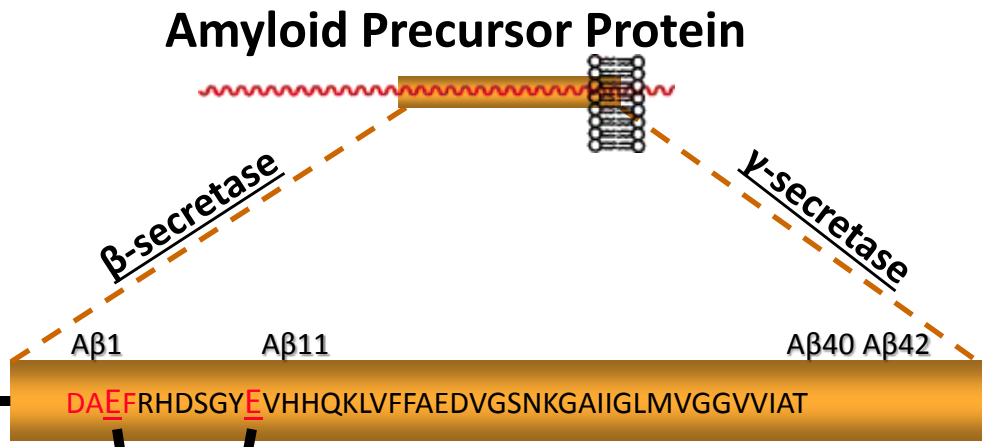
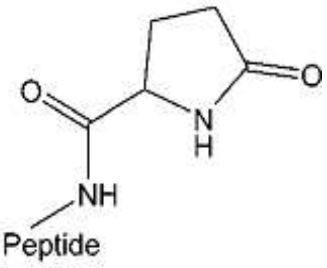
pGluAβ - N-modified Aβ - abundant in AD brain



Human plaques contain

significant amounts of N-truncated and modified Aβ-peptides





Masters, CL et al. *PNAS* 1985
„ragged peptides“

Mori, H *et al. JBC* 1992
25% of Aβ is *p*Glu$_{3/11}$Aβ40/42

Saido, TC et al. *Neurosci Lett* 1996
*p*Glu$_{3/11}$Aβ40/42 major invariantly deposited species

Piccini, A et al *JBC* 2005
*p*Glu$_{3/11}$Aβ dominant in soluble fraction

pGluAβ (N3pE) is abundant in human brain Schilling. Demuth et al. Nature Medicine, 2008

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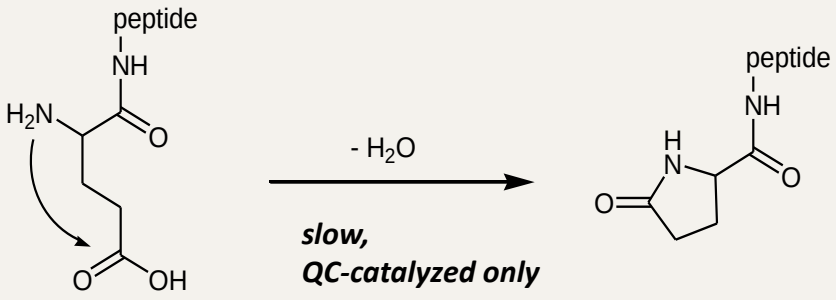
pGlu-Abeta is formed by Glutaminyl Cyclase (QC)

QC cyclases N-terminal Glutamate



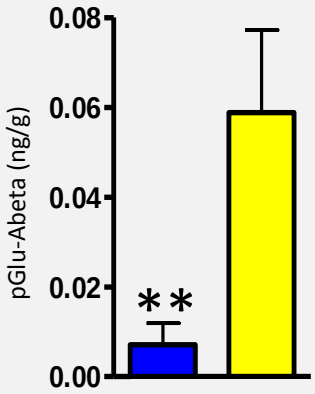
Glutamate (Glu, E) *Pyroglutamate (pGlu, pE)*

- Probiodrug scientists were the first to discover the role of QC in Alzheimer's disease pathology
- QC level as well as pGlu-Abeta level in brain increase with AD progression

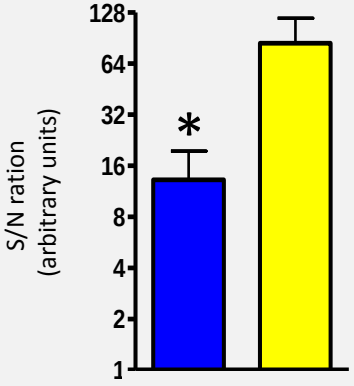
QC overexpression drives to disease development

Elevated QC expression increases pGlu-Abeta → **Increased pGlu-Abeta enhances oligomer formation** → **Abeta oligomers lead to synapse loss in brain** → **Neuronal dysfunction results in memory deficits**

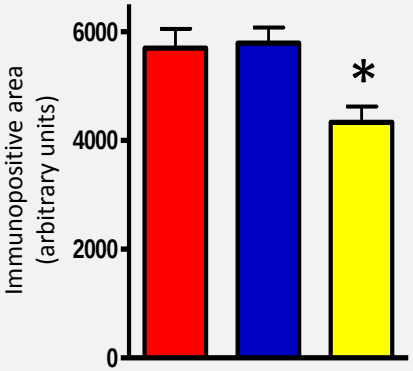
pGlu-Abeta levels in normal AD vs QC-AD model



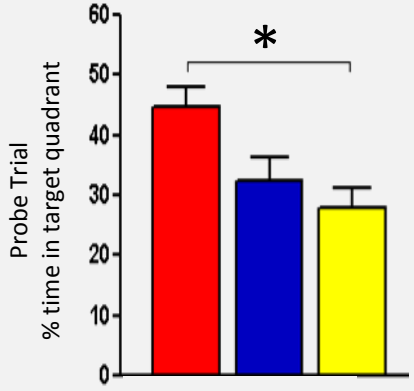
Abeta Oligomers presence in normal AD vs QC-AD model



Synaptophysin levels in wild type, normal AD & QC-AD model



Behavioural Effects in wild type, normal AD & QC-AD model



Based on mouse model

- Wild Type
- APP_{SL} (Alzheimer's model)
- APP_{SL} x hQC (Alzheimer's model plus human QC overexpression)

*-** Statistically significant

- Enhanced QC expression increase pGlu-Abeta and oligomer level
- Enhanced pGlu-Abeta associated with hippocampal synapse loss and impairment of spatial memory

Source: Company data

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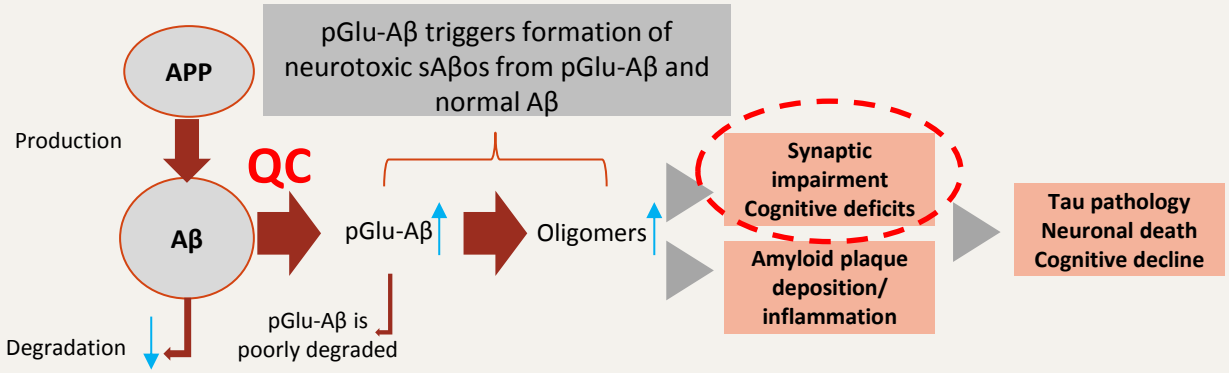
AD is a synaptic failure*

Oligomers impact synapses



Effects on Synapses as the initial target

- Induce (e.g. via NMDR2B receptors)
- Impairs long term potentiation
- Reduces Baseline synaptic transmission
- Decrease spontaneous network activity
- Retraction of synaptic contacts
- Activation of microglia

Subtle impairment e.g. memory

Phase 2 measurements:

- Sensitive domains of NTB
- EEG & fMRI measures synaptic plasticity and neuronal connectivity

QC-I should reverse subtle impairments and show an acute improvement

* Source: D. Selkoe 2002

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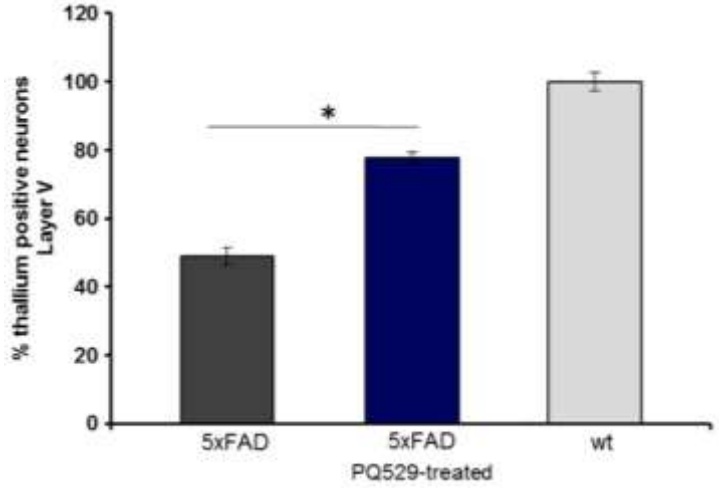
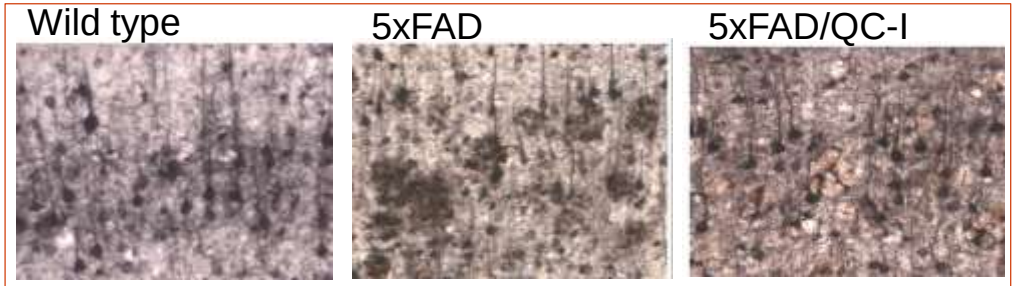
QC-Inhibition improves Network Function

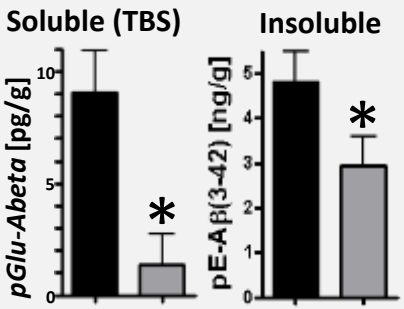
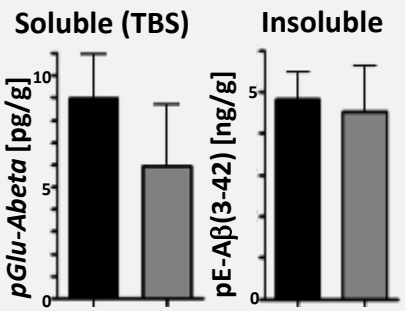
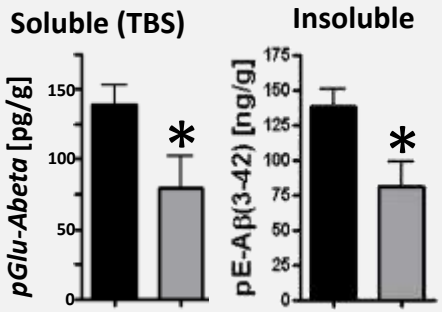




- QC-I improves uptake of Thallium in neurons
- Uptake as measure of Na/K pump activity and thus, neuronal network function

- Electrical activity of pyramidal cells breaks down long before cell death occurs.
  - shown in **a tg mouse model single cell resolution mapping** of neuronal thallium
- This specific disruption of normal cross-laminar cortical processing coincided with a decline of contextual fear learning
- Treatment with **a glutaminyl cyclase inhibitor inhibited the decline of pyramidal cell activity**,
- indicating pyroglutamate-modified forms, potentially mixed oligomers of Aβ, are contributing to neuronal impairment.

PQ912: efficacy in pre-clinical AD model

	Preventive		Therapeutic		
Start+:	3 months		7.5 months		8 months
Duration:	6 months		0.75 - 1.5 months		4 months
					
	✓ Behavioural improvement		✓ Behavioural improvement		• Behaviour not assessed

- Preventive long-term and early therapeutic treatment reduces soluble and insoluble pGlu-Abeta and improves behavior
- Very late therapeutic treatment has no effect

+ Age at the start of treatment; *significant compared to control
Source : Company data

Biogen's Aducanumab (BIIB037) shows strong data targeting aggregated Abeta, ***including soluble oligomers***

Biogen's Aducanumab is a high affinity antibody against specific Abeta targets

- Aducanumab's differs from other antibodies in development for AD, such as crenezumab and gantenerumab, due to high affinity to bind to aggregated forms of beta amyloid, **including soluble oligomers** and insoluble fibrils

Aducanumab phase Ib results

- In March 2015 Biogen published positive interim results from phase 1B study for Aducanumab
- Data from the double-blind placebo controlled study in 166 patients with prodromal or mild Alzheimer's disease showed Aducanumab:
  - Had a dose- and time-dependent reduction of amyloid plaque in the brain over 54 weeks of treatment
  - Significantly slowed cognitive decline on the MMSE as well as a statistically significant clinical decline on the CDR-SB
  - Demonstrated an acceptable safety and tolerability profile

*"This is the **first time an investigational drug for AD has demonstrated a statistically significant reduction** on amyloid plaque as well as a statistically significant … …we are advancing the aducanumab clinical program to phase 3 with plans to initiate enrollment later this year."*

Alfred Sandrock, M.d., Ph.D., group senior vice president and chief medical officer at Biogen

Source: Biogen Media, March 20 2015

Content

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3. Results January – June 2015
4. Outlook
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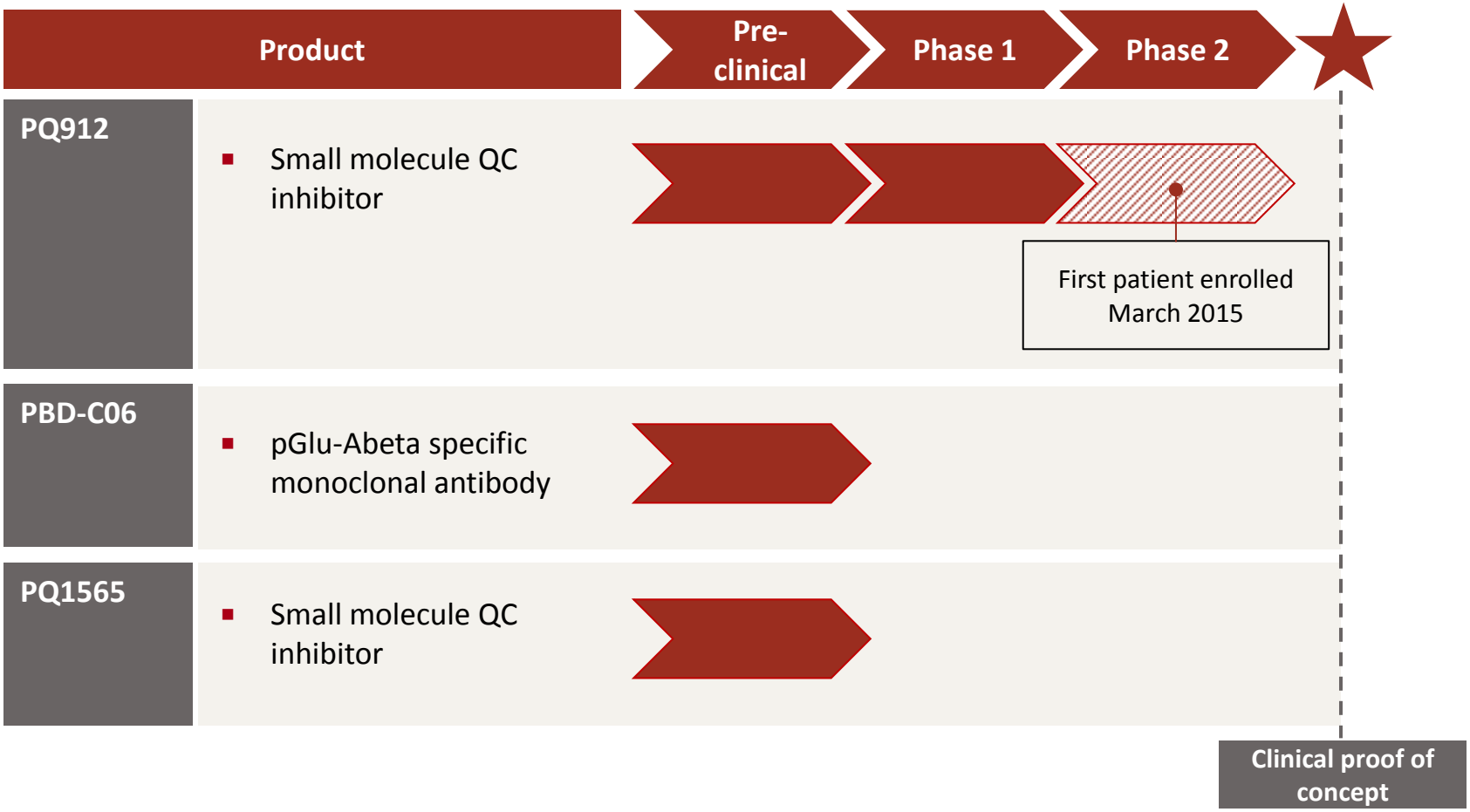
Focused proprietary pipeline



PQ912: the first QC inhibitor against AD in the clinic

Phase 1 results

- PQ912 was tested in a phase 1 clinical study on 200 young and elderly volunteers
- **Safe and well tolerated**, maximum tolerated dose not reached
- Good pharmacokinetic profile resulting in **effective brain concentrations**
  - CSF* half-life 6 hours
  - >90% QC inhibition in CSF at doses to be used in phase 2
  - Twice daily dosing

Ongoing

- 2015 / 2016 phase 2a clinical trial (SAPHIR)
- Considerations
  - Acute cognitive benefits – to be tested in phase 2a trial
  - Chronic disease modification – to be tested in future clinical studies

* Cerebrospinal fluid

Ongoing SAPHIR Phase 2a trial in early AD patients for safety and early efficacy

SAPHIR Phase 2a trial design

- Trial ongoing
- Six European countries
- First patient enrolled March 2015
- 110 patients:
  - Early stage Alzheimer's disease
    - MMSE*: 21-30 inclusive
    - Abeta level in CSF below cut-off 638 ng/L
    - p-tau level in CSF above cut-off >52 ng/L
  - "Treatment naïve": no other Alzheimer drug as co-medication
- 1:1 randomization
- 12 weeks treatment, 4 weeks follow up

Objectives and read-outs

- **Primary objective:** To assess safety and tolerability of PQ912 compared with placebo
- **Exploratory objectives:** set of readouts tailored by Probiodrug to optimize basis for capturing efficacy signals – which will determine further development route
- **Cognitive readouts:** Neuropsychological Test Battery to test short term memory improvements
- **Physiological function assessments:** EEG and rested state functional MRI to measure synaptic plasticity and neuronal connectivity
- **Molecular biomarkers in CSF:** pGlu-Abeta, Abeta pattern, Abeta oligomers and inflammatory markers

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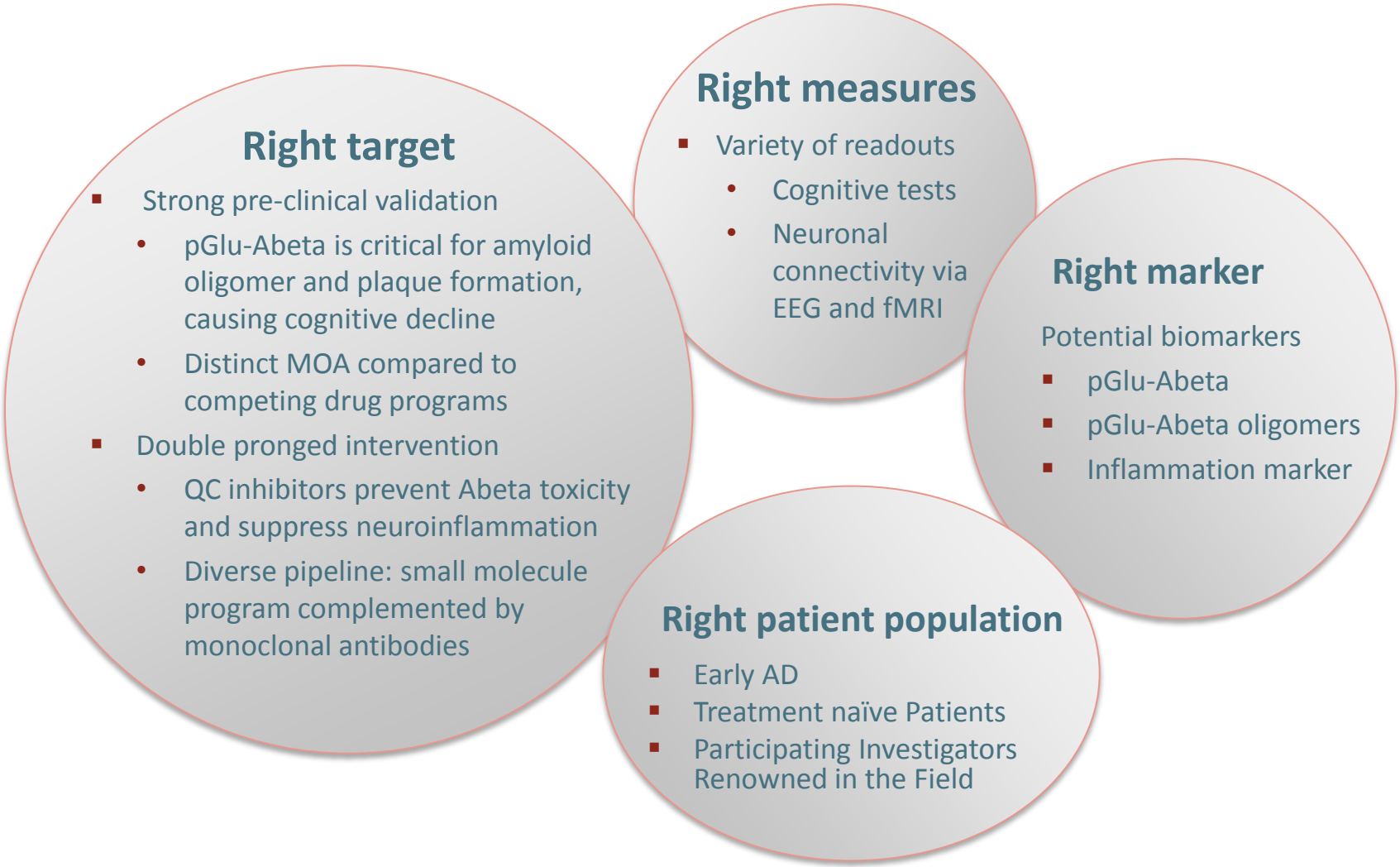
* Mini-Mental State Examination

pGlu-Abeta approach



PBD-C06: a selective antibody targeting pGlu-Abeta

Pre-clinical evidence

- Recognizes pGlu-Abeta with very high selectivity and affinity to multiple forms
- Promising studies performed in Alzheimer's mouse models:
  - Demonstrated the ability to reduce pGlu-Abeta
  - Rescue of short-term memory deficits
  - Showed significant improvement of learning and memory after chronic treatment

Update

- Important next step by selecting a suitable IgG variant as development candidate taken
- Ramp up CMC development activities

PBD-C06 aims to clear existing pGlu-Abeta, leaving the normal non-toxic Abeta untouched

Lilly's pGlu-Abeta antibody data outlines rationale for combination therapy

Background: Eli Lilly has a pGlu-Abeta specific Antibody in Phase 1

- In May 2013, Lilly started a phase 1 study in 100 people on the spectrum of mild cognitive impairment due to AD or mild AD in the United States and Japan
- The data of Lilly's N3pG-Aß Monoclonal Antibody expected to further **validates pGlu-Abeta** as target
- Recent Lilly **poster underlines rationale for combination therapy** with BACE inhibitor

Pre-clinical combination therapy experiment

- Combination of two therapies:
  - Lilly's **pGlu-Abeta specific Antibody and a BACE Inhibitor**, used in Transgenic Mice

Conclusion:

- "Combination therapy targeting multiple steps of the amyloid cascade (both synthesis and clearance) results in dramatic Abeta lowering in PDAPP transgenic mice"
- "These results may have a significant impact on the future of Alzheimer's disease therapies as they support the clinical rationale for using future testing of combination therapy against the a-beta protein in the clinical practice"

Source: 2014 Alzheimer's Association International Conference Presentation number: O1-10-03 R DeMattos et al.; Eli Lilly and Company Annual Report 2014

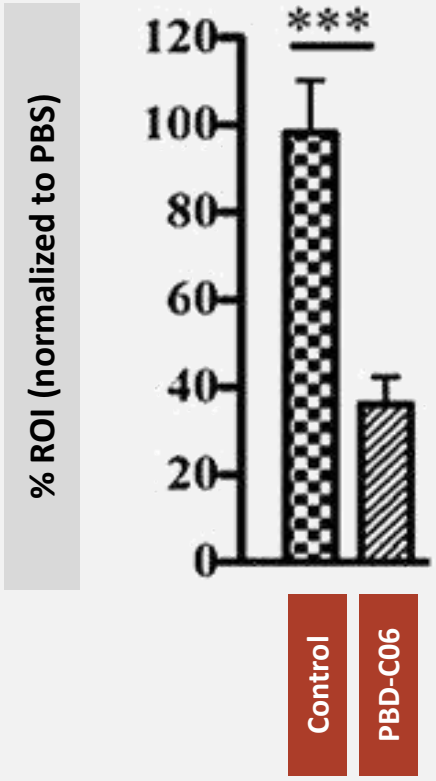
PBD-C06: “Capture and Clear”

PBD-C06 reduces pGlu-Abeta

Control

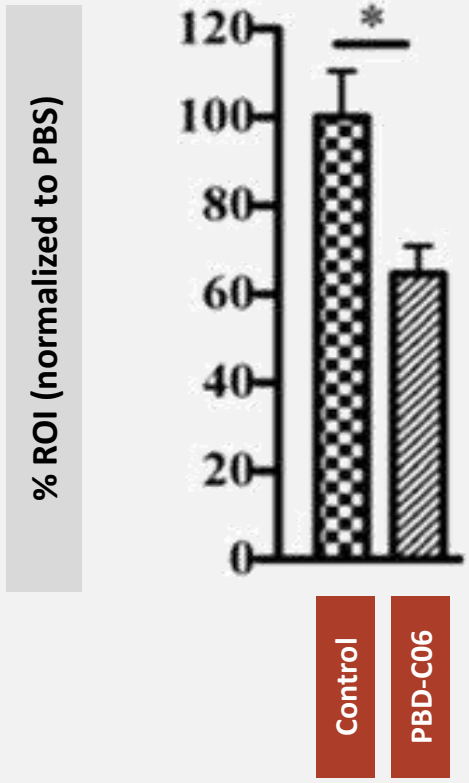
Post PBD-C06 treatment

Hippocampal brain section of AD-like mouse model, stained for pGlu-Abeta



pGlu-Abeta is reduced by 60% post PBD-C06 treatment

PBD-C06 reduces total Abeta



Total Abeta is reduced by 40% post PBD-C06 treatment

Source: Company data presented AD/PD 2013 – The 11th International Conference on Alzheimer’s and Parkinson’s Disease March 6, 2013 – March 10, 2013

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Key Highlights January to June 2015

- Phase 2a study of novel treatment for Alzheimer's disease, the SAPHIR trial, initiated
- Additional data on Glutaminyl Cyclases (QCs) in Alzheimer's disease published in *Acta Neuropathologica*
- Data on Probiodrug`s Anti-pGlu-3 Abeta monoclonal antibody presented at the 12th AD/PD™ 2015, Nice
- Key patents on Glutaminyl Cyclase (QC) inhibition for the treatment of AD granted in Japan
- Winner of the European Mediscience Award 2015 for Best Technology
- Funding of Alzheimer research at Brigham and Women's Hospital, affiliated with Harvard Medical School
- Cash and cash equivalents of EUR 14.8 million as of 30 June, 2015
- Net loss of EUR 6.2 million for the first six-months period compared with EUR 3.4 million in 2014 - in line with company expectations
- Annual General Meeting held in June, all resolutions proposed by Management and Supervisory Board approved
- New members of the Supervisory Board with distinguished industry expertise appointed

Annual General Meeting 2015 (AGM 2015)

On 10 June 2015, Probiodrug held its AGM 2015. All resolutions proposed were approved at the meeting including:

- Adoption of the annual financial statements and the management report of Probiodrug AG for the financial year 2014
- Ratification of the actions of the Executive and Supervisory Board members for financial year 2014
- Appointment of the statutory auditor for the annual financial statements for the financial year 2015
- Appointment of Ms Charlotte Lohmann and Mr Kees Been as new members of the Supervisory Board
- Re-election of Dr Erich Platzer, Dr Dinnies von der Osten, Dr Jörg Neermann and Dr Olivier Litzka as member of the Supervisory Board
- Remuneration of the Supervisory Board
- Authorization to acquire treasury shares
- Authorization to issue option bonds and/or convertible bonds
- Resolution on the adjustment of the Stock Option Program 2014

Key financial figures January – June 2015 (according to IFRS)

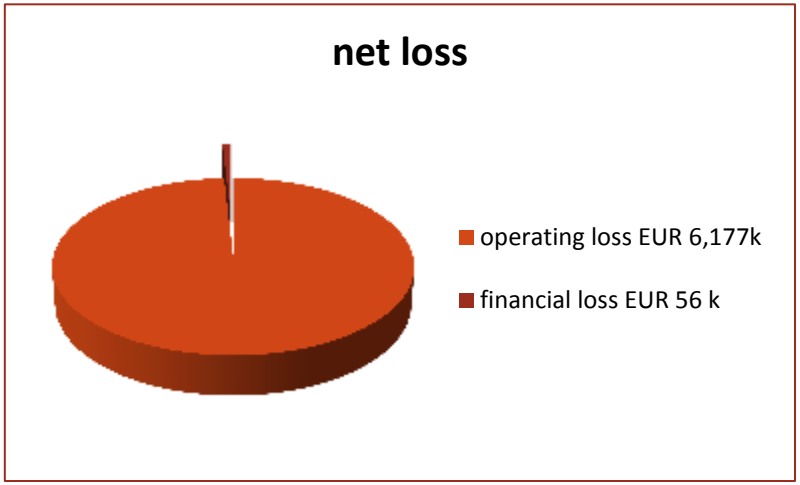
In EUR k	Jan – Jun 2015	Jan – Jun 2014 (unconsolidated*)	Jan – Dec 2014 (unconsolidated*)
Earnings, Financial and Net Assets Position			
Revenues	0	0	0
Operating profit/loss	-6,177	-3,738	-11,267
Net loss for the period	-6,233	-3,761	-11,437
Equity (end of the reporting period)	10,160	n.a.**	15,971
Equity ratio (end of the reporting period) (in %)	66.0 %	0	74.4 %
Balance sheet total (end of the reporting period)	15,383	n.a.**	21,480
Cash flows from operating activities (reporting period)	-6,119	-3,522	-10,589
Cash flows from operating activities (monthly average)	-1,020	-587	-882
Cash flows from financing activities (net)	0	4,276	25,762
Cash and cash equivalents at the end of period	14,793	5,200	20,920
Personnel			
Total number of employees (incl. Board of management) (end of the reporting period)	16	12	13
Probiodrug-Share			
Earnings per share (basic/diluted) (in EUR)	-0.92	-0.88	-2.35
Number of shares issued (end of the reporting period))	6,766	25,529	6,766

* While the semiannual financial statements 2014 where prepared on a consolidated basis, the semiannual financial statements 2015 were prepared on an unconsolidated basis, since the subsidiary Ingenium was sold in July 2014. For comparison reasons the 2014 semiannual financials are also shown in an unconsolidated manner, leaving out Ingenium.
** Acc. to IFRS not applicable

Details of the Financial Results January – June 2015 (according to IFRS)

Net loss

- Net loss in line with expectations
- Operating loss primarily driven by R&D expenses, in particular the SAPHIR study
- Increase in operating expenses reflects primarily administrative costs and post-listing requirements

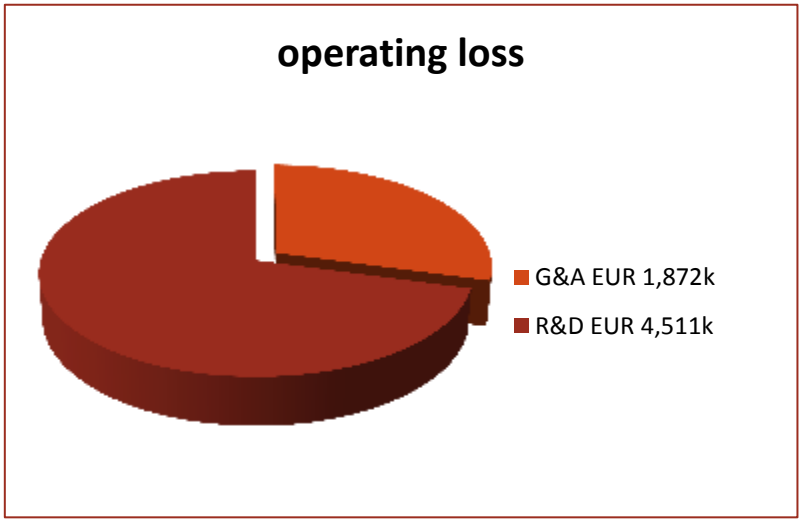


Equity

- The equity amounts to EUR 10,160k (Year 2014: EUR 15,971k), corresponding to an equity ratio of 66.0% (Year 2014: 74.4%).

Cash

- Cash and cash equivalents were EUR 14,793k, compared with EUR 20,920k as at 31 December 2014.



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Outlook 2015

The mid-term focus of Probiodrug's business activities can be summarised as follows:

- Further preclinical and clinical testing of the development candidate PQ912, in particular execution of the first patient study in a Phase 2a trial in 2015/ 2016 and the evaluation and design of a longer term treatment either as an extension of the SAPHIR study - or a separate study.
- Securing further supporting data and intellectual property protection for the therapeutic concept of QC inhibition as a novel approach for the treatment of Alzheimer's disease and other diseases.
- Further progression of the anti pGlu-Abeta specific anti-body (PBD-CO6) as well as of PQ1565, an additional small molecule QC inhibitor.
- Progressing preclinical studies to evaluate the potential of Probiodrug's medical candidates in combinations and in other indications.

Guidance

- Probiodrug estimates the net loss for the financial year 2015 to be comparable to the net loss of 2014

Anticipated news flow (selection)

2014

- PQ912 CTA submission for phase 2 ✔
- **PQ912 First patient enrolled** ✔

2015

- **PQ912 Publication of complete phase 1 results**
- PBD-C06 Results in inflammatory part of AD pathology in animal model
- PQ912 Assessment of potential in Down syndrome
- **PBD-C06 start of development activities to prepare for phase 1**

2016

- **PQ912 phase 2a results**
- PQ912 POP* combination therapy with BACE Inhibitor; and with PBD-C06

* Pre-clinical proof of Principle

Please note: timing of news flow is indicative

Financial Calendar

May 13th, 2015	Interim Management Statement Q1 2015
June 10th, 2015	Annual General Meeting of Shareholders in Berlin
August 27th, 2015	Interim Report, half year results 2015
November 19th, 2015	Interim Management Statement Q3 2015

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Q & A