UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 6-K

Report of Foreign Private Issuer
Pursuant to Rule 13a-16 or 15d-16 of
the Securities Exchange Act of 1934

October 28, 2016

PROQR THERAPEUTICS N.V.

Zernikedreef 9
2333 CK Leiden
The Netherlands
Tel: +31 88 166 7000
(Address, Including ZIP Code, and Telephone Number,
Including Area Code, of Registrant's Principal Executive Offices)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.

Form 20-F ☒ Form 40-F ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1): ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7): ☐

Other Events

On October 27, 2016, ProQR Therapeutics N.V. (the "Company") held an investor presentation, which was accompanied by a slide presentation. A copy of the slide presentation is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

The information in Exhibit 99.1 (which is furnished only) of this Report of Foreign Private Issuer on Form 6-K shall not be deemed to be "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, and shall not be incorporated by reference into any registration statement or other document filed under the Securities Act of 1933, as amended, or the Exchange Act, except as shall be expressly set forth by specific reference in such filing.

EXHIBITS

Exhibit Number	Description
99.1	Slide Presentation, dated October 27, 2016.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

PROQR THERAPEUTICS N.V.

Date: October 28, 2016

By: /s/ Smital Shah
Smital Shah
Chief Financial Officer

Exhibit 99.1


ProQR
ANALYST & INVESTOR EVENT
NACFC 2016, Orlando, Florida
Date:
October 27th, 2016
Presenters:
Daniel de Boer, Noreen Henig and JP Clancy

Agenda

Overview and introduction
by Daniel de Boer

The relevance of the Nasal Potential Difference test in CF
by JP Clancy, M.D.

Results of the QR-010 NPD study
by Noreen Henig, M.D.

Pipeline and path ahead
by Daniel de Boer

Q&A session
with JP Clancy, M.D., Noreen Henig, M.D., Smital Shah and Daniel de Boer

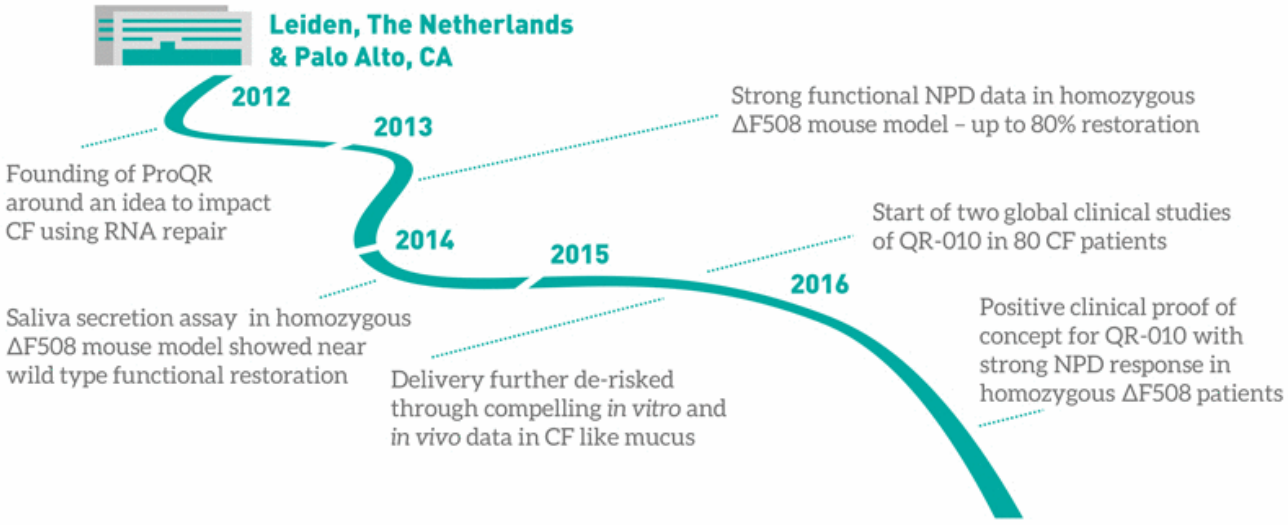
Forward looking statements

This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation, including but not limited to, statements regarding our strategy, future operations, future pre-clinical and clinical trial plans and related timing of trials and results, research and development, future financial position, future revenues, projected costs, prospects, therapeutic potential of our products, plans and objectives of management, are forward-looking statements. The words "aim," "anticipate," "believe," "estimate," "expect," "intend," "may," "plan," "predict," "project," "target," "potential," "will," "would," "could," "should," "continue," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words.

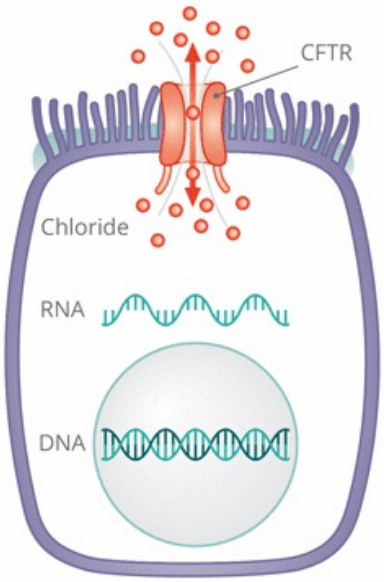
Forward-looking statements represent our management's beliefs and assumptions only as of the date of this presentation. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make. The forward-looking statements contained in this presentation reflect our current views with respect to future events, and we assume no obligation to update any forward-looking statements except as required by applicable law. These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including those that may be described in greater detail in the annual report filed on Form 20-F for the year ended December 31, 2015 that we have filed with the U.S. Securities and Exchange Commission (the "SEC") and any subsequent filings we have made with the SEC. We have included important factors in the cautionary statements included in that annual report, particularly in the Risk Factors section, and subsequent filings with the SEC that we believe could cause actual results or events to differ materially from the forward-looking statements that we make.

ProQR's CF journey

From an idea to clinical proof of concept in 4 years



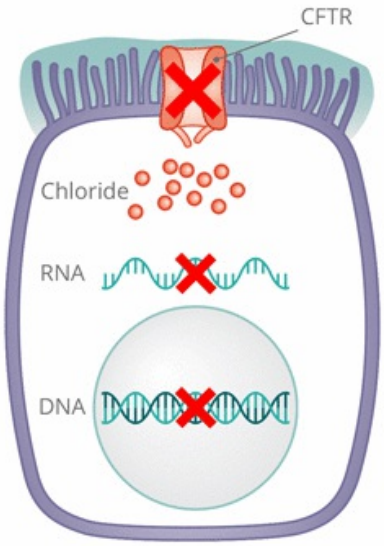
CFTR is hydrating mucus in normal lung cell



CFTR in normal lung cell:

- In healthy people CFTR protein is formed
- CFTR protein acts as a chloride channel
- Due to chloride transport the extracellular mucus is hydrated

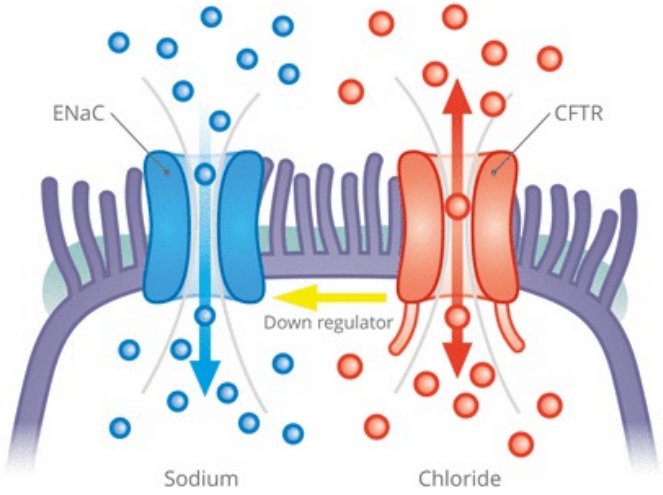
Absence of CFTR is leading to dehydration of mucus



CFTR in CF lung cell:

- In CF patients no functional CFTR protein is formed
- In absence of CFTR chloride can not flow out of the cell
- Due to the lack of chloride transport the extracellular mucus dehydrates

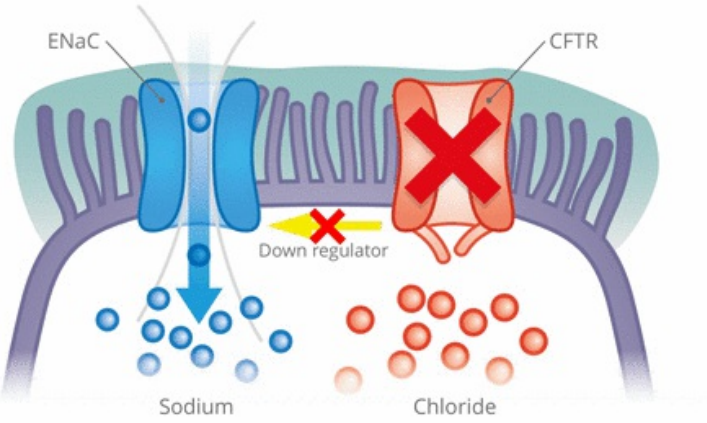
CFTR and ENaC channels in normal lung cell



In healthy people:

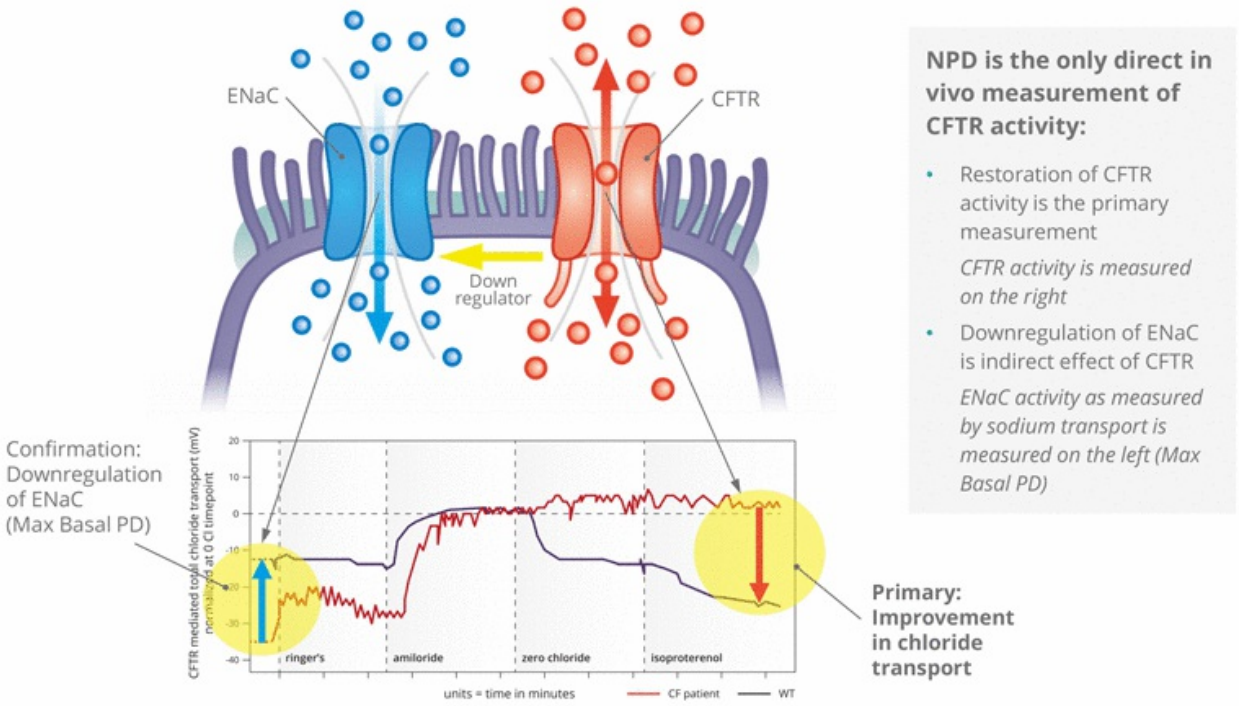
- CFTR and ENaC co-operate to regulate Chloride and Sodium balance
- CFTR is a down regulator of ENaC channel activity

CFTR and ENaC channels in CF lung cell



In CF patients:

- In absence of CFTR protein ENaC is unregulated and thus hyperactive
- This contributes to the CF phenotype



NPD is the only direct in vivo measurement of CFTR activity:

- Restoration of CFTR activity is the primary measurement

  CFTR activity is measured on the right

- Downregulation of ENaC is indirect effect of CFTR

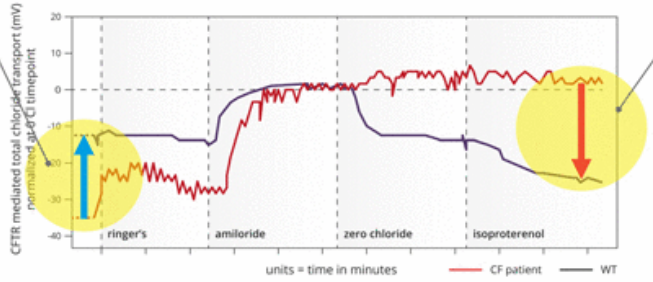
  ENaC activity as measured by sodium transport is measured on the left (Max Basal PD)

CFTR restoration confirmed by ENaC normalization

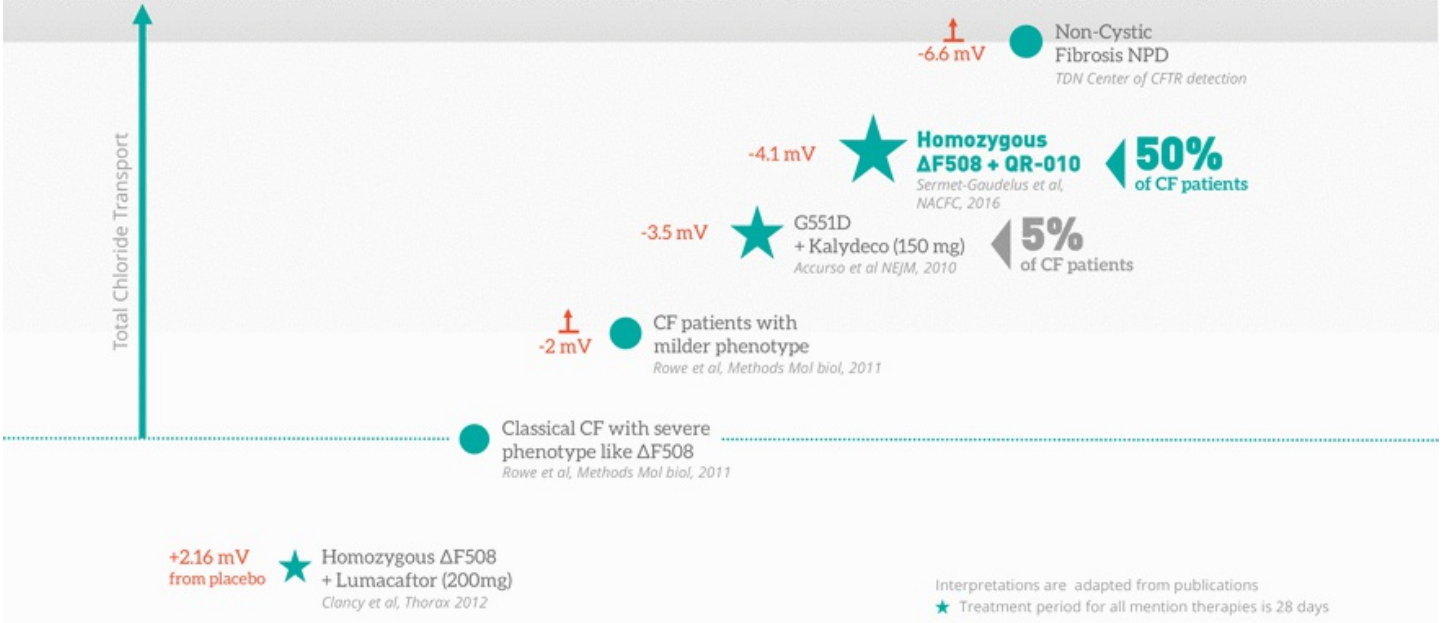
Basal PD (ENaC)
Change from baseline
9.2mV
P=0.0371

CFTR activity is confirmed by normalization of ENaC activity

Total Chloride Transport (CFTR)
change from baseline
-4.1mV
P=0.0389

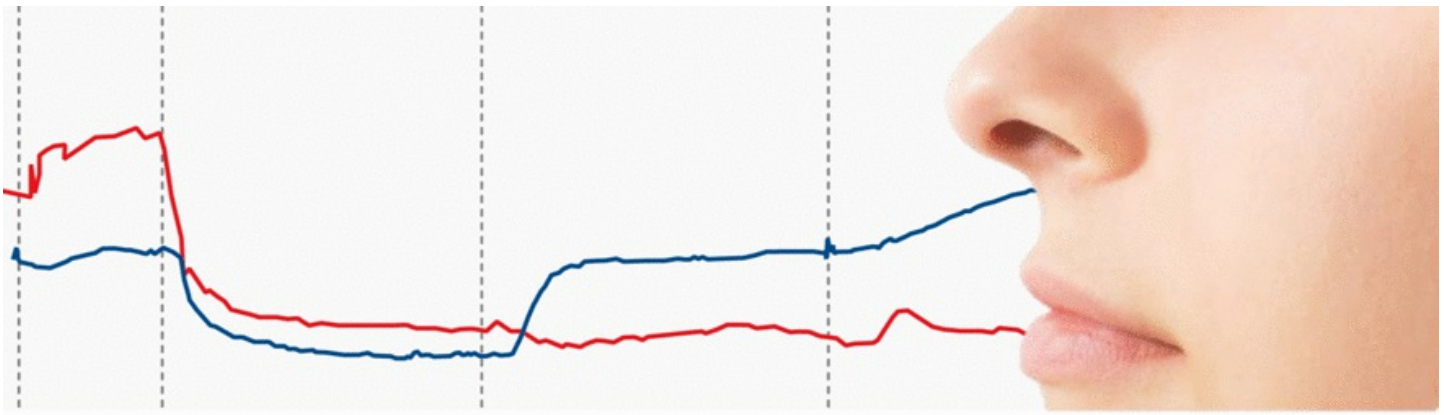


Putting QR-010 NPD results in perspective



+2.16 mV from placebo ★ Homozygous ΔF508 + Lumacaftor (200mg)
Clancy et al, Thorax 2012

Interpretations are adapted from publications
★ Treatment period for all mention therapies is 28 days

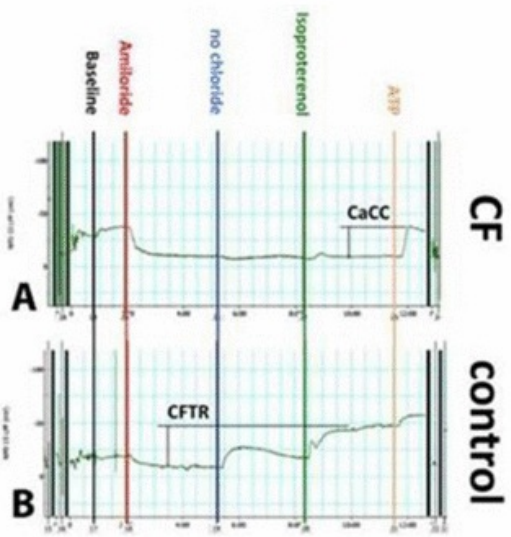


The relevance of the Nasal Potential Difference test in CF

By JP Clancy, MD

Professor of Pediatrics, Research Director Pulmonary Medicine
Cincinnati Children's Hospital

NPD is the only direct measurement of CFTR function



- NPD is only direct measurement of both sodium and chloride channel function
- CFTR downregulation of ENaC is well understood
- A response on both chloride transport and change in basal PD provides validation of a functioning CFTR
- Nasal epithelium well represents (in histology and ion transport) the lung epithelium

NPD methods and interpretation is standardized

Published in final edited form as:
Methods Mol Biol. 2011 ; 741: 69–86. doi:10.1007/978-1-61779-117-8_6.

Nasal Potential Difference Measurements to Assess CFTR Ion Channel Activity

Steven M. Rowe,
Departments of Medicine, Pediatrics, and Physiology and Biophysics MCLM, University of Alabama, 35294-0006, Birmingham, AL, USA

Jean-Paul Clancy, and
Departments of Medicine, Pediatrics, and Physiology and Biophysics MCLM, University of Alabama, 35294-0006, Birmingham, AL, USA

Michael Wilschanski
Respiratory Medicine and Cystic Fibrosis Center, Shaare Zedek Medical Center, 91031, Jerusalem, Israel

Steven M. Rowe: smrowe@uab.edu; Jean-Paul Clancy: john.clancy@cchmc.org

OPEN ACCESS Freely available online

PLOS ONE

Optimizing Nasal Potential Difference Analysis for CFTR Modulator Development: Assessment of Ivacaftor in CF Subjects with the *G551D-CFTR* Mutation

Steven M. Rowe[1], Bo Liu[1], Aubrey Hill[1], Heather Hathorne[1], Morty Cohen[2,a], John R. Beamer[2,b], Frank J. Accurso[3], Qunming Dong[4], Claudia L. Ordoñez[4,c], Anne J. Stone[4], Eric R. Olson[4], John P. Clancy[5,*], for the VX06-770-101 Study Group[*]

Relevance of NPD for the lower airway

Detection of Cystic Fibrosis Transmembrane Conductance Regulator Activity in Early-Phase Clinical Trials

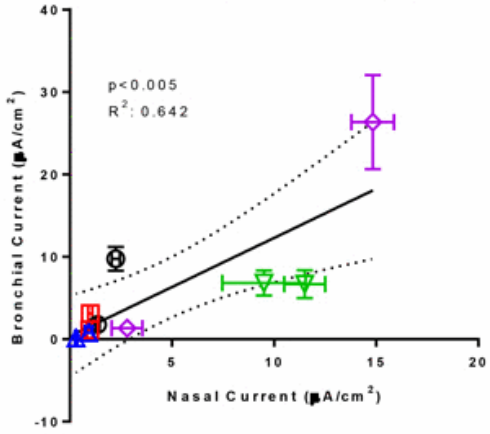
Steven M. Rowe[1,2,3,4], Frank Accurso[5], and John P. Clancy[3,4]

[1]Department of Medicine, [2]Department of Physiology and Biophysics, [3]Department of Pediatrics, and [4]Cystic Fibrosis Research Center, University of Alabama at Birmingham, Birmingham, Alabama; and [5]Department of Pediatrics, University of Colorado, Denver, Colorado

"The nasal epithelium is a faithful representation of the histologic and ion transport features of the pulmonary epithelium, supporting its use as a biomarker for the lower airway."

Rowe, Accurso, Clancy. PATS 2007

CFTR-dependent I_{SC}



Good correlation between bronchial current and Nasal current

155

CHARACTERIZATION OF BRUSHED HUMAN UPPER AND LOWER AECS TO DETECT AND QUANTIFY CFTR FUNCTION

Filbrandt, E.; Ostmann, A.J.; Brewington, J.; Strecker, L.; Clancy, J.P. *Pulmonary Medicine, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA*

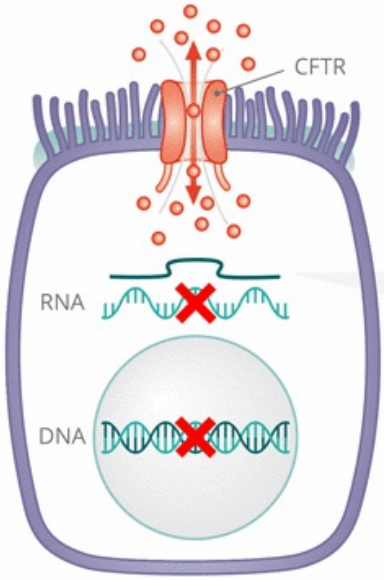
Good correlation in key measures

Cincinnati Children's
changing the outcome together

Results QR-010
Nasal Potential Difference study

By Noreen R. Henig, MD

QR-010 for ΔF508 cystic fibrosis



QR-010

- Single stranded 33-mer RNA oligonucleotide
- P=S and 2'Ome chemically modified for stability and uptake
- Designed to target ΔF508 mutation
- Formulated in saline solution
- Inhaled delivery for efficient lung delivery and systemic uptake
- Delivered by PARI eflow Nebulizer

Pre-clinical data supports QR-010 can restore CFTR function

GLP Tox

28 days in mice

No DLT up to high dose (30mg/kg) for 28 days in monkeys

Inhaled Administration to the Lung

In vitro CF mucus penetration

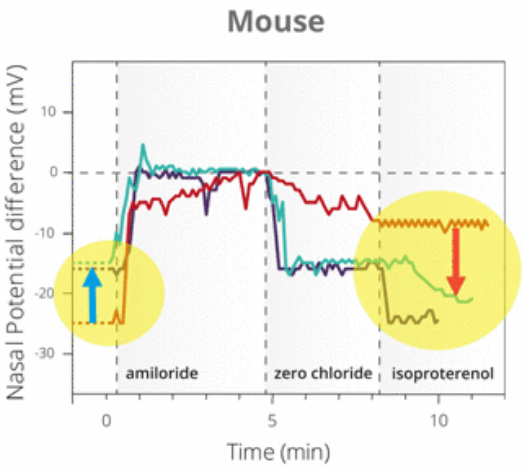
Similar biodistribution between wild-type and mice with CF lung phenotype

Functional Restoration of CFTR Response

Two in vitro models:
- MQAE
- Ussing Chamber

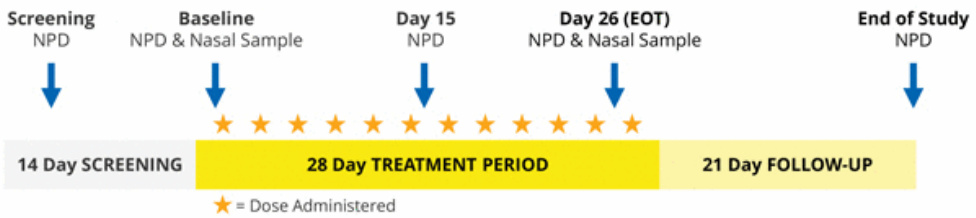
Up to 80% restoration of wild-type CFTR response in two independent ΔF508 mouse assays:
- Saliva Secretion assay
- Nasal Potential Difference assay

Mode of action research ongoing



QR-010

PQ-010-002 Proof of Concept study



Design

- 8 ΔF508 homozygous and 8 compound heterozygous patients ΔF508 CF patients >18yr
- Multiple dose design: 12 doses (3 per week x 4 weeks)
- Intranasal administration
- 5 NPD reference sites in EU (CTN) and North America (TDN)

Endpoints

- CFTR-mediated total chloride transport (primary)
- Sodium transport measured by Basal PD
- Safety, SNOT-22 and NERS assessment
- Sweat chloride test

NPD centers of excellence

- Steve Rowe, MD – Central, blinded reader
- Stuart Elborn, MD – Chairman, Adaptive Design Review Committee
- Marcus Mall, MD – ADRC member

- Marty Solomon, MD – University of Alabama
- David Nichols, MD and Jerry Nick, MD – National Jewish Medical & Research Center
- JP Clancy, MD – University of Cincinnati
- Isabelle Sermet, MD – INSERM U 1151, Hôpital Necker –Enfants Malade
- Christiane de Boeck, MD – KUL

- Standard Operating Procedures of CFF-TDN and ECFS-CTN

NPD: Maximize potential as a useful endpoint

Design

- Controls
- Inclusion criteria
- Well-defined analysis and methods
- Endpoints (chloride response and confirmatory basal PD)

Execution

- Centers of excellence
- Standardized methods (SOP)
- Central supplies
- Minimizing operator variability

Independent analysis

- Blinded central independent NPD reader
- Validation by independent data review committee

Demographics

	Homozygous Cohort (Cohort 1)	Heterozygous Cohort (Cohort 2)
Characteristic	Safety Population (N=10)	Safety Population (N=8)
Age (years)		
Mean (SD)	25.80 (6.7)	36.0 (15.8)
Min, Max	19, 36	18, 63
Sex, n (%)		
Male	6 (60%)	4 (50.0%)
Female	4 (40%)	4 (50.0%)
Race, n (%) Caucasian	10 (100%)	8 (100.0%)
BMI (kg/m^2)		
Mean (SD)	22.8 (2.8)	23.1 (3.3)
Min, Max	19.8, 28.2	19.8, 28.4
Predicted FEV1 (%)		
Mean (SD)	74.2 (17.4)	74.9 (16.9)
Min, Max	45.2, 108.8	52.3, 98.1
Sweat Chloride (mmol/L)		
Mean (SD)	98.7 (15.0)	103.9 (18.0)
Min, Max	78.0, 117.5	86.0, 134,0
Baseline CFTR-Mediated Total Chloride Transport (mV)		
Mean (SD)	-1.2 (5.8)	-2.4 (5.9)
Min, Max	-11.1, 6.4	-13.9, 6.3
Baseline SNOT-22 Total Score		
Mean (SD)	14.9 (5.9)	19.1 (17.7)
Min, Max	8.0, 24.0	5.0, 59.0

Key takeaways:

- Adult subjects with classic ΔF508 phenotype

Preliminary safety & tolerability data

Pooled Cohorts

Treatment-Emergent Adverse Events Occurring in >10% Subjects by Preferred Term	Safety Population (Pooled Cohorts); N=18 N (%)
Subjects with Serious Adverse Events	0 (0)
Subjects with at least one TEAE	15 (83.3)
Gastrointestinal disorders	
Nausea	3 (16.7)
General disorders and administration site conditions	
Fatigue	4 (22.2)
Pyrexia	4 (22.2)
Nervous System Disorders	
Headache	2 (11.1)
Respiratory, thoracic and mediastinal disorders	
Cough	4 (22.2)
Epistaxis	2 (11.1)
Respiratory Tract Congestion	2 (11.1)
Rhinorrhoea	3 (16.7)
Sinus Congestion	2 (11.1)
Nasal Congestion	2 (11.1)

Participation:

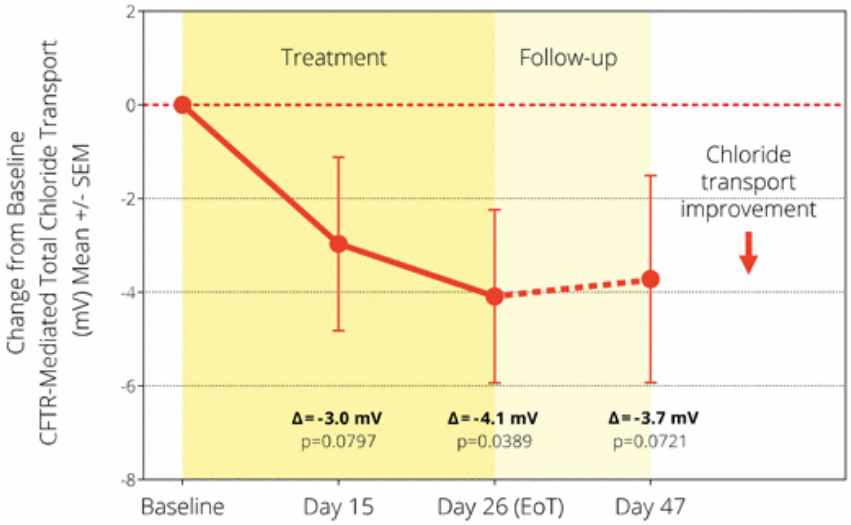
- No discontinuations
- 17 of 18 patients received all 12 doses
- 1 patient received 11 doses

Key takeaways:

- No SAEs observed in treatment and follow up periods
- AE profile consistent with what is expected in CF population

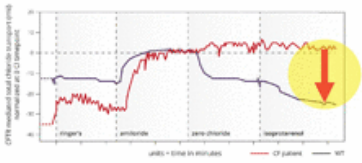
QR-010 meets primary endpoint in homozygous patients

Measured by CFTR mediated total chloride transport

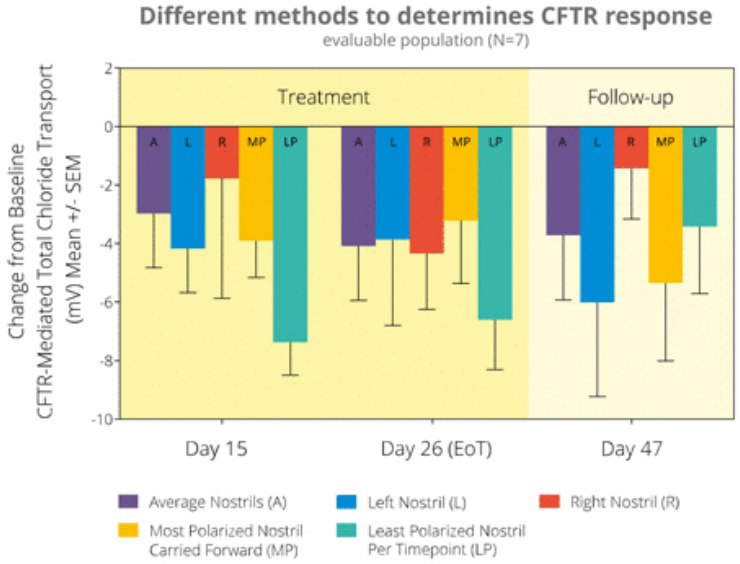


Key takeaways:

- Strong response in CFTR mediated chloride transport
- Statistically significant response per-protocol subjects
- Durable response 21 days post treatment

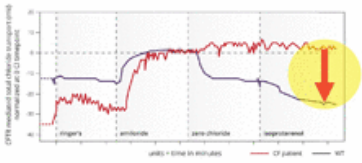


All methods show CFTR response in homozygous patients



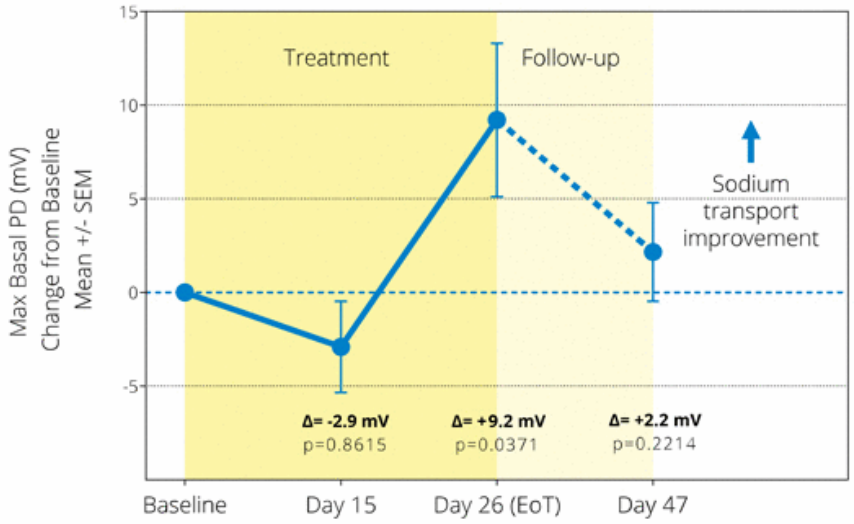
Key takeaways:

- Irrespective of the chosen method of analysis an improvement is observed



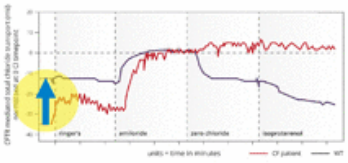
Basal PD change confirms CFTR activity

Sodium transport measured by Basal PD

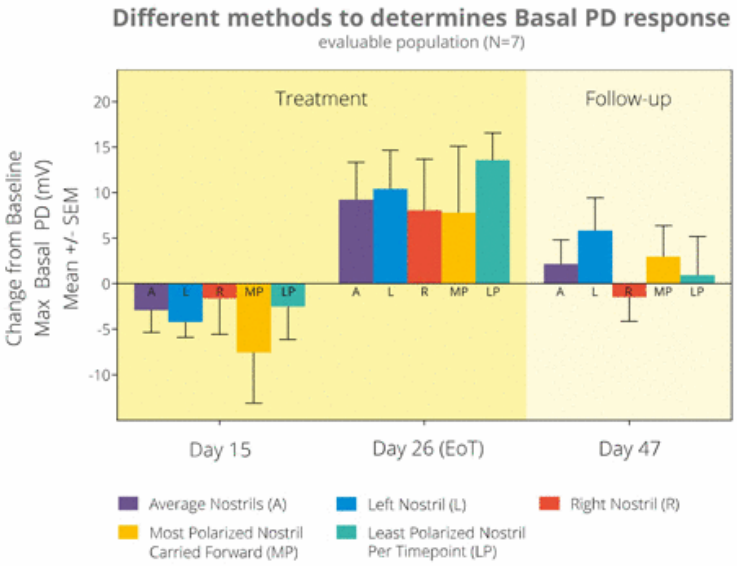


Key takeaways:

- Max Basal PD is direct measurement of ENaC activity as measured by sodium transport
- Basal PD confirms functional data for CFTR activity



All methods show Basal PD response



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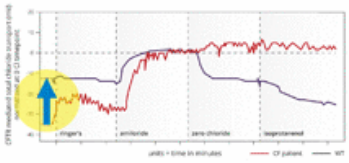
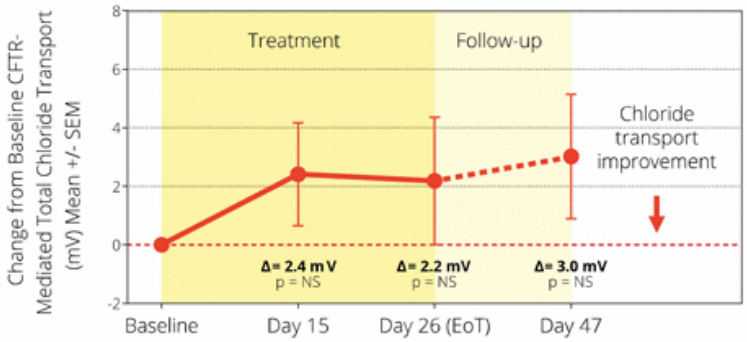


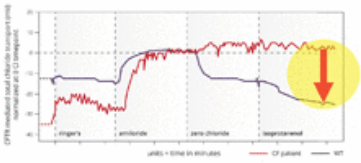
Heterozygous CFTR response



Key takeaways:

- Further pre-clinical research necessary before studying more patients
- Impact of second allele needs to be investigated
  - 8 different second mutations
  - Responder analysis is ongoing

Functional Class	Subject numbers	Mutation	Legacy Nomenclature
I	101202	p.Gln493 [stop]	Q493X
	101204	c.489+1 G>T [splicing]	621+1 G>A
	203203	p.Tyr1092 [stop]	Y1092X
	701203	c.1585-1 G>A [splicing]	1717-1 G>A
II	102202	p.Asn1303Lys	N1303K
	103205	p.Ile336Lys	I336K
	701204	p.Gly628Arg	G628R
V	103208	c.2657+5 G>A [splicing]	2789+5 G>A



PQ-010-002: NPD proof of concept study

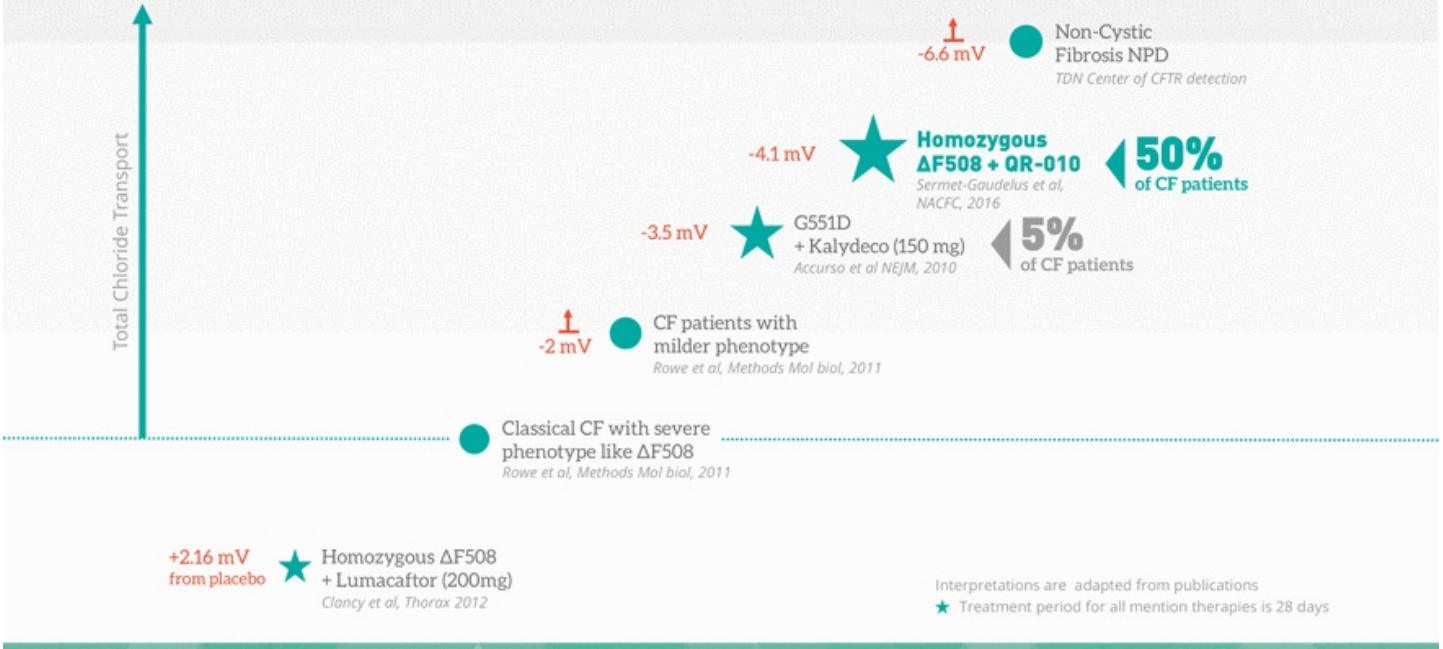
Conclusions

- QR-010 improves CFTR function in subjects with CF due to ΔF508 mutation

Timepoint	CFTR –mediated Total Chloride Response [Mean ± SEM, mV]	p-value
Day 15	- 3.0 ± 1.9	0.0797
Day 26 (EOT)	**- 4.1 ± 1.9**	**0.0389**
Day 47	- 3.7 ± 2.2	0.0721

- A trend for durability of effect was observed 21 days after last dose (day 47)
- QR-010 improvement in CFTR function is also supported a positive sodium transport signal (max basal PD)
- QR-010 changes in nasal potential is comparable to data published for ivacaftor (G551D) and superior to data published on lumacaftor alone (ΔF508)
- First clinical data de-risks development of QR-010 by confirming pre-clinical findings

Putting QR-010 NPD results in perspective



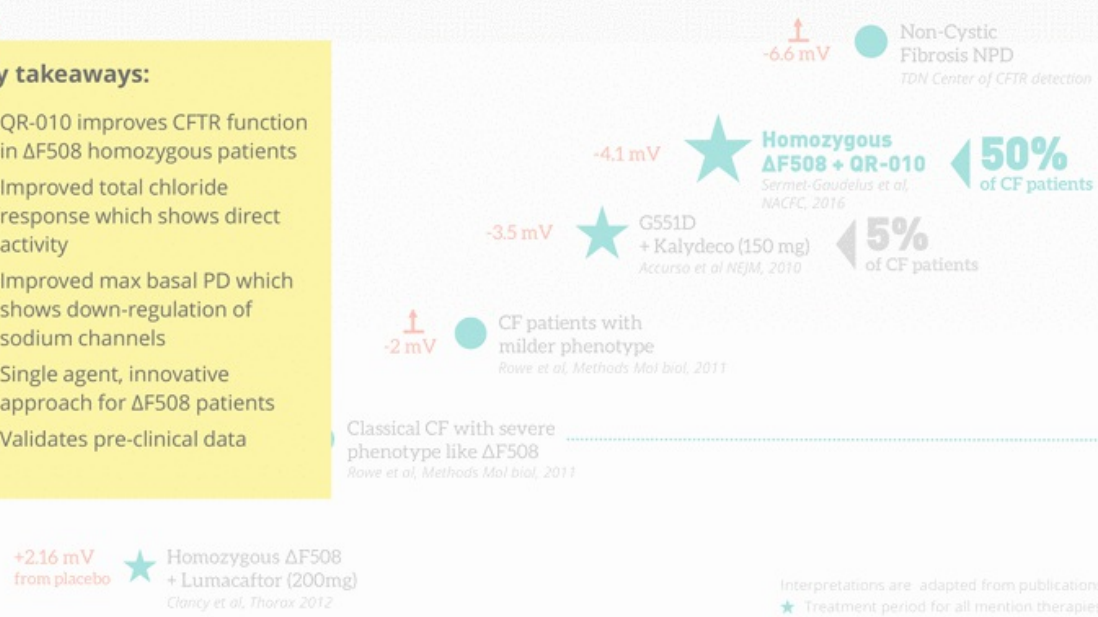
+2.16 mV from placebo ★ Homozygous ΔF508 + Lumacaftor (200mg)
Clancy et al, Thorax 2012

Interpretations are adapted from publications
★ Treatment period for all mention therapies is 28 days

Putting QR-010 NPD results in perspective

Key takeaways:

- QR-010 improves CFTR function in ΔF508 homozygous patients
- Improved total chloride response which shows direct activity
- Improved max basal PD which shows down-regulation of sodium channels
- Single agent, innovative approach for ΔF508 patients
- Validates pre-clinical data



Interpretations are adapted from publications

★ Treatment period for all mention therapies is 28 days

Translating NPD to the lung

Stability

QR-010 is stable in presence of CF lung bacteria
Brinks et al. ECFS, 2016

QR-010 is stable in CF mucus and doesn't degrade
Brinks et al. NACFC, 2015

QR-010 is stable in presence of inhaled CF co-medications
Brinks et al. NACFC, 2015

Uptake

Beta-ENaC mouse with CF lung phenotype shows uptake and bio distribution similar to WT
Brinks et al NACFC, 2014

Significant uptake in lung epithelial cells and plasma in WT rodents and monkeys
Unpublished ProQR Data

QR-010 is detected in blood after intranasal administration
in QR-010 NPD study

Diffusion

QR-010 penetrates pseudomonas biofilm in vitro
Unpublished ProQR Data

Mucus repels QR-010 due to negative charge
Perez- Vilar, JBC, 1999

CF mucus contains DNA, QR-010 has low binding affinity to DNA
Unpublished ProQR Data

QR-010 penetrates rapidly through CF-like mucus in vitro and CF mucus ex vivo
Brinks et al. NACFC, 2015

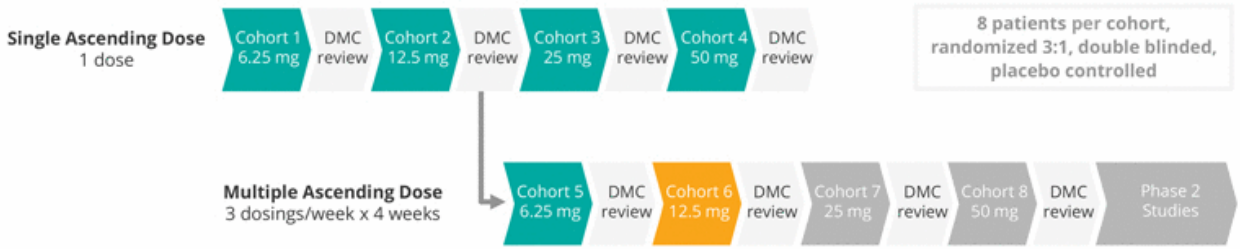
Nebulizer

PARI eFlow is commonly used by CF patients for nebulization of inhaled agents
Unpublished ProQR data

QR-010 in solution is nebulized in desired 3-5 MMAD particle size
Unpublished ProQR data

QR-010 is stable after nebulization by a PARI eFlow nebulizer
Unpublished ProQR data

Phase 1b update



- 64 homozygous ΔF508 CF patients (>18yrs)
- Inhalation through PARI eFlow nebulizer
- Participating sites: 20 sites in EU (CTN) and North America (TDN)

- Endpoints:
  - Safety, tolerability and pharmacokinetics
  - Exploratory efficacy (FEV1, CFQ-R, weight gain, sweat chloride)

Treatment-emergent adverse events - SAD

	6.25 mg N = 8	12.5 mg N = 8	25 mg N = 12	50 mg N = 8	SAD Total N = 36
Subjects with at least one TEAE	4 (50.0)	3 (37.5)	7 (58.3)	5 (62.5)	19 (52.8)
Gastrointestinal disorders	2 (50.0)	0	1 (8.3)	3 (37.5)	6 (16.7)
Abdominal Pain	0	0	0	1 (12.5)	1 (2.8)
Abdominal Pain Upper	0	0	0	1 (12.5)	1 (2.8)
Dry Mouth	2 (25.0)	0	1 (8.3)	0	3 (8.3)
Hypoasthaesia Oral	0	0	0	1 (12.5)	1 (2.8)
Tongue Discolouration	0	0	0	1 (12.5)	1 (2.8)
General disorders and administration site conditions	0	0	1 (8.3)	1 (12.5)	2 (5.6)
	0	0	1 (8.3)	0	1 (2.8)
Chest discomfort	0	0	1 (8.3)	0	1 (2.8)
Chest pain	0	0	0	1 (12.5)	1 (2.8)
Feeling jittery					
Injury, poisoning and procedural complications	0	0	1 (8.3)	0	1(2.8)
Sunburn	0	0	1 (8.3)	0	1(2.8)
Metabolism and nutrition disorders	0	0	1 (8.3)	0	1 (2.8)
Hyperglycaemia	0	0	1 (8.3)	0	1 (2.8)
Musculoskeletal and connective tissue disorders	0	0	0	1 (12.5)	1 (2.8)
Musculoskeletal stiffness	0	0	0	1 (12.5)	1 (2.8)
Neck pain	0	0	0	1 (12.5)	1 (2.8)

Key takeaways:

- Low numbers of treatment emerging AE's
- Independent DSMC
- No safety concerns

Treatment-emergent adverse events - SAD

	6.25 mg N = 8	12.5 mg N = 8	25 mg N = 12	50 mg N = 8	SAD Total N = 36
Nervous system disorders	3 (37.5)	1 (12.5)	2 (16.7)	2 (25.0)	8 (22.2)
Dizziness	0	0	0	2 (25.0)	2 (5.6)
Headache	2 (25.0)	1 (12.5)	1 (8.3)	1 (12.5)	6 (16.7)
			1 (8.3)		
Sinus Headache	1 (12.5)	0	0	0	1 (2.8)
Psychiatric disorders	0	1 (12.5)	0	0	1(2.8)
Agitation	0	1 (12.5)	0	0	1(2.8)
Reproductive system and breast disorders	1 (12.5)	0	0	0	1(2.8)
Menstruation irregular	1 (12.5)	0	0	0	1(2.8)
Respiratory, thoracic and mediastinal disorders	0	1 (12.5)	2 (16.7)	2 (25.0)	5 (13.9)
Cough	0	1 (12.5)	1 (8.3)	1 (12.5)	3 (8.3)
Pulmonary congestion	0	0	0	2 (25.0)	2 (5.6)
Throat irritation	0	0	0	1 (12.5)	1 (2.8)
Wheezing	0	0	1 (8.3)	0	1 (2.8)
Skin and subcutaneous tissue disorders	0	1 (12.5)	0	0	1 (2.8)
Pruritus generalised	0	1 (12.5)	0	0	1 (2.8)

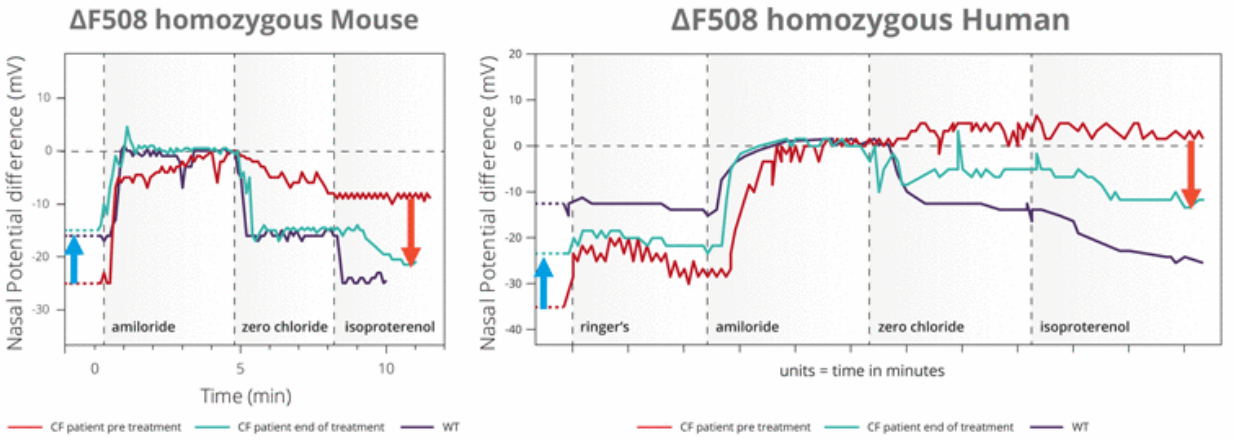
Key takeaways:

- Low numbers of treatment emerging AE's
- Independent DSMC
- No safety concerns

Phase 1b update

- QR-010 in doses tested to date is observed to be safe and well tolerated
  - 4 Single dose cohorts completed
    - 8 patients per cohort, randomized 3:1
    - 6.25, 12.5, 25 and 50 mg by inhalation
  - All cohorts reviewed by independent DSMC
  - No SAEs reported
  - No dose limiting toxicity identified
- Multiple dose cohorts
  - 4 dose escalating repeated dose cohorts
    - 8 patients per cohort, randomized 3:1
    - 12 doses of QR-010 by inhalation
  - Cohort 5 completed
    - 12 doses of QR-010 were well tolerated
    - Reviewed by DSMC, no safety signal, no dose-limiting toxicity
  - Cohort 6 currently enrolling
  - Data is blinded until study is completed
  - Top-line data is expected in mid 2017

QR-010 pre-clinical results translated to patients



- ✓ Pre-clinical data supports QR-010 can restore CFTR function in homozygous CF mice
- ✓ Pre-clinical PoC translated into patients
- ✓ Proof of Concept achieved in homozygous CF patients
- ✓ Phase 1b to read out in mid-2017

Pipeline Update

By Daniel de Boer

Innovation

In-house discovery engine

RNA based
RNA modulation to restore wild-type functionality

Product focused
High unmet needs

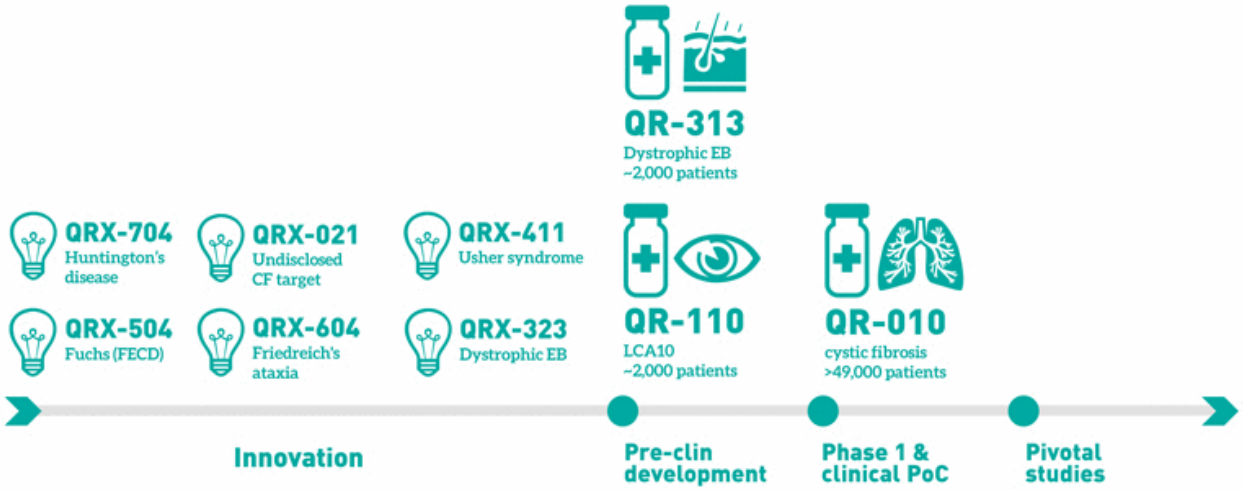
Feasible delivery
Feasible delivery route to target organ

Well understood causality
Genetic defect leading to disease manifestation well understood

Intellectual property
Agressive patenting strategy
Broad IP portfolio

Product selection
Thorough selection process before a candidate goes in development

Research and development pipeline



QR-110 for LCA10

mRNA profile restoration

mRNA profile restored to wild-type

Restoration CEP290 protein levels

Significant increase in CEP290 protein levels

Local (intravitreal) delivery to the eye

Eye well validated target for oligo's

Efficient delivery to outer nuclear layer in the retina

Regulatory

Regulatory discussions supportive of development plan

Toxicology

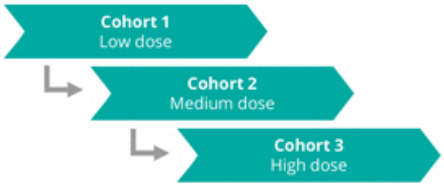
GLP tox in two species up to 3 months

Phase 1b

Start of a repeated dose first in human clinical trial in H1 2017

QR-110 for LCA10

PQ-110-001 Phase 1b clinical study



4 patients per cohort,
Open-label

Ascending Doses
4 doses (every 3 months)

- 12 homozygous or compound heterozygous p.Cys998X LCA10 patients
- Adults and children (>6yrs) intravitreal injections in one eye, other eye serves as control
- Participating sites: major sites in EU and US
- Primary endpoints:
  - Safety, tolerability and pharmacokinetics
- Exploratory efficacy:
  - FST, mobility testing, visual acuity, OCT, PRO, ERG, nystagmus tracking, pupillometry)
- **Expected to dose first patient in H1 2017**
- **Expected top-line data in 2018**

QR-313 for DEB

mRNA profile restoration

mRNA profile restored to produce active protein

Local delivery to the skin

Oligo in hydrogel for application to wounds

Delivery to C7 producing fibroblasts and keratinocytes

Restoration C7 protein functionality

Functional protein restored forming anchoring fibrils

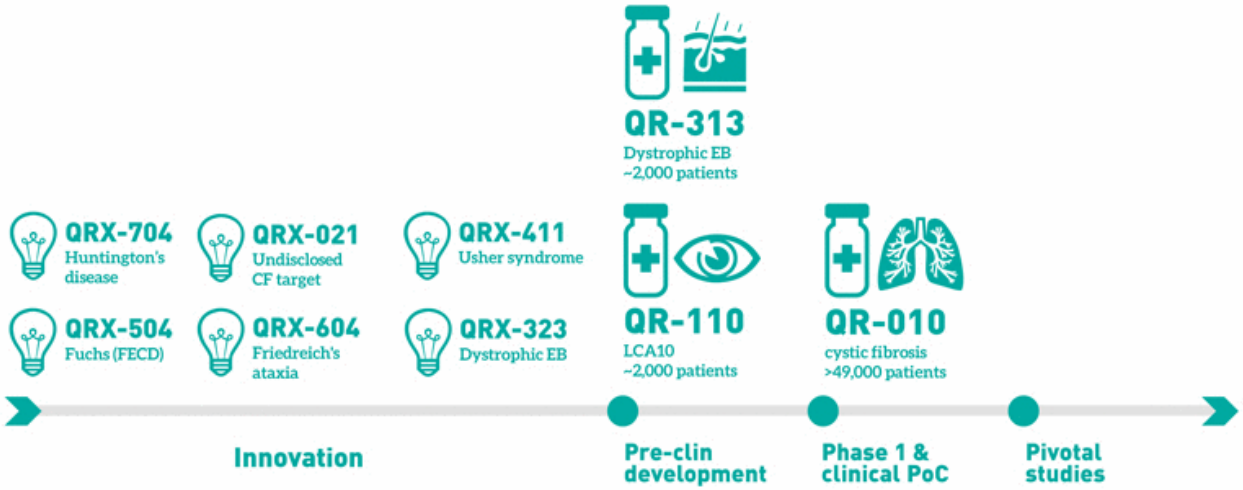
Toxicology

GLP tox in two species

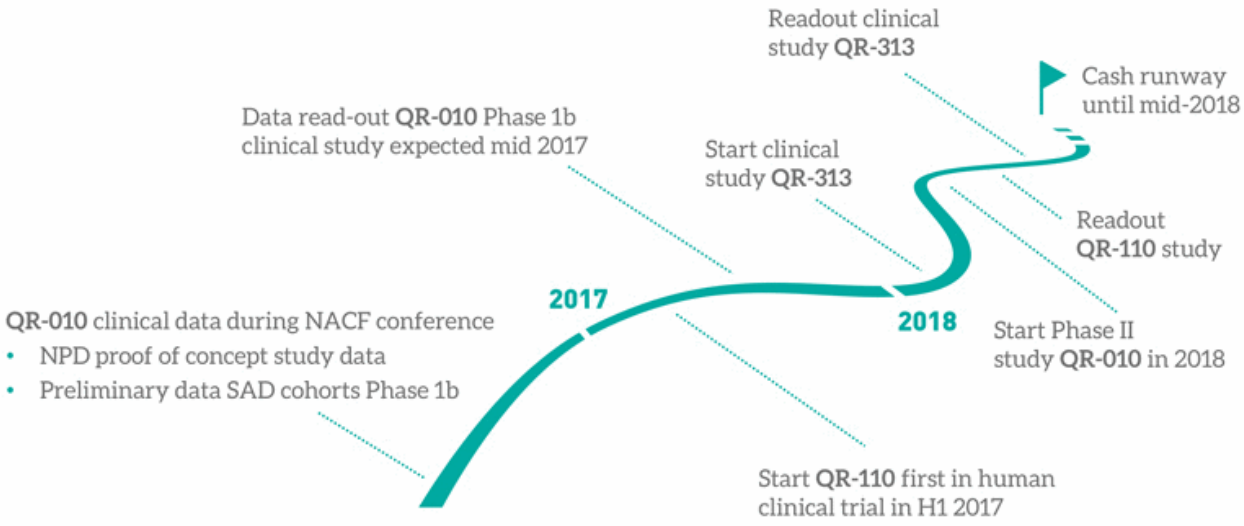
Phase 1b

Preparations ongoing
Study to start in 2018

Research and development pipeline



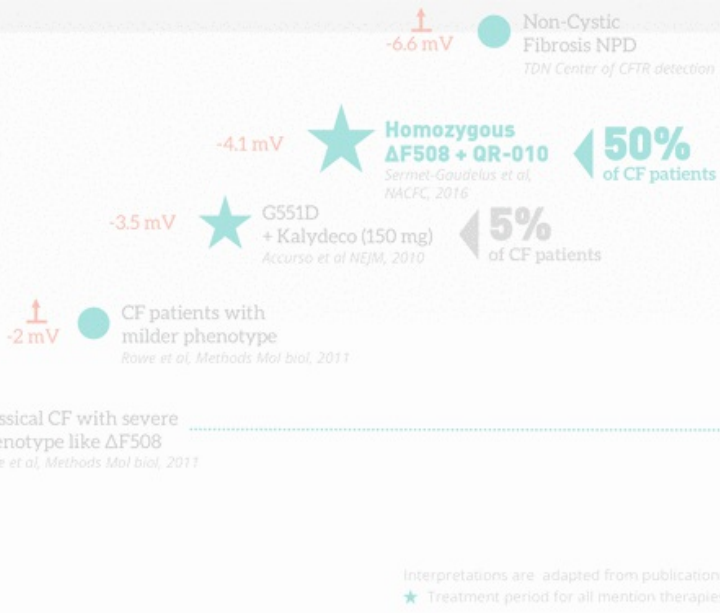
ProQR Therapeutics - What's next?



Putting QR-010 NPD results in perspective

Key takeaways:

- QR-010 improves CFTR function in ΔF508 homozygous patients
- Improved total chloride response which shows direct activity
- Improved max basal PD which shows down-regulation of sodium channels
- Single agent, innovative approach for ΔF508 patients
- Validates pre-clinical data



Interpretations are adapted from publications

★ Treatment period for all mention therapies is 28 days


ProQR®
IT'S IN
OUR RNA