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

FORM 6-K

REPORT OF FOREIGN PRIVATE ISSUER
PURSUANT TO SECTION 13a-16 OR 15d-16
UNDER THE SECURITIES EXCHANGE ACT OF 1934

For the month of January, 2017

Commission File Number: 001-36815

Ascendis Pharma A/S
(Exact Name of Registrant as Specified in Its Charter)

Tuborg Boulevard 5
DK-2900 Hellerup
Denmark
(Address of 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): ☐

Spokespersons of Ascendis Pharma A/S (the "Company") plan to present the information in the presentation slides attached hereto as Exhibit 99.1 at various investor and analyst meetings scheduled during the week of January 9, 2017.

The furnishing of the attached presentation is not an admission as to the materiality of any information therein. The information contained in the presentation is summary information that is intended to be considered in the context of more complete information included in the Company's filings with the Securities and Exchange Commission (the "SEC") and other public announcements that the Company has made and may make from time to time by press release or otherwise. The Company undertakes no duty or obligation to update or revise the information contained in this report, although it may do so from time to time as its management believes is appropriate. Any such updating may be made through the filing or furnishing of other reports or documents with the SEC, through press releases or through other public disclosures.

SIGNATURES

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

Ascendis Pharma A/S

Date: January 9, 2017

By: /s/ Michael Wolff Jensen
Michael Wolff Jensen
Chairman and Senior Vice President, General Counsel

EXHIBIT INDEX

Exhibit No.	Description
99.1	Company Presentation.

Exhibit 99.1

Ascendis Pharma A/S

Jan Mikkelsen
President & CEO

January 2017

Cautionary Note On Forward-Looking Statements:

This presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, such as statements regarding our future results of operations and financial position, including our business strategy, prospective products, availability of funding, clinical trial results, product approvals and regulatory pathways, collaborations, timing and likelihood of success, plans and objectives of management for future operations, and future results of current and anticipated products, are forward-looking statements. These forward-looking statements are based on our current expectations and beliefs, as well as assumptions concerning future events. These statements involve known and unknown risks, uncertainties and other factors that could cause our actual results to differ materially from the results discussed in the forward-looking statements. These risks, uncertainties and other factors are more fully described in our reports filed with or submitted to the Securities and Exchange Commission, including, without limitation, our most recent Annual Report on Form 20-F, particularly in the sections titled “Risk Factors” and “Management’s Discussion and Analysis of Financial Condition and Results of Operations” as well as our Report on Form 6-K filed with the SEC on October 18, 2016. In light of the significant uncertainties in our forward-looking statements, you should not place undue reliance on these statements or regard these statements as a representation or warranty by us or any other person that we will achieve our objectives and plans in any specified timeframe, or at all.

Any forward-looking statement made by us in this presentation speaks only as of the date of this presentation and represents our estimates and assumptions only as of the date of this presentation. Except as required by law, we assume no obligation to update these statements publicly, whether as a result of new information, future events or otherwise after the date of this presentation.

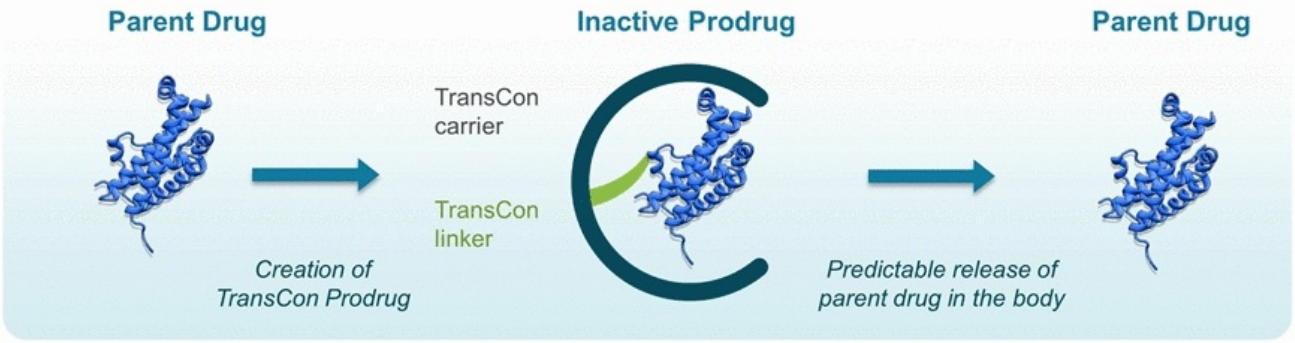
This presentation concerns product candidates that are or have been under clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration, European Medicines Agency or other foreign regulatory authorities. These product candidates are currently limited by U.S. Federal law to investigational use, and no representations are made as to their safety or effectiveness for the purposes for which they are being investigated.

Company Overview

- Create best-in-class rare disease products addressing unmet medical needs
  - Apply TransCon technology to parent drugs with clinical proof-of-concept
  - Expect higher development success rate compared to traditional drug development
- Endocrinology rare disease wholly-owned pipeline and expected milestones
  - TransCon Growth Hormone for pediatric GHD: heiGHt Trial full enrollment Q4 2017
  - TransCon PTH for hypoparathyroidism: IND/equivalent in 2Q 2017; planning pivotal trial in 2018
  - TransCon CNP for achondroplasia: IND/equivalent in 4Q 2017
- Commercial focus on the U.S. market with multi-billion dollar revenue potential
- Established high-value collaborations with Roche/Genentech in ophthalmology and Sanofi in diabetes
- Pro forma September 30, 2016 cash of ~ €190 million[1]

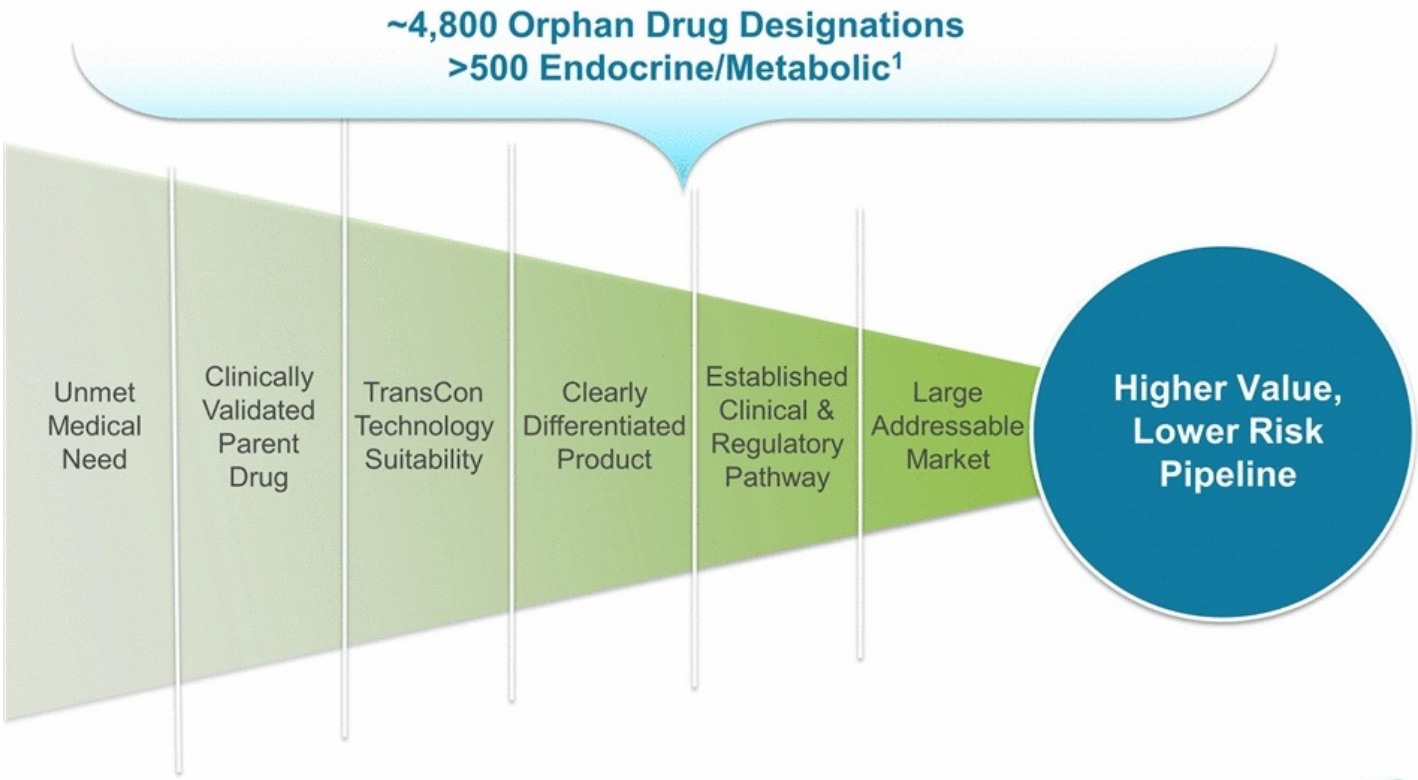
[1] Based on cash and cash equivalents as of September 30, 2016 and approximately $127.4 million in estimated net proceeds from the public offering of our ADSs that was completed in 4Q 2016.

TransCon Technology



- Predictable release of unmodified parent drugs
  - Parent drugs can be proteins, peptides or small molecules
  - Linker release only dependent on pH and temperature
  - TransCon carrier enables both systemic and localized drug exposure
- Maintains same mode-of-action of parent drug molecule
- Release of parent drug supporting up to half-yearly administration
- TransCon products eligible for new composition of matter IP claims

Ascendis' Approach to Product Innovation



[1] Totals from FDA and EMA Orphan List, public & proprietary databases, advisors and conferences

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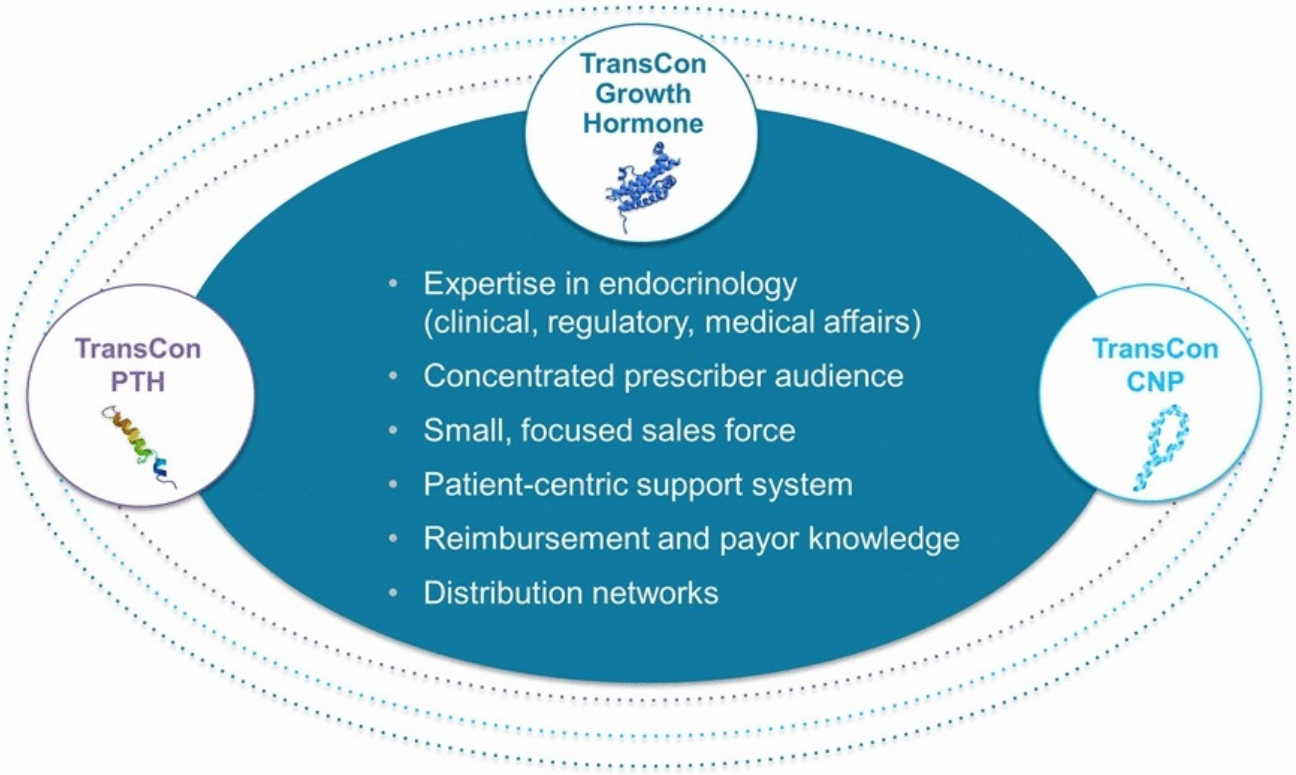
Expected Synergies of Therapeutic Focus



Building a Leading Company in Rare Diseases

Internal Rare Disease Endocrinology Pipeline

Product Candidate	Primary Indication	Development Stage	Potential WW Market[1]	WW Commercial Rights
TransCon Growth Hormone	Growth hormone deficiency	Phase 3	> $3 billion[2]	ascendis pharma
TransCon PTH	Hypoparathyroidism	Pre-IND	> $2 billion[3]	ascendis pharma
TransCon CNP	Achondroplasia	Pre-IND	> $1 billion	ascendis pharma

Current/Potential Strategic Collaborations

TransCon Ranibizumab	Ophthalmology	Not disclosed	> $7 billion	Genentech
TransCon Peptides	Diabetes	Not disclosed	> $1 billion	SANOFI
TransCon Treprostinil	PAH	Phase 1	> $1 billion	Partnering Opportunity

Endocrinology | Ophthalmology | CV

[1] Based on market data and company estimates.
[2] Includes all indications.
[3] Based on treatment of ~25% of the U.S. patient population of ~77,000 patients.

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TransCon Growth Hormone:
Once-Weekly Replacement
Therapy

Long-Acting Growth Hormones

- Human Growth Hormone (hGH) is a growing ~$3 billion market
  - Largest indication is Growth Hormone Deficiency (GHD), representing ~50% of the market
  - Pediatric indications comprise up to 90% of the market
- Current growth hormone therapies require daily injections, which often results in suboptimal compliance and treatment response[1]
- No successful commercialization of a long-acting growth hormone despite ~ 30 years of multiple attempts with different technologies
  - Molecular enlargement technologies – GH protein fusions, permanent PEGylation of GH
  - Depot formulations – release of unmodified GH from polymer microspheres
- TransCon Growth Hormone capitalizes on lessons from previous programs
  - Delivers physiological levels of unmodified growth hormone
  - Safety, tolerability, efficacy and immunogenicity shown to be comparable to daily hGH therapy

[1] PLoS ONE 2011, 6(1), e16223

GHD – Not Only About Growth

- Children with GHD suffer beyond growth failure[1]
  - Increased and abnormal fat distribution (especially truncal fat mass)
  - Abnormal metabolic profile
  - Impaired exercise capacity
  - Decreased quality of life
- Adults with GHD[2]
  - Trunk fat accumulation and decrease in lean body mass
  - Decreased bone mineral density
  - Dyslipidemia
  - Increased cardiovascular mortality and morbidity
  - Decreased quality of life
- Daily hGH addresses all the symptoms of the disease

Long-acting GH must fully mimic daily hGH to address the totality of the disease

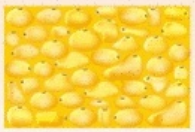
[1] BMC Endocrine Disorders 2012, 12:26
[2] J Clin Endocrinol Metab 2006, 91: 1621–1634

Tissue Specific Effects of GH and IGF-1

- GH receptors are expressed on virtually every cell of the body[1]
- IGF-1 in serum is primarily derived from the liver and regulated by GH[1]

BONE

Optimal growth is achieved *via* co-stimulation of GH and IGF-1 receptors in bone[1]



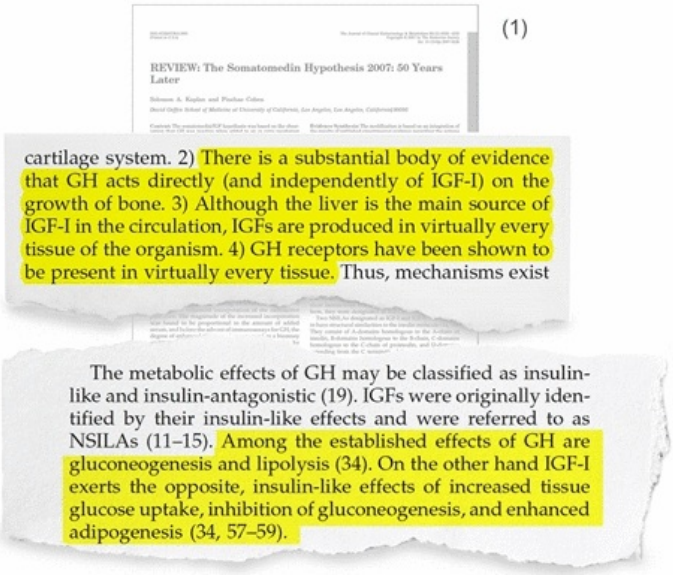
ADIPOSE TISSUE

GH breakdown fat *via* stimulation of GH receptors in fat, counteracting the lipogenesis effect of IGF-1[1]

- Increasing molecular size through permanent protein modification alters the ability to distribute outside the blood compartment compared to unmodified drug[2]
- Data continue to support that limiting the distribution of GH alters the therapeutic effect on trunk fat and growth velocity compared to unmodified/daily hGH

[1] J Clin Endocrinol Metab 2007, 92(012): 4529-4535
[2] Digestive and liver disease, 2004, 36 suppl.3, S334-39

Tissue Distribution Associated with Optimal Profile

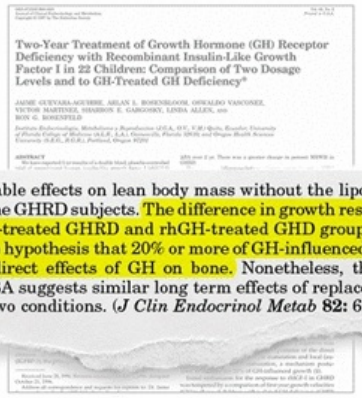


REVIEW: The Somatomedin Hypothesis 2007: 50 Years Later

cartilage system. 2) There is a substantial body of evidence that GH acts directly (and independently of IGF-I) on the growth of bone. 3) Although the liver is the main source of IGF-I in the circulation, IGFs are produced in virtually every tissue of the organism. 4) GH receptors have been shown to be present in virtually every tissue. Thus, mechanisms exist

(1)

The metabolic effects of GH may be classified as insulin-like and insulin-antagonistic (19). IGFs were originally identified by their insulin-like effects and were referred to as NSILAs (11–15). Among the established effects of GH are gluconeogenesis and lipolysis (34). On the other hand IGF-I exerts the opposite, insulin-like effects of increased tissue glucose uptake, inhibition of gluconeogenesis, and enhanced adipogenesis (34, 57–59).



Two-Year Treatment of Growth Hormone (GH) Receptor Deficiency with Recombinant Insulin-Like Growth Factor I in 22 Children: Comparison of Two Dosage Levels and to GH-Treated GH Deficiency*

comparable effects on lean body mass without the lipolytic effects of GH in the GHRD subjects. The difference in growth response between rhIGF-I-treated GHRD and rhGH-treated GHD groups is consistent with the hypothesis that 20% or more of GH-influenced growth is due to the direct effects of GH on bone. Nonetheless, the comparable ΔHA/ΔBA suggests similar long term effects of replacement therapy in the two conditions. (*J Clin Endocrinol Metab* **82:** 629–633, 1997)

(2)

Additional published data show that long-acting GH formulations based on unmodified hGH and daily hGH are associated with reduction in fat mass[3-4]

1 J Clin Endocrinol Metab 2007,12: 4529-4535
2 J Clin Endocrinol Metab1997, 82(2): 629-633
3 J Clin Endocrinol Metab 2005 90: 6431–40
4 Pituitary. 2013 Sep;16(3):311-8

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TransCon GH Designed to Address Issues Raised by Academic and Regulatory Experts[1]

Commentary
Open Access
J S Christiansen and others | Long-acting growth hormone development | 174:6 | C1–C8

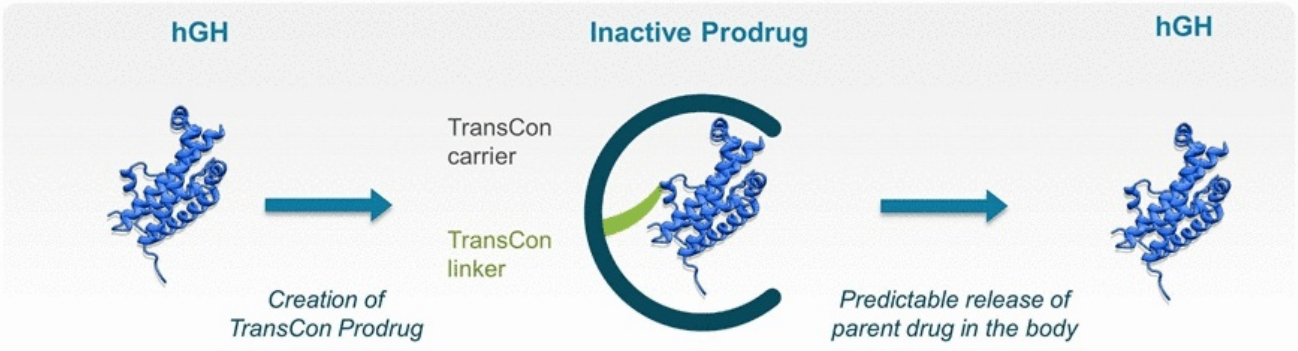
Growth Hormone Research Society perspective on the development of long-acting growth hormone preparations

Jens Sa
Beverly
Philipp
Laurie I
Yukihir
Jeff M I
Gudmu
John J I
Bradley
Judith I
Michae

Potential issues include: (i) supraphysiological elevations of GH and/or IGF1, (ii) trough GH concentrations above normal physiological levels, (iii) fluctuating IGF1 levels, (iv) elevated IGF1 in the absence of GH bioactivity, (v) nonphysiological tissue distribution due to distinct biological features of the products, and (vi) the specific chemical composition of each LAGH product may confer unique safety issues.

Once-weekly prodrug releasing unmodified GH designed to mimic daily hGH:

- ✓ Tissue distribution
- ✓ Physiological levels
- ✓ Therapeutic effects



[1] *European Journal of Endocrinology* (2016) **174**, C1–C8

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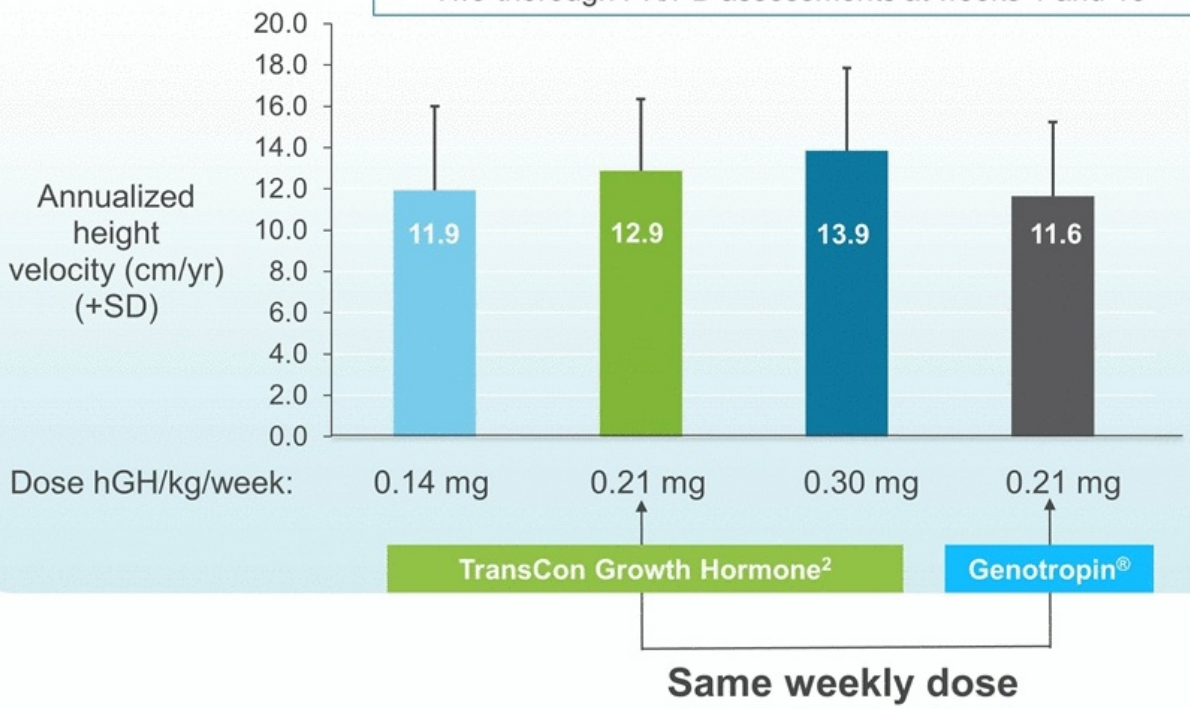
Growth Comparable to Daily hGH in Phase 2 Study[1]

- 26-week treatment period
- Two thorough PK/PD assessments at weeks 1 and 13



[1] Intergroup differences not statistically significant.
[2] Conducted with a previous lower strength version of TransCon Growth Hormone.

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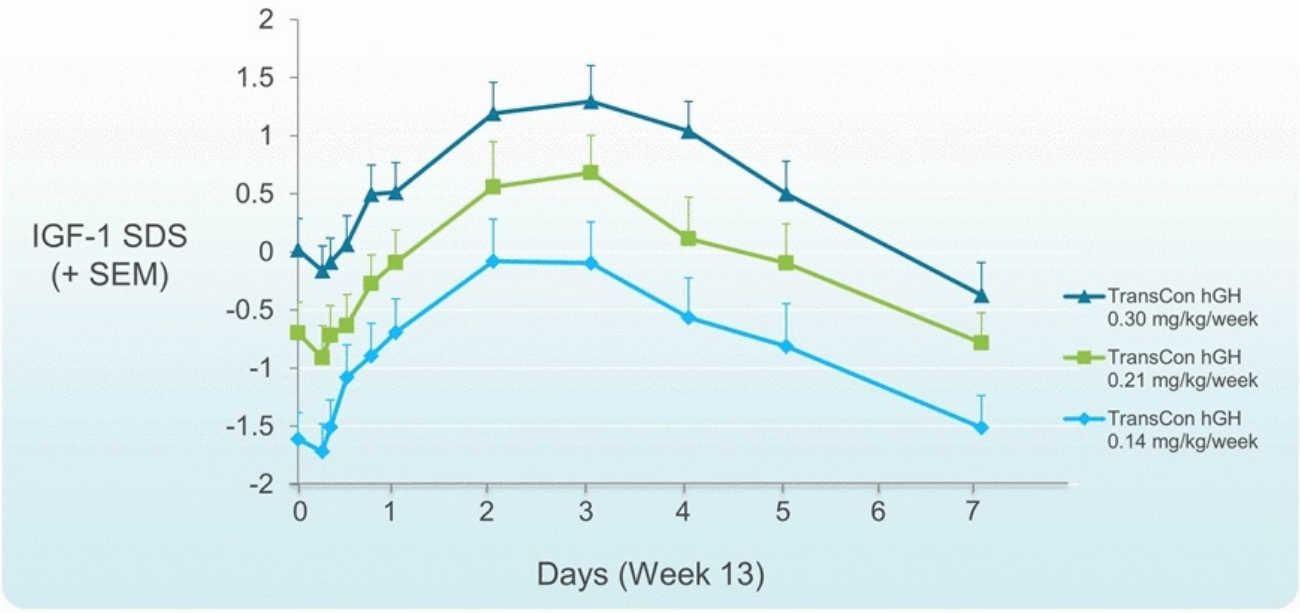
Dose Proportional IGF-1 Elevation Observed into the Normal Range in Phase 2 Study



Transient point values of IGF-1 SDS > +2.0 have been observed in a small number of patients primarily at the highest dose level

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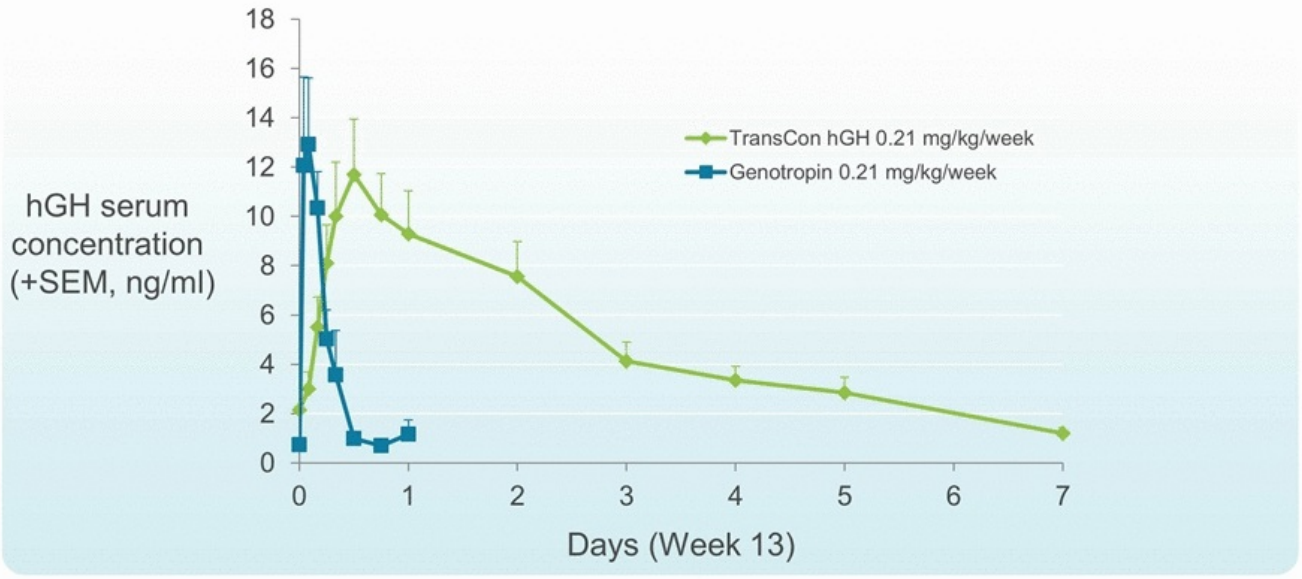
Comparable hGH Levels for TransCon Growth Hormone and Daily hGH in Phase 2 Study



Maximum hGH blood concentration comparable between equivalent weekly doses of TransCon Growth Hormone and daily hGH

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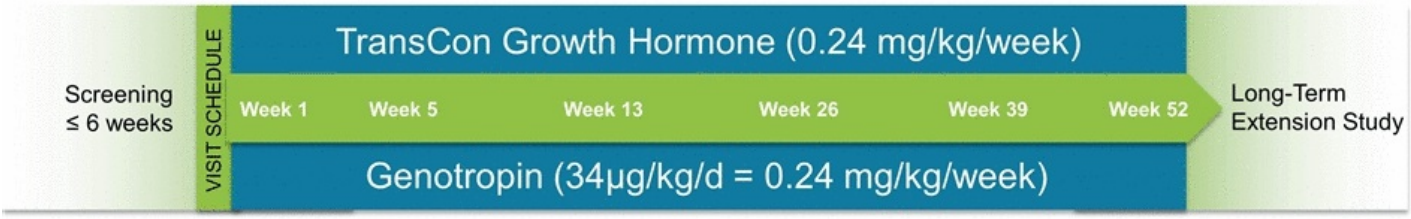
Comparable Safety to Daily hGH in Phase 2

No Serious Adverse Events (SAEs) related to study drug	• Adverse events consistent with daily hGH therapy observed and not different between cohorts
Immunogenic profile comparable to daily hGH	• Favorable immunogenic profile comparable to daily hGH • No occurrence of neutralizing antibodies
Injection site tolerability comparable to daily hGH	• >1100 TransCon Growth Hormone injections administered in the Phase 2 pediatric study • No reports of lipoatrophy or nodule formation

Phase 3 Study - heiGHt Trial Ongoing

heIGHt TRIAL

~ 150 treatment-naïve children with GHD (2:1 randomization)



Key Inclusion Criteria

- Prepubertal children with GHD
- Height SDS ≤ -2.0
- IGF-1 SDS ≤-1.0
- GHD with 2 GH stim. tests (GH ≤10 ng/mL)
- Bone age ≥ 6 months behind chronological

Key Endpoints

- Annualized height velocity (HV) at 52 weeks (primary endpoint)
- Annualized HV at earlier time points
- Change in HT SDS over 52 weeks
- Change in serum IGF-1/IGFBP-3 levels
- Change in IGF-1 SDS and IGFBP-3 SDS
- Normalization of IGF-1 SDS

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Easy to Use Device with Optimal Product Features

Key Device Features

- Simple operation with few user steps
- A single low-volume injection for all patients (<0.6 mL)
- Small needle comparable to daily hGH (31G, 4mm)
- Room temperature storage
- No waste due to empty-all design
- Enabled for Bluetooth® connectivity to IT health care solutions
- Device lifetime at least 4 years

Auto-injector for commercialization to be used in extension study

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TransCon Growth Hormone Target Product Profile

- ✓ Efficacy
- ✓ Safety (including immunogenicity)
- ✓ Tolerability

=

Comparable to Daily Growth Hormone

- ✓ Weekly subcutaneous administration
- ✓ Single injection/dose
- ✓ Convenience
  - ✓ 31G needle
  - ✓ Room temperature storage
- ✓ Device
  - ✓ Easy to use
  - ✓ Empty-all design (controlled substance)

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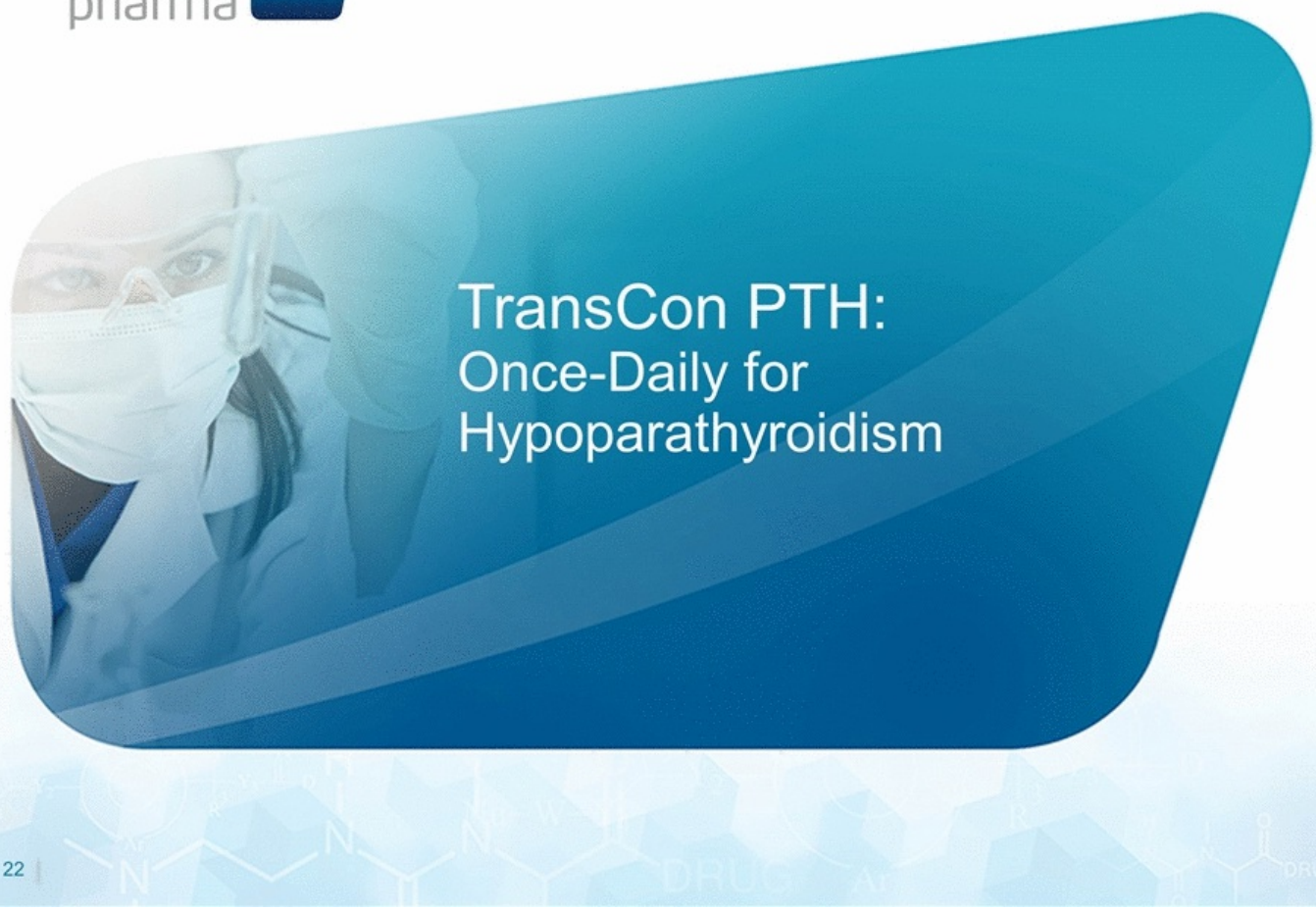
TransCon Growth Hormone: Highlights

- Potential best-in-class long-acting growth hormone product profile
- Phase 3 heiGHt Trial recruiting and initial patients dosed; full enrollment expected 4Q 2017
- Commercial scale manufacturing and supply chain established
- Auto-injector developed and on-track for launch
- Multiple patent concepts provide potential protection into 2036


TransCon PTH:
Once-Daily for
Hypoparathyroidism

Hypoparathyroidism – Not Only About Serum Calcium

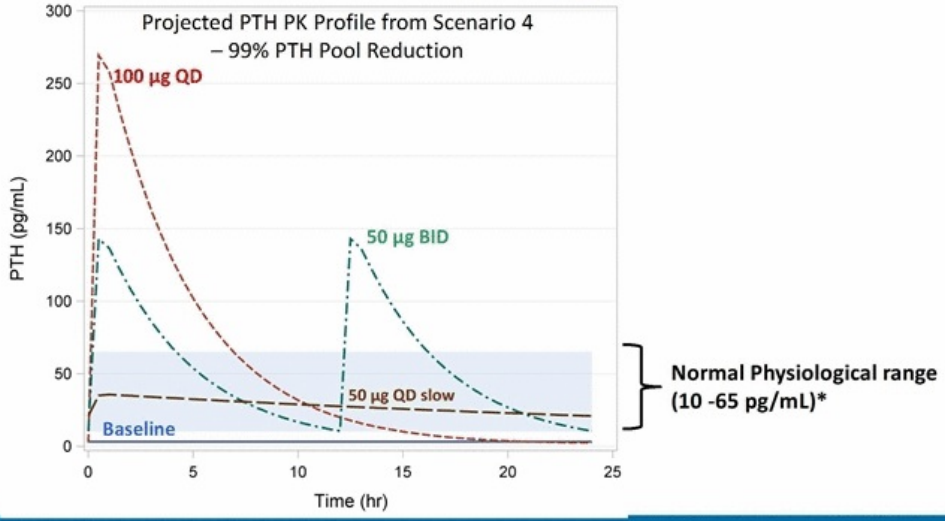
- Hypoparathyroidism affects 77,000 patients in the U.S.[1]
- Patients suffer numerous comorbidities:
  - Hypocalcemia and hypercalcemia
  - Hypercalciuria (stones, nephrocalcinosis)
  - Psychiatric disorders, depression
  - Reduced QOL
  - Basal ganglia/CNS calcifications
  - Lenticular calcifications/cataracts
  - Arterial calcifications/atherosclerosis
- Until recently, hypoparathyroidism remained among the few hormonal insufficiency states not treated by the replacement of its missing hormone
- Natpara® [PTH(1-84)] launched in 2015 as once-daily administration but incompletely addresses all aspects of the disease

[1] J Clin Endocrinol Metab 2016, 101(6): 2284-2299

FDA Perspective on Optimal PTH PK[1]

U.S. Food and Drug Administration
Protecting and Promoting Public Health
www.fda.gov

Altering Regimen (QD to BID) or Release Profile Bring PTH Levels Close to the Physiological Levels



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[1] FDA presentation Natpara Advisory Committee September 12, 2014

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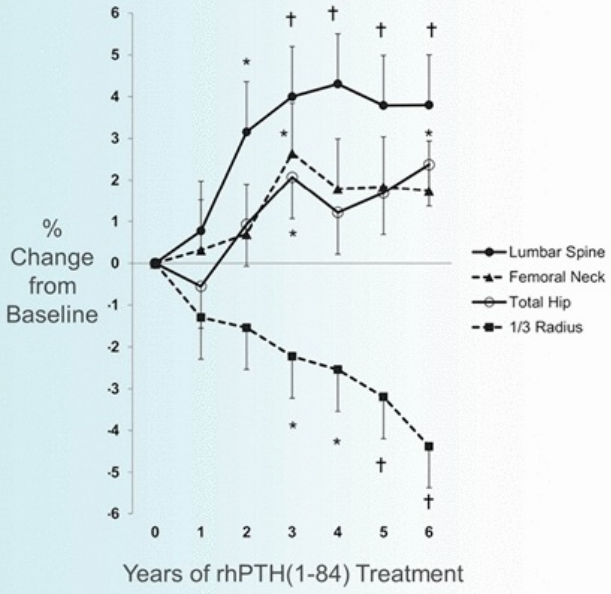
Intermittent PTH Caused Decrease in Cortical BMD

- Six years of therapy with Natpara was associated with a progressive decrease in cortical BMD[1]
- Continuous exposure to PTH(1-34) restores bone turnover to normal levels while avoiding the overstimulation of daily or twice daily injections[2]
- TransCon PTH is expected to normalize bone turnover and BMD, due to continuous PTH exposure

6 years of Natpara therapy decreases cortical bone BMD



[1] J Clin Endocrinol Metab 2016 doi: 10.1210/jc. 2015-4135
[2] J Clin Endocrinol Metab 2012, 97(2): 391-399

Optimal PTH PK Improved Treatment Outcomes

Physiological Effect in Hypoparathyroidism	Natpara Once-daily[1, 2]	PTH (1-34) Infusion[3-6]
Increase serum calcium	✓	✓
Reduce pill burden	✓	✓
Normalize urinary calcium excretion	X	✓
Reduce clinical hypercalcemia	X	✓
Reduce clinical hypocalcemia	X	✓
Normalize serum phosphate	✓ (high-normal range)	✓
Normalize bone turnover	X (cortical bone loss)	✓

NIH clinical trials demonstrated superiority of continuous infusion > twice daily injections > once daily injections[3-6]

[1] Natpara Product Label
[2] J Clin Endocrinol Metab 2016, 101(7): 2742-2750
[3] J Clin Endocrinol Metab 2009, 93(9): 3389-3395
[4] J Clin Endocrinol Metab 1998, 83(10): 3480-3486
[5] J Clin Endocrinol Metab 2013, 88(9): 4214-4220
[6] JAMA 1996, 276(8): 631-636

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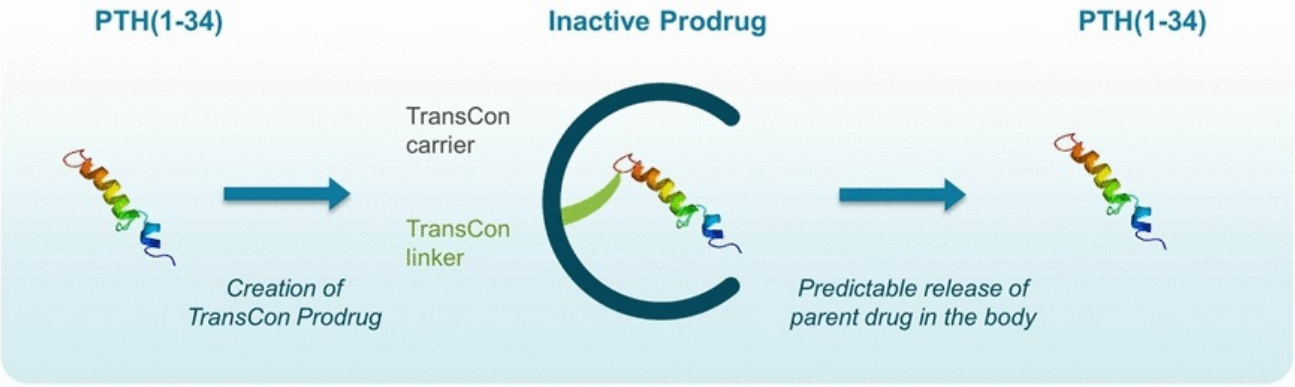
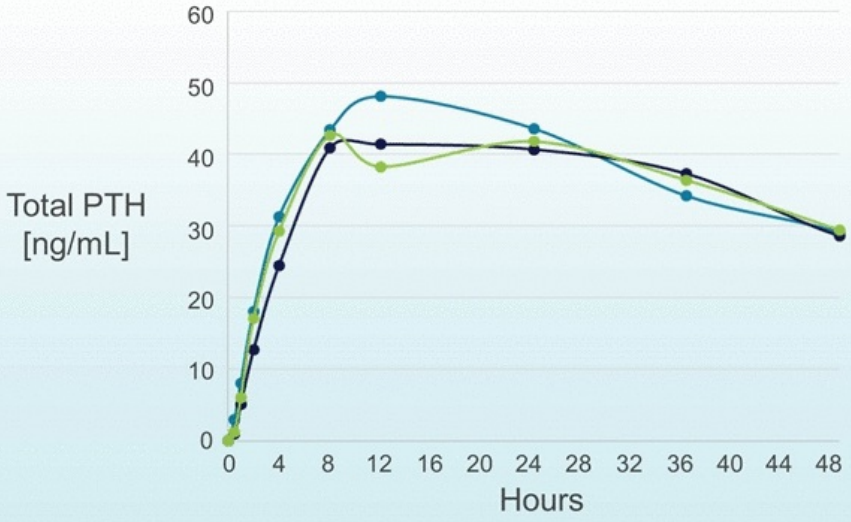
Designed Specifically for Hypoparathyroidism



- TransCon PTH is a sustained-release prodrug, providing free PTH levels in the physiological range over an entire 24 hours
- TransCon PTH designed to address limitations of current therapies to normalize:
  - Serum and urinary calcium levels
  - Serum phosphate levels
  - Bone turnover

Desired PK Profile Confirmed in Primates

PTH levels following SC injections in cynomolgus monkeys
(TransCon PTH 5µg/kg, n=3)



PK profile of TransCon PTH in cynomolgus monkeys supports infusion-like profile following daily administration

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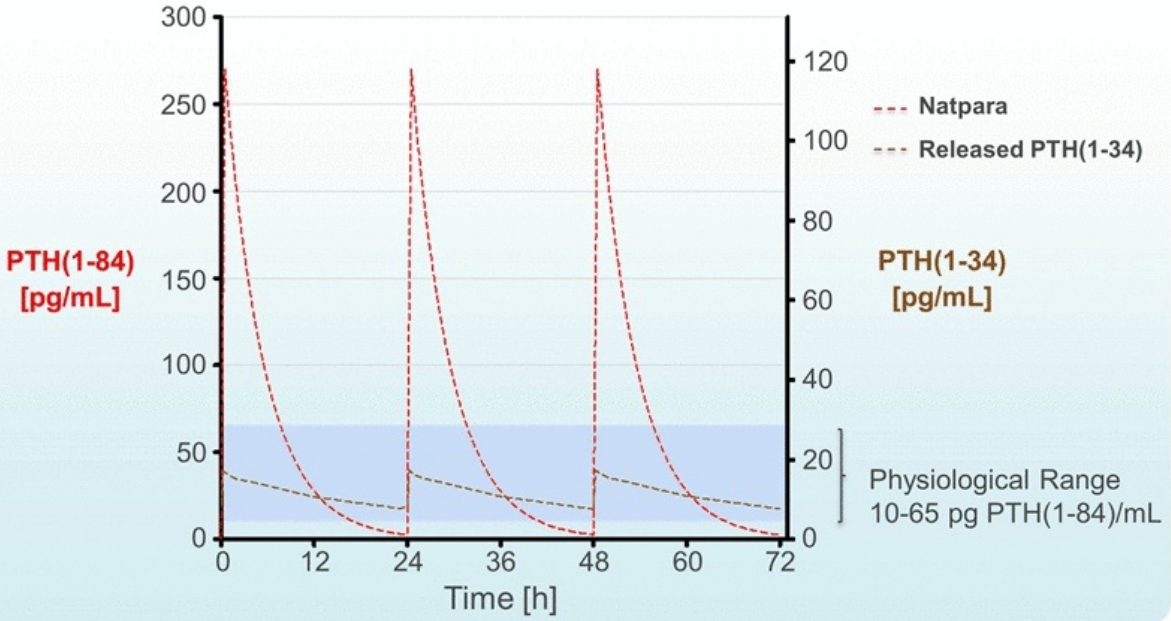
Designed to Provide Physiological PTH

TransCon PTH Modeling Based on Primate PK Data[1]



TransCon PTH is designed to maintain serum PTH levels in the physiological range, mimicking physiological exposure

[1] Simulation based on 100 µg QD Natpara and 70 µg QD TransCon PTH; Simulation of 3 repeat doses of Natpara and TransCon PTH at steady state

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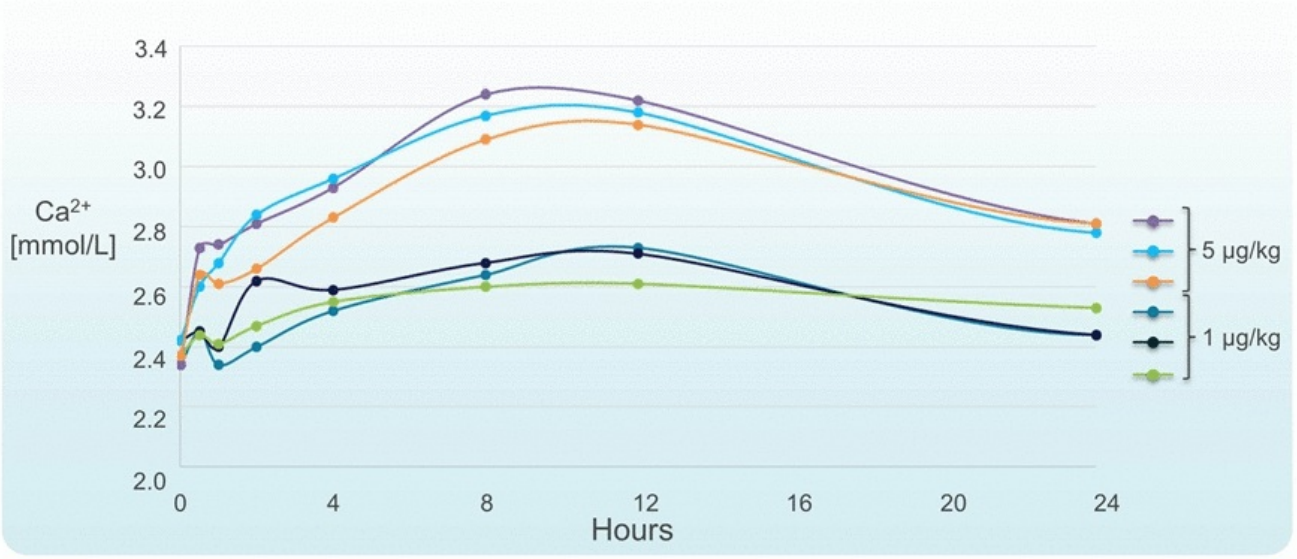
Dose-Dependent Increase of Serum Calcium

Ca^{2+} following SC injections in cynomolgus monkeys
(TransCon PTH 1µg/kg and 5µg/kg, n=3/group)



TransCon PTH leads to sustained elevation of serum calcium lasting more than 24 hours with low inter-individual variability

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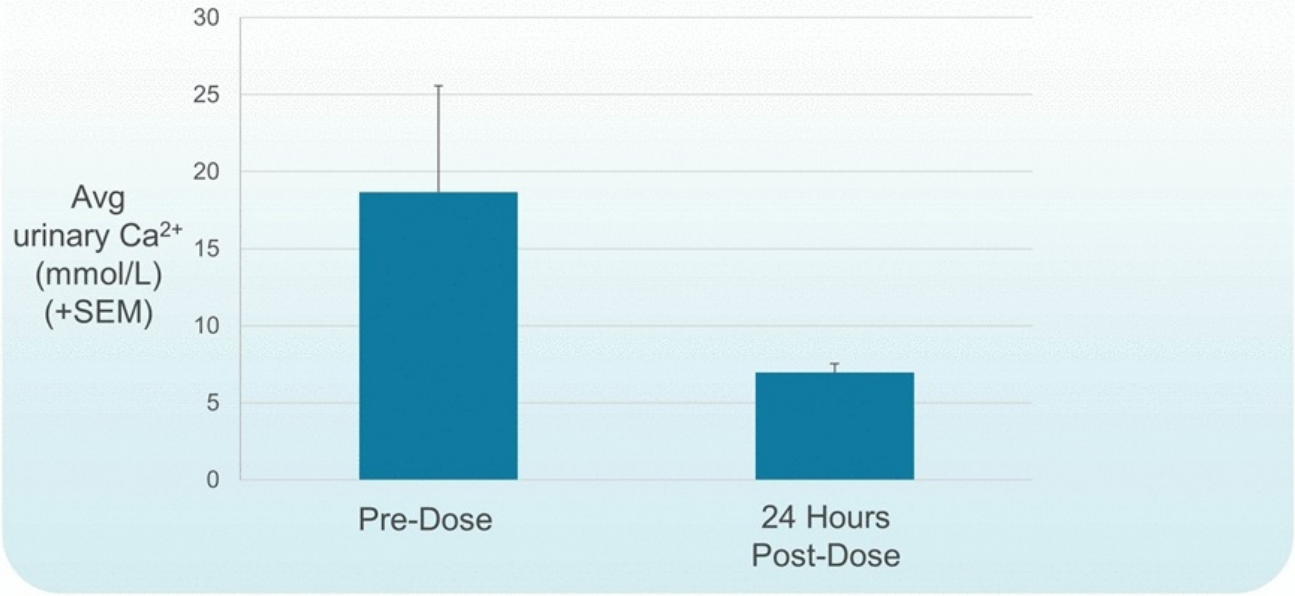
Reduction in Urinary Calcium Excretion

Urinary Ca^{2+} following SC injections in cynomolgus monkeys
(TransCon PTH 1µg/kg, n=3)



TransCon PTH administration reduces urinary calcium excretion in cynomolgus monkeys

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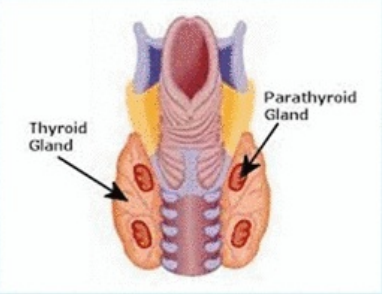
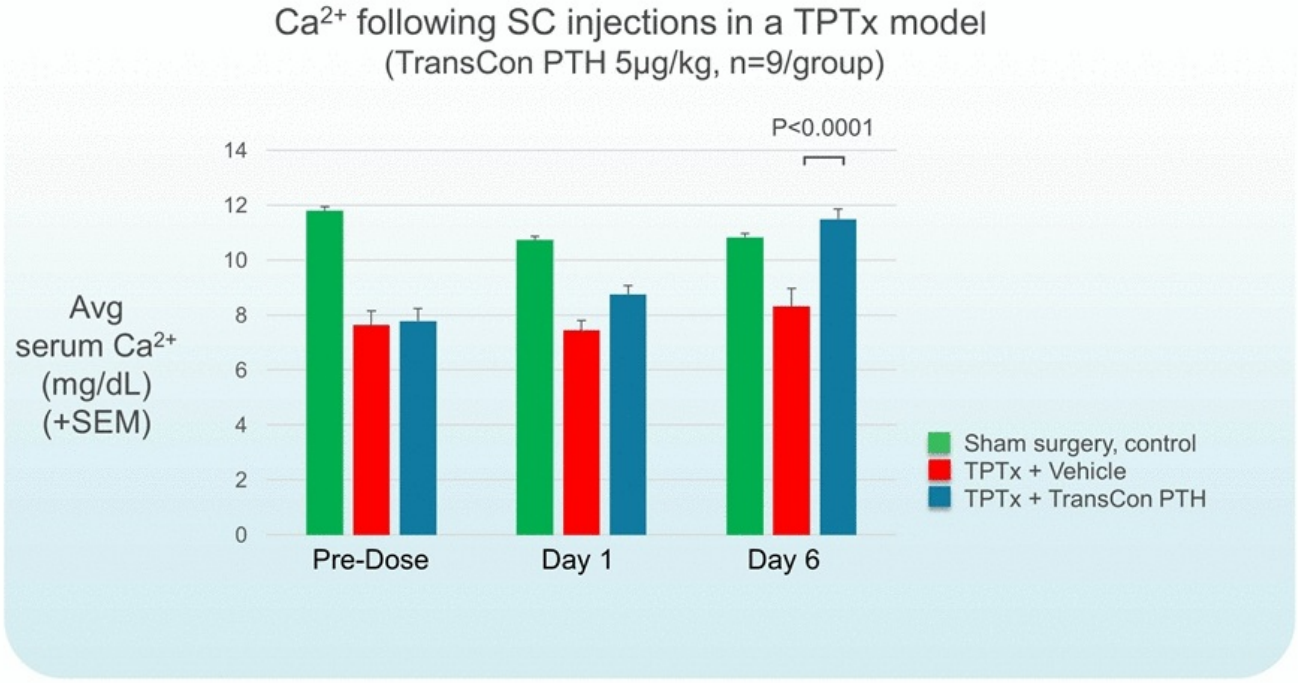
Thyroparathyroidectomy (TPTx) Model

- Hypoparathyroidism disease model
  - Rats subjected to thyroparathyroidectomy (TPTx) by removal of the thyroid and parathyroid glands and allowed to stabilize prior to dosing
  - Model characterized by decreased serum calcium and increased serum phosphorus



Normalized Calcium in Hypoparathyroidism Model



TransCon PTH normalized serum calcium in a relevant disease model of hypoparathyroidism

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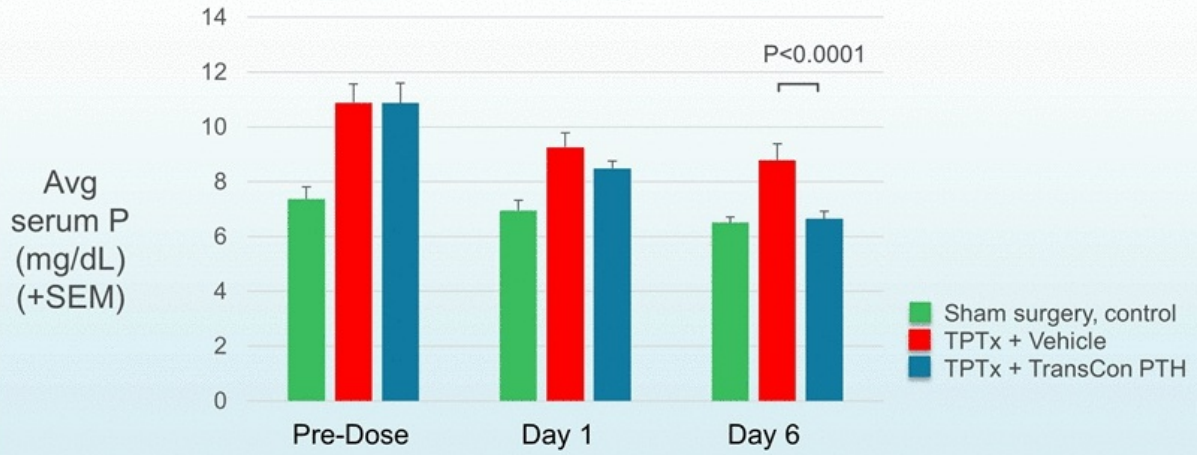
Reduced Phosphorous in Hypoparathyroidism Model

Serum Phosphorous following SC injections in a TPTx model
(TransCon PTH 5µg/kg, n=9/group)



TransCon PTH normalized serum phosphorous in a relevant disease model of hypoparathyroidism

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TransCon PTH: Highlights

- Development risk lowered as TransCon PTH based on parent drug [PTH(1-34)] with clinical proof of principle and proven TransCon technology
- Preclinical data mimic [PTH(1-34)] infusion and demonstrate once-daily TransCon PTH address the limitations of current therapies
- Accelerated clinical development plan
  - IND or equivalent filing expected 2Q 2017
  - Combined Phase 1 single and multiple ascending dose trial
  - Planning initiation of pivotal trial in 2018
- Multiple patent concepts provide potential protection into 2037


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TransCon CNP:
Once-Weekly CNP for
Achondroplasia

Achondroplasia – Not Only a Skeletal Disease

Autosomal dominant genetic disorder

- Most common form of human dwarfism
- Approximately 250,000 patients worldwide[1]
- 80% born to average-sized parents

Patients suffer numerous comorbidities

- Back/spine/cord compression
- Cardiovascular complications
- Dental complications
- Ear infections/sleep apnea
- Obesity
- Bowed legs

No FDA-approved therapy

- Only option to improve height is surgical limb lengthening

[1] Lancet 2007, 370: 162-172

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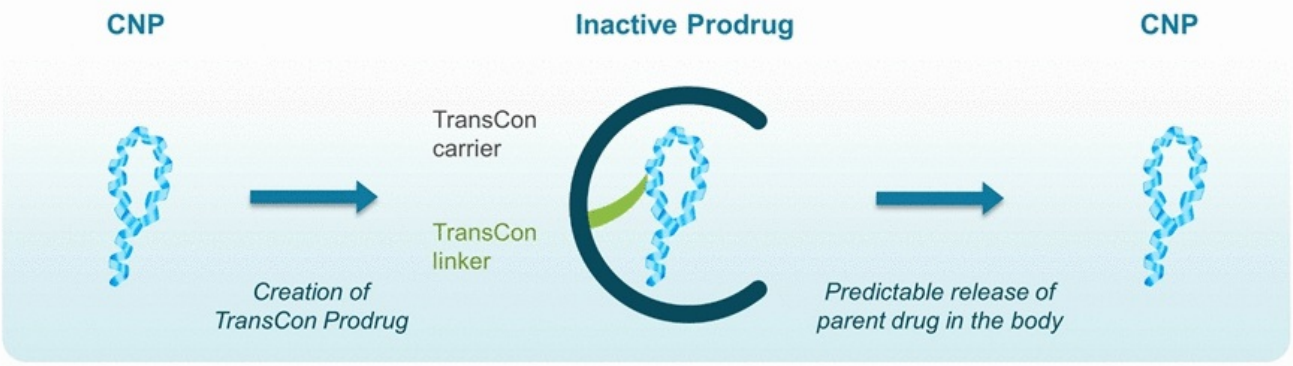
Clinical Proof of Principle in Achondroplasia

- Vosoritide (CNP analog) in Phase 3 for achondroplasia; reported promising height velocity data
  - Effects on growth at 12 months with 46-65% improvement from baseline in mean annual growth velocity[1]
  - Vosoritide well tolerated, but hypotension observed in 40% of subjects receiving 15 µg/kg/day[1]
  - Therapeutic coverage limited by the half-life of vosoritide (~20 min)

Therapeutic Goal: Optimize CNP efficacy with a well tolerated and convenient dosage form

[1] Biomarin R&D Day, April 2016

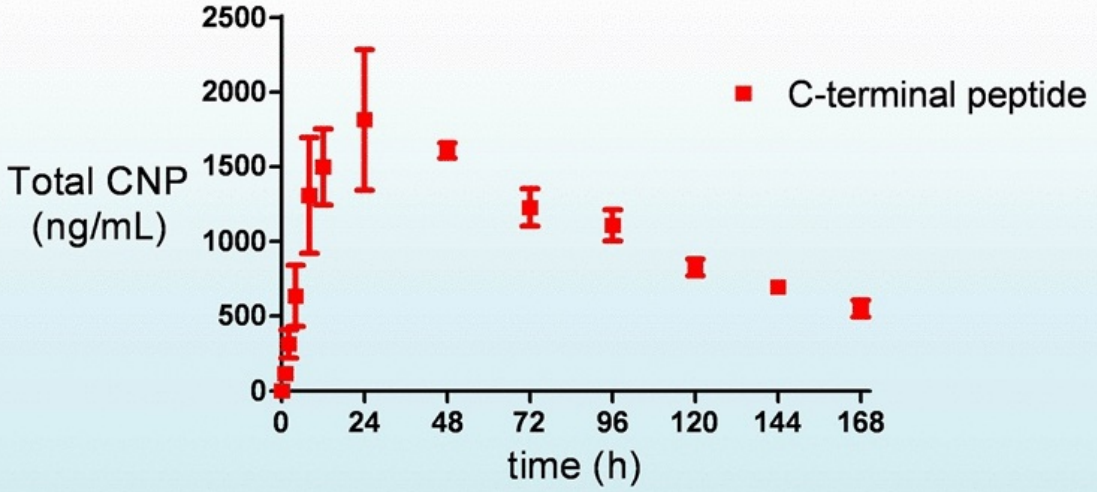
TransCon Technology Offers Potential Solution



- Effective shielding of TransCon CNP
  - From neutral endopeptidase degradation in subcutaneous tissue and blood compartment
  - Minimize binding of TransCon CNP to the NPR-C receptor to decrease clearance
  - Reduce binding of TransCon CNP to the NPR-B receptor to avoid hypotension
- Unmodified CNP liberated from TransCon CNP maintains small enough size to allow penetration into growth plates

TransCon CNP Weekly Profile Confirmed in Primates

TransCon CNP following SC injections in cynomolgus monkeys (n=3)



- Data from ongoing and completed preclinical studies supports weekly dosing in patients with achondroplasia
- No cardiovascular adverse effects observed in preclinical models

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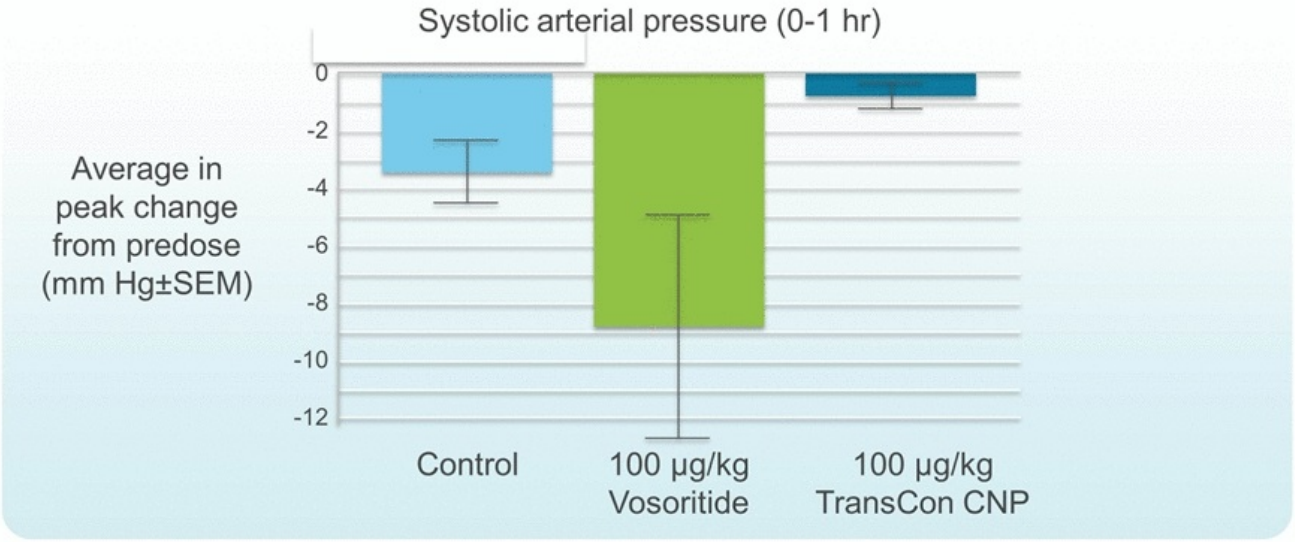
No CV Safety Signals Observed for TransCon CNP[1]



- No hypotension or increased heart rate observed in telemetrized cynomolgus monkeys (n=4/group) receiving TransCon CNP
- BP changes in this study comparable to previous vosoritide study[2]

[1] "Vosoritide" refers to a synthesized molecule with the same amino acid sequence prepared by Ascendis Pharma.
[2] J Pharmacol Exp Ther, 2015; 353, 132–149

Continuous Infusion More Effective Than Daily[1]

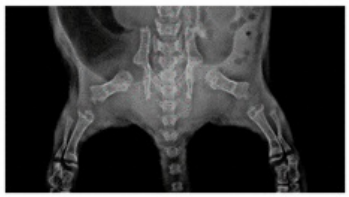
Mouse Growth Study Right Tibia Length
50 nmol/kg/day (n=9/group)

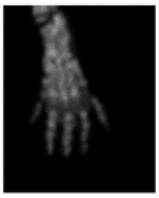
- Same amount of CNP given as continuous infusion in mice is more efficacious than daily SC injection over 35 days
- Same effect demonstrated for Ascendis' CNP peptide

[1] "Vosoritide" refers to a synthesized molecule with the same amino acid sequence prepared by Ascendis Pharma.

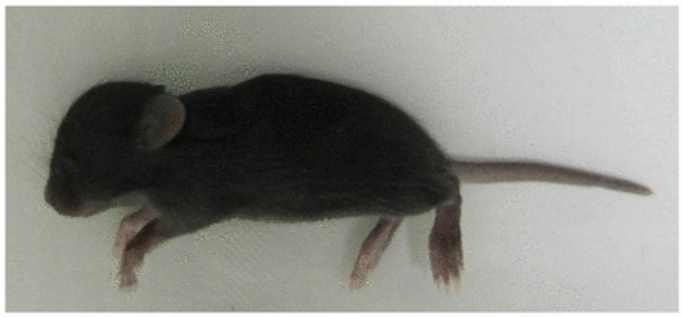
TransCon CNP Improved Phenotype of Fgfr3$^{Y367C/+}$ Mouse Model of Achondroplasia

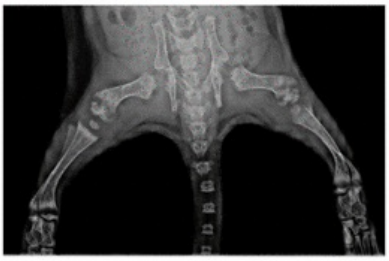
Fgfr3$^{Y367C/+}$ + Vehicle

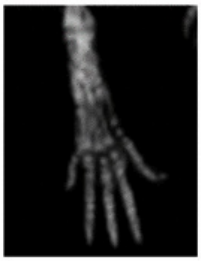




Fgfr3$^{Y367C/+}$ + TransCon CNP







In collaboration with imagine INSTITUT DES MALADIES GÉNÉTIQUES

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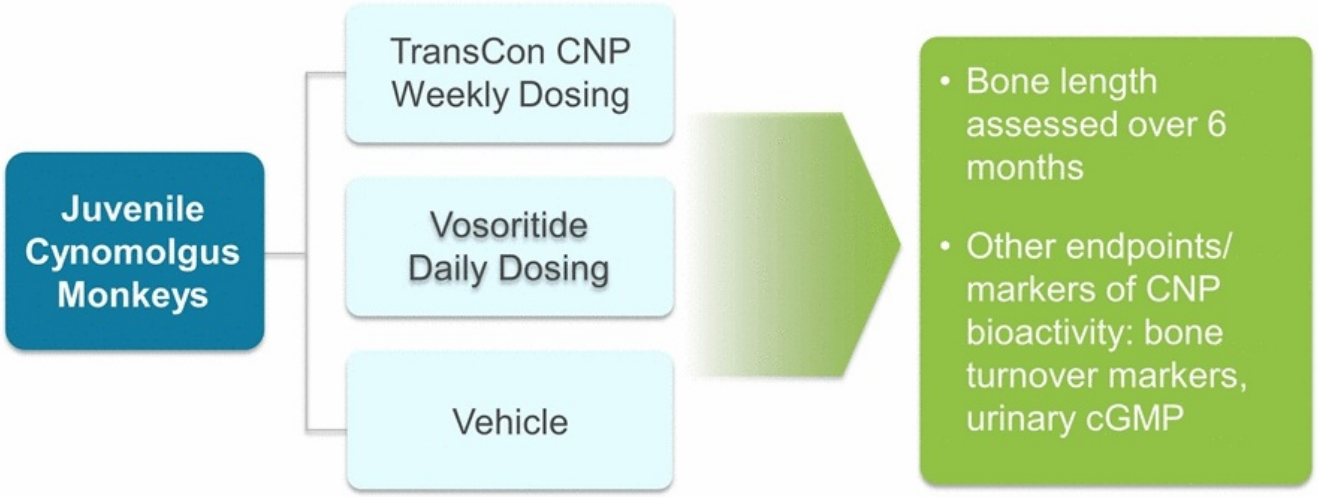
Juvenile Cynomolgus Growth Study Ongoing



- Comparing weekly TransCon CNP to a higher cumulative daily dose of vosoritide
- 6-month data expected in 1Q 2017

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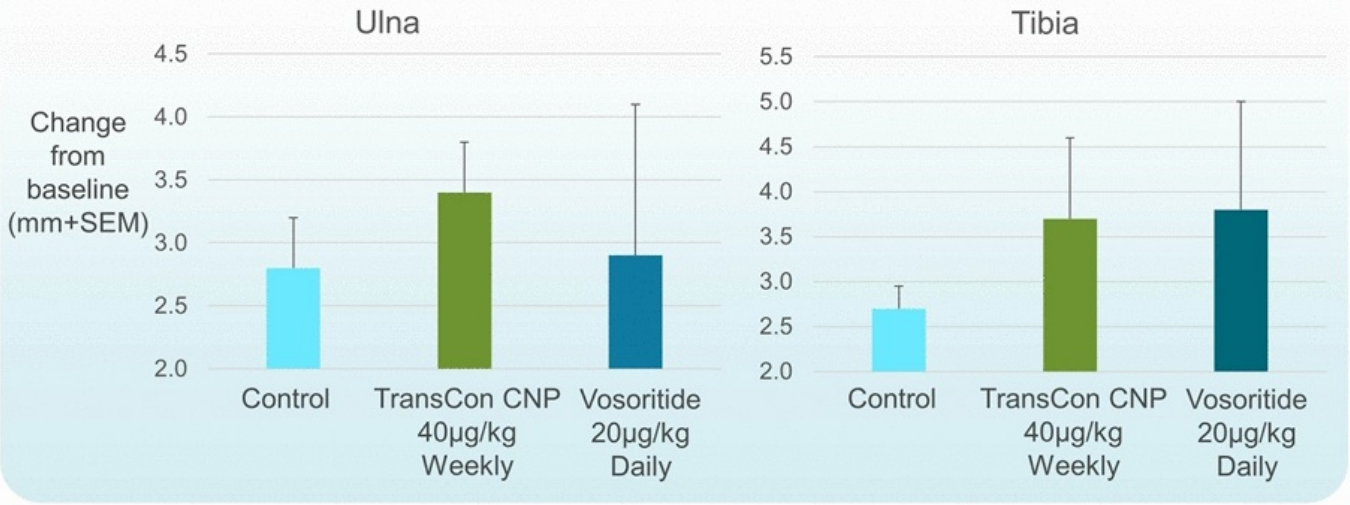
Juvenile Cynomolgus Growth Study – Interim Data[1]

Growth following SC injections in
juvenile cynomolgus monkeys at week 8 (n=4)

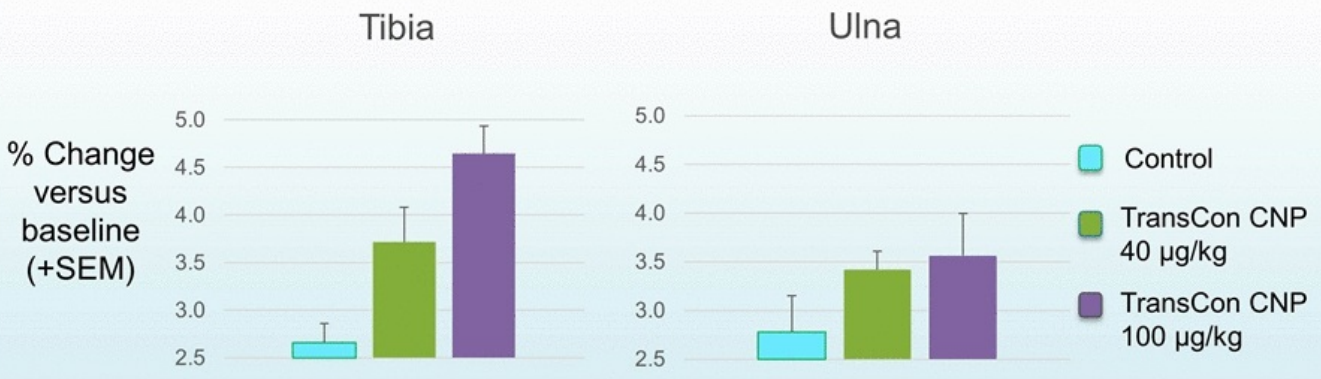


- TransCon CNP dose ~1/3 of vosoritide on a weekly basis
- Both TransCon CNP and vosoritide demonstrated a trend of bone growth over control-treated monkeys

[1] "Vosoritide" refers to a synthesized molecule with the same amino acid sequence prepared by Ascendis Pharma.

Juvenile Cynomolgus Growth Study – Dose Effect

Growth following SC injections in
juvenile cynomolgus monkeys at week 8 (n=4)

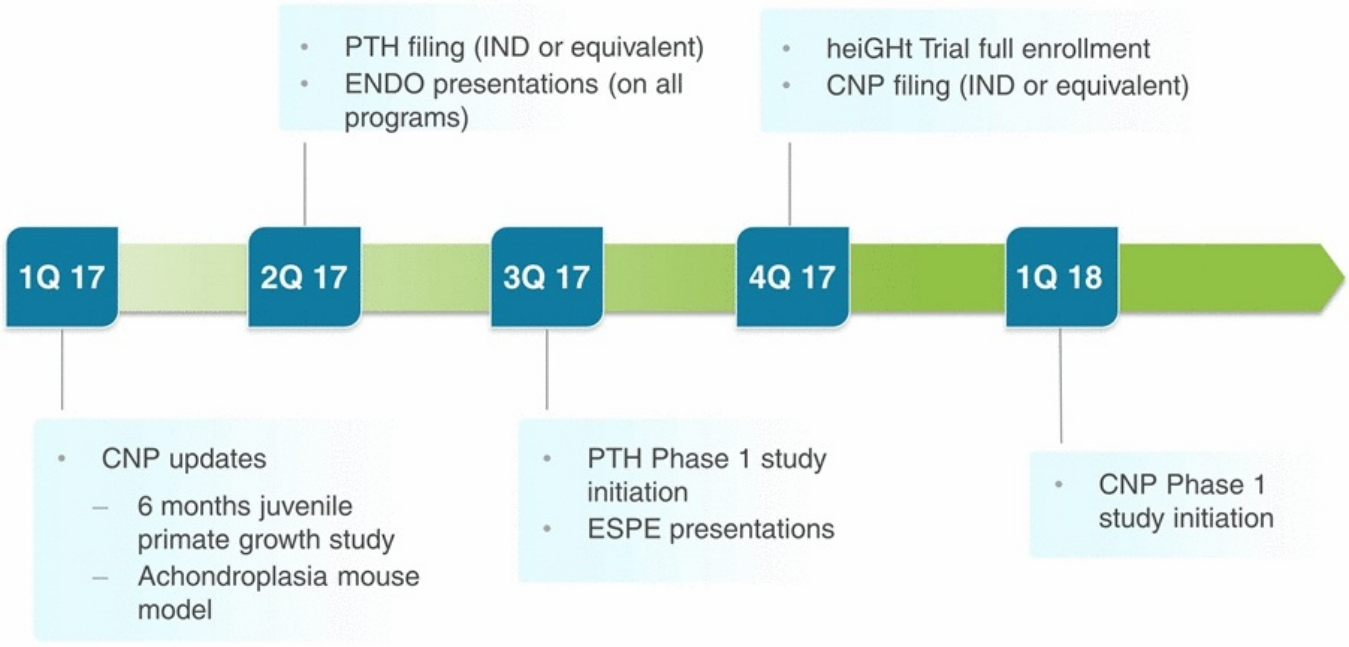


- Trend towards dose-proportional increase in growth with once-weekly TransCon CNP administration

TransCon CNP: Highlights

- TransCon CNP leverages Ascendis' technology platform to develop a once-weekly administration, without dose limiting cardiovascular adverse effects
  - Shields CNP from NPR-C receptor clearance and NPR-B induced-hypotension
  - Prolonged half-life extension and efficacy trend observed in cynomolgus monkeys
  - Reversion of phenotypical traits and comorbidities in mouse model of achondroplasia
- IND or equivalent filing expected 4Q 2017
- Phase 1 planned in healthy volunteers to establish tolerable dose range
- Multiple patent concepts provide potential protection into 2037

Selected Expected Milestones & News Drivers



Building a Leading Rare Disease Company

- Create best-in-class rare disease products addressing unmet medical needs
  - Apply TransCon technology to parent drugs with clinical proof-of-concept
  - Expect higher development success rate compared to traditional drug development
- Endocrinology rare disease wholly-owned pipeline and expected milestones
  - TransCon Growth Hormone for pediatric GHD: heiGHt Trial full enrollment Q4 2017
  - TransCon PTH for hypoparathyroidism: IND/equivalent in 2Q 2017; planning pivotal trial in 2018
  - TransCon CNP for achondroplasia: IND/equivalent in 4Q 2017
- Commercial focus on the U.S. market with multi-billion dollar revenue potential
- Established high-value collaborations with Roche/Genentech in ophthalmology and Sanofi in diabetes
- Pro forma September 30, 2016 cash of ~ €190 million[1]

[1] Based on cash and cash equivalents as of September 30, 2016 and approximately $127.4 million in estimated net proceeds from the public offering of our ADSs that was completed in 4Q 2016.