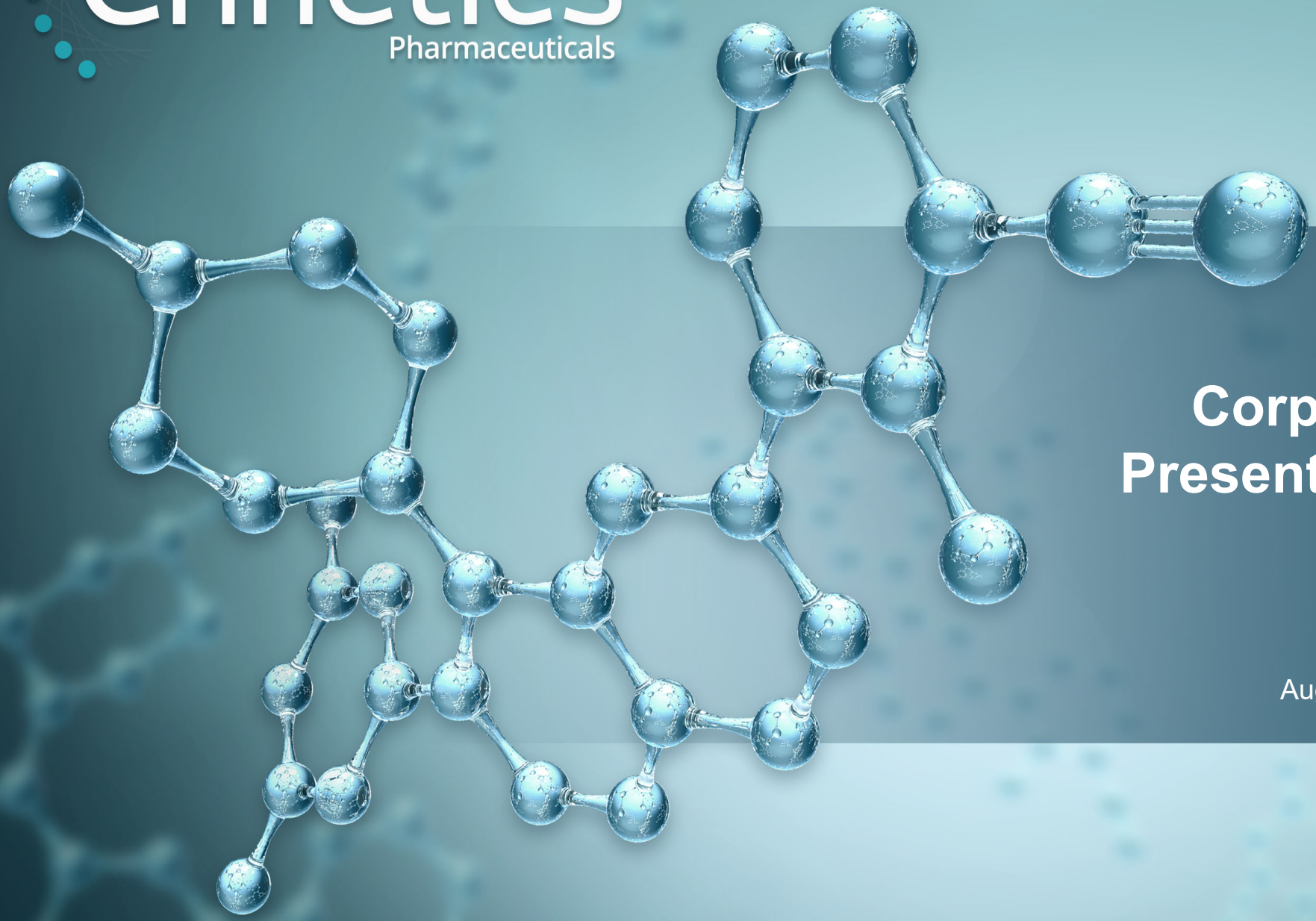
Crinetics Pharmaceuticals



Corporate Presentation

August 2020

SAFE HARBOR STATEMENT

This presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation are forward-looking statements, including statements regarding: the potential for interim results to be consistent with final results, once available; the potential for any of our ongoing clinical trials to demonstrate safety or efficacy; the potential benefits of paltusotine for acromegaly patients; the potential to initiate a pivotal Phase 3 trial of paltusotine in acromegaly based on interim results to date and the timing thereof; the planned expansion of the paltusotine development program to include the treatment of patients with NETs and the expected timing thereof; the anticipated timing of topline data for Edge and PK/PD data for its other development programs and initiation of trials thereafter; the potential benefits of our ACTH agonist in patients across multiple indications and the expected timing of the advancement of such program; the potential benefits of our SST5 agonist in patients with congenital hyperinsulinism and the expected timing of the advancement of such program; and the company's anticipated cash runway. In some cases, you can identify forward-looking statements by terms such as "may," "believe," "anticipate," "could," "should," "estimate," "expect," "intend," "plan," "project," "will," "forecast" and similar terms. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements, including, without limitation: the risk that interim results of a clinical trial do not necessarily predict final results and that one or more of the clinical outcomes may materially change as patient enrollment continues, following more comprehensive reviews of the data, and as more patient data become available; potential delays in the commencement, enrollment and completion of clinical trials and the reporting of data therefrom; the COVID-19 pandemic may disrupt our business and that of the third parties on which we depend, including delaying or otherwise disrupting our clinical trials and preclinical studies, manufacturing and supply chain, or impairing employee productivity; our dependence on third parties in connection with product manufacturing, research and preclinical and clinical testing; the success of our clinical trials and nonclinical studies for paltusotine and our other product candidates; regulatory developments in the United States and foreign countries; unexpected adverse side effects or inadequate efficacy of our product candidates that may limit their development, regulatory approval and/or commercialization; we may use our capital resources sooner than we expect; and other risks described under the heading "Risk Factors" in documents we file from time to time with the Securities and Exchange Commission. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995 and, except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

OUR VISION

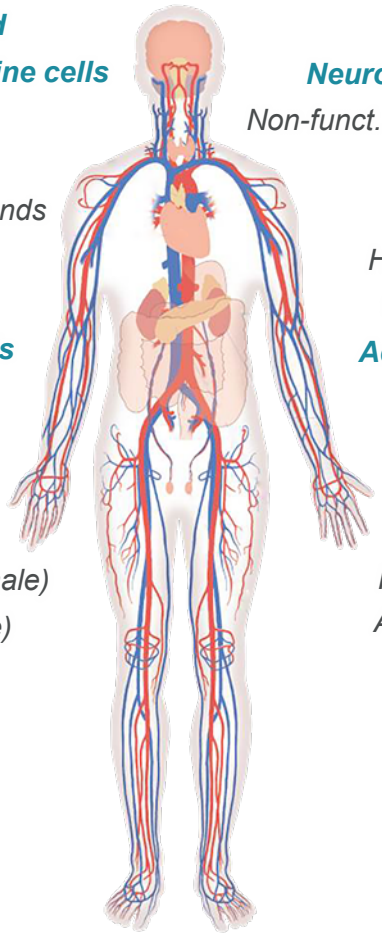
To build the leading endocrine company that consistently pioneers new therapeutics to help patients better control their disease and improve their daily lives

Our strategy: Discover, develop and commercialize across multiple rare endocrine diseases and endocrine-related tumors

- Ongoing in-house discovery of novel drug-candidates
- Focus on endocrine diseases and related tumors with:
  - High unmet medical need
  - Established biology
  - Biomarker endpoints
  - POC in Phase 1
  - Small registration trials
- Advance clinical pipeline of multiple drug candidates in parallel
- Retain commercialization rights in core therapeutic areas and regions
- Nurture an entrepreneurial, scientifically rigorous, collaborative and inclusive company culture

The endocrine therapeutic area

Endocrine system:	Multiple indications:
Pituitary gland	***Acromegaly***
Enteroendocrine cells	***Neuroendocrine tumors***
Hypothalamus	*Non-funct. pituitary adenomas*
Pineal gland	*GH deficiency*
Parathyroid glands	*Graves' disease*
Thyroid gland	*Hyperparathyroidism*
Thymus	***Cushing's disease***
Adrenal glands	***Adrenal hyperplasia***
Kidneys	*Adrenal cancer*
Pancreas	***Hyperinsulinism***
Liver	*Insulinoma*
Placenta	*Thyroid cancer*
Ovaries (in female)	*Hypoparathyroidism*
Testes (in male)	*Androgen deficiency*
	Infertility



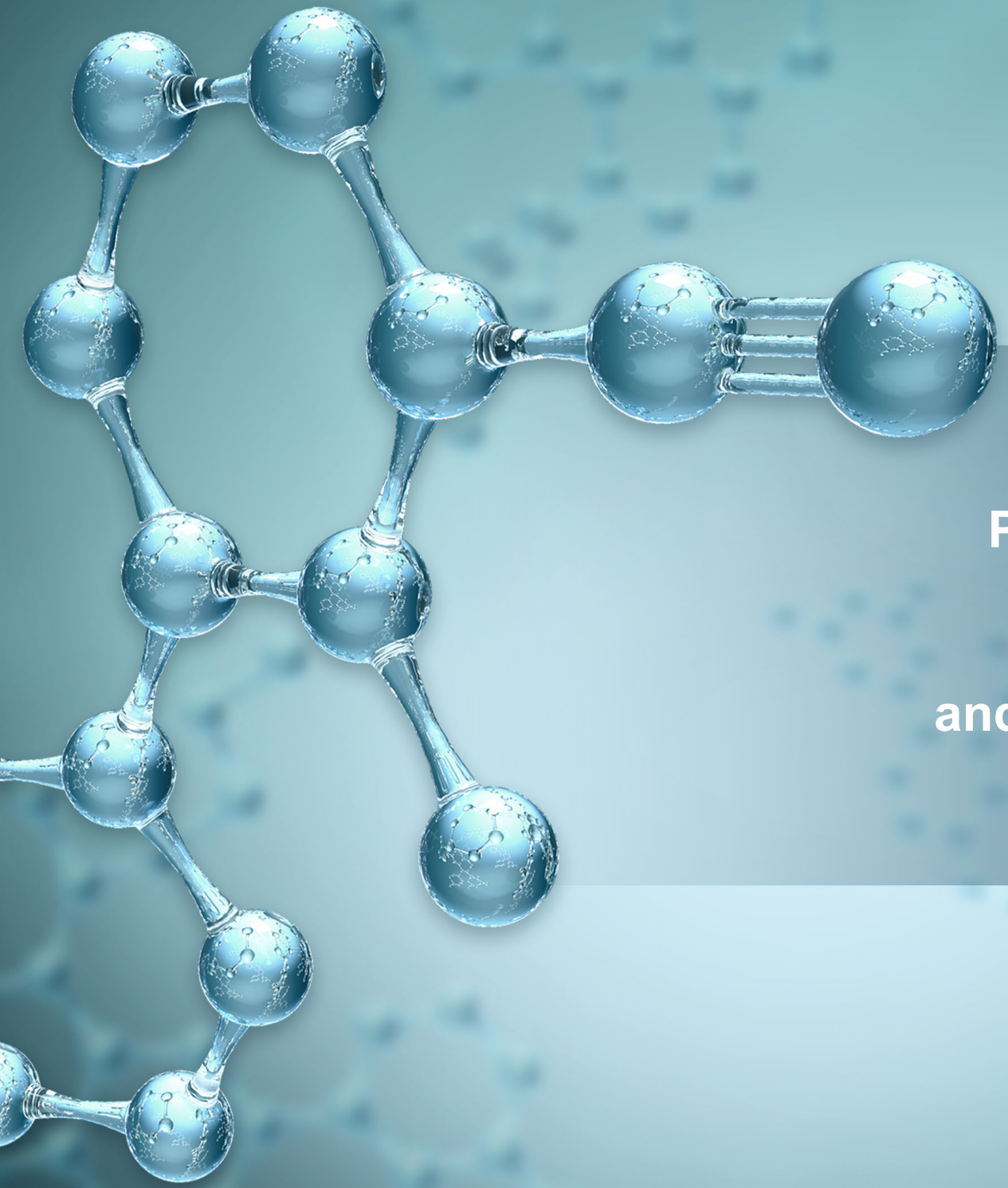
Targeting today */ Future opportunity*

Pipeline

Building a rare disease franchise in endocrinology and endocrine oncology

PROGRAM	DISCOVERY	PRECLIN	PHASE 1	PHASE 2	PHASE 3	NEXT MILESTONE
Paltusotine Acromegaly						Topline data from Phase 2 acromegaly trials in 4Q 2020 Initiate acromegaly Phase 3 trial in 1H 2021
Neuroendocrine Tumors (NETs)						Initiate NETS Phase 2 trial in 2021
Oral ACTH Antagonist Cushing's Disease, Congenital Adrenal Hyperplasia						Initiate Phase 1 trial late 2020/early 2021
Oral SST5 Agonist Hyperinsulinism						Initiate Phase 1 trial late 2020/early 2021

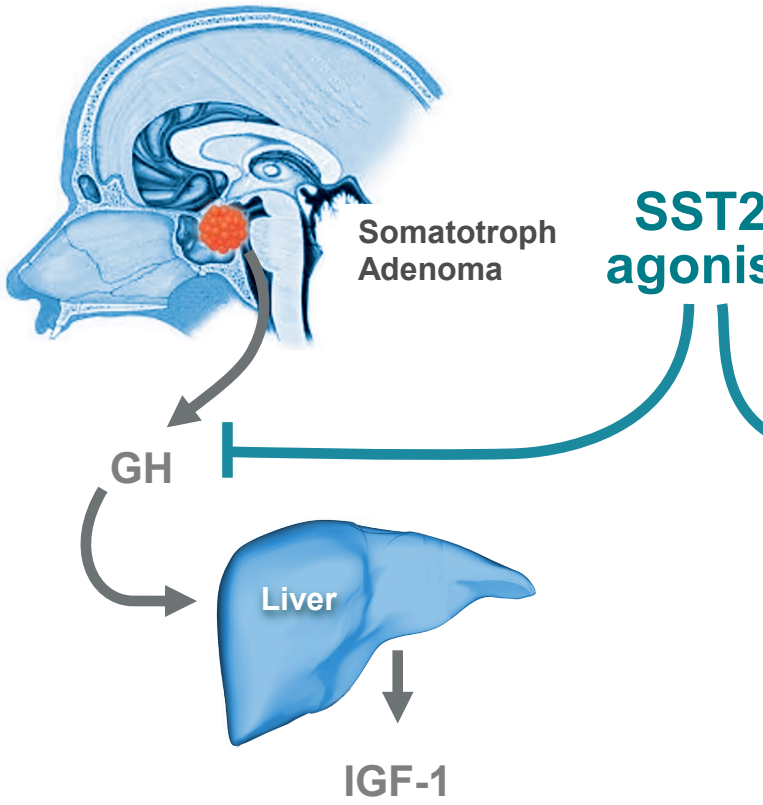
All product candidates discovered and developed internally
Global rights retained and no licensing obligations
Composition of matter for paltusotine through 2037



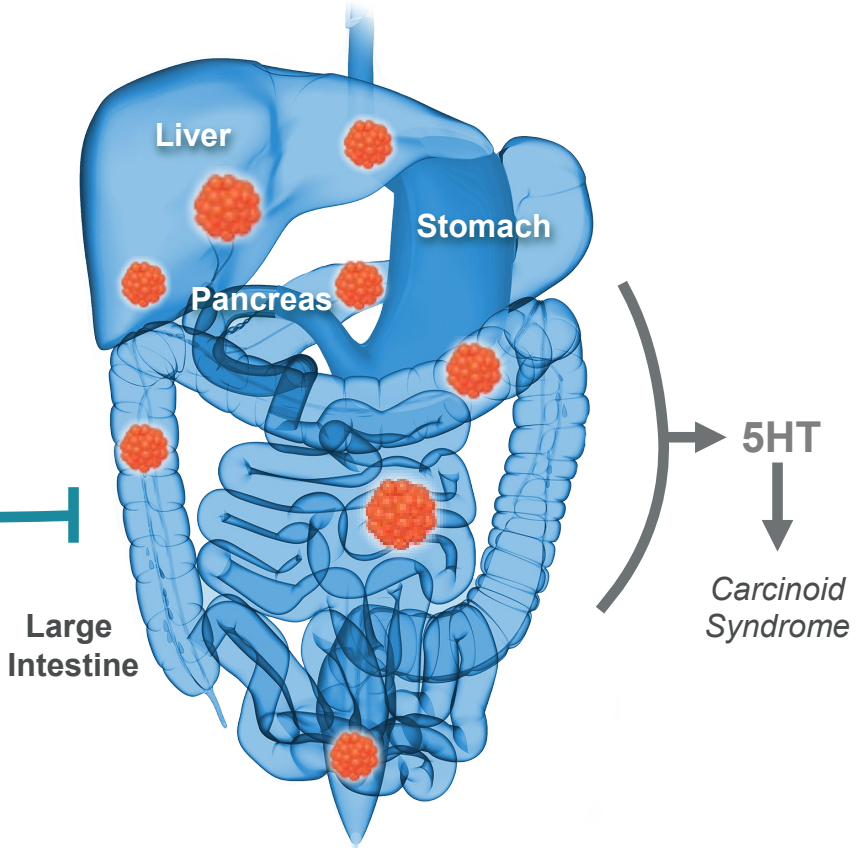
Paltusotine: a nonpeptide SST2 agonist for the treatment of acromegaly and neuroendocrine tumors

Somatostatin receptor type 2 (SST2) peptide agonists are the standard of care for acromegaly and many NETs

Acromegaly



Neuroendorine Tumors (NETs)



Prevalence: more than 25,000 people with acromegaly in the U.S.

Prevalence: ~171,000 people with NETs in the U.S.

Established commercial opportunity for injectable somatostatin peptides despite significant limitations

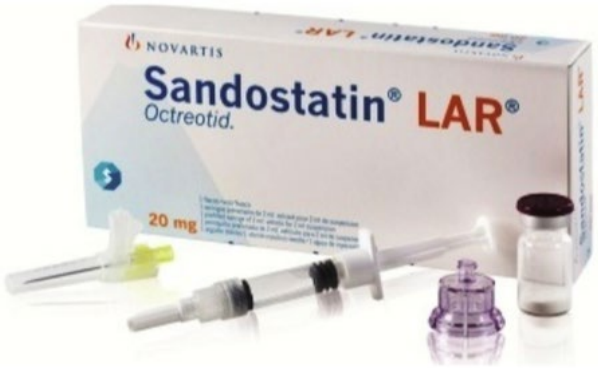
2019: $3.1 billion in global sales

RECORDATI
Signifor®
(pasireotide)
$75M

Pfizer
Somavert®
(pegvisomant)
$264M

IPSEN
Innovation for patient care
Somatuline®
(lanreotide)
$1.2B
(Ipsen #1 drug)

NOVARTIS
Sandostatin®
(octreotide)
$1.6B
(Novartis #6 drug)



High unmet medical need

Daily injections

- Patients buy a second refrigerator for storage
- Travel is difficult

Painful intramuscular/deep sc injections every month (octreotide, lanreotide)

- Hardness, bruising and swelling at injection site

Inconvenient

- Monthly visits to physician's office interrupts normal life

Limited efficacy

- Many patients do not achieve disease control
- Return of symptoms near end of the month

Sources: Company earnings and equity research analyst reports
[1] Includes acromegaly, neuroendocrine tumors and other uses

For most patients, acromegaly is not a solved problem

25-50% of IM injections are unsuccessful

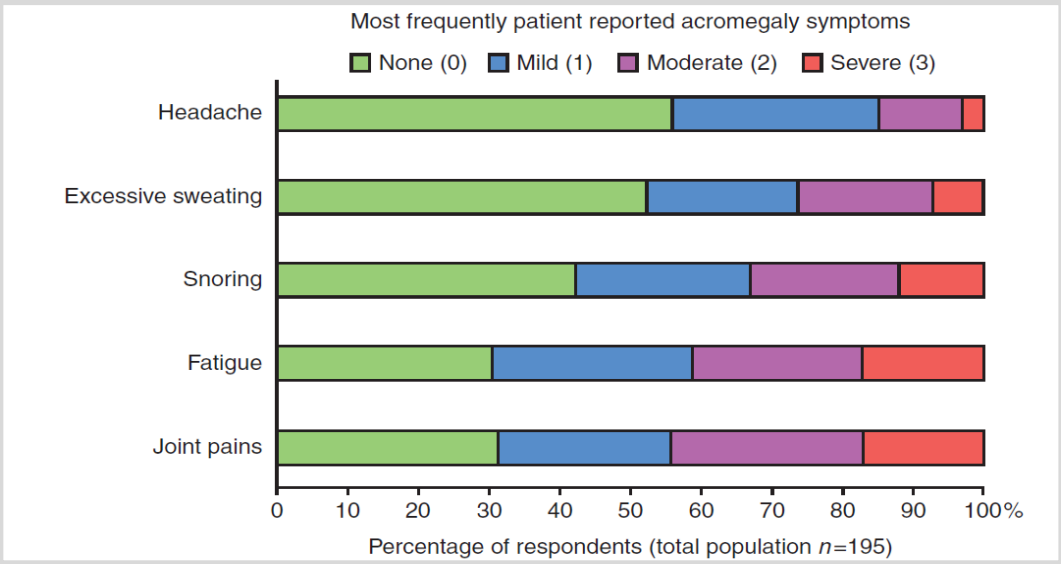
Pancreas. 2013 Jul;42(5):878-82. doi: 10.1097/MPA.0b013e318279d552.

Improving the success rate of gluteal intramuscular injections.

Boyd AE[1], DeFord LL, Mares JE, Leary CC, Garris JL, Dagohoy CG, Boving VG, Brook JP, Phan A, Yao JC.

Results: At baseline 52% of injections were successfully delivered…
After instruction, the success rate increase from 52 to 75%...
...Successful injection was associated with better control of flushing among those with carcinoid syndrome (P=0.005).

>70% of treated patients have symptoms



Strasburger et al. Eur J Endo. 2016, 174, 355-362; Boyd et al. Pancreas 2013; 42: 878-82

What patients are telling us

- "Not a day goes by when I 'feel good'"
- "Mood disturbances, I would say *<are the most frequent symptom>*. Or inability to find the joy, I guess. You know, I start to have a difficult time processing emotions right before I get my injections."
- "The doctor didn't warn me about this."
- "I had to schedule it [*injection*] sometimes after 3:30 because where I teach and where I was getting the injection was almost 40 minutes away. I had to make an appointment and my principal would let me leave early."
- "Don't plan on going someplace the next day because you may end up with diarrhea - I had diarrhea for three solid days. You don't go anywhere. I got it [*injection*] last night and the hip that she gave it in hurts so bad."

Paltusotine (CRN00808) – Target product candidate profile

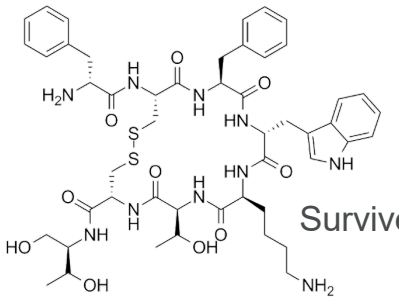
- A new class of oral selective nonpeptide SST2 biased agonist designed for the treatment of acromegaly
- First agent in its class with reported clinical results

POTENTIAL ATTRIBUTES	POTENTIAL BENEFITS
Orally bioavailable nonpeptide (small molecule)	Lack of injections/pain Administration at home Rapid dose optimization Consistent exposure over time Lower COGS and admin costs
Long half life (42-50 hrs.) observed in Phase 1 study	Once daily dosing
Reduced desensitization of SST receptors observed in preclinical studies	Potential for improved responder rates
Selectivity for SST2	Glucose control (avoid SST5 mediated hyperglycemia)

PRODUCT CANDIDATE DESIGNED TO DELIVER KEY BENEFITS

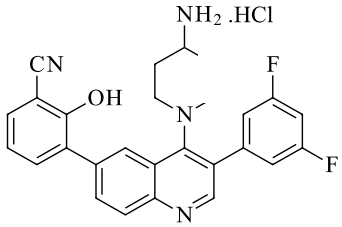
Paltusotine is designed to be intrinsically gut permeable just like other traditional oral small molecule drugs

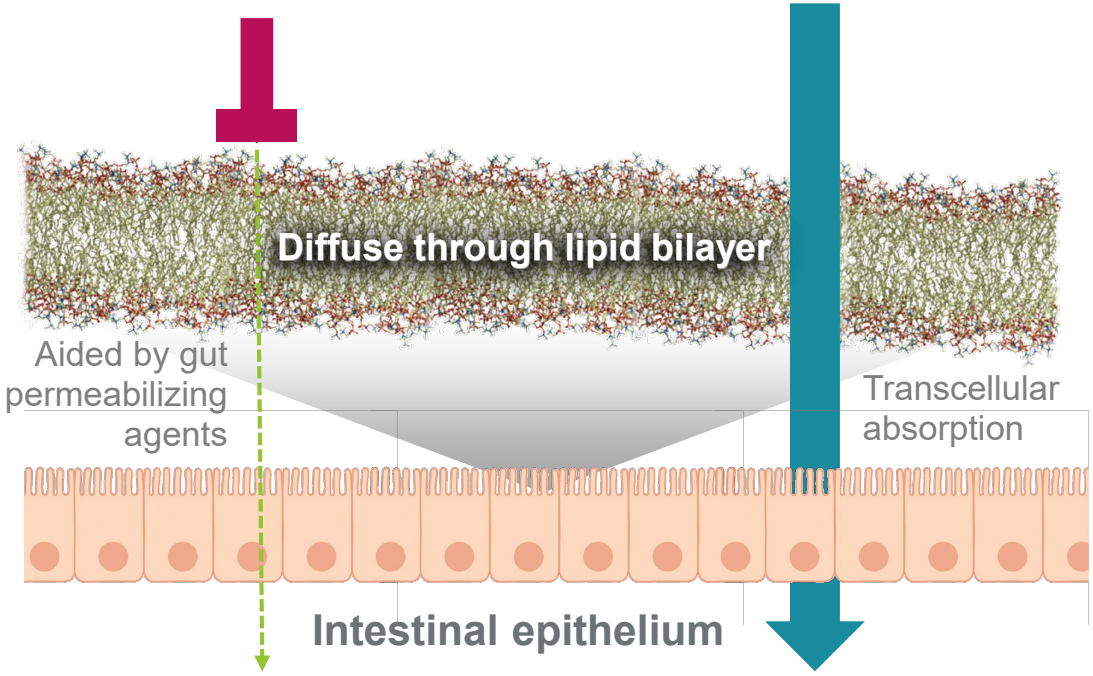
Peptide: Octreotide

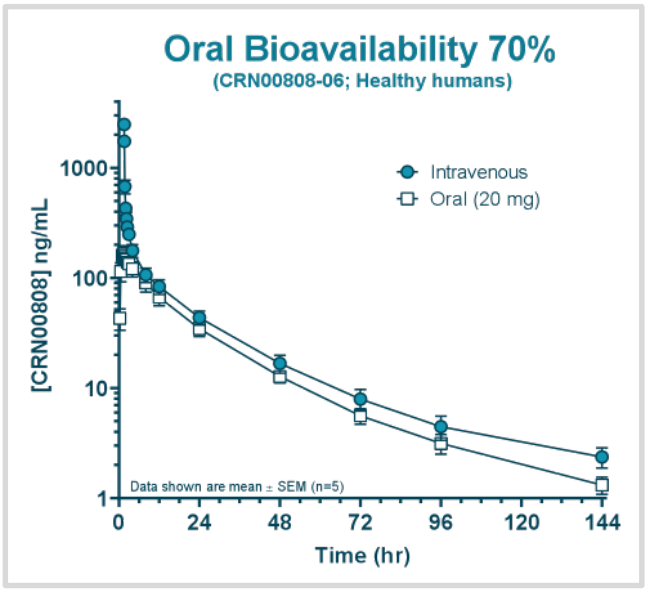


Survive proteolysis

Nonpeptide: paltusotine







	Paltusotine (CRN00808)
Estimated Oral Bioavailability	70%
Observed Half Life	42-50 hr.

Acromegaly clinical development strategy: Core studies so far

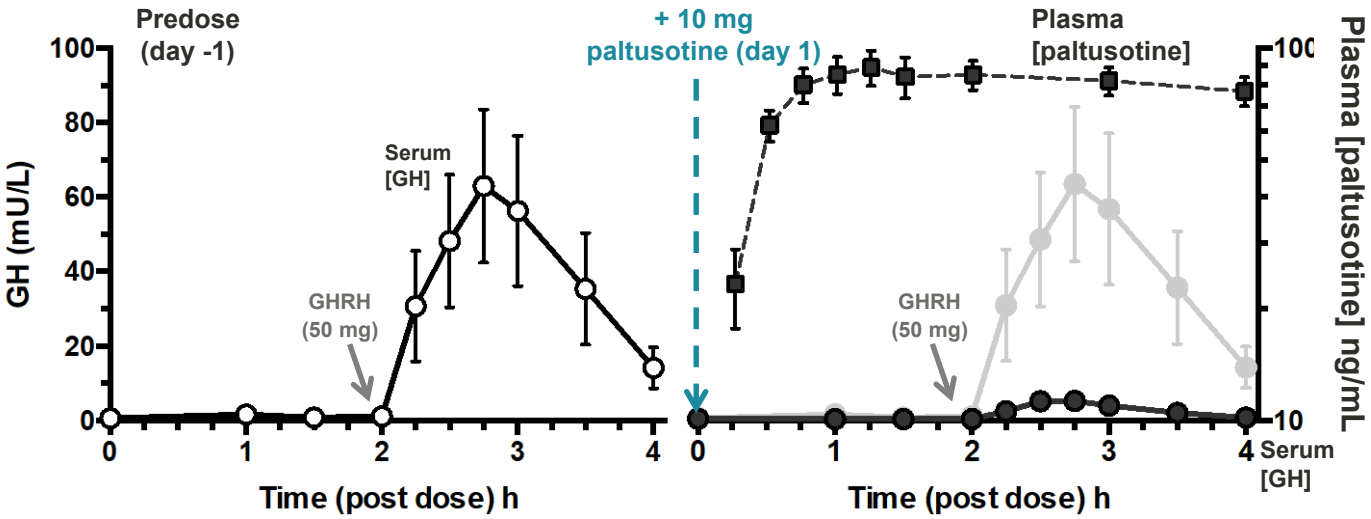
Phase 1 FIM	Healthy Volunteers	Proof-of-concept Define PK/PD Preliminary safety	QD dosing GH/IGF suppression = peptide SSAs
ACROBAT EDGE	Patients not fully controlled on oct/lan monotherapy (>65% of patients)	Can patients switch to oral and maintain IGF control? Demonstrate efficacy vs washout	IGF control on oral = peptide SSAs Treated IGF < washout IGF
ACROBAT EVOLVE	Patients fully controlled on oct/lan monotherapy (20-30% of patients)(1)	Can patients switch to oral and maintain IGF control? Demonstrate efficacy vs randomized withdrawal	Responder rate > pbo
ACROBAT ADVANCE	Edge & Evolve patients	Long-term patient experience for NDA submission	Durable safety and IGF suppression

(1) New enrollment in the Evolve study has been discontinued. The 12 patients enrolled will continue in the study.

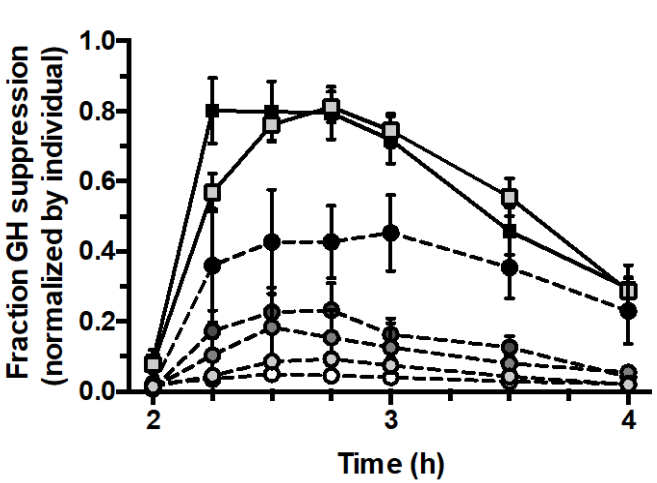
Colao et al, J Clin Endo Metab (2013); Strasburger et al, Eu J Endo (2016); Ezzat et al, Annals of Internal Medicine (1992)

Phase 1 SAD arm: PK/PD analysis

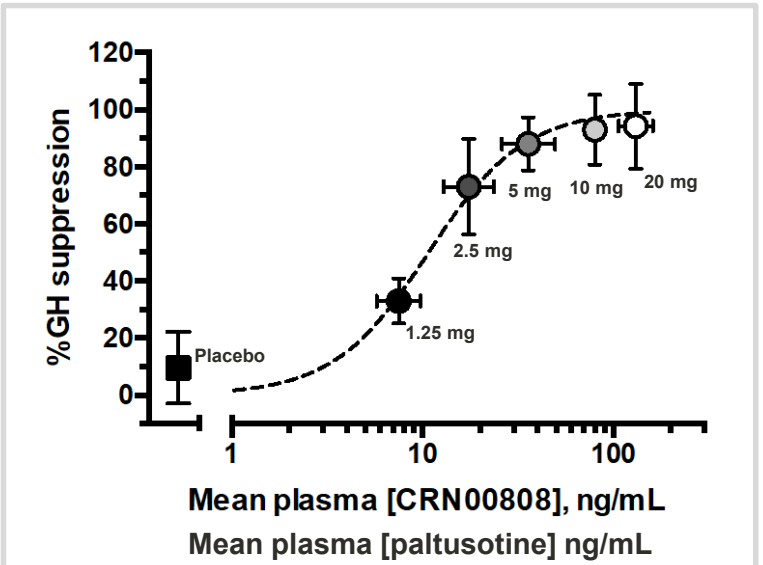
Suppression of GHRH stimulated GH secretion by 10 mg of paltusotine



Dose response of GH suppression

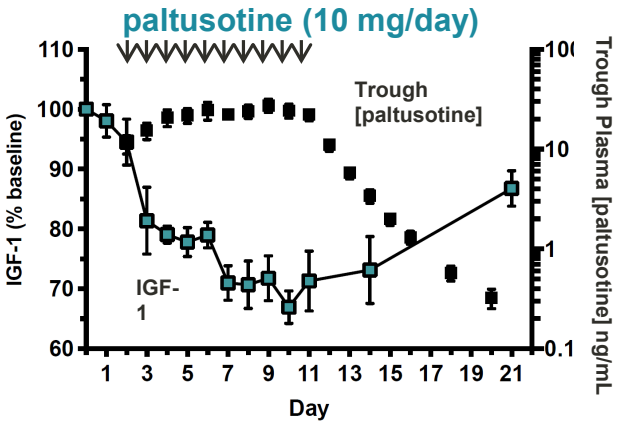


Exposure response of GH suppression

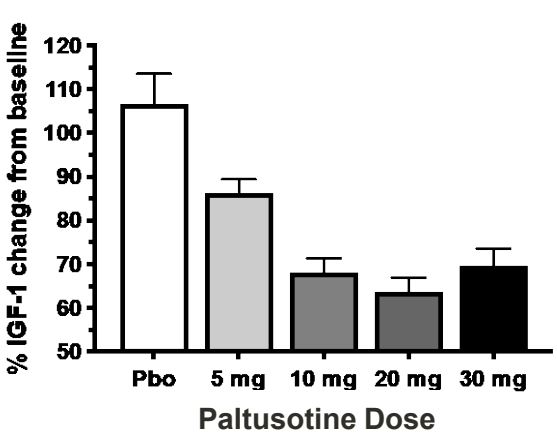


Phase 1 MAD arm: PK/PD analysis

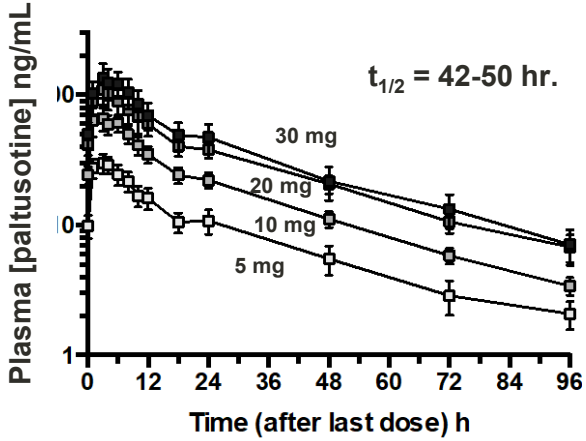
Time-course of plasma paltusotine trough and IGF-1 concentrations



Dose response of IGF-1 suppression



Plasma concentration on the last day of dosing

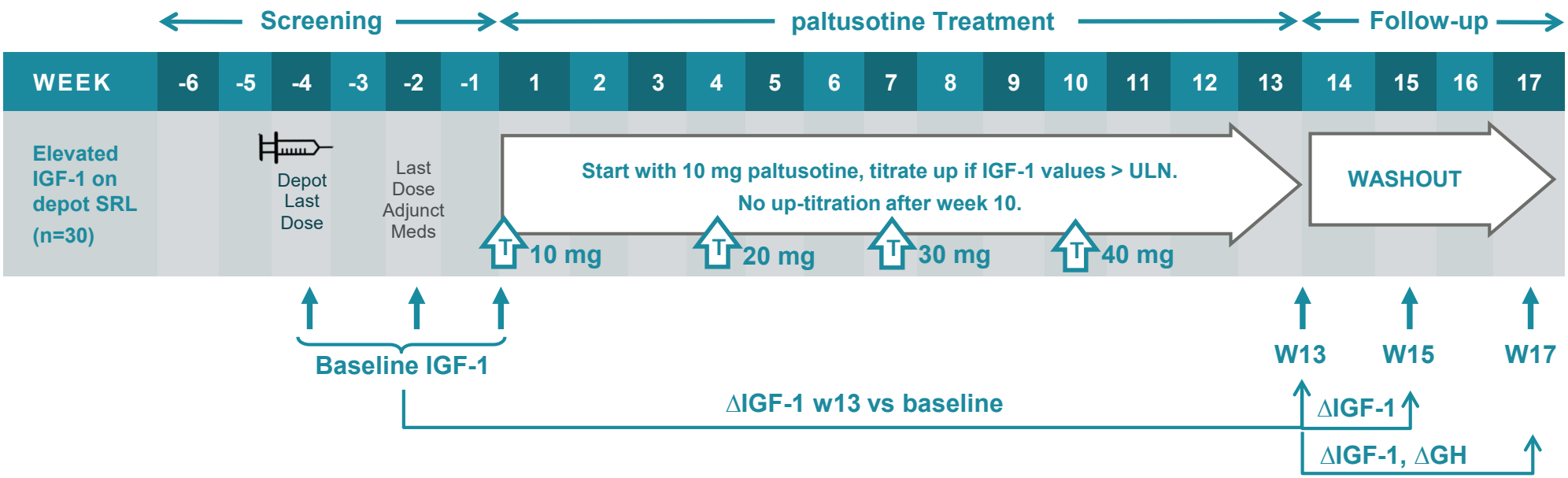


Safety and tolerability results observed in phase 1

- Tolerability and AE profile was generally consistent with approved peptide somatostatin analogs (abdominal pain, flatulence, abdominal distension, and diarrhea in approximately 30% of subjects and mild elevations of pancreatic enzymes in approximately 10% of subjects)
- Additional adverse events included: headache, dizziness and cardiac rhythm abnormalities (including NSVT). One serious adverse event of moderate NSVT was observed following a single 1.25 mg dose and was considered unlikely to be related to paltusotine. These AEs were not dose dependent and were also observed in placebo subjects and/or prior to dosing.

10 mg selected as the initial dose in Phase 2 trials

ACROBAT Edge design



Group	Patient Groups	IGF-1 range	# of Patients
1	Octreotide LAR or lanreotide depot	> 1.0x ULN to ≤ 2.5x ULN	At least 30
2	Dopamine agonist + octreotide LAR or lanreotide depot	> 1.0x ULN to ≤ 2.5x ULN	
3	Dopamine agonist + octreotide LAR or lanreotide depot	≤ 1.0x ULN	Max 15
4	Pasireotide LAR	≤ 1.0x ULN	
5	Pegvisomant + octreotide LAR or lanreotide depot	≤ 1.0x ULN	

Key inclusion/rescue criteria

- Patients on stable approved monthly dose of SRL for at least 3 mo.
- 18 to 75 years of age
- Criteria for rescue with standard acromegaly medication: significant worsening of acromegaly symptoms

Edge interim data as of February 23, 2020

Data available for interim analysis

- As of February 23, 2020, 32 patients have enrolled in the study and either completed participation, or continue to receive paltusotine
- 17 patients have completed participation in the study.
  - 13 of these initial patients were treated with lanreotide depot (n=8) or octreotide LAR (n=5) monotherapy and entered the trial with IGF-1 above the upper limit of normal (Group 1)
  - 10 of the 11 (91%) patients in group 1 who completed paltusotine treatment maintained IGF-1 levels within 15% of their respective baseline levels at week 13
- Efficacy Evaluation Set (n=13)
  - All available data from Group 1 patients who completed participation in the trial as of February 23, 2020
- Safety Evaluation Set (n=32)
  - All patients dosed in all subgroups as of February 23, 2020 (n=32) including those that had not yet completed the study

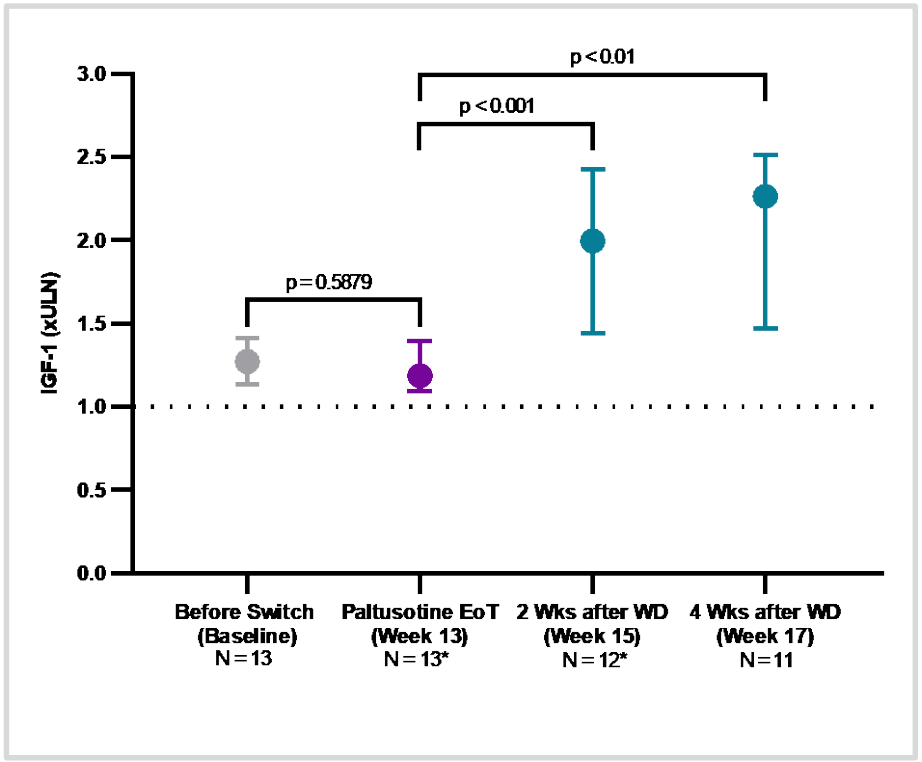
Patient baseline characteristics

	Group 1 (N=13)	Total (N=32)
Median Age, years (Min, Max)	53.0 (34, 68)	51.5 (34, 70)
Sex		
Female	7 (53.8%)	19 (59.4%)
Male	6 (46.2%)	13 (40.6%)
Ethnicity		
Hispanic or Latino	0	6 (18.8%)
Not Hispanic or Latino	13 (100%)	26 (81.3%)
Race		
White	13 (100%)	29 (90.6%)
Black or African American	0	1 (3.1%)
Other	0	2 (6.3%)
Median Weight, kg (Min, Max)	97.9 (63, 155)	89.3 (57.3, 155)

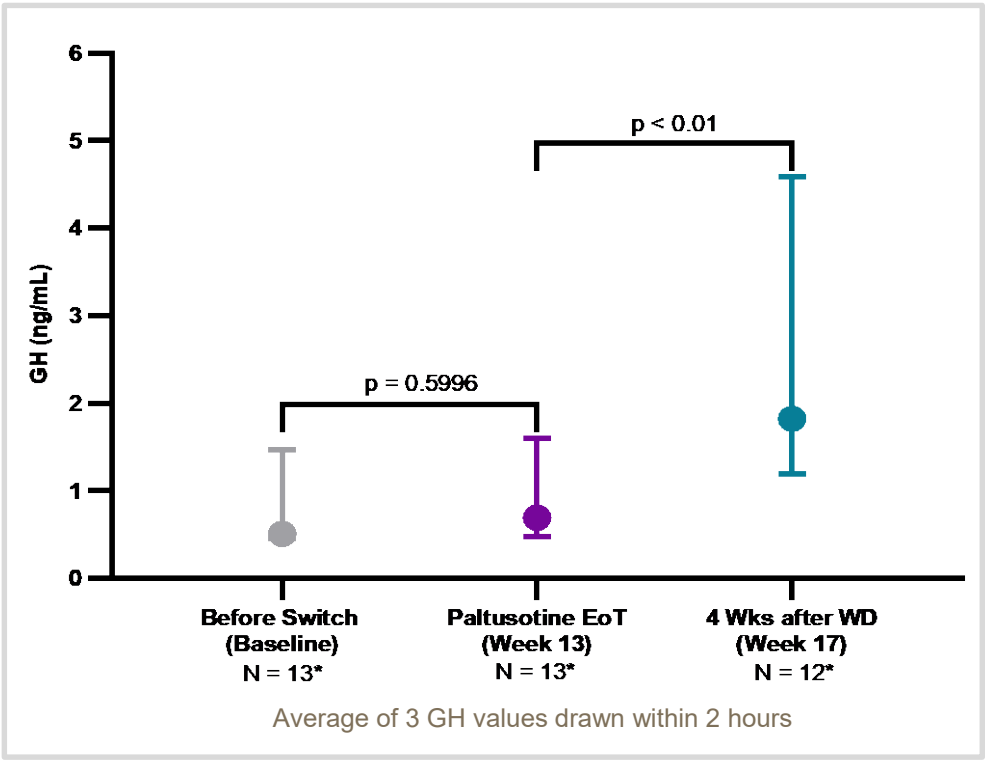
Paltusotine maintained baseline IGF-1 and integrated GH levels 13 weeks after switching from injected depot therapy

Both IGF-1 and GH levels promptly rose after withdrawal of paltusotine

Serum IGF-1 changes at end of treatment and after withdrawal of paltusotine



Serum GH changes at end of treatment and after withdrawal of paltusotine



Data shown are median (25th percentile, 75th percentile) for Group 1 patients

- *Data includes two early termination (ET) patients who discontinued for non-study drug related reasons: (1) use of prohibited concomitant medication and (2) inability to complete study visits. Their final treatment values were used for EoT. The 2 ET patients had 16 days (~2 weeks) and 23 days (~3 weeks) of paltusotine exposure. One ET patient had final assessment 2 weeks after EoT (GH data from this subject was included in the 4 wk GH WD data), the washout data for the other was not available
- p-values are based on non-parametric Wilcoxon Sign Rank test of whether the median change is different from zero.
- EoT=End of Treatment; WD=Withdrawal

Exploratory interim statistical analysis of Group 1 patients

Interim Efficacy Results

1. IGF-1 and GH levels after 13 weeks of paltusotine treatment were not different than baseline (while treated with SRL depot)
2. IGF-1 rose significantly within 2 weeks of paltusotine withdrawal. GH hormone also rose significantly (GH measured only 4 weeks after withdrawal)

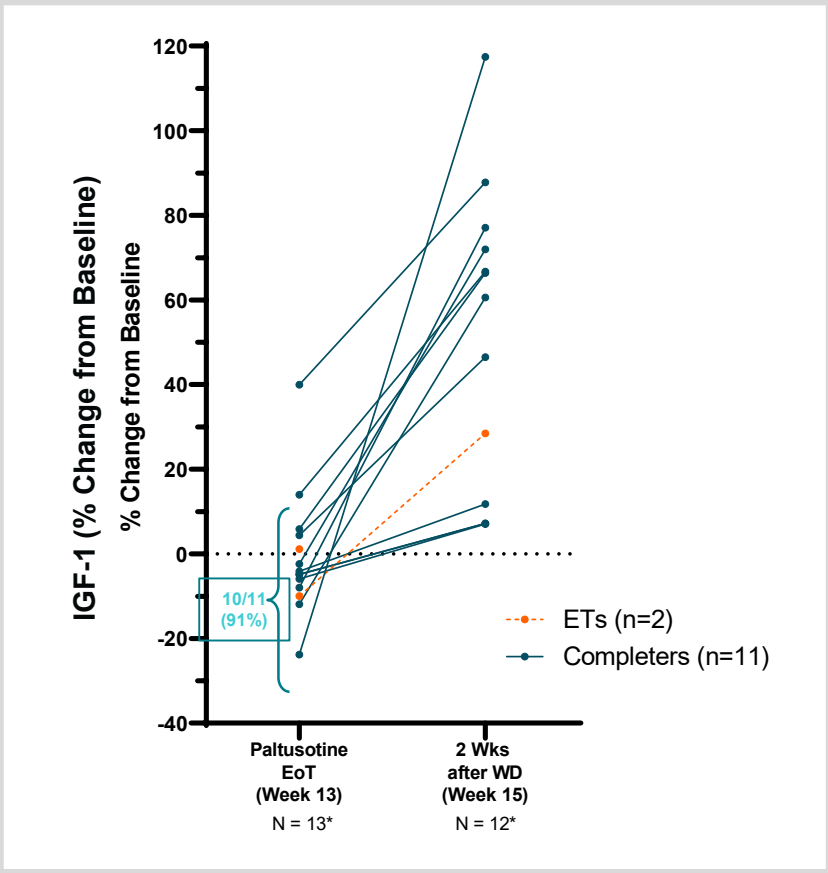
Parameter (units)	Change in Serum Hormone Levels		
	Change from Baseline to EoT (Week 13)	Change from EoT to Post Withdrawal Visit 2 Weeks	4 Weeks
IGF-1 (×ULN)	**N=13**	**N=12**	**N=11**
Mean (95% CI)	-0.015 (-0.123, 0.092)	0.739 (0.394, 1.083)	0.767 (0.379, 1.155)
Median (25th, 75th percentiles)	-0.047 (-0.102, 0.050)	0.574 (0.297, 1.083)	0.782 (0.371, 1.250)
p-value^	0.5879	< 0.001	< 0.01
GH (ng/mL)	**N=13**	**Not Measured**	**N=12**
Mean (95% CI)	0.054 (-0.285, 0.394)		1.612 (0.452, 2.772)
Median (25th, 75th percentiles)	0.011 (-0.100, 0.240)		0.891 (0.358, 2.716)
p-value^	0.5996		< 0.01

^p-values are based on non-parametric Wilcoxon Sign Rank test
EoT=End of Treatment;

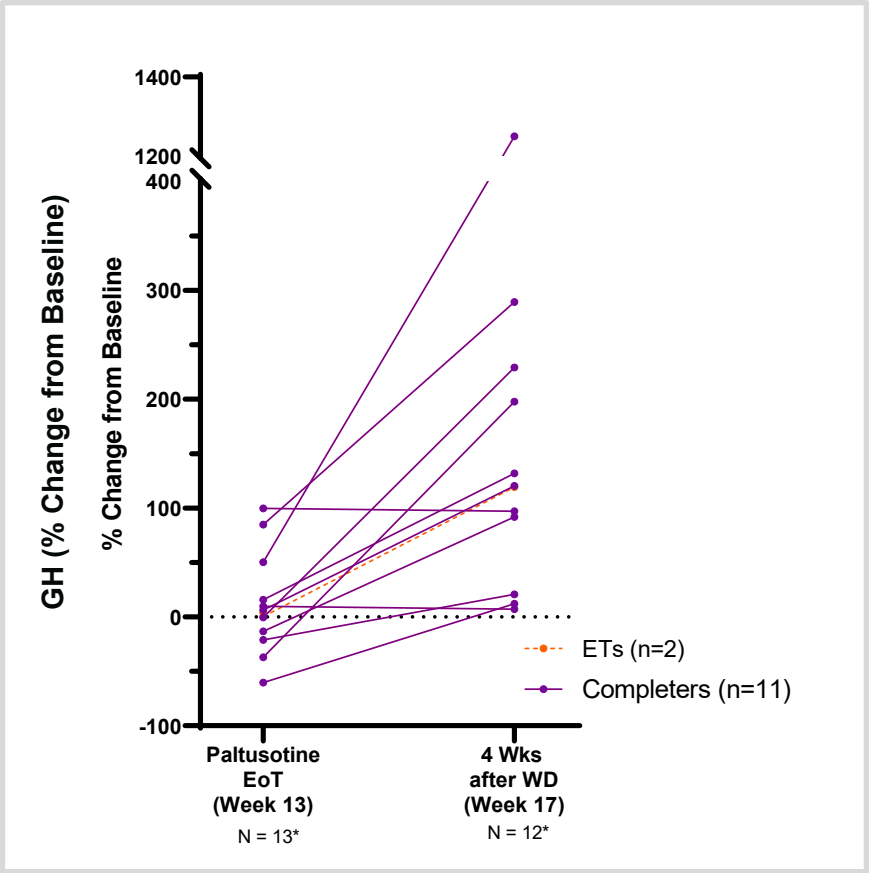
Switching to once daily oral paltusotine from injected depot SRLs maintained IGF-1 levels in > 90% of Group 1 completers

IFG-1 and GH level changes after withdrawal are consistent with the approximately 2-day half-life previously measured in phase 1 trial

Individual IGF-1 changes at end of treatment and 2 weeks after withdrawal of paltusotine



Individual GH changes at end of treatment and 4 weeks after withdrawal of paltusotine



- EoT=End of Treatment; WD=Withdrawal
- *Data includes two early termination (ET) patients who discontinued for non-study drug related reasons and had 16 days (~2 weeks) and 23 days (~3 weeks) of paltusotine exposure. 1 ET patient had final assessment 2 weeks after EoT (GH data from this subject was included in the 4 wk GH WD data), the washout data for the other was not available. These were not included in calculation of 91% who maintained IGF-1 levels within 15% of baseline values.
- Pre-trial therapy SRL median concentrations at W13 compared to baseline: [Lanreotide] ↓ by 70%; [Octreotide] ↓ by 100%; paltusotine median concentrations at W15 compared to W13 (last dose) were decreased by >99.9%

Paltusotine was well tolerated

Safety data in all patients dosed with paltusotine as of February 23, 2020 (n=32) in Edge

Adverse events on treatment regardless of causality in 2 or more patients

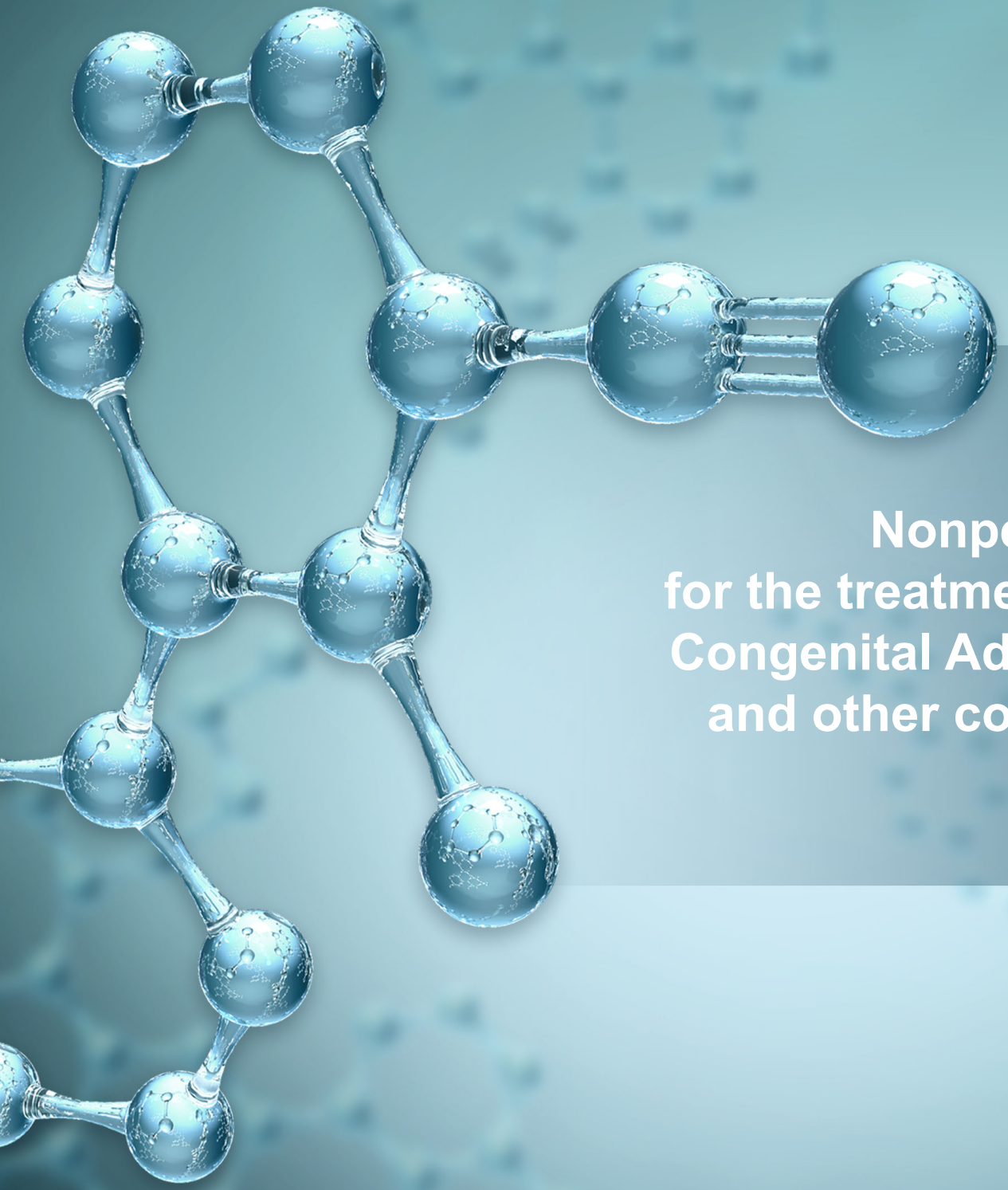
Preferred Term	Group 1 (N=13) n (%)	Total (N=32) n (%)
Number (%) of Patients with any TEAEs	9 (69%)	14 (44%)
Arthralgia	3 (23%)	6 (19%)
Headache	4 (31%)	5 (16%)
Abdominal discomfort	1 (8%)	3 (9%)
Peripheral swelling	2 (15%)	3 (9%)
Back pain	2 (15%)	2 (6%)
Diarrhoea	0	2 (6%)
Flatulence	0	2 (6%)
Hyperhidrosis	2 (15%)	2 (6%)
Palpitations	1 (8%)	2 (6%)

- No discontinuations due to adverse events
- No patients have required “rescue treatments” with standard acromegaly medications
- 1 SAE--Headache--non-treatment related (admission for diagnostic evaluation)
- No safety signals as of the interim data cut off date with vital signs, clinical safety laboratories (including amylase/lipase, fasting glucose, liver function tests), HbA1c, ECGs
- Safety and tolerability results have been generally consistent with those observed in >100 healthy volunteers dosed with paltusotine to date

Paltusotine Program Next Steps

- Complete study conduct for all patients already enrolled in Edge and Evolve and report top-line data in 4Q 2020
- Prepare for initiation of paltusotine Phase 3 acromegaly program:
  - Finalize study design for Phase 3 with regulatory and KOL feedback using end of Phase 2 data
  - Prepare drug product using to-be-marketed formulation for Phase 3
- Initiate NETs clinical development in 2021

Crinetics is committed to a commercialization strategy that unlocks the full potential of paltusotine across acromegaly and NETs



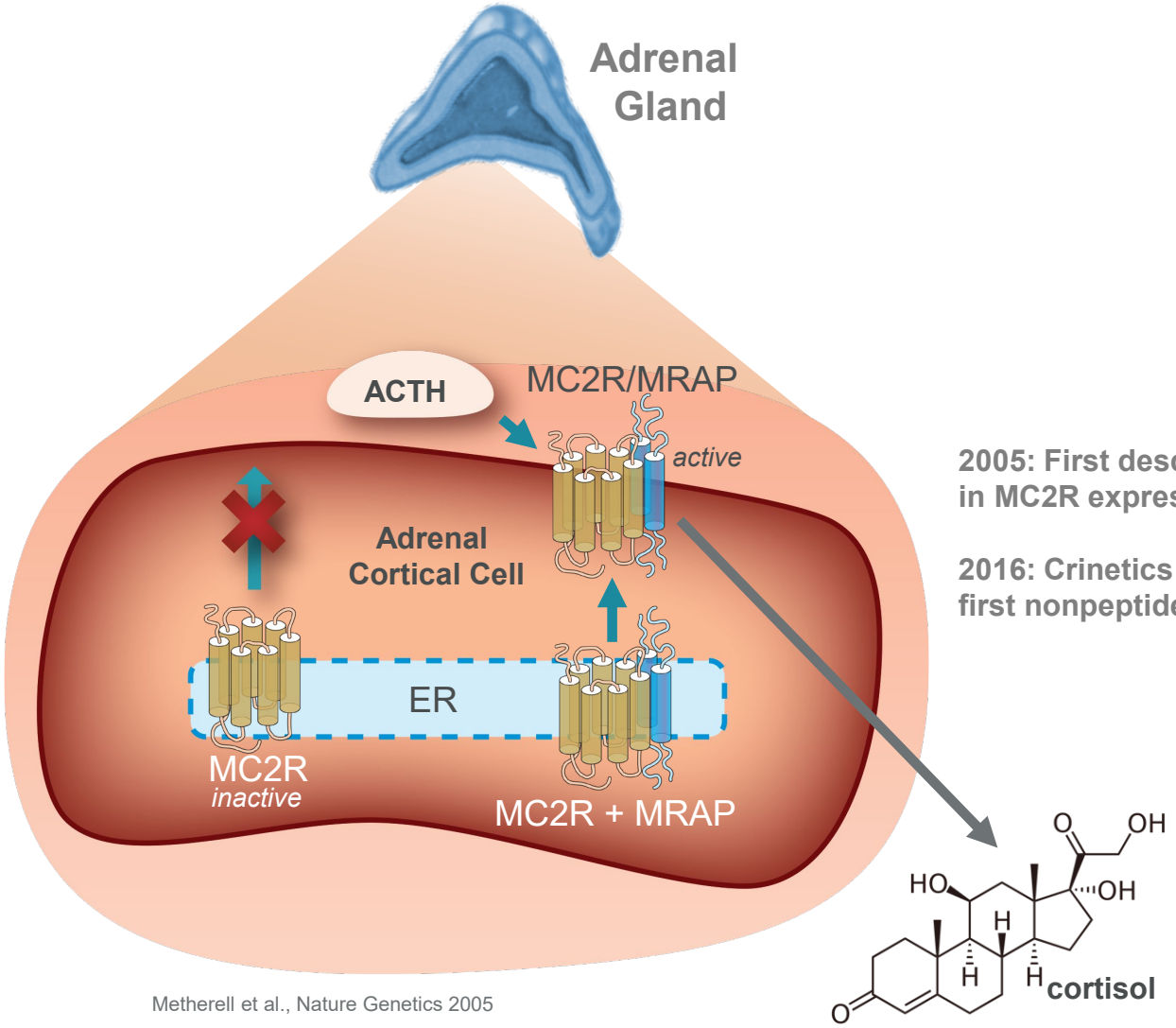
Nonpeptide ACTH Antagonists for the treatment of Cushing's Disease, Congenital Adrenal Hyperplasia (CAH), and other conditions of ACTH excess

Multiple markets of entry are possible for an ACTH antagonist, all are areas of high unmet medical need

Condition	Defect	Impact	ACTH antagonist target
Congenital Adrenal Hyperplasia (CAH)	Genetic defects that prevent production of cortisol by the adrenal	Loss of negative feedback causes over production of ACTH from the pituitary and build up of steroid precursors	✓
Cushing's Disease (CD)	Pituitary tumor	Over production of ACTH leading to hypercortisolemia	✓
Ectopic Cushing's Syndrome	Non-pituitary tumor	Over production of ACTH leading to hypercortisolemia	✓

Guraraldi and Salvatore, JABFM, 2012

ACTH Antagonists: A potential breakthrough in endocrinology



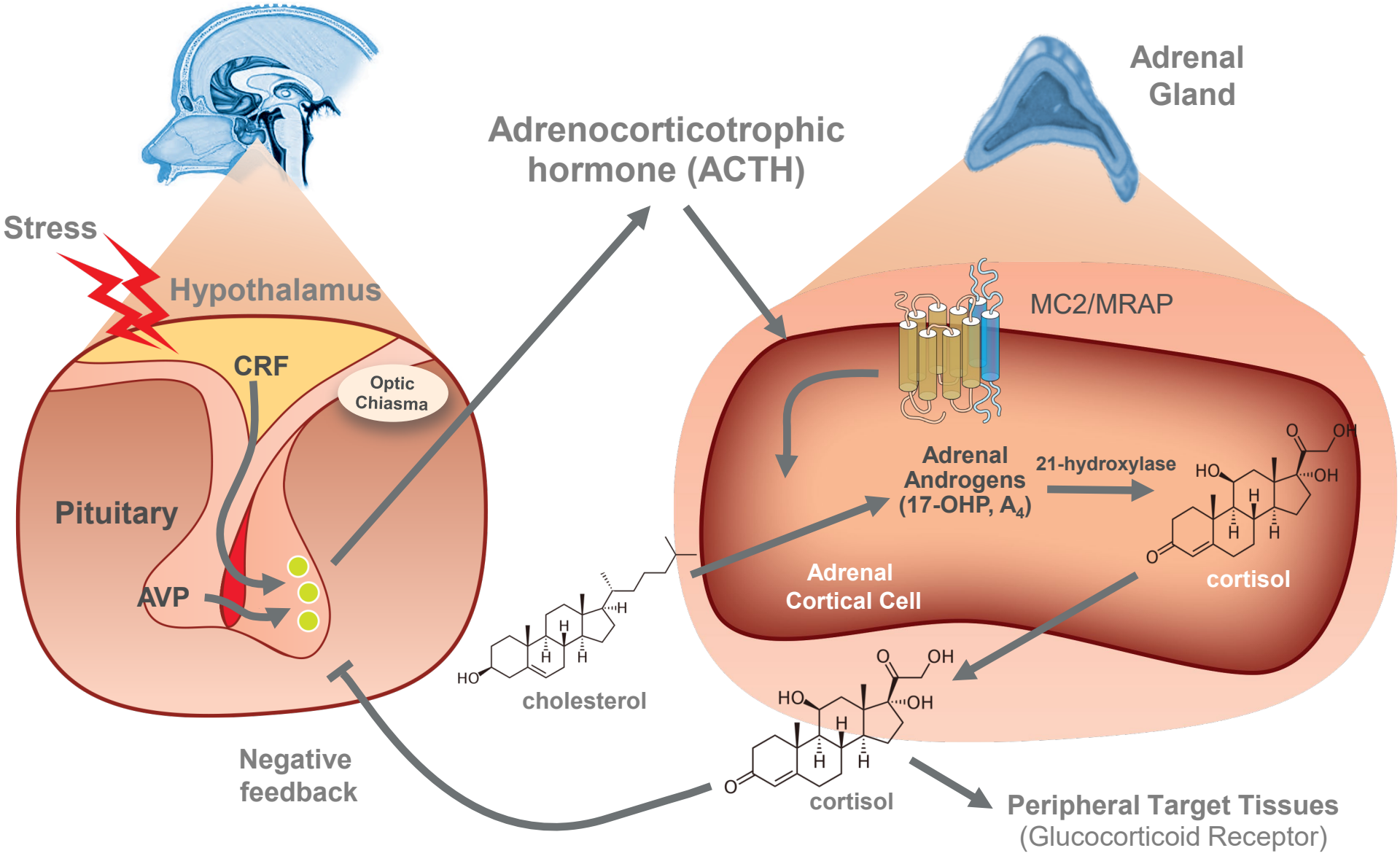
Metherell et al., Nature Genetics 2005

2005: First description of MRAP's role in MC2R expression and ACTH binding

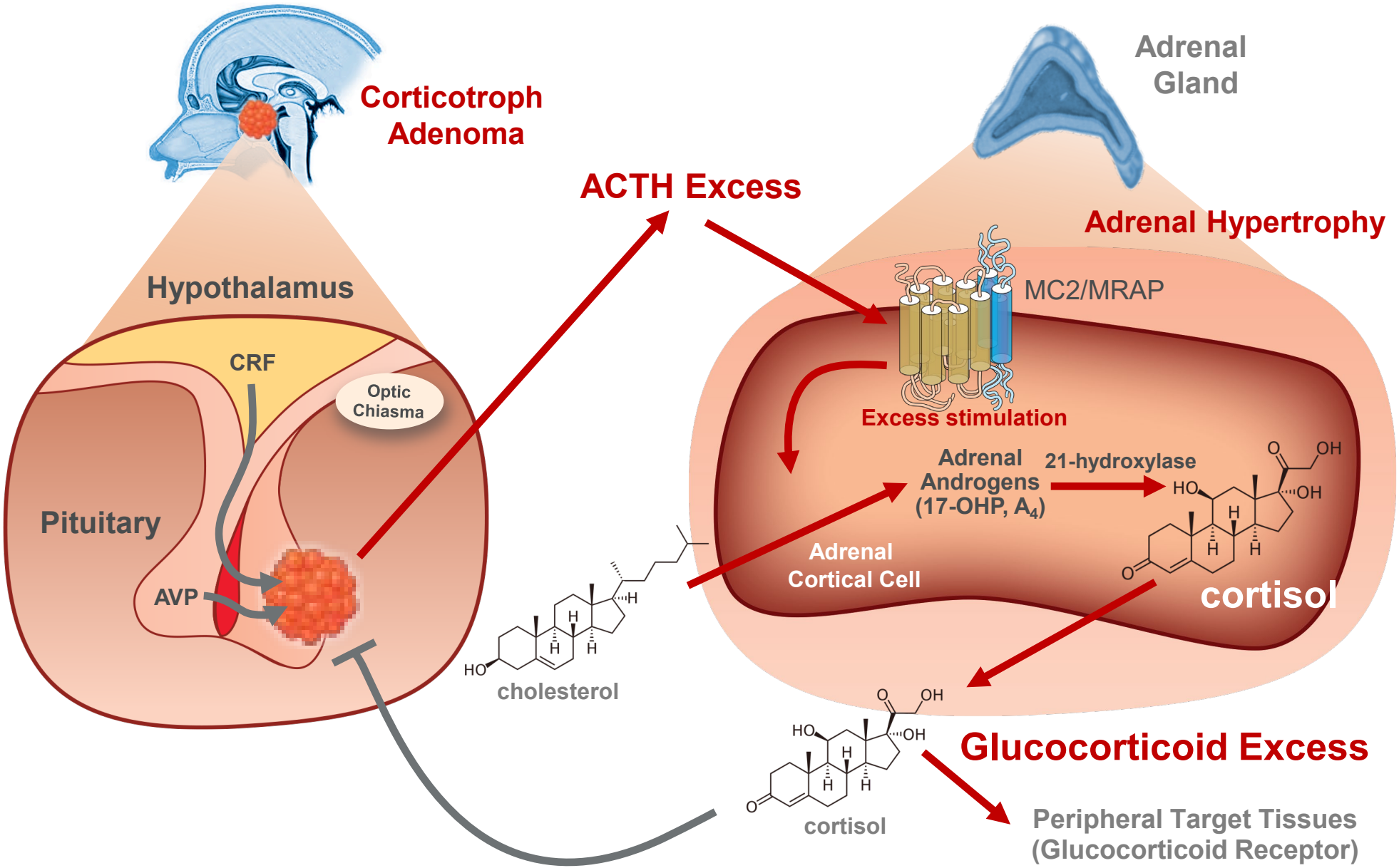
2016: Crinetics initiates effort to create first nonpeptide ACTH antagonist

Recent discoveries in peptide hormone GPCR regulation enabled discovery of first-in-class nonpeptide drug candidates

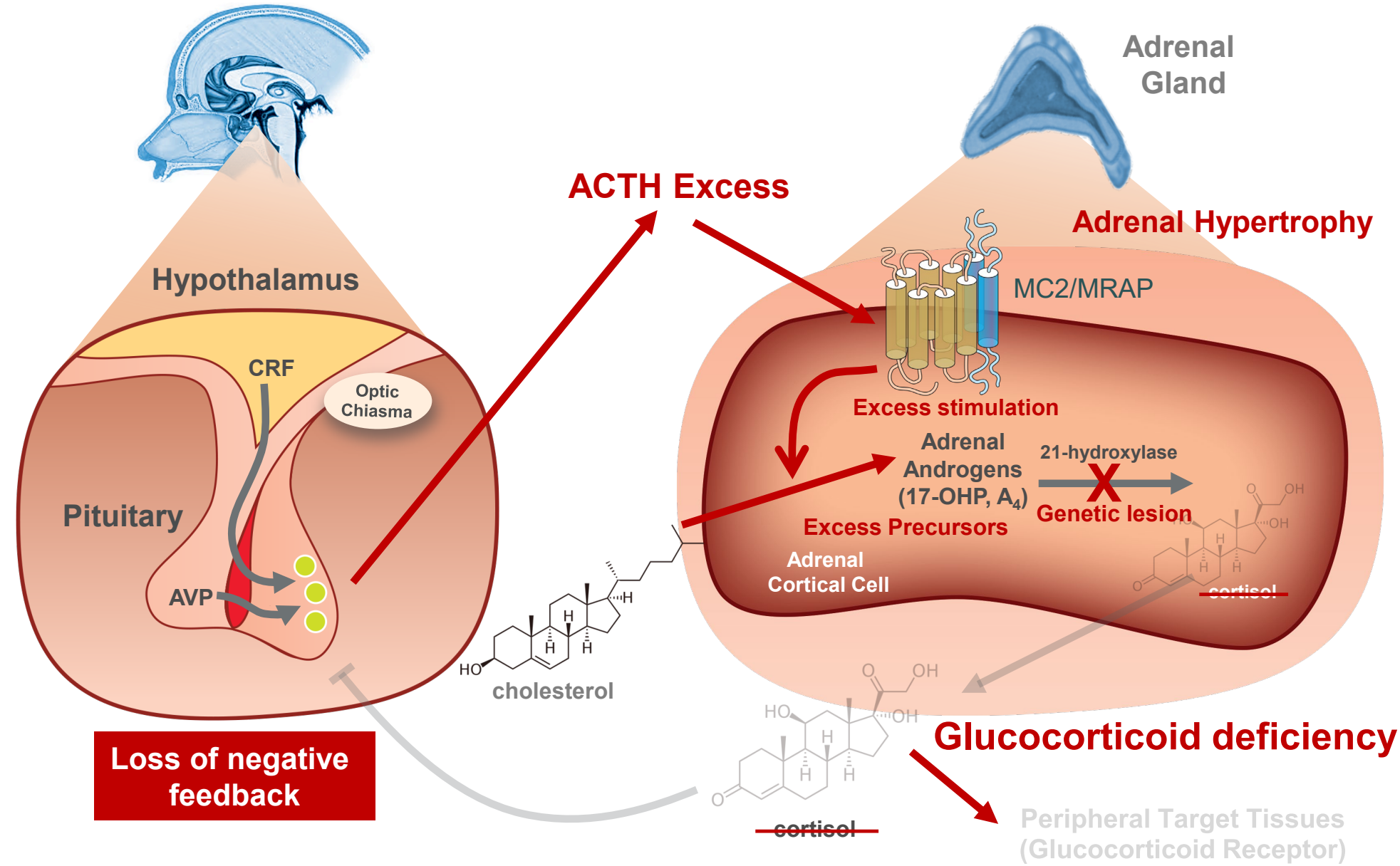
The hypothalamic-pituitary-adrenal (HPA) axis



Cushing's Disease Etiology

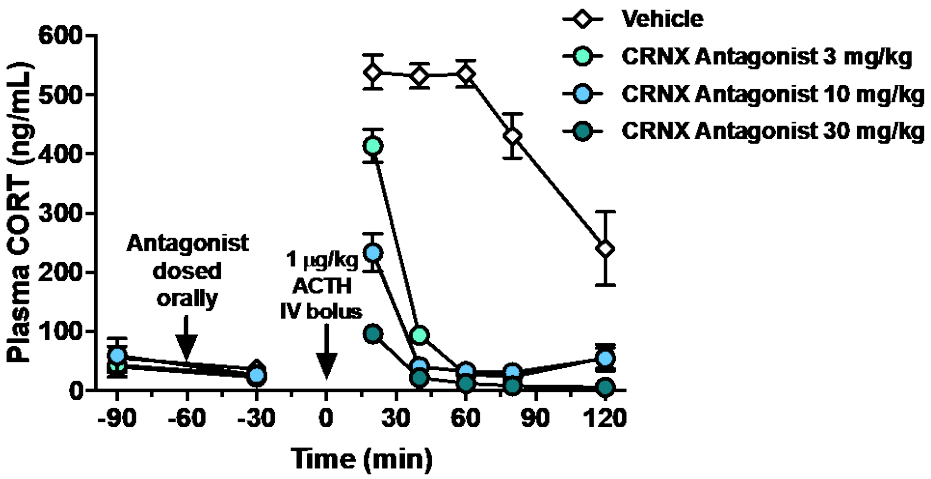


Congenital Adrenal Hyperplasia (CAH) Etiology

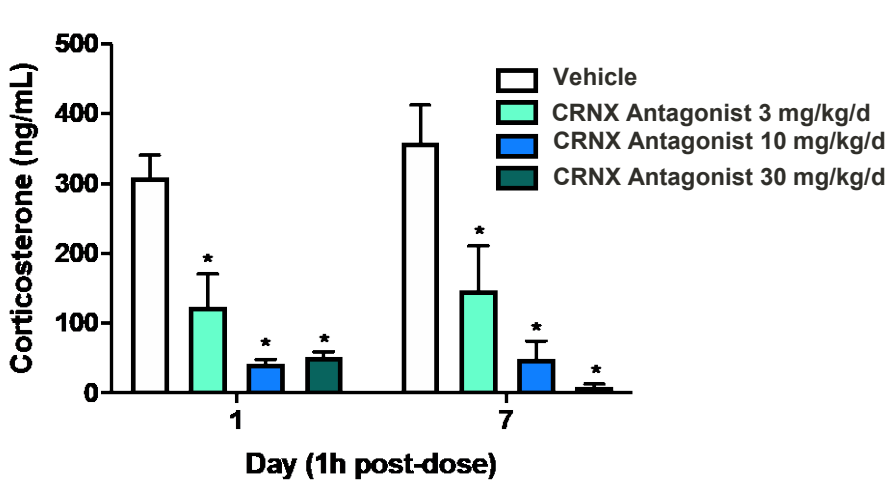


Nonpeptide ACTH antagonists demonstrated activity in rat models that mimic Cushing's and CAH

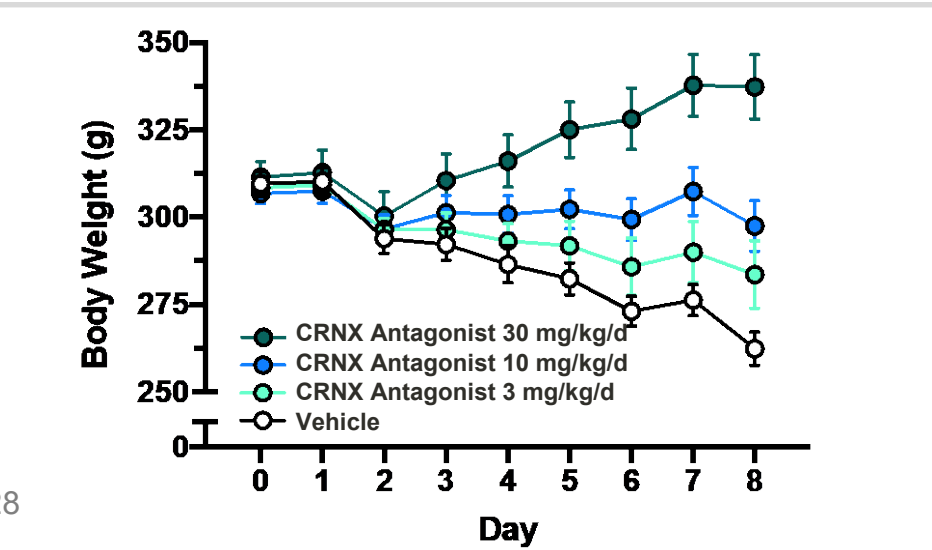
Acute suppression of ACTH-induced corticosterone observed in rats



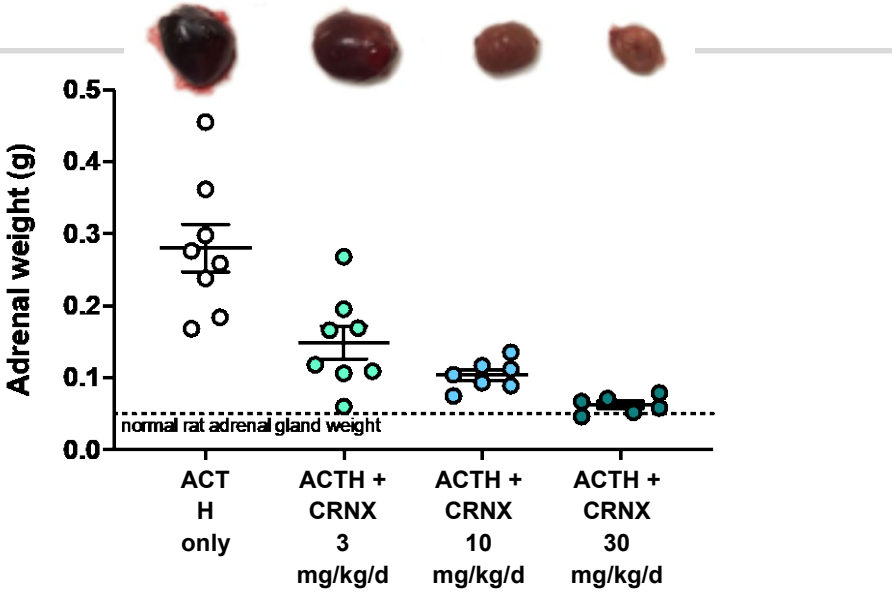
Repeat antagonist dosing (7d) suppressed corticosterone from chronic ACTH infusion



Repeat antagonist dosing (7d) rescued body weight loss from chronic ACTH infusion

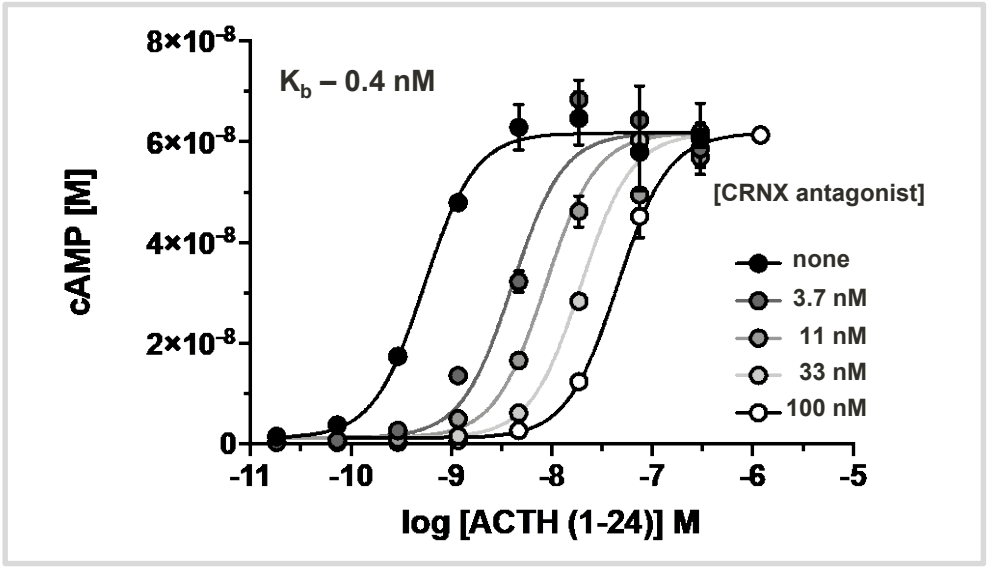


Repeat antagonist dosing (7d) rescued adrenal hypertrophy from chronic ACTH infusion



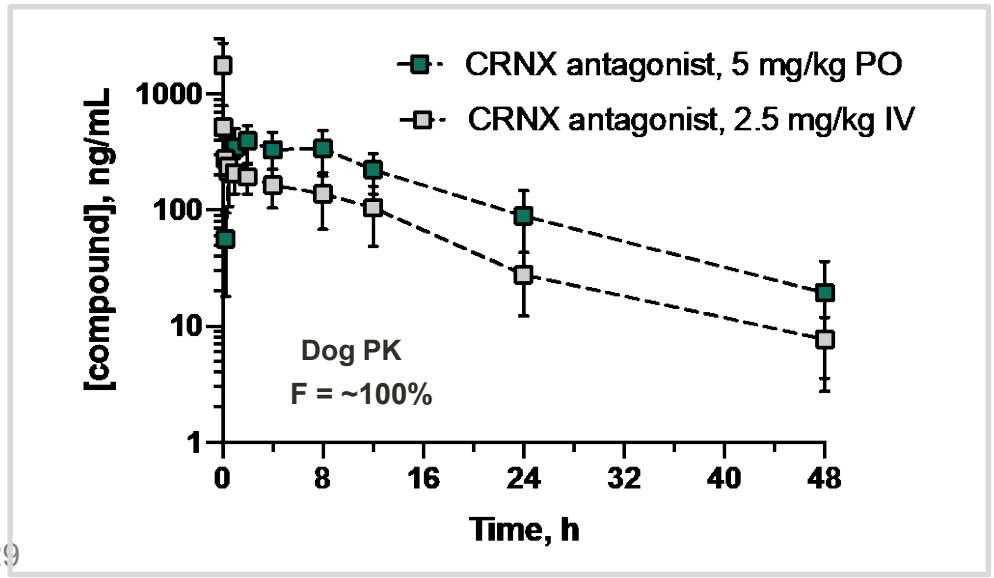
Crinetics ACTH antagonist now in preclinical development is designed to be a high-quality drug candidate

in vitro pharmacology results

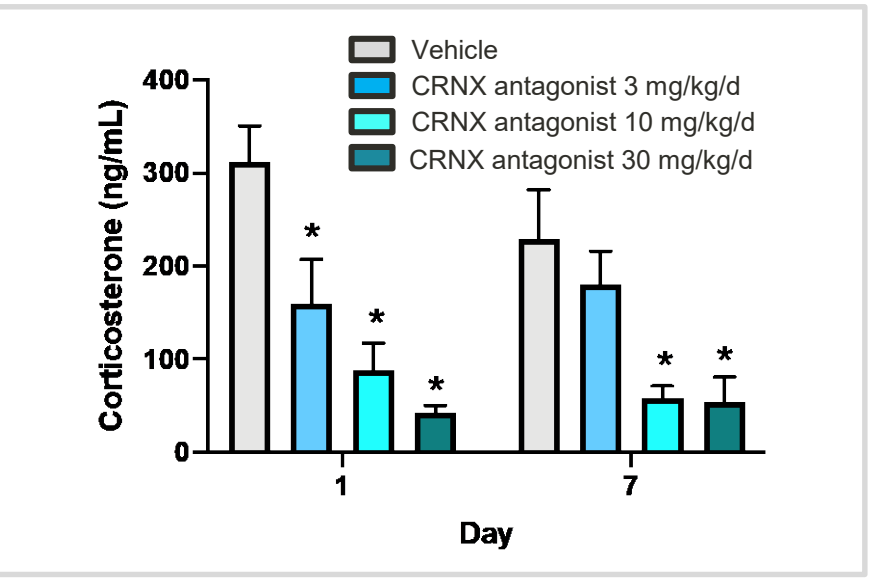


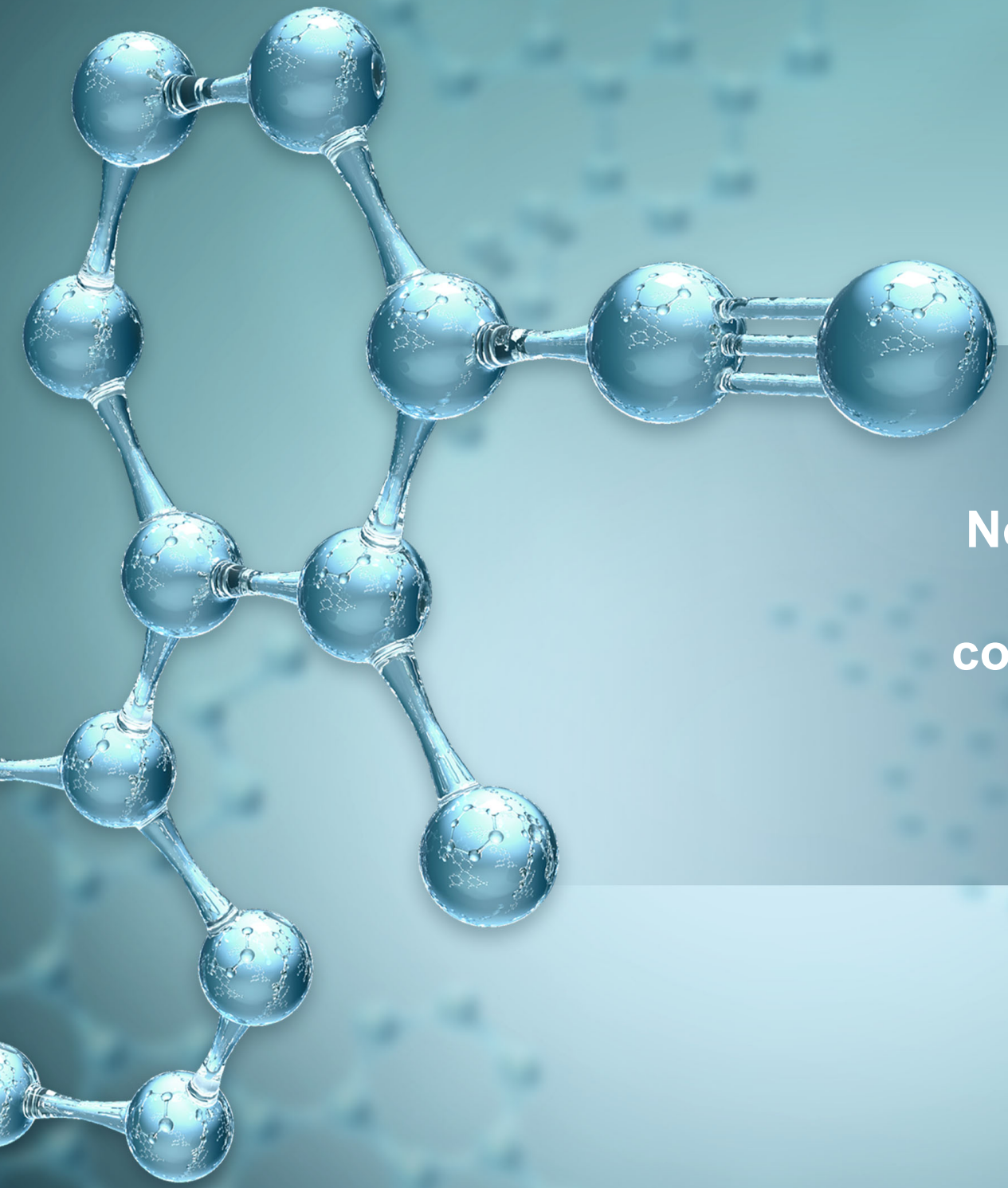
Property	CRNX ACTH Antagonist Candidate Preclinical Results
MW	~600
Solubility @ pH 7.4	1 mg/mL
human MC2R [K_B]	0.4 nM
rat MC2R [K_B]	3.3 nM
hMC1,3,4,5 [K_i]	>1 μM
CYP inhibition	No Inhibition
CYP induction	No Induction
Species differences in metabolism	No human unique metabolite
Rat PK	$t_{1/2}$ = 2.9 h F = 47 %
Dog PK	$t_{1/2}$ = 8.7 h F = ~100 %
Genotoxicity	Negative

Favorable bioavailability findings



Potent in vivo activity observed





Nonpeptide SST5 agonists for the treatment of congenital hyperinsulinism

Congenital Hyperinsulinism (CHI): disease overview and treatment limitations

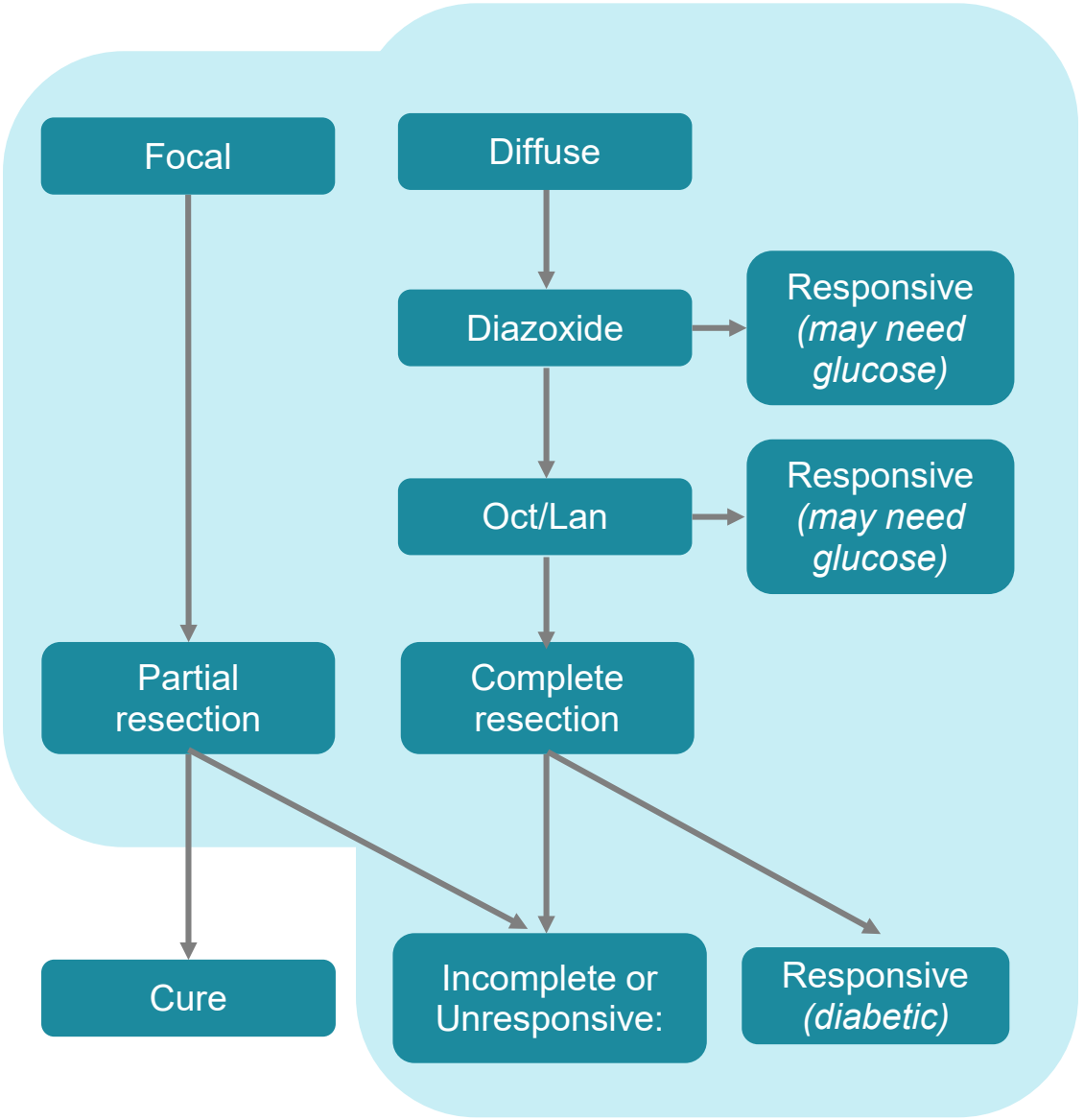
Indications

- Congenital hyperinsulinism (CHI)
  - Genetic defects (e.g. K_{ATP} channel) results in excess insulin secretion and profound hypoglycemia
- Incidence:
  - 1:30,000 to 1:50,000 births (U.S.)
  - Treated at a handful of specialty centers world-wide (e.g. Children's Hospital of Philadelphia)

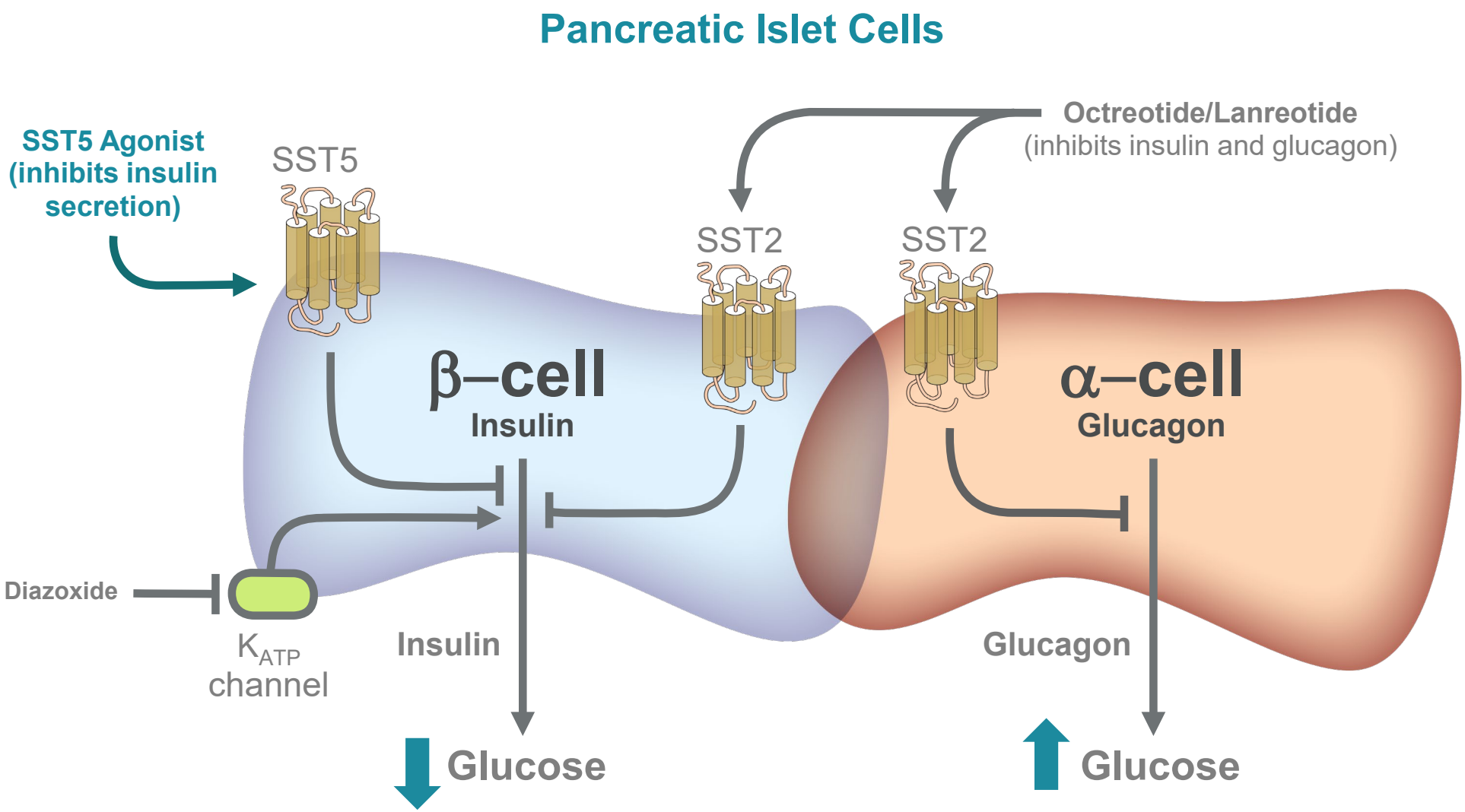
Patient and parent goals

- Avoid pancreatectomy
- Prevent cognitive / developmental problems
- Reduce injections and glucose sticks
- Medical management until HI is resolved
- Live a normal life

Amenable to SST5 agonist

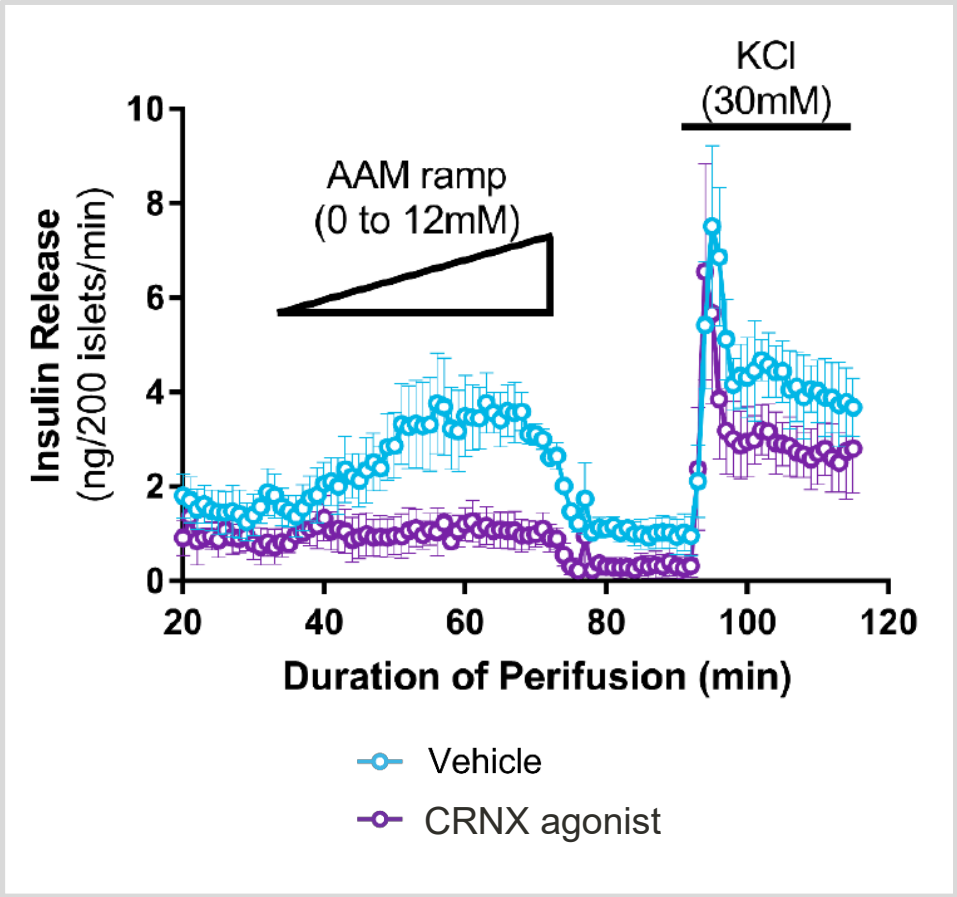


Our hypothesis: an oral, selective SST5 agonist is the optimal strategy for treating all HI patients

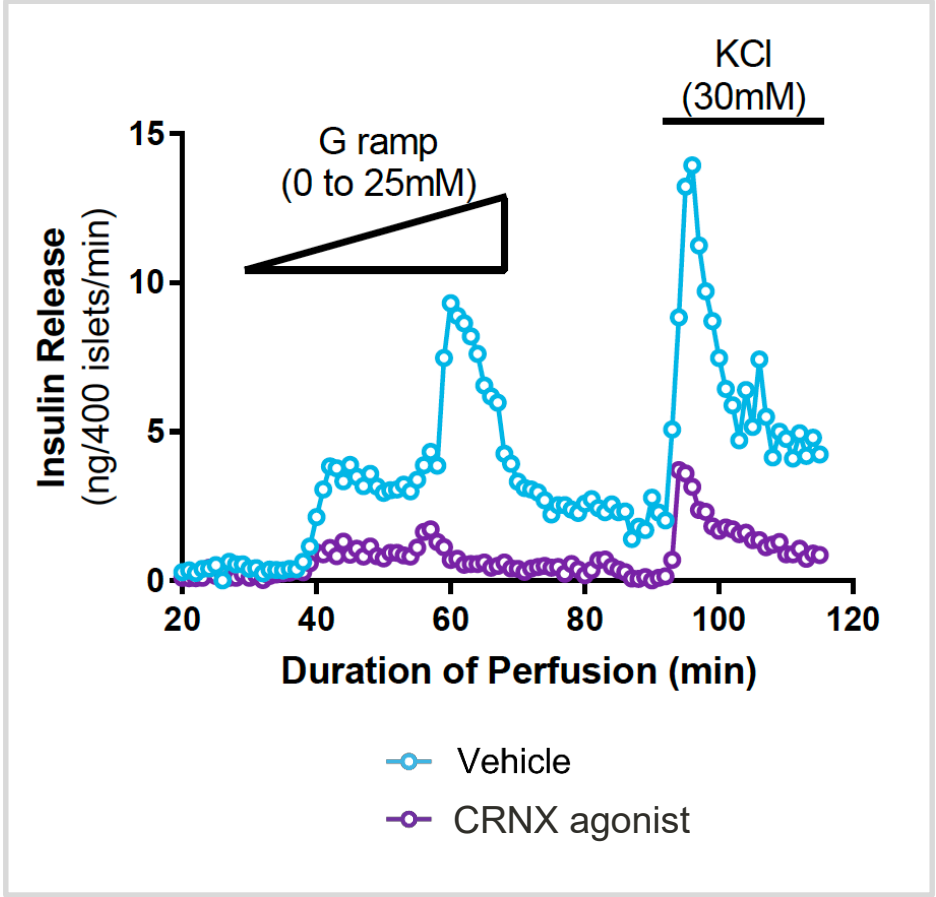


Mechanistic islet studies demonstrate Crinetics' SST5 agonists potently suppressed insulin secretion in disease models

In isolated SUR1$^{-/-}$ knockout mouse islets
(Mice mimic ~50% of CHI patients)

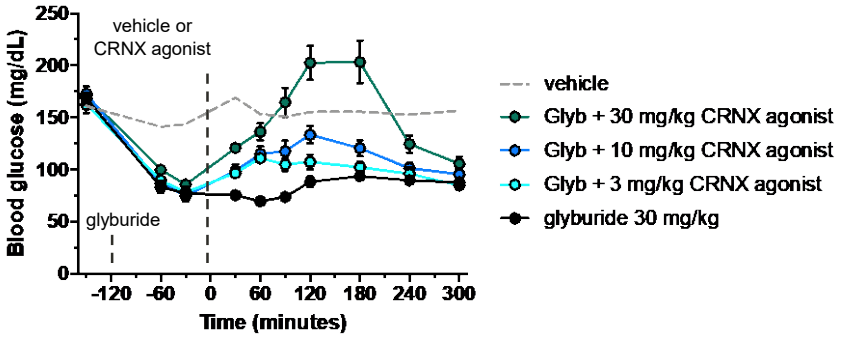


In isolated Beckwith Wiedemann Syndrome
patient islets

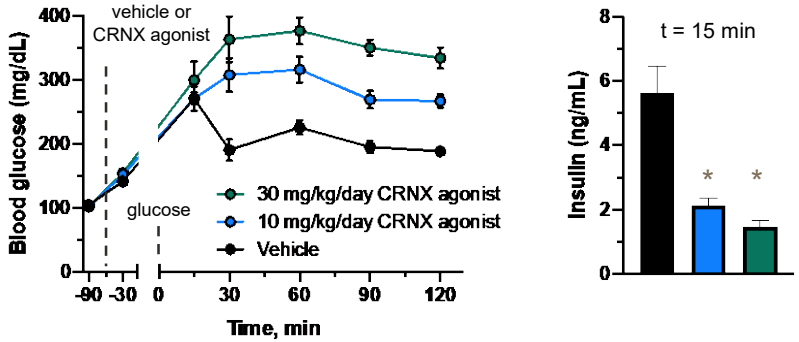


Crinetics' SST5 agonist now in preclinical development is designed to be a high-quality drug candidate

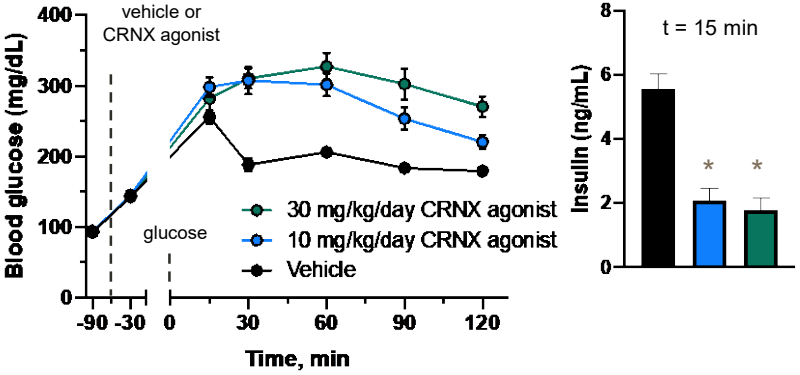
Rescue of glyburide-induced hypoglycemia



Day-1 OGTT Glucose increased insulin suppressed



Day-7 OGTT Effects maintained



Property	CRNX SST5 Agonist Candidate Preclinical Results
MW	< 500
Solubility @ pH 7.4	0.8 mg/mL
human SST5 [EC_{50}]	0.4 nM
rat SST5 [EC_{50}]	6.2 nM
hSST1 [EC_{50}] hSST2 [EC_{50}] hSST3 [EC_{50}] hSST4 [EC_{50}]	>10000 nM 770 nM 540 nM 4700 nM
CYP inhibition	No Inhibition
CYP induction	No Induction
hERG [IC_{50}]	>10 μM
Rat PK	$t_{1/2}$ = 3.3 h F = 30 %
Dog PK	$t_{1/2}$ = 9.9 h F = 57 %
Genotoxicity	Negative

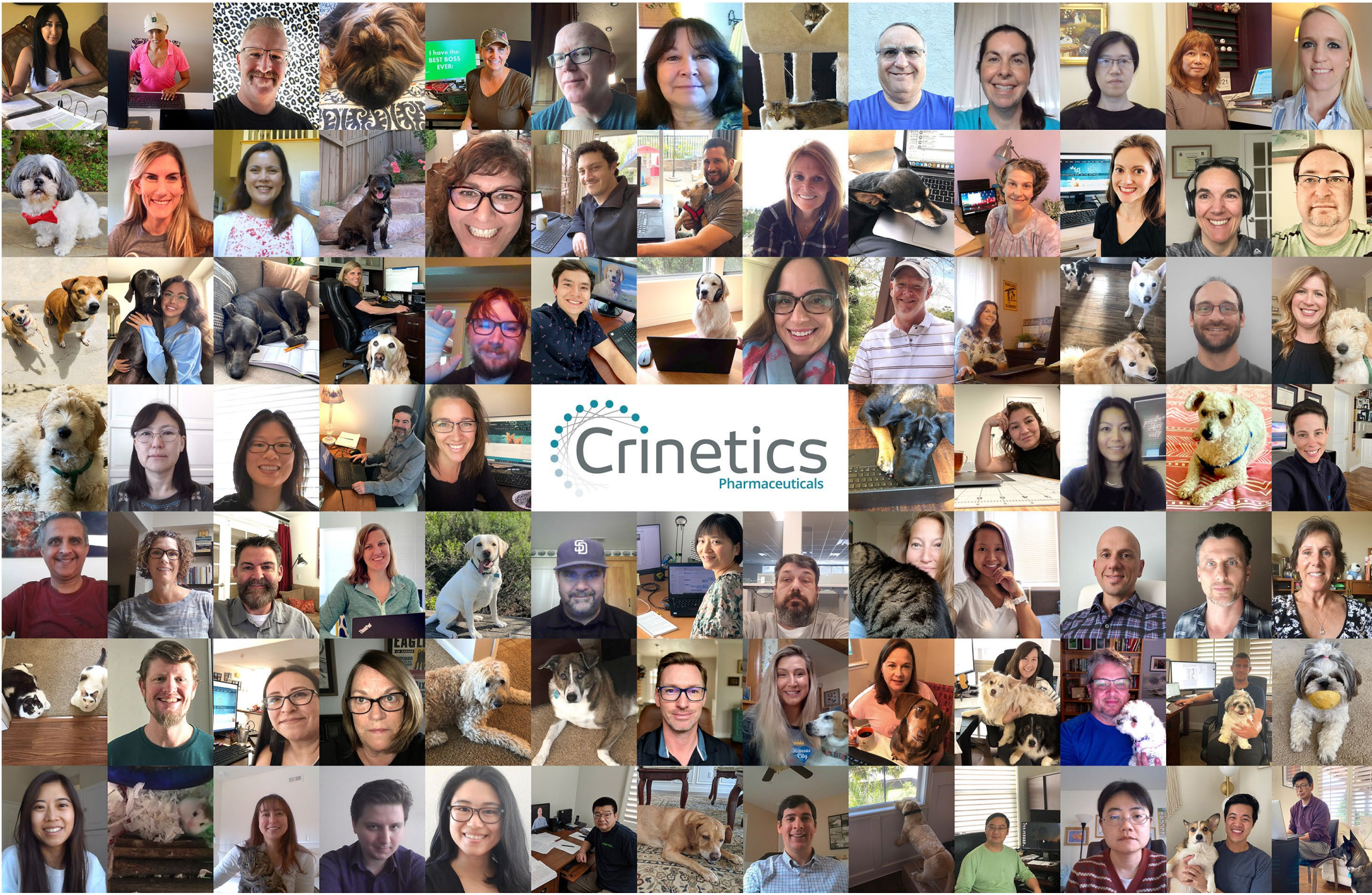
Financial Overview

- $205.2 million cash, cash equivalents and investment securities as of June 30, 2020
- Strong balance sheet with cash runway into 2023

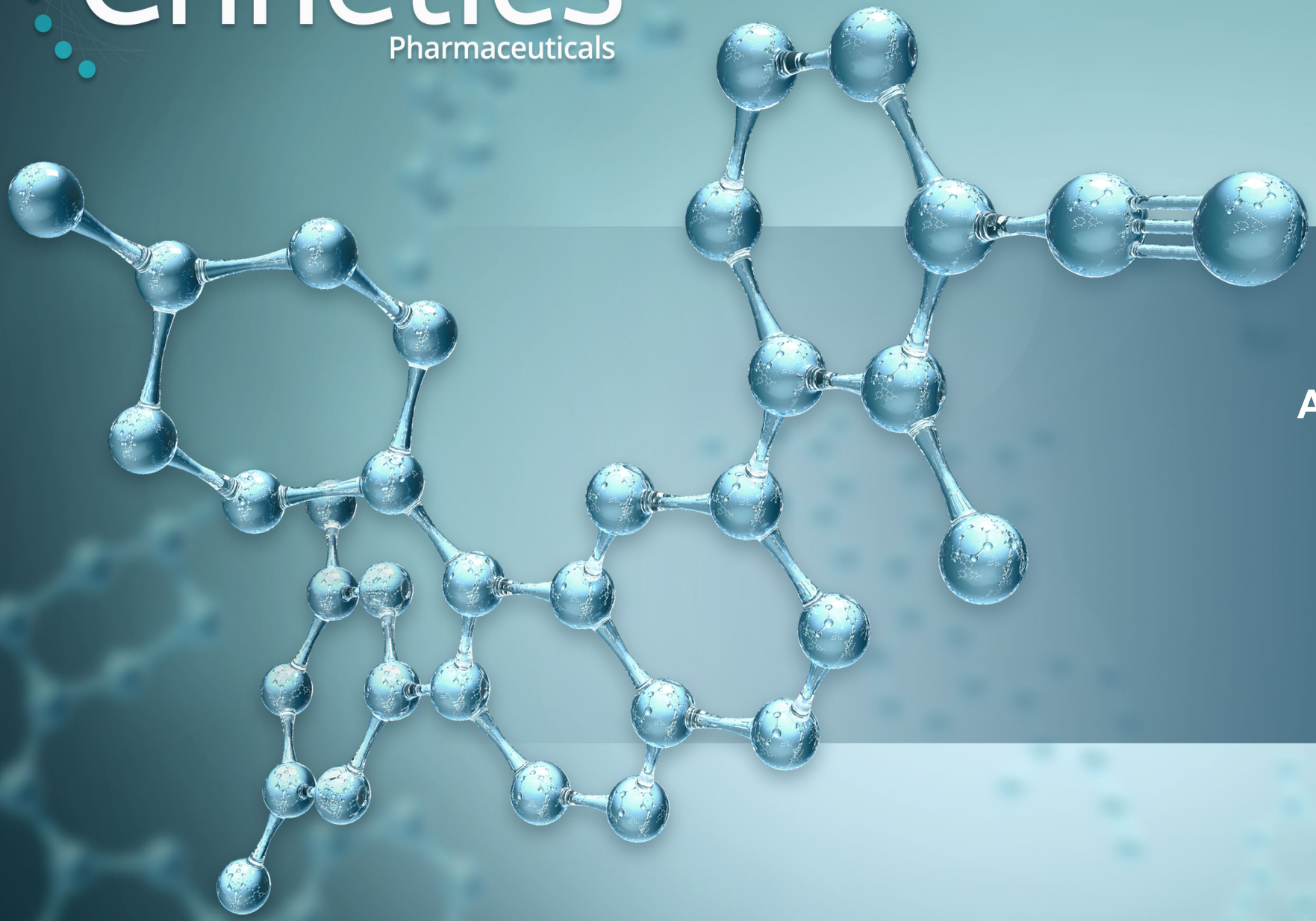
Expected near-term milestones

Program	Milestone	Expected Timing
Paltusotine: nonpeptide SST2 agonist	Topline data from Edge and Evolve acromegaly Phase 2 trials	4Q 2020
	Initiation of acromegaly Phase 3 program	1H 2021
	Initiation of NETs Phase 2 program	2021
Nonpeptide ACTH Antagonist	Initiation of Phase 1 clinical study	4Q 2020 or early 2021
Nonpeptide SST5 agonist	Initiation of Phase 1 clinical study	4Q 2020 or early 2021

Pipeline consisting of a maturing rare disease franchise in endocrinology and endocrine oncology


I have the BEST BOSS EVER!
Crinetics
Pharmaceuticals

Crinetics
Pharmaceuticals



Appendix

Leadership Team

Scott Struthers, PhD	President & CEO, Founder	Neurocrine Biosciences; ScienceMedia; Biosym Technologies; salk Where cures begin.
Frank Zhu, PhD	VP of Chemistry, Founder	Neurocrine Biosciences; UC San Diego; Shanghai Institute of Organic Chemistry, Chinese Academy of Sciences
Steve Betz, PhD	VP of Biology, Founder	Neurocrine Biosciences; Abbott; DuPont Merck
Ajay Madan, PhD	Chief Development Officer	Neurocrine Biosciences; UC San Diego; XenoTech Uncommon Science | Uncommon Service; American Board of Toxicology, Inc.
Marc Wilson	Chief Financial Officer	Cidara Therapeutics; Trius Therapeutics; Neurocrine Biosciences; pwc
Alan Krasner	Chief Medical Officer	Shire; Biodel; Pfizer; Johns Hopkins School of Medicine
Gina Ford	VP, Corporate Strategy & Commercial Planning	AcelRx Pharmaceuticals, Inc.; Ipsen Innovation for patient care; Solstice Neurosciences; elan
Adriana Cabré	VP, Human Resources	National University; Medimpact; Amylin; CooperVision

Directors and Advisory Board

BOARD OF DIRECTORS	
Wendell Wierenga, PhD	Chairman (Former EVP R&D, Santarus)
Scott Struthers, PhD	Founder & CEO
Steve Kaldor, PhD	Former CEO, Quanticel
Matt Fust, M.B.A.	Former CFO, Onyx
Weston Nichols, PhD	Founder & Managing Partner, Lynx1 Capital
Stephanie Okey, M.S	Former SVP, Genzyme Corporation
SCIENTIFIC ADVISORY BOARD	
David Clemmons, MD	Professor of Medicine at UNC, Chapel Hill
Anne Klibanski, MD	President & CEO, Partners Healthcare Former Chief of Neuroendocrine Unit at MGH & Professor of Medicine at Harvard
Philip Harris, FRCP, PhD	Chief Medical Officer, Isotopen Technologien München