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

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of report (Date of earliest event reported): October 1, 2018

CRINETICS PHARMACEUTICALS, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-38583**	**26-3744114**
(State or other jurisdiction of incorporation or organization)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

10222 Barnes Canyon Road, Bldg. #2
San Diego, California 92121
(Address of principal executive offices) (Zip Code)

(858) 450-6464
(Registrant's telephone number include area code)

N/A
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☒

Item 7.01 Regulation FD Disclosure.

During the week of October 1, 2018, representatives of Crinetics Pharmaceuticals, Inc. (the "Company") will be attending meetings with investors, analysts and other parties in connection with the Leerink Partners Roundtable Series: Rare Disease & Oncology in New York, New York. During these meetings, the Company will present the corporate slide presentation attached as Exhibit 99.1 to this Current Report on Form 8-K, which is incorporated herein by reference.

The Company's updated corporate presentation has been posted to the Company's website, www.crinetics.com. The Company plans to use its website to disseminate future updates to its corporate presentation and does not intend to file or furnish a Form 8-K alerting investors each time the presentation is updated.

The information set forth in this Item 7.01 is being furnished pursuant to Item 7.01 and shall not be deemed "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 it shall not be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or under the Exchange Act, whether made before or after the date hereof, except as expressly provided by specific reference in such a filing.

By filing this Current Report on Form 8-K and furnishing the information in this Item 7.01, the Company makes no admission as to the materiality of Item 7.01 in this report or the presentation available on the Company's website. The information contained in the presentation is summary information that is intended to be considered in the context of the Company's filings with the Securities and Exchange Commission (the "SEC") and other public announcements that the Company makes, by press release or otherwise, from time to time. The Company undertakes no duty or obligation to publicly update or revise the information contained in this Current Report on Form 8-K, although it may do so from time to time as its management believes is appropriate or as required by applicable law. Any such updating may be made through the filing of other reports or documents with the SEC, through press releases, by updating its website or through other public disclosure.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Slide Presentation

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 hereunto duly authorized.

CRINETICS PHARMACEUTICALS, INC.

Date: October 1, 2018

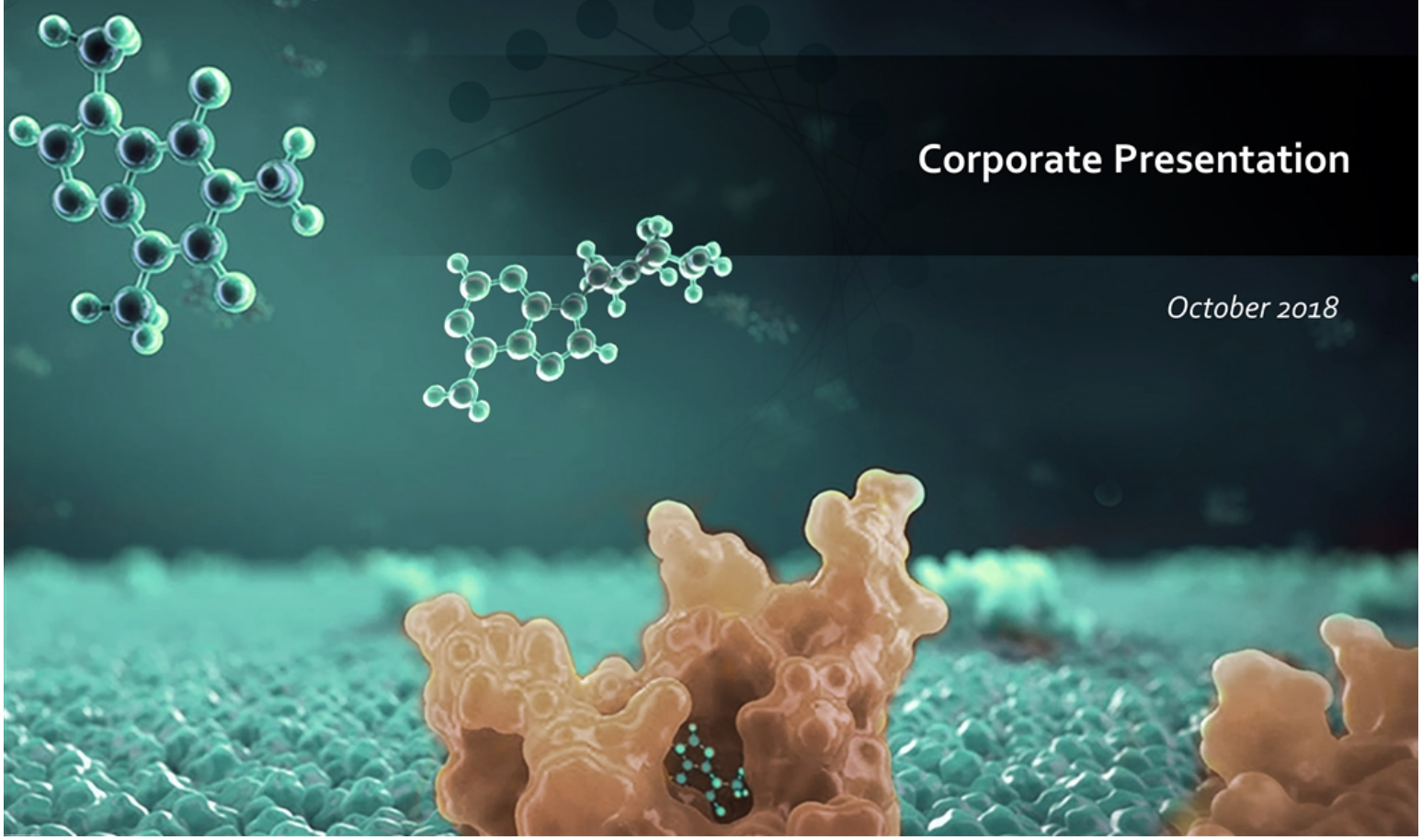
By: /s/ Marc Wilson
Marc Wilson
Chief Financial Officer

Exhibit 99.1

Crinetics
Pharmaceuticals

Corporate Presentation

October 2018



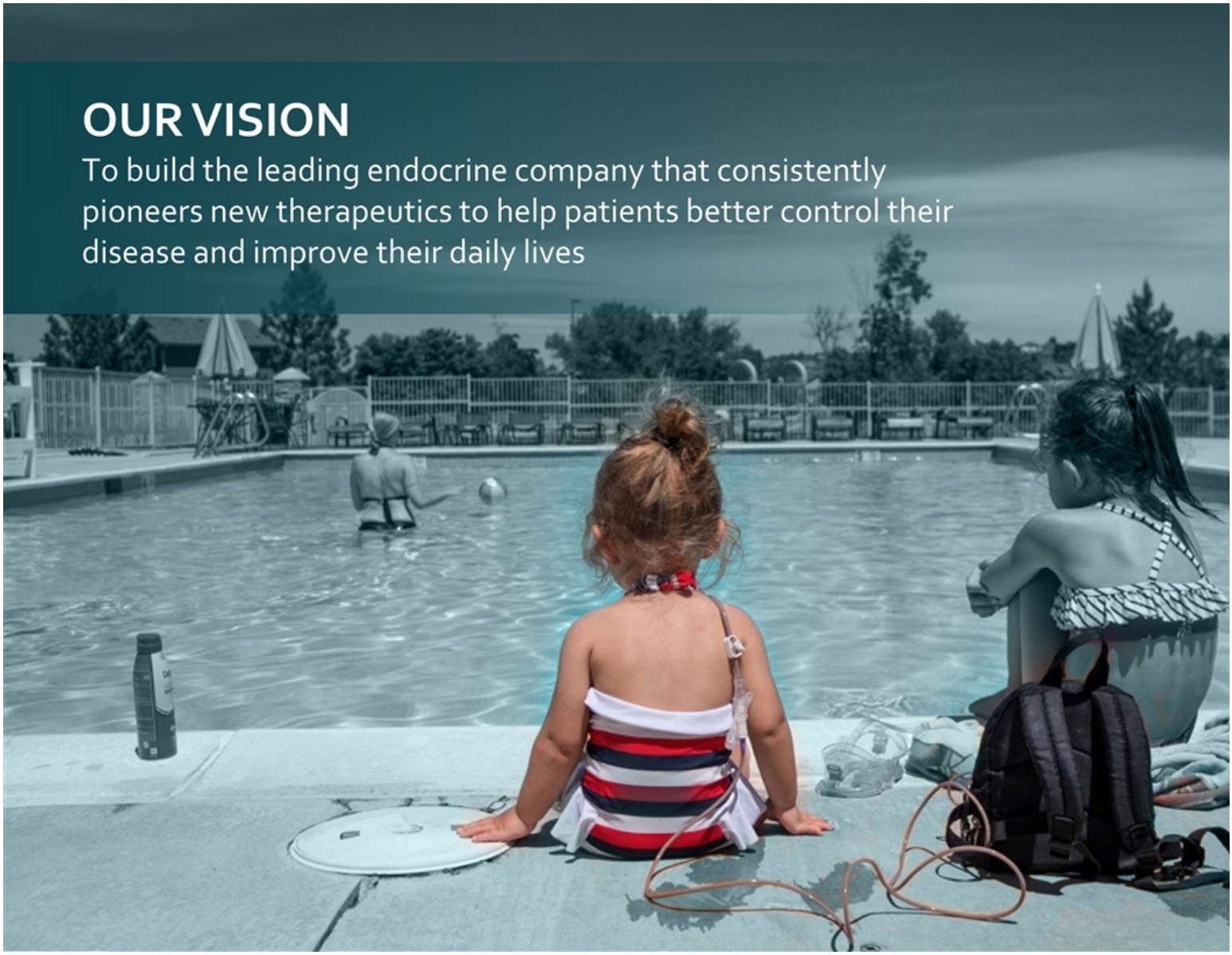
Safe harbor statement

This presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, prospective products, product approvals, research and development costs, 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. 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. These known risks and uncertainties are described in detail in our filings made with the Securities and Exchange Commission from time to time. 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



Investment highlights

Focused on developing oral nonpeptide therapeutics for rare endocrine diseases and related tumors

- Targeting peptide G-protein coupled receptors (GPCRs) with validated biomarkers
- Unlocking new targets with high therapeutic potential

Lead product candidate: CRN00808

- Potential first-in-class oral, nonpeptide, selective, somatostatin receptor type 2 (sst2) biased agonist designed to treat acromegaly
- Potent suppression of the growth hormone (GH) axis observed in Phase 1 trial
- Observed tolerability and AE profile consistent with approved somatostatin agonists
- U.S. IND in effect

Pipeline of novel therapeutics addressing diseases with capital efficient approval paths

- Neuroendocrine tumors (NETs)
- Hyperinsulinemia
- Cushing's disease

Product candidate pipeline addresses multi-billion aggregate market with high unmet need

- Injected somatostatin peptide drugs to treat predominantly acromegaly and neuroendocrine tumors generated sales of ~$2.7 billion in 2017
- Additional disease targets provide significant further upside

Retain global rights to commercialize our product candidates and have no licensing obligations

Strong management team with key leadership roles in the development of Orlissa and Ingrezza at Neurocrine Biosciences

Successful July 2018 IPO

- IPO net proceeds of $106.4 million

Experienced leadership team

Scott Struthers, PhD
CEO, Founder & Director

Frank Zhu, PhD
VP of Chemistry & Founder

Steve Betz, PhD
VP of Biology & Founder

Ajay Madan, PhD
VP of Development

Marc Wilson
CFO

Alan Krasner, MD
CMO

Neurocrine Biosciences | Neurocrine Biosciences | Neurocrine Biosciences | Neurocrine Biosciences | CIDARA Therapeutics | Shire

ScienceMedia | UC San Diego | Abbott | UC San Diego | Trius Therapeutics | BIODEL

BIOSYM Technologies | Shanghai Institute of Organic Chemistry, Chinese Academy of Sciences | DU PONT MERCK | XENOTECH Uncommon Science | Uncommon Service | Neurocrine Biosciences | Pfizer

SALK INSTITUTE FOR BIOLOGICAL STUDIES | pwc | JOHNS HOPKINS SCHOOL of MEDICINE

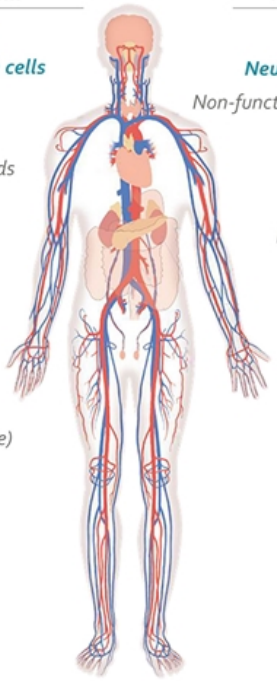
We have a long history of successfully discovering and developing important new therapeutics

Significant opportunities across multiple rare endocrine diseases and endocrine-related tumors

Endocrine pathways function to maintain **homeostasis** and commonly use **peptide hormones acting through GPCRs** to regulate many aspects of physiology including growth, energy, metabolism, gastrointestinal function and stress responses

Opportunities in the endocrine space

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



Our indication criteria:

- ✓ High unmet need
- ✓ Established biology
- ✓ Biomarker endpoints
- ✓ POC in Phase 1
- ✓ Small registration trials

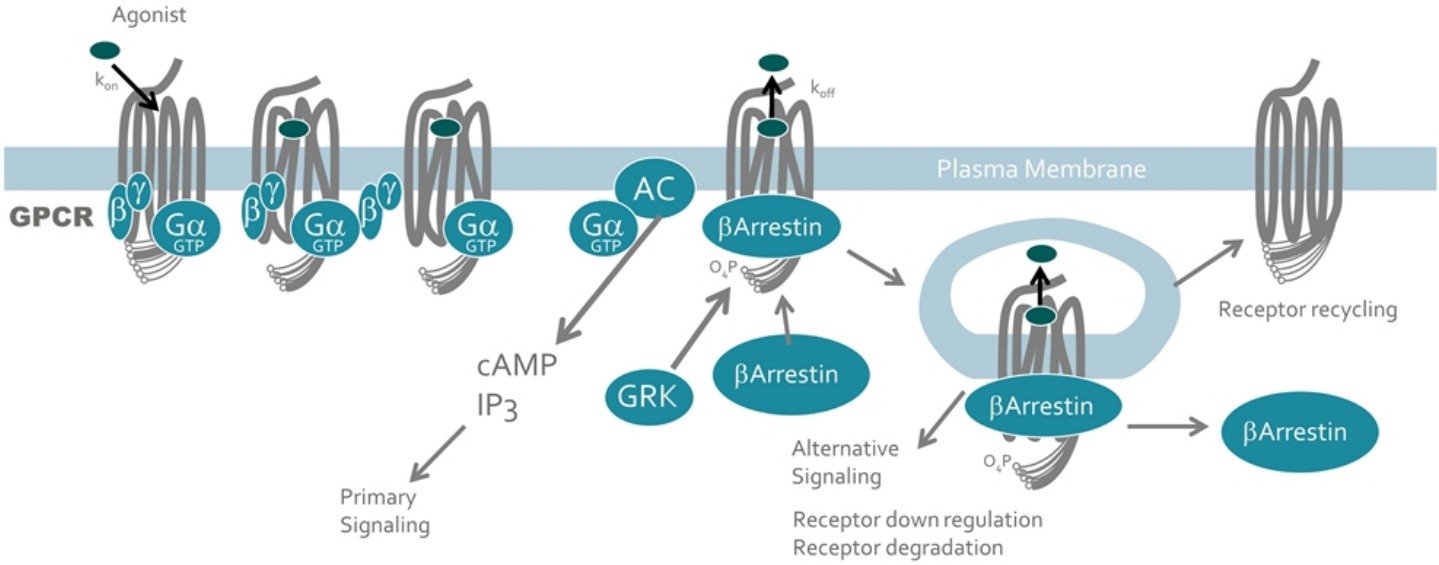
Our focus:

- ✓ Oral nonpeptide drug candidates that target peptide GPCRs
- ✓ Ability to generate small molecules that can replicate the complex interactions between peptides and their cognate GPCRs
- ✓ Overcome the formulation challenges and injection inconvenience of peptide drugs

Our discovery capabilities:

- ✓ Significant experience influencing the dynamic behaviors of GPCRs
- ✓ Developed a number of proprietary methods, techniques and tools to evaluate newly synthesized molecules
- ✓ Specifically tailor a product candidate to be highly optimized for its interaction with its specific GPCR target
- ✓ Iterative strategy where compounds are designed and rapidly characterized for pharmacologic and pharmaceutical properties

Our approach: Tailor ligands to regulate dynamic GPCR behaviors and ultimately improve patient outcomes



Ligand binding kinetics conformational changes

Receptor signaling

Receptor trafficking

Our understanding of the complex GPCR signaling pathways enables us to develop oral, small molecule, product candidates that modulate GPCR dynamic behaviors

The somatostatin receptor family of peptide GPCRs

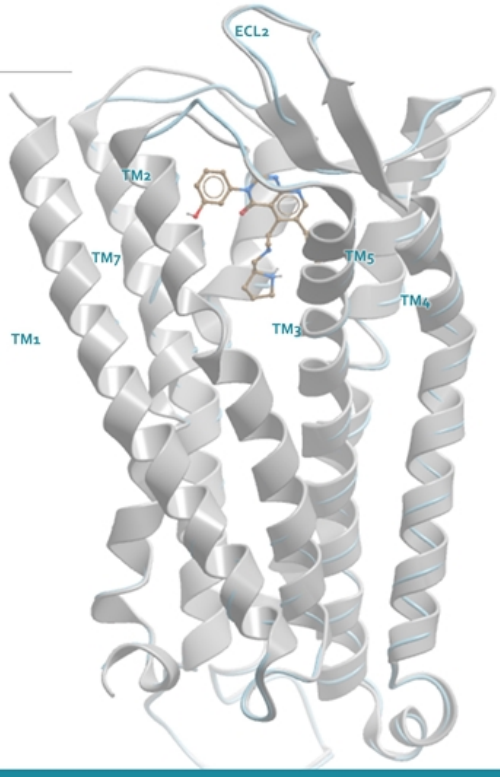
Overview of somatostatin (sst) receptors

The peptide hormone somatostatin is produced by a variety of cell types and has pleiotropic effects throughout the body, many of which are related to the ***inhibition of secretion of other hormones or neurotransmitters***

The effect of somatostatin are mediated by ***five different receptor proteins (sst1-sst5)***, each of which is expressed in different subsets of tissues

sst2 is the most widely expressed subtype in NETs and is the dominant receptor by which GH secretion is suppressed in the pituitary

The ***sst5 receptor*** is expressed by pancreatic islet cells where its activation ***potently inhibits insulin secretion***



Somatostatin agonism is a well-established, commercially validated mechanism

Crinetics Pharmaceuticals

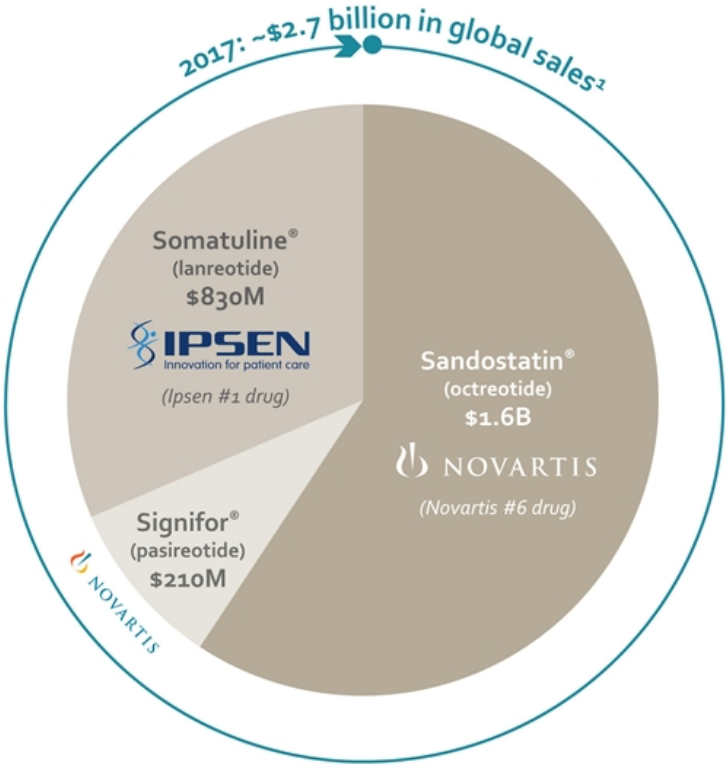
Pipeline:

Potential to build a rare disease franchise in endocrinology

PROGRAM	DISCOVERY	PRECLIN	PHASE 1	PHASE 2	PHASE 3
CRN00808 (Oral sst2 Agonist) Acromegaly					
CRN01941 (Oral sst2 Agonist) Neuroendocrine Tumors (NETs)					
CRN02481 Oral sst5 Agonist Hyperinsulinemia					
Oral ACTH Antagonist Cushing's Disease					


CRN00808
for the treatment of acromegaly

Established commercial opportunity for injectable somatostatin peptides despite significant limitations



Limitations of current somatostatin peptide analogs

- ***Painful*** intramuscular/deep sc injections every month (octreotide, lanreotide)
  - Hardness, bruising and swelling at injection site
- ***Inconvenient*** / frequent physician office visits
  - Complex reconstitution of depot dosing regimens and prone to error
- Limited efficacy – ***only half of patients are fully controlled*** (reduce excess GH secretion and normalize IGF-1 levels) throughout the treatment period
- A majority of ***patients experience increased glucose levels*** within the first 2-3 weeks of treatment with pasireotide

CRN00808 pioneers a new class of oral selective non-peptide sst2 biased agonists

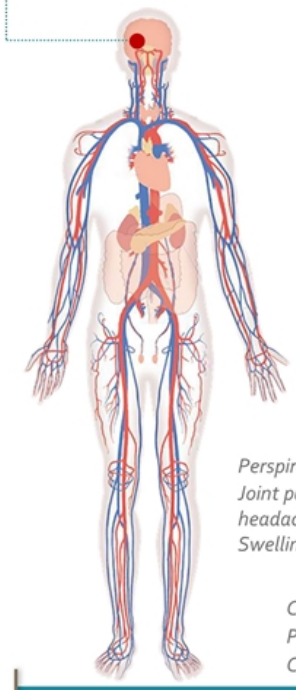
Crinetics Pharmaceuticals

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

Acromegaly: disease overview and treatment paradigm

~25,000 people in the U.S. suffer from acromegaly

Acromegaly is caused by a benign pituitary tumor



Increased GH results in ***excess downstream secretion of IGF-1*** from the liver

GH and IGF-1 promote tissue growth and other metabolic effects

Perspiration, Joint pain, Severe headache, Swelling

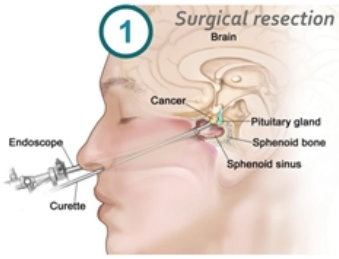
Hypertension, Hepatomegaly, Impaired glucose, Thyroid hypertrophy

Changed facial features, Prognathism, Enlarged hands, Carpal tunnel, Arthritis

Uncontrolled acromegaly is debilitating and increases the risk of early death

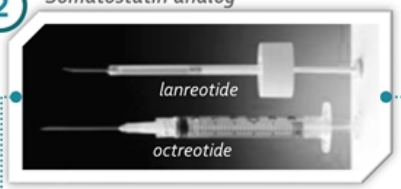
Current treatment options for patients

1 ***Surgical resection*** of tumor for eligible patients



40-60% of acromegaly patients are candidates for chronic pharmacological intervention

2 ***Somatostatin analog***



Not controlled

3 ***Dopamine Agonist***
GH Receptor Antagonist
+/- Somatostatin Analog

Controlled

Controlled

Estimated 10-15K acromegaly patients in the U.S. on life long therapy

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

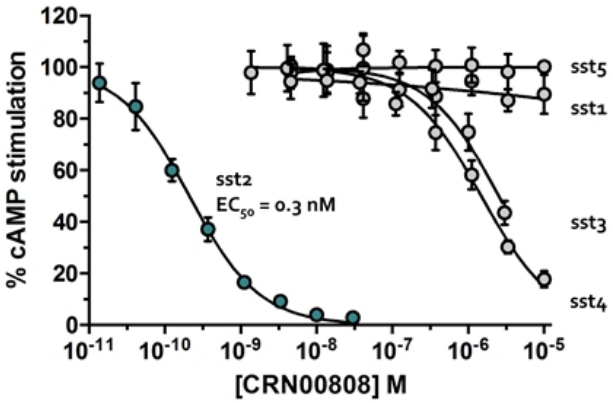
PRODUCT CANDIDATE TAILORED TO DELIVER KEY BENEFITS

CHARACTERISTICS	PRIMARY 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)	Once daily dosing
Reduced desensitization	Potential improved responder rates
Selectivity for sst2	Glucose control (avoid sst5 mediated hyperglycemia)

Crinetics Pharmaceuticals

CRN00808 is a potent and selective nonpeptide sst2 agonist…

In vitro selectivity profile at all five somatostatin receptor subtypes for CRN00808



In vitro selectivity profile at all five somatostatin receptor subtypes for CRN00808 and SSA peptides

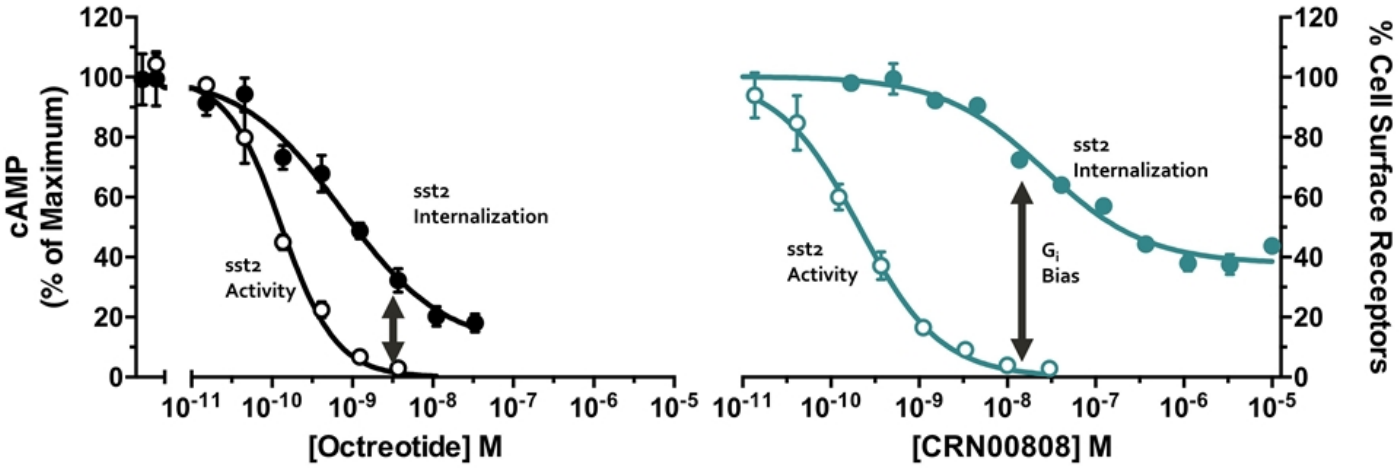
	Human EC_{50} (nM)				
AGONIST	sst2	sst1	sst3	sst4	sst5
CRN00808	0.25	>10000	3300	1100	>10000
lanreotide	0.10	1900	200	820	19
octreotide	0.061	> 10000	7.9	470	2.1
pasireotide	0.59	23	0.78	6300	0.076
native SS14	0.14	0.83	0.17	0.21	0.065

Potency was measured by inhibition of cAMP in cells stably expressing the indicated human receptor

CRN00808's potency for sst2 is 4,000 times greater than for other subtypes

…designed with G_i bias to reduce internalization and desensitization of sst2

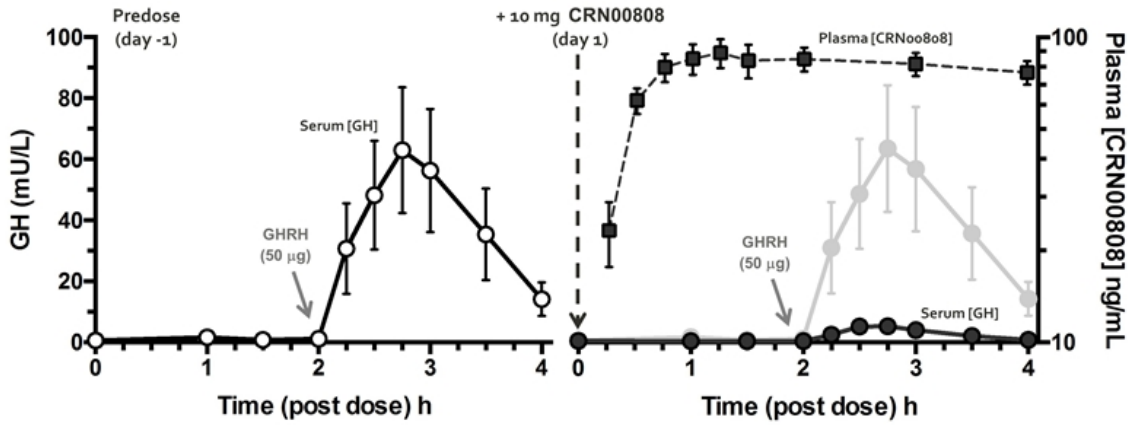
> In vitro studies have shown CRN00808 was *75 times more potent* for cAMP inhibition than receptor internalization



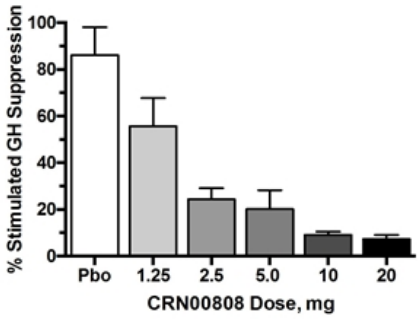
Dose response curves are shown from individual representative experiments. All points are the mean ± standard error of either triplicate or quadruplicate readings. White circles are from a cAMP assay measuring sst2 activation. Filled circles are from an internalization assay measuring the amount of cell surface receptors.

Phase 1 SAD arm: PK/PD analysis

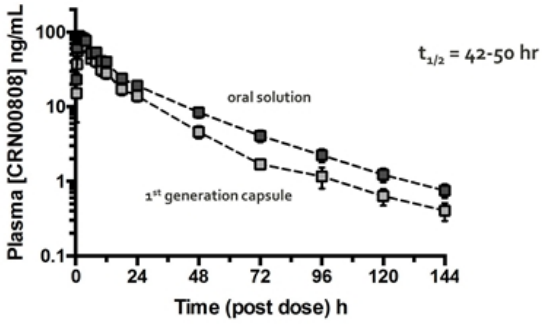
Suppression of GHRH stimulated GH secretion by 10 mg of CRN00808



Dose response of GH suppression



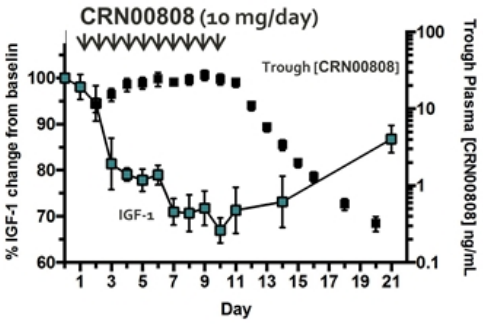
Comparison of oral solution vs. first-generation capsule



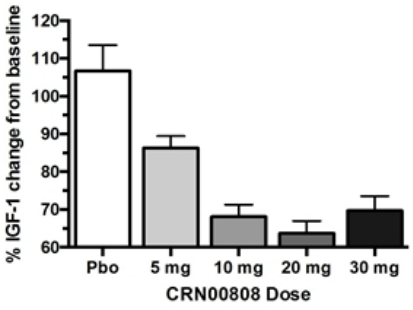
PK/PD analysis of the single-ascending dose arm. Top chart: Suppression of GHRH stimulated GH by 10 mg of CRN00808 administered as an oral solution on day 1 (filled circles, right), compared to day -1 (open circles, left). The plasma exposure of CRN00808 is shown in black squares. Lower left chart: Dose response of GH suppression of CRN00808 (data excludes an outlier in the 1.25 mg cohort which was likely related to variability in method of GHRH administration. The methodology was corrected for cohorts 2.5 mg and higher). Lower right chart: Comparison of CRN00808 plasma exposure following oral administration of 10 mg CRN00808 as an oral solution (black squares) and as a first-generation capsule (white squares). h = hour, Pbo=placebo. All data are mean ± standard error. When the capsule was administered with a standardized high fat meal, plasma AUC was reduced by approximately 83% (data not shown).

Phase 1 MAD arm: PK/PD analysis

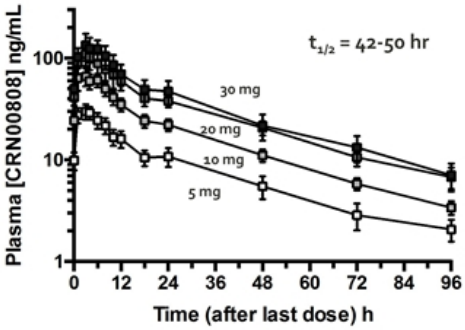
Time-course of plasma CRN00808 trough and IGF-1 concentrations



Dose response of IGF-1 suppression



Plasma concentration on the last day of dosing



PK/PD analysis of the multiple-ascending dose arm. Left chart: Time-course of plasma CRN00808 trough concentrations (black squares) and IGF-1 concentrations (green squares) in the 10 mg MAD cohort. Middle chart: Dose response of IGF-1 suppression on last day of dosing for MAD cohorts compared to placebo (Pbo). All data are mean ± standard error. Right chart: Plasma concentration of CRN00808 on last day of dosing. 30 mg (black circles), 20 mg (white circles), 10 mg (black squares), 5 mg (white squares).

10 mg selected as the initial dose in Phase 2 trials

CRN00808: Conclusions from Phase 1

Potent suppression of GH axis in Phase 1 provided clinical POC

- Clear exposure response relationship observed for GH suppression
- Maximum GH and IGF-1 suppression observed with 10 mg dose

Pharmacokinetics data suggest suitability for once daily oral administration

- CRN00808 was well absorbed in fasted subjects with low variability between subjects
- Exposure reduced when first generation capsule taken with food
- Half-life (42-50 hr) consistent with once daily dosing
- No CYP inhibition observed: No change in midazolam exposure with co-administration

Safety

- 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 CRN00808. These AEs were not dose dependent and also observed in placebo subjects and/or prior to dosing.

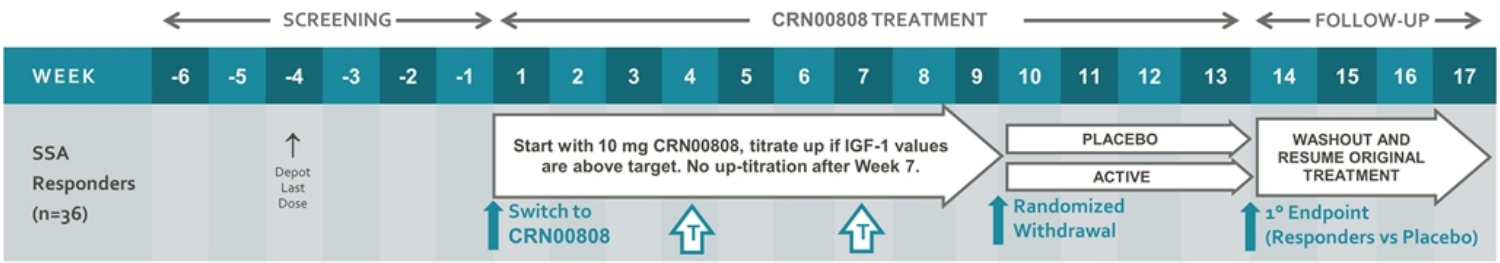
Supports starting dose of 10 mg/day in planned Phase 2 acromegaly trials

Crinetics Pharmaceuticals

CRN00808: Planned Acromegaly Phase 2 Trials: EVOLVE

ACROBAT EVOLVE

ACROBAT EVOLVE Study: Evaluation of CRN00808 vs placebo in patients controlled on injected SSA monotherapy



Entry Criteria: IGF ≤ ULN on SSA depot monotherapy (octreotide LAR or lanreotide)

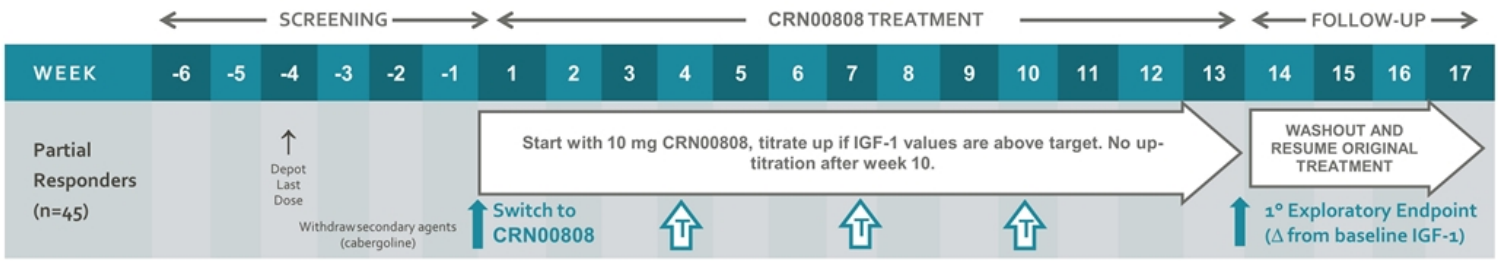
Titrate up if two consecutive IGF-1 values are above ULN.

Crinetics Pharmaceuticals

CRN00808: Planned Acromegaly Phase 2 Trials: EDGE

ACROBAT EDGE

ACROBAT EDGE Study: Exploration of CRN00808 in patients inadequately controlled on injected SSA monotherapy



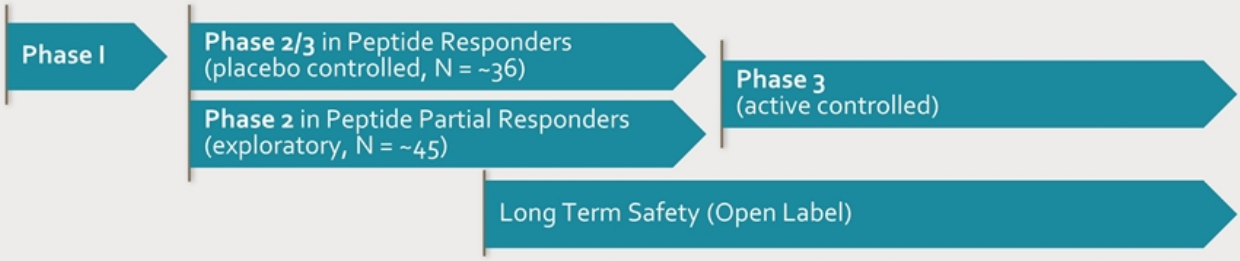
Entry Criteria: Partial responders on SSA monotherapy or when combined with dopamine agonist. Complete and partial responders on combination regimens will also be studied

Titrate up if two consecutive IGF-1 values are above ULN.

Crinetics Pharmaceuticals

CRN00808: Established clinical development strategy based on other approved products

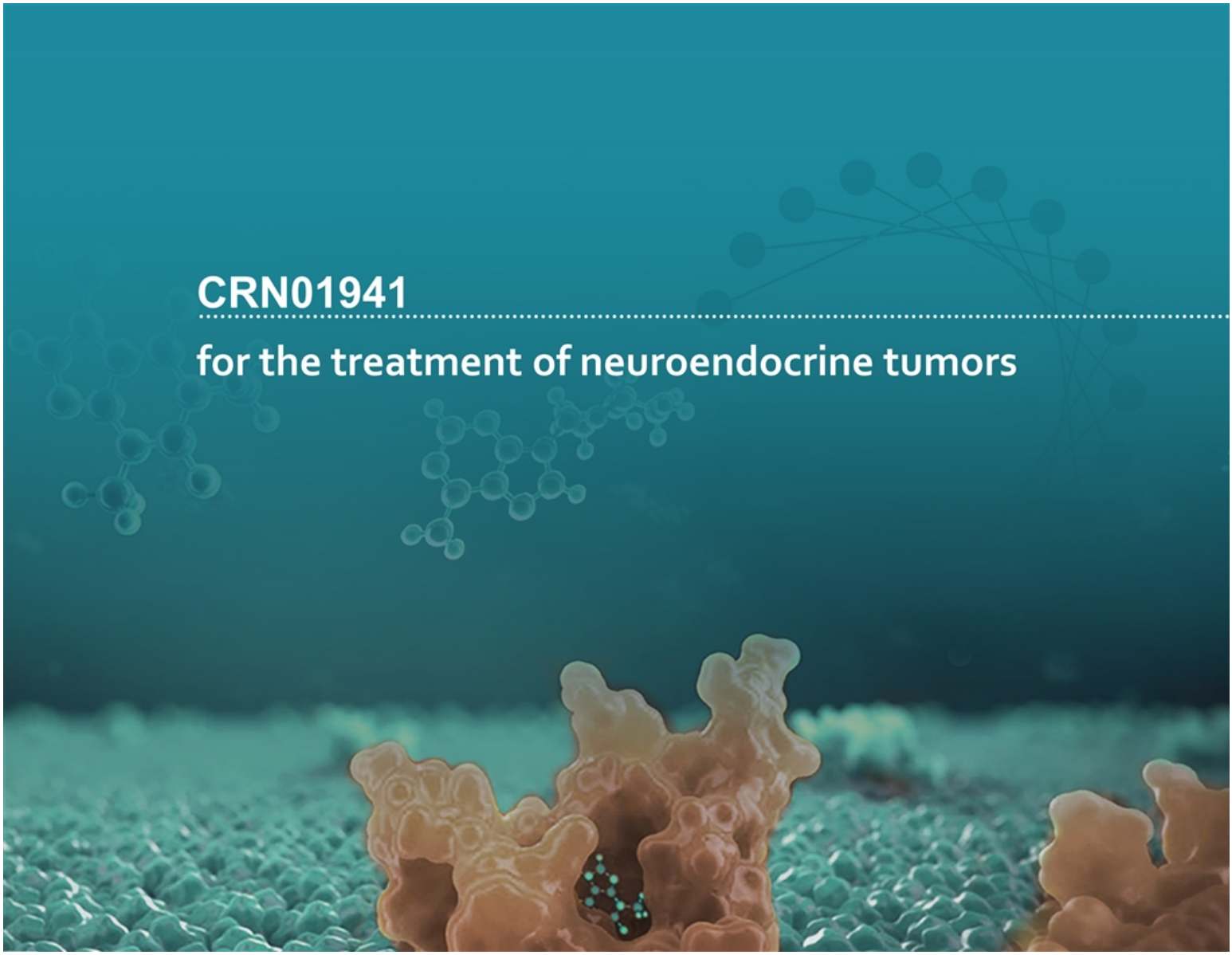
Planned clinical development path outline



Summary of acromegaly registration trials

DRUG (TRIAL)	COMPARATOR	N	PRIMARY ENDPOINT
Somatuline® Depot (lanreotide) Injection	placebo	107	50% GH ↓ @ 4 weeks
	none	63	IGF normalization @ week 48
Oral octreotide	baseline	155	IGF normalization @ month 7
	placebo	50	IGF normalization @ month 9
	octreotide/lanreotide	130	TWA IGF-1 over 9 months
Signifor® (pasireotide) Injection	octreotide/lanreotide	198	GH +IGF normalization @ week 12
	octreotide	358	GH + IGF normalization @ month 12
SOMAVERT® pegvisomant	placebo	112	IGF reduction / normalization @ week 12

Crinetics Pharmaceuticals


CRN01941
for the treatment of neuroendocrine tumors

SSAs: a growing standard of care in NETs

Neuroendocrine tumors (NETs)

Prevalence: ~171,000 patients (U.S.)[1]

Arise from enteroendocrine cells in the GI tract, lung, more rarely, the pancreas

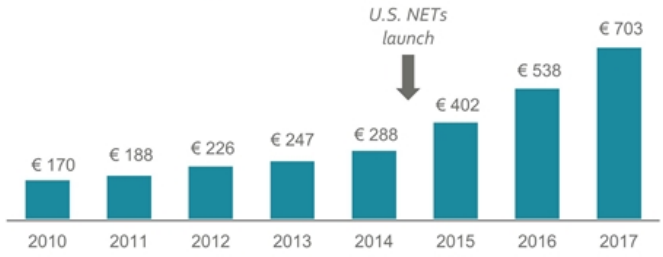
NETs commonly overexpress sst2 receptors

Somatostatin analogs (SSAs) are a standard of care - NCCN guidelines outline SSAs as first line for a large segment of NETs patients

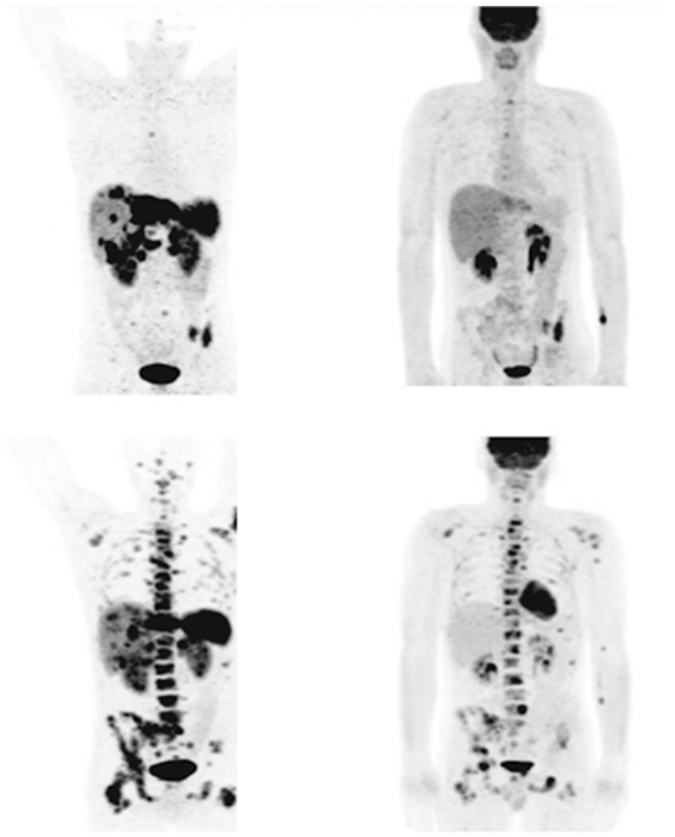
Use of SSAs has been increasing over the last 5 years

- Historically indicated only for carcinoid syndrome
- Data emerging for positive impact of SSAs on progression free survival
- Somatuline NETs treatment label launch 2015

Somatuline® sales – growth driven by NETs label[2]



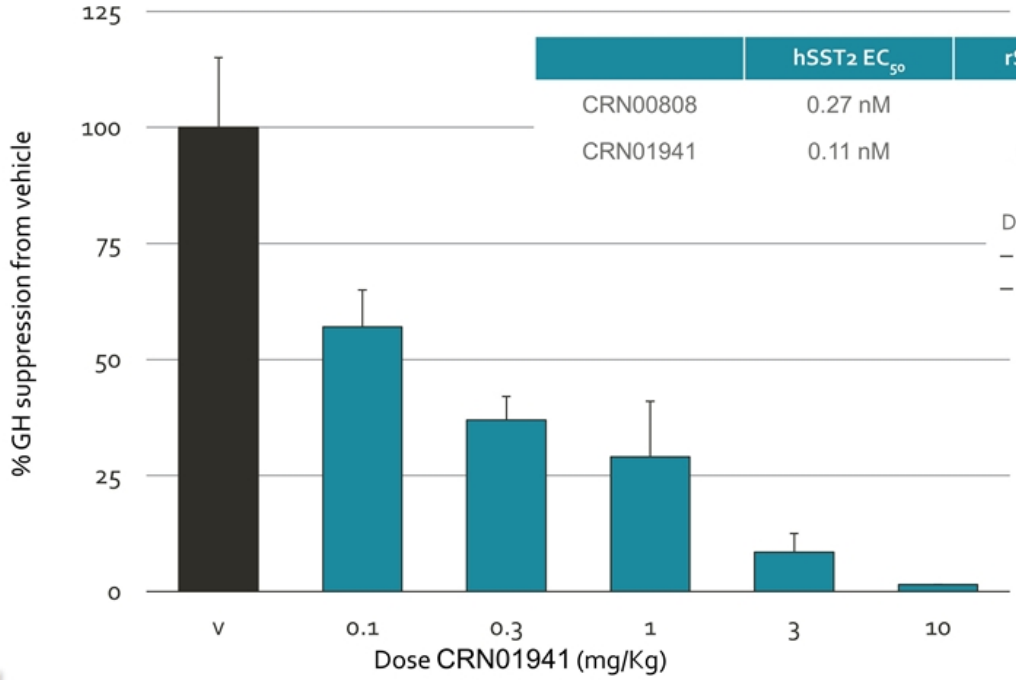
PET scans of NETs patients



Crinetics Pharmaceuticals

Sources: [1] Desari, JAMA Onc 2017; [2] Ipsen financial reports

CRN01941:
Suppression of GHRH-induced GH in preclinical models



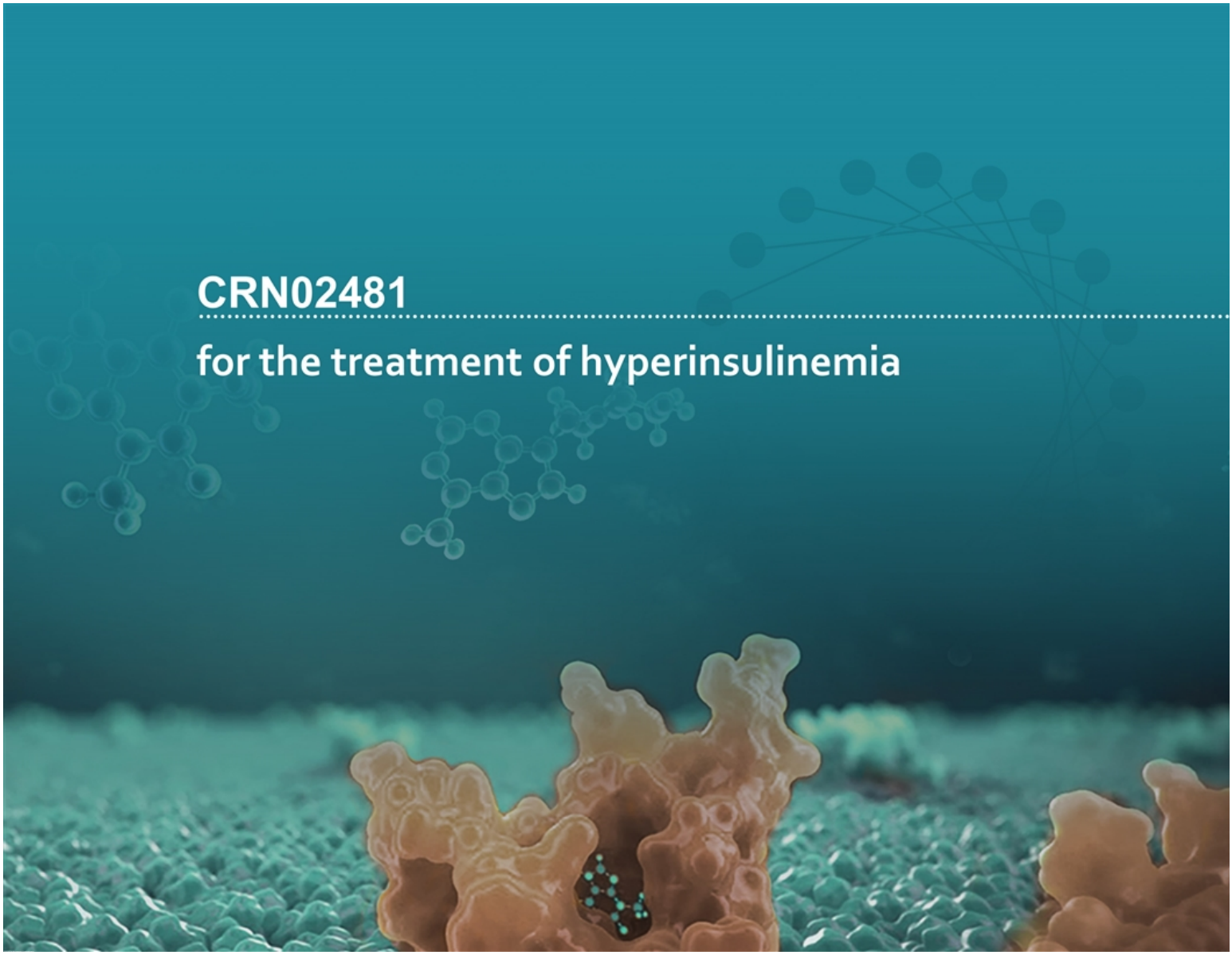
	hSST2 EC_{50}	rSST2 EC_{50}	In vivo EC_{50}
CRN00808	0.27 nM	1.1 nM	11 ng/mL
CRN01941	0.11 nM	0.38 nM	1 ng/mL

Dose CRN01941 at t=oh
- GHRH (iv) at t=3h
- Blood draw at t=3h 10m (GH and PK)

Goal: Initiate a Phase 1 human proof-of-concept clinical trial in the first half of 2019 with results expected in late 2019 / early 2020

CRN02481

for the treatment of hyperinsulinemia



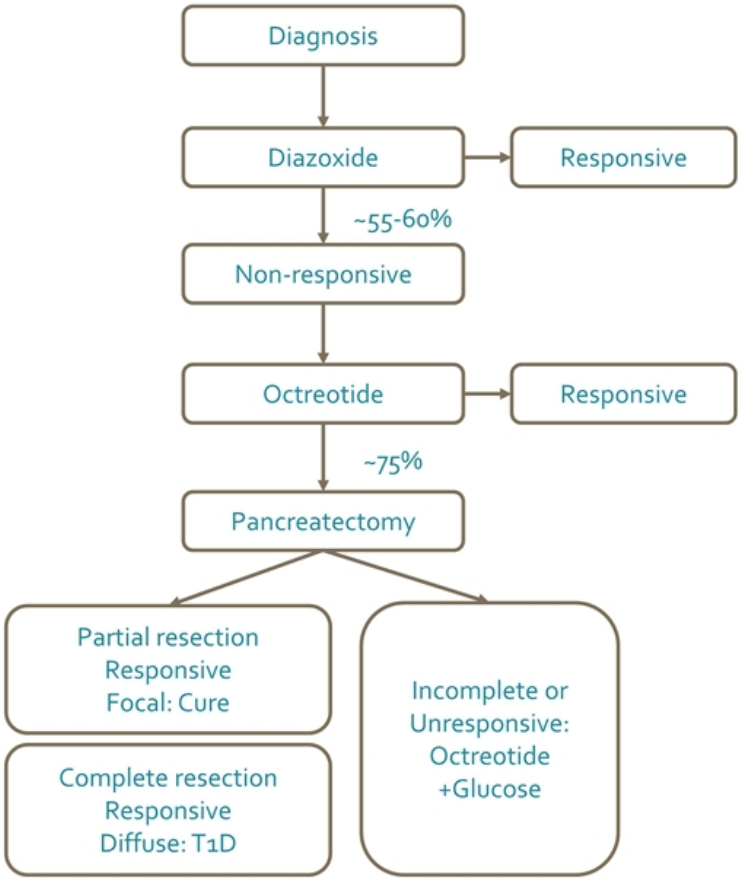
CHI: disease overview and treatment limitations

Indications

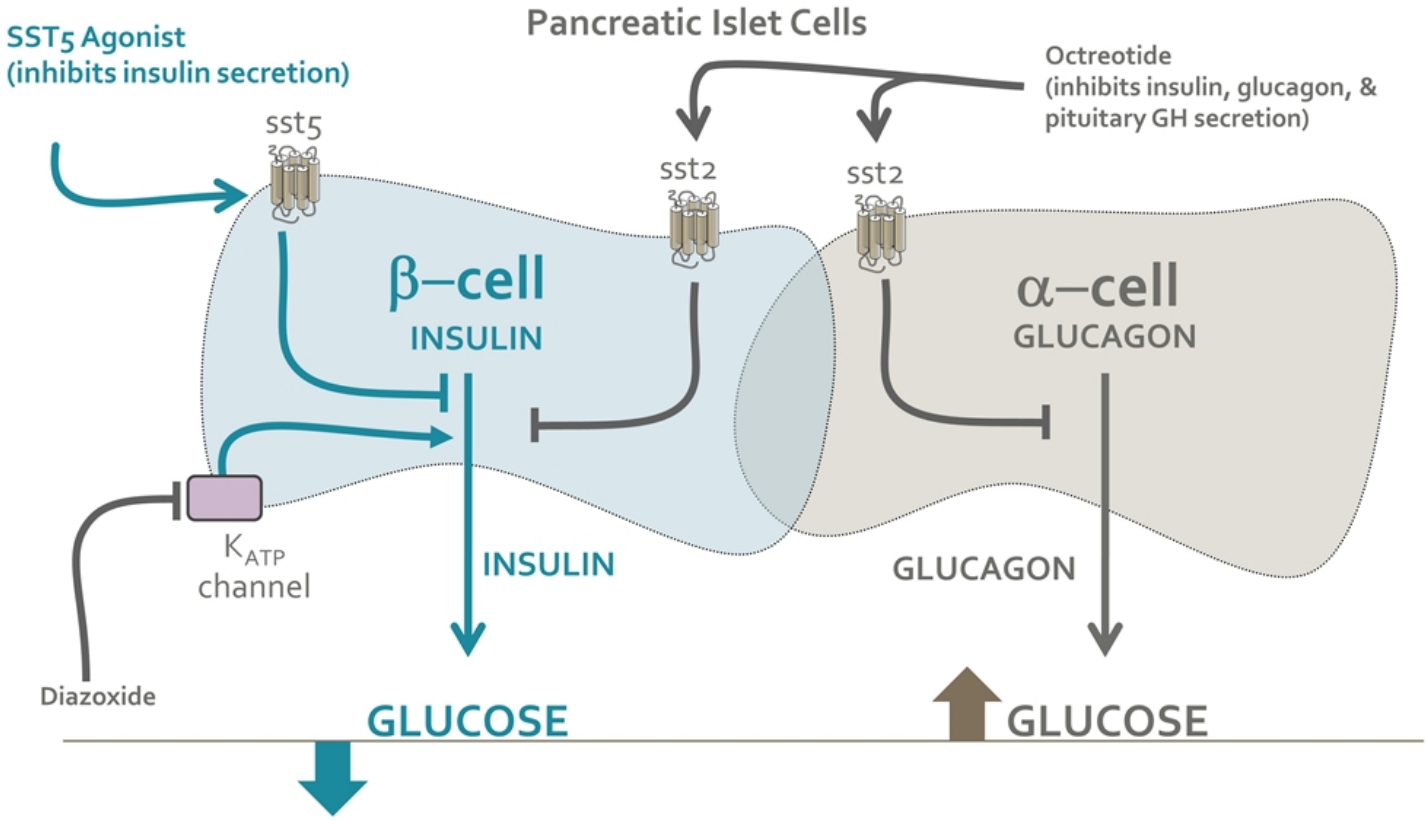
- Congenital hyperinsulinism (CHI)
  - Genetic defects (eg. K_{ATP} channel) results in excess insulin secretion and profound hypoglycemia
- Incidence:
  - 1:30,000 to 1:50,000 births (U.S.)
- Post-bariatric surgery hypoglycemia
- Insulinoma
  - Insulin secreting neuroendocrine tumor
  - Ultra-rare

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

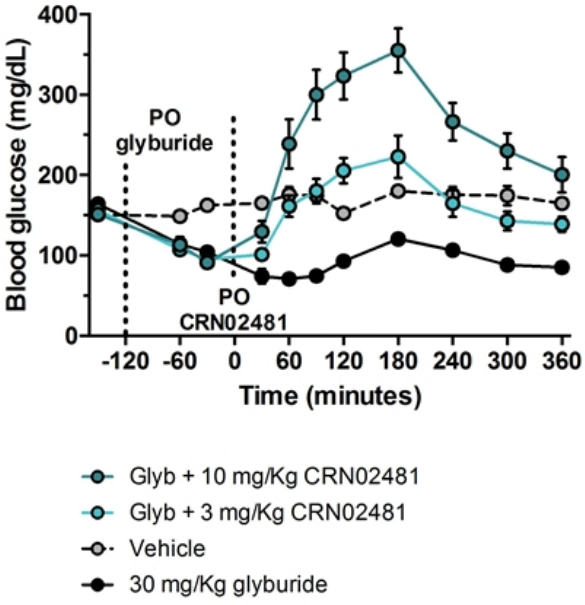


CHI hypothesis: an oral, selective-sst5 drug is the optimal strategy for treating all HI patients



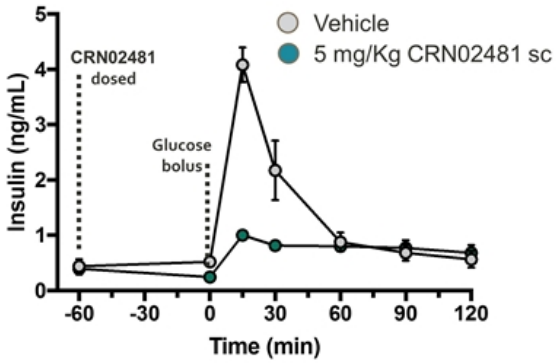
Preclinical results

Rescue of hypoglycemia in rats induced by treatment with sulfonylurea glyburide

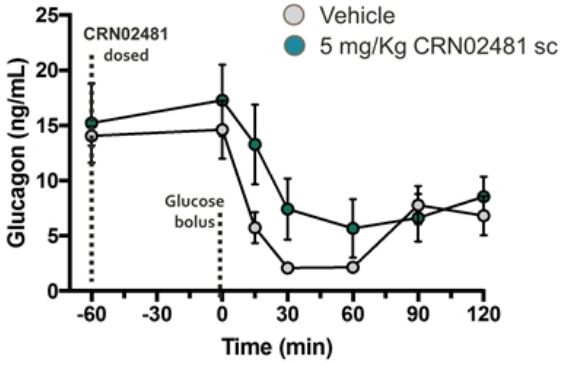


1 mg/Kg ACTH
Iv bolus

In an OGTT, CRN02481 suppressed insulin…



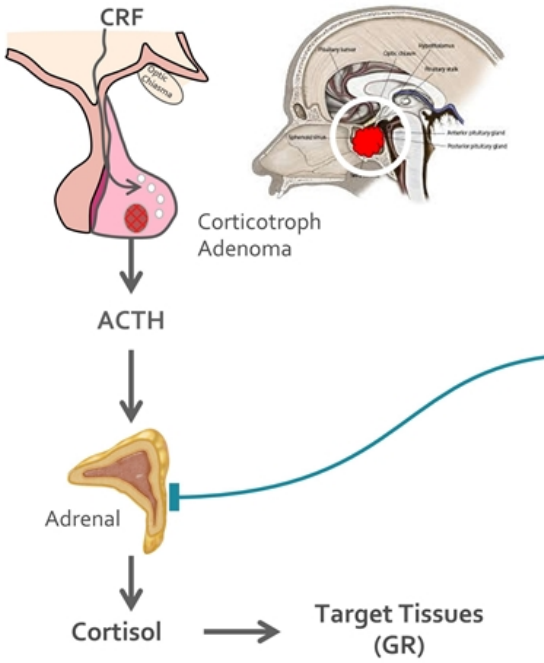
…while maintaining glucagon levels




Cushing's Disease

Cushing's disease overview

Cushing's disease is caused by a benign pituitary adenoma with **excess adrenocorticotropic hormone (ACTH) secretion, resulting in excess cortisol**



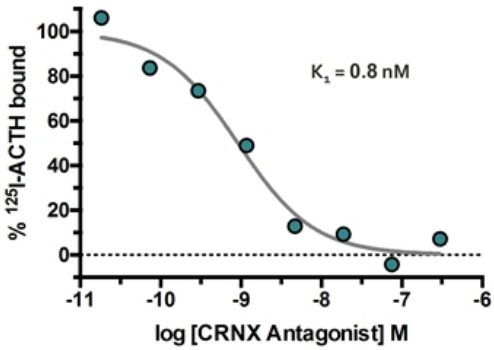
Standardized Mortality Ratio = 2.4
(95% CI, 1.2-3.9)

Source: Dekkers et al (2007) 1210, 2006-2012

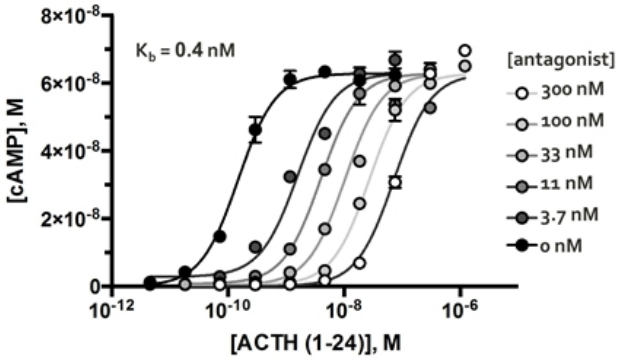
TARGET	EXAMPLES	ISSUES / OPPORTUNITIES
SST5 Agonist	Pasireotide	Limited Efficacy Hyperglycemia
CRF Antagonist	NBI-74788 SPR001	For CAH only
ACTH Antagonist	**Crinetics Pharmaceuticals**	**Potential best-in-class mechanism** **Potential for CAH & other diseases of ACTH excess**
Cortisol Synthesis Inhibitors	Metyrapone Ketoconazole LCI-699 ATR101	Loss of negative feedback Adrenal insufficiency Hyperandrogenism
Glucocorticoid Receptor Antagonists	Mifepristone	Difficult to dose and monitor Anti-progesterone activity

A prototype antagonist of ACTH with high potency in vitro observed in preclinical studies

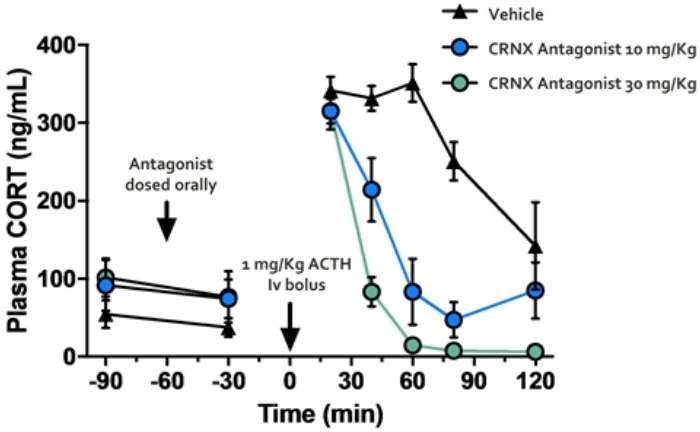
Competition radio-ligand binding assay



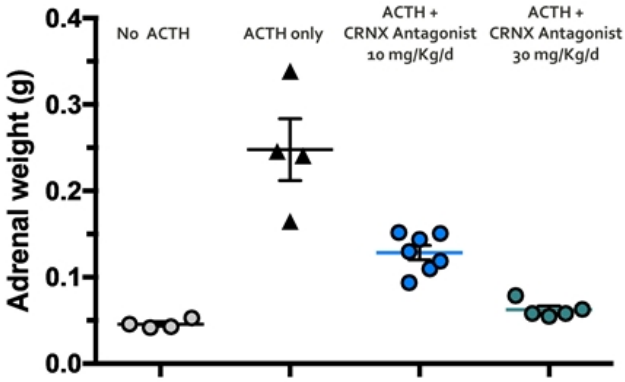
Schild analysis of functional antagonism



In vivo POC: acute suppression of ACTH-induced corticosterone in rats



In vivo POC: repeat antagonist dosing (7d) rescues adrenal hypertrophy from chronic ACTH infusion



Crinetics Pharmaceuticals

Financial Overview

Strong financial position

- $174.8 million pro forma cash and cash equivalents based on
  - $68.4 million in cash and cash equivalents as of June 30, 2018
  - $106.4 million net proceeds from IPO in July 2018
- No debt
- 24,024,231 common shares outstanding as of August 24, 2018

Crinetics
Pharmaceuticals


Crinetics
Pharmaceuticals
Appendix

Directors and Advisory Board

BOARD OF DIRECTORS

Wendell Wierenga, PhD	Chairman (Former EVP R&D, Santarus)	   
Scott Struthers, PhD	Founder & CEO	 
Mason Freeman, MD	Venture Partner, 5AM; Endocrinologist at MGH	   
Steve Kaldor, PhD	Former CEO, Quanticel	   
Matt Fust	Former CFO, Onyx	   
Jack Nielsen	Managing Director, Vivo Capital	  
Weston Nichols, PhD	Analyst, Perceptive Advisors	   

SCIENTIFIC ADVISORY BOARD

David Clemmons, MD	Professor of Medicine at UNC, Chapel Hill	  
Anne Klibanski, MD	Chief of Neuroendocrine Unit at MGH & Professor of Medicine at Harvard