UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, DC 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): June 14, 2019

REZOLUTE, INC.
(Exact Name of Registrant as Specified in its Charter)

Delaware	000-54495	**27-3440894**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

201 Redwood Shores Pkwy, Suite 315, Redwood City, CA	94065
(Address of Principal Executive Offices)	(Zip Code)

Registrant's Telephone Number, Including Area Code: (650) 206-4507

(Former Address, 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))

Securities registered or to be registered pursuant to Section 12(b) of the Act.

Item 9.01 Financial Statements and Exhibits

Exhibit No.	**Description**
99.1	Corporate Presentation 3Q 2019

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.

REZOLUTE, INC.

Date: June 14, 2019

By: /s/ Keith Vendola
Name: Keith Vendola
Title: Chief Financial Officer

EXHIBIT INDEX

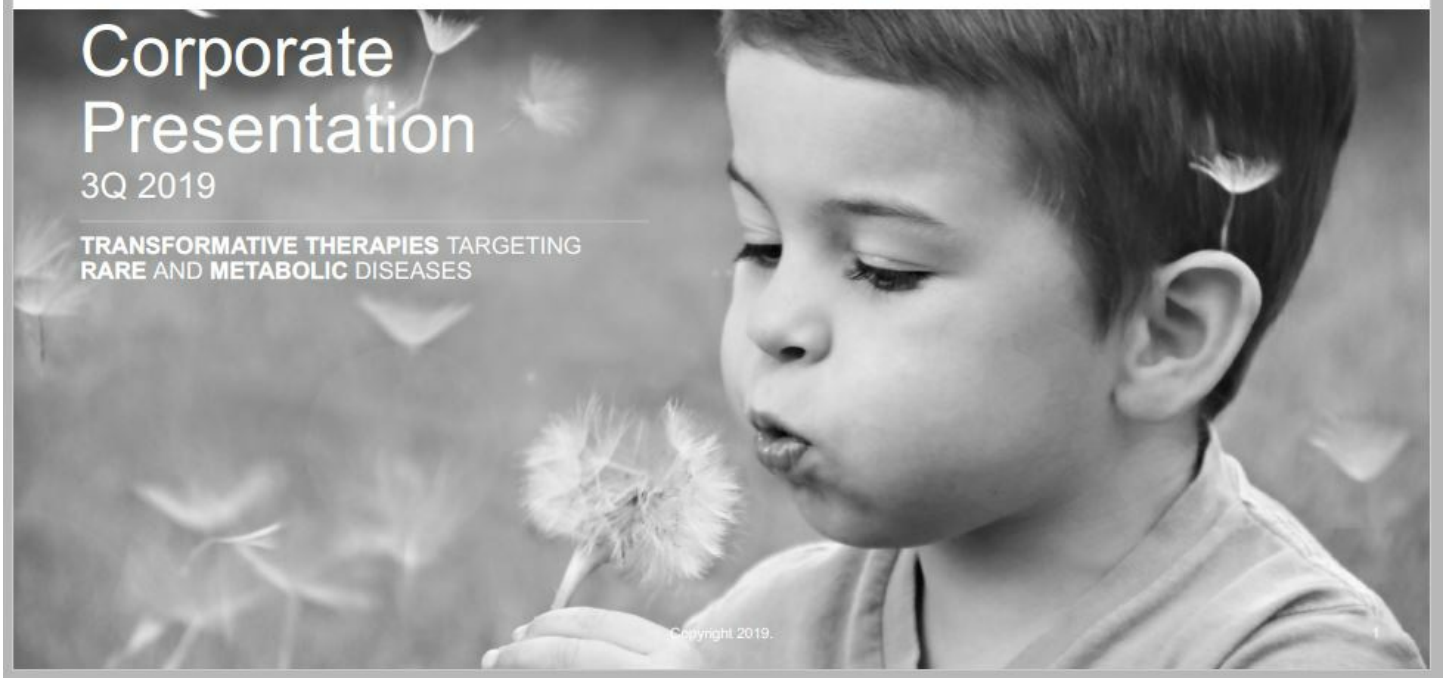
Exhibit No.	**Description**
99.1	Corporate Presentation 3Q 2019

REZOLUTE

Corporate Presentation

3Q 2019

TRANSFORMATIVE THERAPIES TARGETING
RARE AND **METABOLIC** DISEASES



Forward-Looking Statements

Statements in this presentation that are not descriptions of historical facts are forward-looking statements relating to future events, and as such all forward-looking statements are made pursuant to the Securities Litigation Reform Act of 1995. Statements may contain certain forward-looking statements pertaining to future anticipated or projected plans, performance and developments, as well as other statements relating to future operations and results. Any statements in this presentation that are not statements of historical fact may be considered to be forward-looking statements. Words such as "may," "will," "expect," "believe," "anticipate," "estimate," "intends," "goal," "objective," "seek," "attempt," or variations of these or similar words, identify forward-looking statements.

These forward-looking statements by their nature are estimates of future results only and involve substantial risks and uncertainties, including but not limited to risks associated with the uncertainty of future financial results, additional financing requirements, development of new products, successful completion of the Company's proposed restructuring, the impact of competitive products or pricing, technological changes, the effect of economic conditions and other uncertainties detailed from time to time in our reports filed with the Securities and Exchange Commission.

There can be no assurance that our actual results will not differ materially from expectations and other factors more fully described in our public filings with the U.S. Securities and Exchange Commission, which can be reviewed at www.sec.gov.

Rezolute: a Metabolic & Orphan Disease Company

RZ358	Antibody for an **ultra-orphan** indication, Congenital Hyperinsulinism (CHI), potential first in class and best in class therapy
RZ402	**Oral** Plasma Kallikrein Inhibitor (PKI) for Diabetic Macular Edema (DME) with in vivo pharmacology demonstrating comparable efficacy relative to anti-VEGF therapies
AB101	**Weekly** insulin with the potential to transform insulin management by reducing therapeutic burden and improving compliance

Transformative therapies targeting well-known genetic pathways and mechanisms of action

Diversified Pipeline at Multiple Stages of Development

Program	Description	Preclinical	Phase 1	Phase 2
RZ358	Antibody for CHI	Phase 2b dosing anticipated 2H'19		
RZ402	Oral PKI for DME	IND anticipated mid-year '20		
AB101	Weekly insulin	Top-line results anticipated 2H'19		

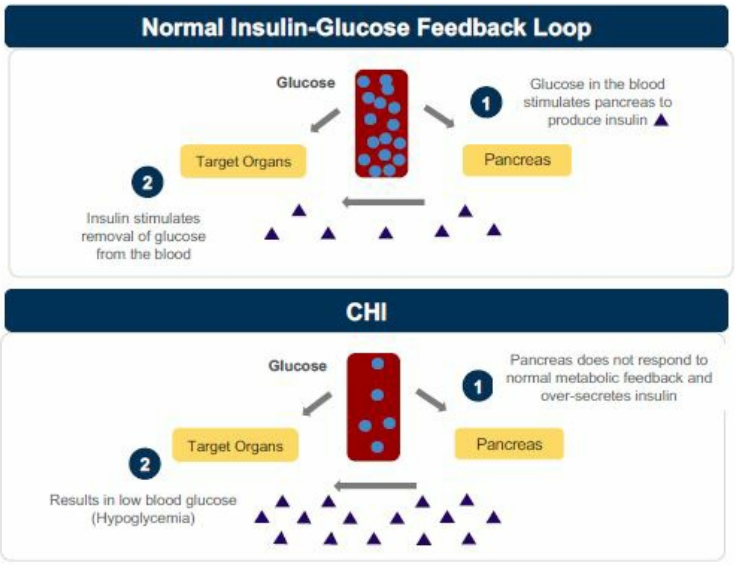
Multiple near-term inflection points anchored by a late-stage orphan program

RZ358

RZ402
AB101

Congenital Hyperinsulinism (CHI)

- Ultra-rare disease
- Caused by one of 11 known mutations, leading to excessive insulin secretion
- Most common cause of persistent hypoglycemia in infants and children
- Persistent hypoglycemia increases risk of neurologic complications, coma and death
- Signs/symptoms often not recognized until life-threatening
- Patients and families live in fear of hypoglycemia
- Existing therapies are suboptimal



Observational Study Demonstrates Unmet Need

Conducted in partnership

Continuous Glucose Monitoring (CGM) for Two Weeks: Summary of Results

- Blood glucose <70 mg/dL is hypoglycemia
- On average, patients had ~3 hours / day (~180 min) of hypoglycemia, even on standard of care (SOC) medications
- Younger subjects are particularly vulnerable

Glucose Threshold (mg/dL)	All Patients		Patients on SOC Medication	
	All Ages (N = 22)	2-6 Year Olds (N = 12)	All Ages (N = 15)	2-6 Year Olds (N = 9)
<70	174	207	174	223
<60	56	74	54	81
<50	15	22	14	24

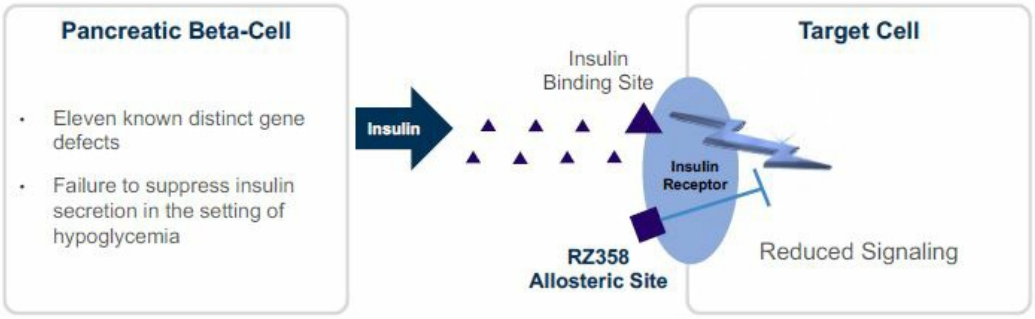
CGM reveals current therapies are ineffective at controlling hypoglycemia

Potential First and Best in Class Therapy for CHI

RZ358

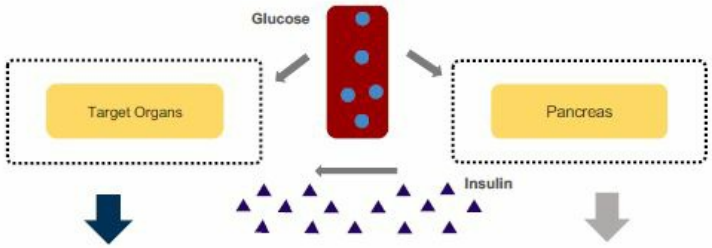
Mechanism	Preclinical	Clinical	CMC	Market
• Fully humanized monoclonal antibody • Allosteric mechanism of action (MOA) down modulates insulin effects when insulin is elevated • MOA is uniquely suited for CHI	• Multiple animal models demonstrate dose-dependent improvement in hypoglycemia with no hyperglycaemias • Chronic toxicology complete	• Safe and well-tolerated • Phase 1 and 2 studies show proof of concept • Phase 2b-ready	• Fully enabled • Phase 2b-ready	• Ultra-orphan • Priority review voucher

Unique Mechanism Attenuates Insulin Effects



- High affinity binding to the insulin receptor at the allosteric site
- Highly selective to the insulin receptor (no IGF-1 interaction)
- Insulin still binds and signals
- Dims the insulin signal when insulin is elevated

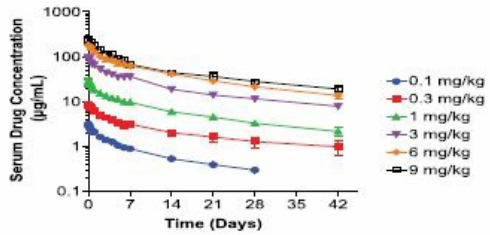
Potential to Address Limitations of Standard of Care



	RZ358 (Broad Focus)	Standard of Care (Narrow Focus)
Development	▲ Tailored for CHI	▼ Not developed for CHI
Targeting	▲ Insulin receptor/signal on insulin-dependent target tissues	▼ Beta cell only
Relevancy	▲ Potentially universal treatment	▼ Genetics-dependent narrow targeting
Impact	▲ Reversibly counteracts insulin only when insulin is elevated	▼ Marginally effective, invasive, and/or significant side effects

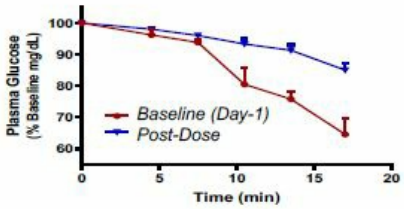
Phase 1 – Completed Proof of Mechanism

PK



- Single IV doses 0.1 to 9 mg/kg to healthy volunteers
- Dose-dependent PK with a half-life of 15 days

Hyperinsulinism: Insulin Tolerance Test



- RZ358 uniformly prevents hypoglycemia following insulin administration
- PK-PD correlation (dose-response)
- No hyperglycemia
- Effect persists for 2 weeks

Safe and well-tolerated

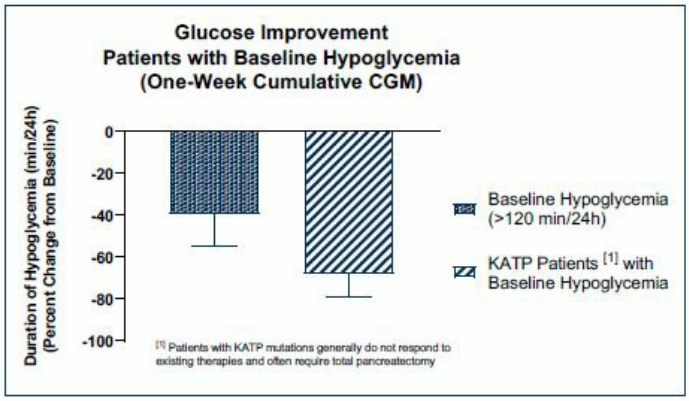
Phase 2a – Completed Proof of Concept

Design

- Single IV doses of 1 to 9 mg/kg in patients with CHI
- Ages ≥ 12 in Europe and ≥ 18 in the US

Results

- PK comparable with healthy volunteers
- Baseline and post-treatment CGM
  - **Up to 70% improvement in patients with baseline hypoglycemia**
  - No hyperglycemia
  - Effect persisted for 2 weeks, consistent with Ph1 PK/PD
  - Established proof-of-concept in CHI patients
- Safe and well-tolerated



2H '19 Phase 2b Study Overview

- **Design**: Open-label, repeat-dose study in 4 sequential dosing cohorts
- **Population**: CHI ≥ 2 years old with baseline hypoglycemia (<70 mg/dL for ≥ 2 hours per day by CGMS)
- **Principal assessments / endpoints**: Duration of hypoglycemia measured by CGMS
- **Interim Analysis**: Open label design provides opportunity for interim discussions with health authorities

Dosing Cohort	Induction				Extended Dosing	
	Weekly RZ358 for 4 weeks (mg/kg)				RZ358 for 8 weeks	
	Week 1	Week 2	Week 3	Week 4	mg/kg	Interval
1	3	3	3	3	3	14 Days
2	6	6	6	6	6	14 Days
3	9	9	9	9	9	14 Days
4	3	6	9	12	9	14 Days

RZ358

RZ402

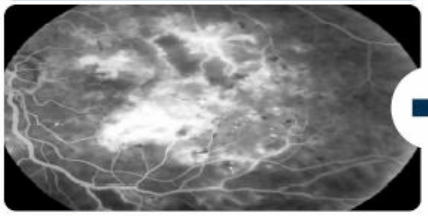
AB101

Diabetic Macular Edema (DME)

Primary Vision Complication Associated with Diabetes

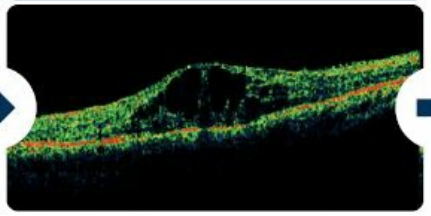
Increased Retinal Vascular Permeability

Leakage of fluid from vascular compartment into the retina



Macular Edema

Swelling of the central macula



Progressive Vision Loss and Blindness

Risk factors

- Elevated blood glucose
- High blood pressure
- Duration of diabetes

Large and growing population

- 750,000+ people in US
- 21M+ worldwide

Leading cause of blindness in working age adults

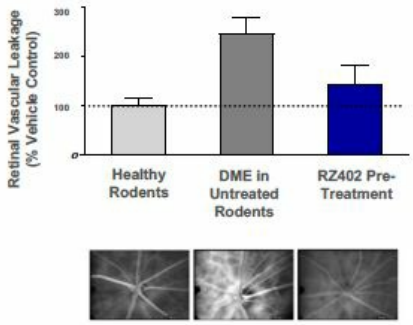
Potential Best and First in Class Therapy for DME

RZ402

Mechanism	Preclinical	Clinical	Target Profile
• Plasma kallikrein inhibitor targeting vascular leakage • Potent target inhibition in rat, monkey, and human plasma	• Suppresses vascular leakage by 90% or more • Animal toxicology pending	• Pre-IND meeting completed • Clear path towards Phase 1 initiation	• Oral dosage • Daily dosing • Large market

Systemic Administration Reduces Retinal Vascular Leakage

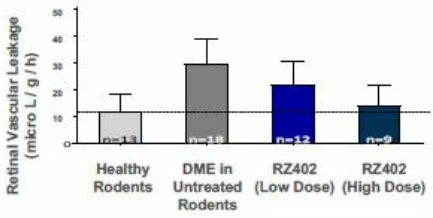
Dosing with RZ402 *at Onset* of Model Induction Prevents Formation of DME



Efficacy Achieved

- Up to 90% reduction in DME Measures
- Works in both a preventive and interventional setting
- On par with VEGF therapies in similar models

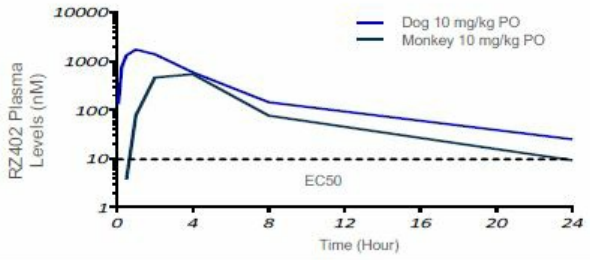
Dosing with RZ402 *after Onset* of DME Reverses DME

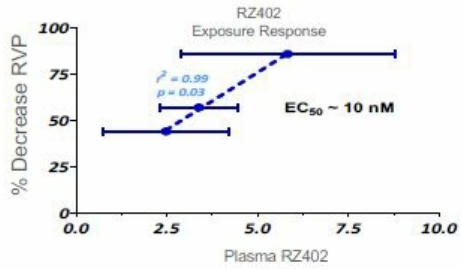


Dose-dependent effect

Exposure-Response Predicts Once Daily Dosing

ANIMAL MODEL	END POINT	RZ402 DOSE (SC ROA)	INHIBITION
Hypertension	**Retinal Plasma Leakage**	0.2 – 0.4 mg/kg/day	**60-90%**
Diabetes	**Retinal Plasma Leakage**	0.25 – 0.6 mg/kg/day	**43-86%**
Diabetes	**Hematoma Expansion**	0.4 mg/kg/day	**85%**
Retinal Hemorrhage	**Retinal Leukostasis**	1 mg/kg/day	**>90%**



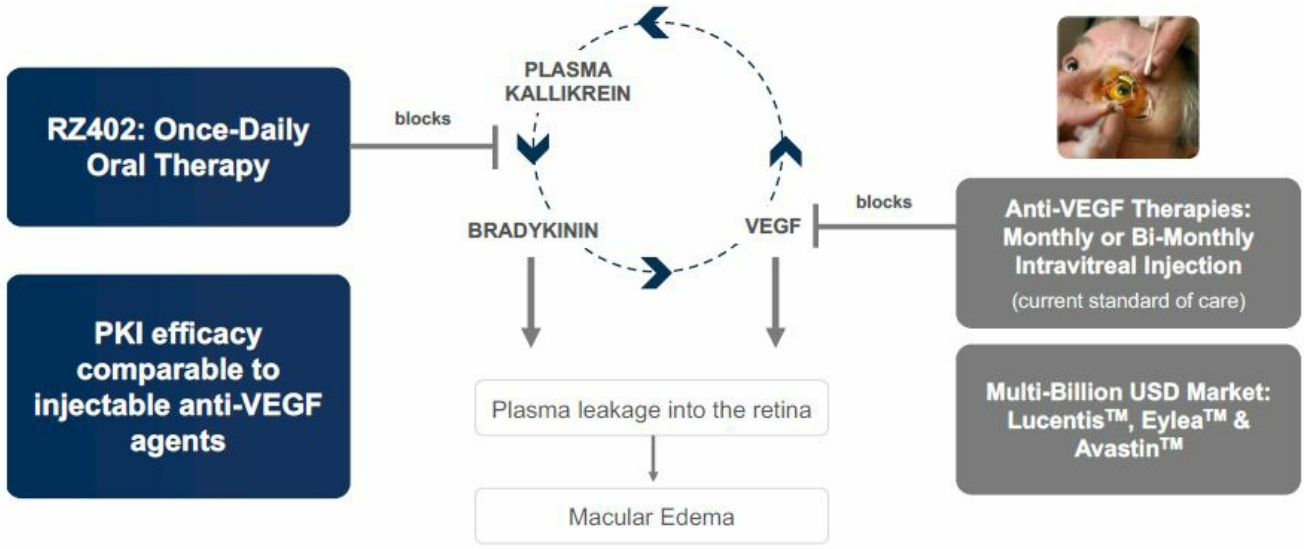


Efficacious concentrations exceeded for the full 24 hour dosing interval after oral administration to dogs and monkeys

Common Pathways with Different Routes of Administration

RZ402: Once-Daily Oral Therapy

PKI efficacy comparable to injectable anti-VEGF agents



Anti-VEGF Therapies: Monthly or Bi-Monthly Intravitreal Injection
(current standard of care)

Multi-Billion USD Market: Lucentis™, Eylea™ & Avastin™

RZ358
RZ402

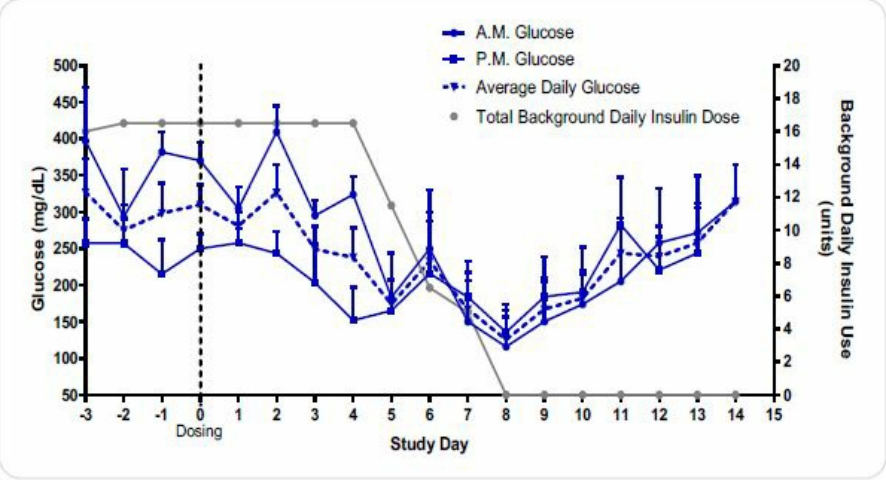
AB101

Potential Best and First in Class Super Long Acting Basal Insulin

AB101

Mechanism	Preclinical	Clinical	Target Profile
• Uniform pegylated-insulin microspheres enabling controlled and sustained long-acting release • Proprietary microsphere platform	• Proof-of-concept in 3 species presented at ADA • Peak-less steady-state profile supports weekly dosing • Efficacious doses in animals translatable to human dosing	• Phase 1 clinical study dosing initiated in 3Q '17 • Data read out anticipated 2H '19	• >$20 billion insulin market • No therapies >24-36 hours • Weekly subcutaneous injection

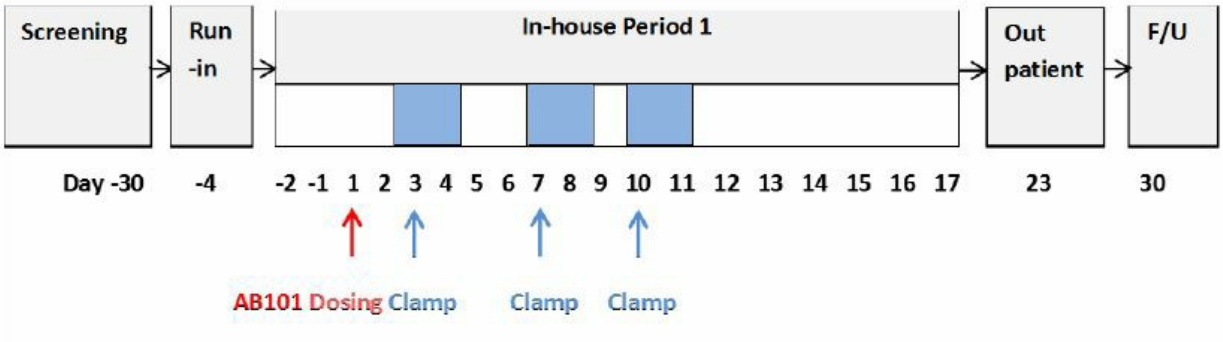
In Vivo Proof of Concept – AB101 Dosing in Diabetic Mini Pigs



- Near-normalization of both fasting AND post-meal glucose
- Reduction and removal of background insulin
- Reduction in glucose variability (intra-day and inter-day

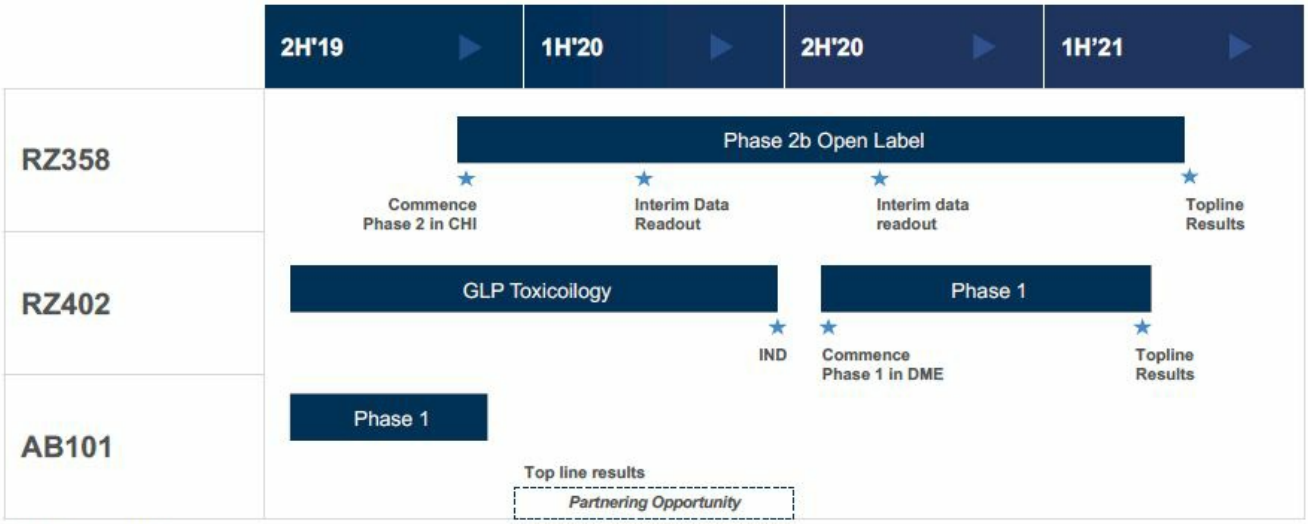
Phase 1 – First-in-Human Study Overview

- Single ascending dose study underway assessing the safety and tolerability, pharmacokinetics, and pharmacodynamics of AB101 in subjects with Type 1 Diabetes Mellitus
- Objective is to show a safe, sustained, and relatively peak-less PK-PD profile that enables extended release dosing



MILESTONES

Anticipated Near Term Milestones



Milestones +/- a quarter

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