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): **March 29, 2017**

vTv Therapeutics Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-37524**	**47-3916571**
(State or other jurisdiction of incorporation)	(Commission File No.)	(IRS Employer Identification No.)

4170 Mendenhall Oaks Pkwy
High Point, NC 27265
(Address of principal executive offices)

(336) 841-0300
(Registrant's telephone number, including area code)

NOT APPLICABLE
(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 (*see* General Instruction A.2. below):

☐ 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))

Item 7.01 Regulation FD Disclosure

Representatives of vTv Therapeutics Inc. (the "Company") are scheduled to display a poster entitled "TTP399: A Liver-Selective and Therapeutically Viable Glucokinase Activator: Results from a 6-Month Phase 2 Study" at the Seventeenth Annual Levine-Riggs Diabetes Research Symposium in Orlando, FL on March 29, 2017. The poster presentation is attached hereto as Exhibit 99.1 and the associated abstract is attached hereto as Exhibit 99.2.

The information in Item 7.01 of this Form 8-K (including Exhibit 99.1 and 99.2) shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference under the Securities Act of 1933, as amended, except as expressly set forth by specific reference in such a filing, regardless of any general incorporation language in any such filing, unless the Company expressly sets forth in such filing that such information is to be considered "filed" or incorporated by reference therein. The information set forth in the exhibits to this Form 8-K relating to this item 7.01 shall not be deemed an admission as to the materiality of any information in this report that is required to be disclosed solely to satisfy the requirements of Regulation FD.

Item 9.01 Financial Statements and Exhibits

(d) Exhibits

Exhibit No.	Description
99.1	Poster Presentation entitled "TTP399: A Liver-Selective and Therapeutically Viable Glucokinase Activator: Results from a 6-Month Phase 2 Study"
99.2	Abstract entitled "TTP399: A Liver-Selective and Therapeutically Viable Glucokinase Activator: Results from a 6-Month Phase 2 Study"

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.

VTV THERAPEUTICS INC.

By:	/s/ Rudy C. Howard
Name:	Rudy C. Howard
Title:	Chief Financial Officer

Dated: March 29, 2017

EXHIBIT INDEX

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TTP399: A Liver-Selective and Thera

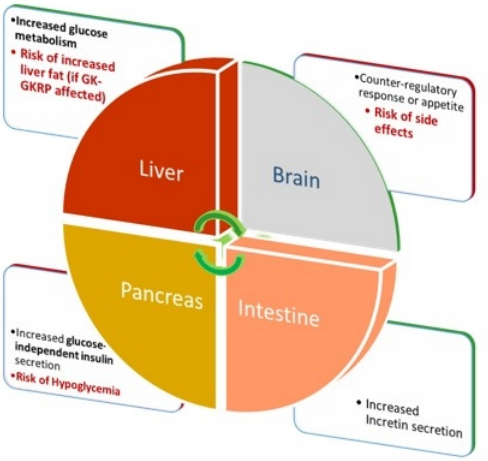
Adrian Vella[1], Jenn

[1]Mayo

Introduction

The therapeutic promise of glucokinase activators (GKAs) for type 2 diabetes has been limited by adverse events such as hypoglycemia and steatohepatitis, and lack of durability.
The clinical characteristics of patients with GK-activating mutations or GK regulatory protein (GKRP) loss of function mutations suggest that liver-selective GKAs that do not activate GK in β-cells or affect the GK-GKRP interaction would present a superior profile. Guided by this evidence, we discovered and have developed TTP399.

- **Liver selective:** TTP399 does not activate GK in the beta-cells, reducing risk of hypoglycemia
- MOA preserves the natural interaction of **GK-GKRP** in the liver, reducing risk of increase in lipids



Aim

The goals of this 6 month, randomized, double-blind, placebo- and active-controlled, parallel group trial in type 2 diabetics on stable doses of metformin were to prove:

1. Clinically relevant reduction in HbA1c
2. Sustained effect over six months of treatment
3. Negligible hypoglycemia and hyperlipidemia

AGATA Study Design

- 190 subjects randomized
- 7.0-9.5% baseline HbA1c
- Arms (1:1:1:1)
  - Placebo
  - TTP399 400mg once daily
  - TTP399 800mg once daily
  - Active comparator (sitagliptin / DPP-4 inhibitor)
- **Primary endpoint:** Change from baseline in HbA1c at 6 months

Disposition o

	Total	Plac
Subjects Randomized	**190**	**4**
Subjects Treated	189	4
Subjects completing dosing period	**124**	**36 (7**
Subjects completing the study (dosing period+follow-up visit)	115	3
Subjects terminated prematurely	**74**	**1**
Lost to follow-up	22	4
Withdrawal by subject	9	1
Adverse event	8	1
Protocol Deviations	2	2
Rescued:		
According to Protocol criteria	24	5
Inadequate glucose control (Investigator decision)	3	1
Other	6	2

Demography – Baseli

	Placebo (n=48)	
Age (years): Mean	56	
Gender: males; females (% males)	27; 21 (56%)	
Ethnicity: Not Hispanic or Latino (%)	27 (56%)	
Race: White (%)	39 (81%)	
Weight (kg): Mean	91.6	
Height (cm): Mean	168	
BMI (kg/m2): Mean	32	
Baseline HbA1c: Mean (Median)	7.9 (7.6)	
Metformin Dose: Mean (Median)	1693 (2000)	

Safety Ov

	Placebo (n=48)	4
Subjects with any treatment-emergent adverse event (TEAE)	29 (60%)	
Subjects with any serious AE	0	
Subjects with any related TEAE	4	
Subjects with TEAE leading to discontinuation from the study	1	
Subjects with related TEAE leading to discontinuation *- Related TEAEs leading to discontinuation of study*	0 ---	
Subjects meeting DILI criteria	0	
ALT increased	0	
AST increased	0	
Bilirubin increased	0	
Alkaline phosphatase increased	0	
Hypertension and blood pressure increase	1	
Subjects with ICH E14 criteria: QTc>480 msec	0	
Subjects with ICH E14 criteria: QTc change > 60 msec	0	

TTP399: A liver-selective and Therapeutically Viable Glucokinase Activator. Results for a 6-Month Phase 2 Study

Adrian Vella[1], Jennifer Freeman[2], Chris Dvergsten[2], Imogen Dunn[2] and Carmen Valcarce[2].

[1]Mayo Clinic, Rochester, MN, USA. [2]vTv Therapeutics LLC, High Point, NC, USA

The critical role of glucokinase (GK) in the regulation of glucose homeostasis is reinforced by the fact that mutations in the gene encoding GK can cause both hyper-and hypoglycemia. Furthermore, direct and indirect evidence suggest that a defect in hepatic glucose phosphorylation might underlie the lack of suppression of hepatic glucose production during hyperglycemia in human diabetes. Based on this, strategies to increase the activity of GK have been proposed as a novel approach for the treatment of type 2 diabetes. However, the therapeutic promise of glucokinase activators (GKAs) for type 2 diabetes has been limited by adverse events such as hypoglycemia and steatohepatitis, and lack of durability.

The clinical characteristics of patients with GK-activating mutations or GK regulatory protein (GKRP) loss of function mutations suggest that liver-selective GKAs that do not activate GK in β-cells or affect the GK-GKRP interaction would present a superior profile. Moreover, data from transgenic animals shows that a selective increase in hepatic GK is the only requirement for the normalization of the metabolic profile in insulin deficient animals. Guided by this evidence, we discovered and have developed TTP399, a liver-selective GKA that does not appear to disrupt the interaction between GK and GKRP.

In the Phase 2 AGATA study, a six-month, randomized, double-blind, placebo-and active-controlled parallel group trial of TTP399 in type 2 diabetes, 190 patients with type 2 diabetes on stable doses of metformin were randomized (1:1:1:1) to TTP399 400 mg once daily; TTP399 800 mg once daily; sitagliptin 100 mg once daily; or placebo once daily. The patient population was approximately half male (53%), predominantly white (82%), and majority white non-Hispanic or non-Latino (59%). The mean (±SD) age, HbA1c and BMI at baseline were 55 years ±10, 8.0% ±0.7% and 32.8 kg/m^2 ± 5.6 kg/m^2, respectively.

Placebo-subtracted changes in HbA1c at month 6 were –0.9% for TTP399 800 mg ($p<0.01$), –0.2% for TTP399 400 mg, and –1.0% for sitagliptin. Treatment with TTP399 800 mg, but not with placebo, sitagliptin or TTP399 400mg, was associated with a significant increase in HDL-C (3.2 mg/dL; $p< 0.05$), decrease in fasting plasma glucagon (19.6 pg/dL; $p=0.012$), and decrease in weight (–3.4kg, $p < 0.05$, in patients weighing ≥100 kg). More significantly, TTP399 did not cause hypoglycemia or adversely affect blood pressure or lipid profiles.

The totality of the evidence from our preclinical and clinical data suggest that TTP399 may be the first therapeutically viable member of this class. These findings also demonstrate the importance of tissue selectivity and the preservation of physiological regulation when targeting key metabolic regulators such as GK. Additional research on the clinical effects of TTP399 in a larger clinical trial is needed to confirm these promising results.