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): June 4, 2017

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

Nevada	001-37471	EIN 30-0784346
(State of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

255 State Street, 9th Floor
Boston, MA
United States
(Address of principal executive offices, including zip code)

Registrant's telephone number, including area code: 857-246-8998

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 (17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2).
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.

On June 4, 2017, Pieris Pharmaceuticals, Inc. presented Phase 1b study results for its anemia program, PRS-080#022-DP, at the 54th European Renal Association & European Dialysis and Transplant Association (ERA-EDTA) Congress in Madrid, Spain. The poster is furnished as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated by reference herein.

Attached hereto as Exhibit 99.2 and incorporated by reference herein is press release regarding the presentation at ERA-EDTA.

The information set forth under this "Item 7.01. Regulation FD Disclosure," including the exhibits attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, nor shall it be deemed incorporated by reference into any filing under the Securities Act of 1933, as amended, except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits

(d) *Exhibits.*

99.1 Poster of Pieris Pharmaceuticals, Inc.
99.2 Press Release dated June 4, 2017.

SIGNATURE

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.

Dated: June 5, 2017

PIERIS PHARMACEUTICALS, INC.

By: /s/ Lance Thibault
Name: Lance Thibault
Title: Acting Chief Financial Officer

EXHIBIT INDEX

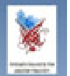
Exhibit No. Description

99.1 Poster of Pieris Pharmaceuticals, Inc.
99.2 Press Release dated June 4, 2017.

A phase Ib study investigating the safety, tolerability, pharmacokinetics, and pharmacodynamics of the hepcidin antagonist PRS-080#022-DP in anemic chronic kidney disease patients undergoing hemodialysis

Lutz Renders, MD[1], Ming Wen, MD[1], Frank Dellanna, MD[2], Heinrichs, Sven, MD[3], Klemens Budde, MD[3], Christian Rosenberger, MD[3], Christiane Erley, MD[4], Birgit Bader, MD[5], Claudia Sommerer, MD[5], Schaier, Matthias, MD[5], Werner Feuerer, MD[6], Edgar Fenzl, MD[7], Rachel van Swelm, PhD[8], Dorine Swinkels, MD PhD[8], Klaus Kutz, MD[9], Louis Matis, MD[10], Ulrich Moebius, PhD[11]

[1]Klinikum Rechts der Isar, Department Nephrology, Munich, Germany; [2]DaVita Düsseldorf, Germany; [3]Charité Berlin, Germany; [4]St. Joseph Krankenhaus, Berlin, Germany; [5]University Hospital Heidelberg, Germany; [6]Nuvisan Pharma Services, Neu-Ulm, Germany; [7]FGK Clinical Research, Munich, Germany; [8]Radboud University Medical Center, Nijmegen, The Netherlands; [9]AccelPharm, Basel, Switzerland; [10]Pieris Pharmaceuticals, Inc., Boston, Massachusetts; [11]Pieris Pharmaceuticals, Inc., Freising, Germany



Introduction

The hepatic hormone hepcidin was identified as an important regulator of iron metabolism in chronic diseases and offers a new target to treat anemia of chronic disease (Figure 1). Elevated levels of hepcidin contribute to functional iron deficiency and anemia by restricting iron to the reticulo-endothelial system and thereby reducing its availability for erythropoiesis. Thus, antagonizing hepcidin has the potential to improve iron availability and erythropoiesis, while avoiding overload with exogenous iron and reducing the administered levels of ESAs (1). PRS-080#022-DP is an Anticalin® drug candidate derived from the naturally occurring human neutrophil gelatinase-associated lipocalin. The 20kD protein is linked to a 30kD linear poly-ethylene-glycol that specifically binds to human hepcidin 25, thereby inhibiting its activity.

Here we report first data on safety, pharmacokinetics (PK) and pharmacodynamic (PD) of single doses of PRS-080#022-DP in anemic patients with chronic kidney disease (CKD) requiring hemodialysis.

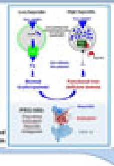


Figure 1: Iron metabolism regulated by hepcidin/ferroportin

Methods and Study design

In this multi-center, placebo-controlled, double-blind Phase Ib study, 24 anemic stage 5 CKD patients were treated with single ascending doses of PRS-080#022-DP in 3 cohorts at 2, 4, and 8 mg/kg body weight. Male (17) and post-menopausal female patients (7) of 55 ±14 years and 77 ±14 kg body weight, on hemodialysis for at least 90 days, on stable ESA dose, with Hb value of 9-12g/dL, ferritin ≥300 ng/mL, TSAT ≤40% and hepcidin of 5-75 nmol/L were included.

Iron treatment was not allowed from 7 days before until 7 days after study treatment. 6 patients per cohort received PRS-080#022-DP and 2 patients received placebo. Placebo or active treatments were administered by i.v. infusion over 1 h.

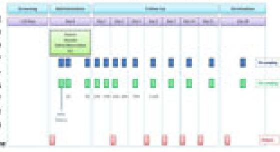


Figure 2: Study outline

Results

Safety

PRS-080#022-DP was safe and well tolerated. In total, 22 treatment-emergent adverse events (TEAEs) occurred in 12 patients (placebo and drug-treated patients).

One serious adverse event (dry gangrene) occurred after dosing with 2 mg/kg but was judged not related to PRS-080#022-DP. PRS-080#022-DP related adverse events (AEs) occurred in 2 patients and included "exercise tolerance decreased" (1 patient in 2 mg/kg dose group) and "abdominal discomfort" and "headache" (1 patient 4 mg/kg dose group). The most frequently reported TEAEs were administration site conditions (edema and swelling) with 4 events, gastrointestinal disorders (abdominal discomfort, anal fissure, nausea, and vomiting) and vascular disorders (dry gangrene, hypertension and hypotension) with 4 events each. Most of the TEAEs were only reported once, except nausea (1 event in 4 and 8 mg/kg dose group) and cough (1 event in 2 and 4 mg/kg dose group). No dose-dependent increase of AEs was observed. Notably, vital signs, temperature and ECG were unchanged following administration.

Table 1: Overview of adverse events reported during the study

	PLACEBO			2 mg/kg			4 mg/kg			8 mg/kg			TOTAL		
	NE	N	%	NE	N	%	NE	N	%	NE	N	%	NE	N	%
AEs	4	3	50.0	8	5	83.3	10	4	66.7	1	1	16.7	23	13	54.2
Pre-TEAEs	1	1	16.7	-	-	-	-	-	-	-	-	-	1	1	4.2
TEAEs[1]	3	2	50.0	8	5	83.3	10	4	66.7	1	1	16.7	22	12	50.0
TEAEs related to study drug	-	-	-	1	1	16.7	2	1	16.7	-	-	-	3	2	8.3

(Treatment: PRS-080#022-DP at 2, 4 and 8 mg/kg)

Results

PK of FREE and TOTAL PRS-080#22-DP

Maximum concentration (C_{max}) and areas under the time curve (AUC) of FREE and TOTAL (free and bound to hepcidin) PRS-080#22-DP show a dose proportional increase.

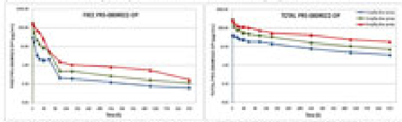


Figure 3: Mean serum concentration of FREE and TOTAL PRS-080#022-DP over time after single administration of different PRS-080#022-DP doses - semi-logarithmic scale

The plasma concentration profile of PRS-080#022-DP can be described by applying a two-exponential model with a fast first distribution phase and a much longer and slower second disposition phase. C_{max} of FREE and TOTAL PRS-080#022 were generally reached within 1 h after start of the infusion and declined dose-dependently with a first disposition phase and dose-independently in the final disposition phase (see half-life in Table 2).

Table 2: Mean pharmacokinetic parameters of FREE and TOTAL PRS-080#022-DP

		2 mg/kg FREE	2 mg/kg TOTAL	4 mg/kg FREE	4 mg/kg TOTAL	8 mg/kg FREE	8 mg/kg TOTAL
N		6	6	6	6	6	6
C_{max}	[µg/mL]	80	85	178[1]	122	164	255
t_{max}	[h]	1	1	1	3	1	1
$AUC_{(0-inf)}$	[µg/mL*h]	564	7370	2038	17095	5020	37688
C_{max}/D	[µg/mL/mg]	14.86	22.33	43.53	27.88	20.52	31.88
$AUC_{(0-inf)}$/D	[µg/mL*h/mg]	251.97	3085.00	509.59	4273.81	627.46	4633.52
$t_{1/2}$	[h]	8.14	9.92	13.49	18.31	16.45	13.95
t_{last}	[h]	284	200	247	196	231	263

Iron mobilization by PRS-080#22-DP

PRS-080#022-DP dose-dependently mobilized serum iron with increases in both serum iron concentration and TSAT following treatment (Figure 4). The serum C_{max} of iron and TSAT was reached 19h after infusion at all 3 doses.

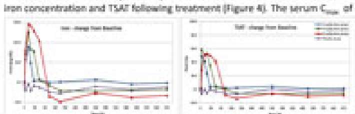


Figure 4: Iron mobilization after single dose PRS-080#022 (means, absolute change from Baseline): serum iron (left panel), and TSAT (right panel)

The duration of elevated serum iron concentration and TSAT increased dose-proportionally as well.

Table 3: Mean AUC values of serum iron profiles at different time intervals after i.v. administration of different doses of PRS-080#022-DP or PLACEBO

Dose group	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
2 mg/kg	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
4 mg/kg	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
8 mg/kg	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
PLACEBO	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

After infusion of 2, 4, and 8 mg/kg PRS-080#022-DP, the mean serum iron concentration reached its baseline value after 2, 3 and between 4 and 5 days after the end of the infusion, respectively. AUCs of serum iron at different time points are shown Table 3.

Additionally, preliminary data of the study show that administration of PRS-080#022-DP resulted in a decrease of free hepcidin shortly after i.v. infusion (data not shown). Serum ferritin levels were largely unaffected by treatment at all three doses. No dose dependency was observed. These findings provide evidence that the serum iron mobilization is independent of the initial serum ferritin and initial TSAT values.

Conclusion

The very good safety profile and the activity of PRS-080#022-DP on iron metabolism observed in anemic dialysis dependent end-stage CKD patients warrant further investigation of PRS-080#022-DP in a multiple dosing regimen to explore potential amelioration of anemia in stage 5 CKD patients.

Topic: Nephrology

PRESS RELEASE

Pieris Pharmaceuticals Announces Presentation of Clinical Data for its Lead Anemia Drug Candidate, PRS-080#022-DP, at the 54th ERA-EDTA Congress

BOSTON, MA — (Marketwired) — June 4, 2017 — Pieris Pharmaceuticals, Inc. (NASDAQ: PIRS), a clinical-stage biotechnology company advancing novel biotherapeutics through its proprietary Anticalin® technology platform for cancer, respiratory and other diseases, announced today the presentation of Phase 1b study results for its anemia program, PRS-080#022-DP, at the 54th European Renal Association & European Dialysis and Transplant Association (ERA-EDTA) Congress, convening in Madrid, Spain June 3–6, 2017.

The poster presentation, entitled “A phase Ib study investigating the safety, tolerability, pharmacokinetics, and pharmacodynamics of the hepcidin antagonist PRS-080#022-DP in anemic chronic kidney disease patients undergoing hemodialysis”, was delivered by Dr. Lutz Renders, Professor at the Klinikum Rechts der Isar, Department of Nephrology, Munich, Germany, and the lead investigator on the clinical trial. The poster is available here.

In this multi-center, placebo-controlled, double-blind study, 24 dialysis-dependent stage 5 chronic kidney disease (CKD5) patients with anemia were treated with single ascending doses of PRS-080#022-DP in 3 cohorts at 2, 4, and 8 mg/kg body weight.

Intravenous administration of PRS-080#022-DP was both safe and well-tolerated at all doses, and resulted in a profound decrease in free hepcidin within one hour after infusion, followed by robust mobilization of serum iron, with dose-proportional increases in both the level and duration of serum iron concentration and transferrin saturation (TSAT) following treatment.

Dr. Renders commented, “PRS-080#022-DP was safe and well tolerated with dose-dependent pharmacodynamic activity. Hepcidin levels are invariably elevated in anemic CKD5 patients, and as the master inhibitory regulator of iron metabolism, hepcidin represents an attractive target for treating the hypoferremia and iron-restricted anemia (IRA) which are often associated with poor prognosis and lower quality of life. Management of IRA using intravenous iron and erythropoiesis stimulating agents is ineffective for a significant subset of patients and may have adverse effects, driving the need for alternative new therapies.”

Louis Matis, M.D., Pieris SVP and Chief Development Officer commented, “Based on the favorable profile of PRS-080#022-DP observed in this study, we look forward to the outcome of our upcoming multi-dose study to further explore the clinical potential of PRS-080#022-DP in hemodialysis-dependent anemic patients, for whom elevated hepcidin is associated with the severity of anemia.”

About PRS-080#022-DP

PRS-080#022-DP is a fully proprietary Anticalin protein that sequesters hepcidin, typically regarded as the master negative regulator of iron metabolism. With a pharmacokinetic profile tuned to remove hepcidin in line with target turnover dynamics, PRS-080 is intended to optimally mobilize iron trapped in iron storage cells, particularly in anemic patients with iron-restricted erythropoiesis due to functional iron deficiency. The research leading to these results initially received funding from the European Community’s Seventh Framework Programme (FP7/2007-2013) under grant agreement n° 278408. Patients with end-stage renal disease almost invariably develop anemia, which is often associated with increased morbidity and mortality, as well as a reduced quality of life.

About Anemias of Chronic Disease

Anemia of chronic disease (ACD), also known as anemia of inflammation (AI), is the most prevalent anemia in hospitalized patients worldwide. It occurs in patients with acute or chronic inflammatory conditions including infections, cancer, rheumatoid arthritis, and chronic kidney disease. ACD is generally characterized by a normocytic anemia, impaired erythropoiesis, low serum iron and low transferrin saturation, but often normal to high body iron stores with iron sequestered in intracellular compartments. The molecular mechanisms and pathogenesis of the iron distribution abnormalities in ACD have been elucidated, and it has now been shown that inflammatory cytokines released during acute infection or chronic disease alter systemic iron metabolism by inducing excess synthesis of the iron regulatory hormone hepcidin. In turn, hepcidin inhibition of iron export from cells by blocking ferroportin activity has been established as the major underlying cause of the hypoferremia and iron-restricted erythropoiesis seen in ACD. Current treatment of the anemia generally includes administration of intravenous iron and erythropoiesis stimulating agents. However, the fact that these approaches do not directly address the high levels of hepcidin responsible for functional iron deficiency, together with concerns over adverse effects from these therapies, have driven the need for alternative treatments.

***About Pieris Pharmaceuticals*:**

Pieris is a clinical-stage biotechnology company that discovers and develops Anticalin® protein-based drugs to target validated disease pathways in a unique and transformative way. Our pipeline includes immuno-oncology multi-specifics tailored

for the tumor microenvironment, an inhaled Anticalin protein to treat uncontrolled asthma and a half-life-optimized Anticalin protein to treat anemia. Proprietary to Pieris, Anticalin proteins are a novel class of therapeutics validated in the clinic and by partnerships with leading pharmaceutical companies. Anticalin is a registered trademark of Pieris. For more information, visit www.pieris.com.

Forward Looking Statements

This press release contains forward-looking statements as that term is defined in Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934. Statements in this press release that are not purely historical are forward-looking statements. Such forward-looking statements include, among other things, references to novel technologies and methods; our business and product development plans; the timing and progress of our studies, our liquidity and ability to fund our future operations; our ability to achieve certain milestones and receive future milestone or royalty payments; or market information. Actual results could differ from those projected in any forward-looking statements due to numerous factors. Such factors include, among others, our ability to raise the additional funding we will need to continue to pursue our business and product development plans; the inherent uncertainties associated with developing new products or technologies and operating as a development stage company; our ability to develop, complete clinical trials for, obtain approvals for and commercialize any of our product candidates; competition in the industry in which we operate and market conditions. These forward-looking statements are made as of the date of this press release, and we assume no obligation to update the forward-looking statements, or to update the reasons why actual results could differ from those projected in the forward-looking statements, except as required by law. Investors should consult all of the information set forth herein and should also refer to the risk factor disclosure set forth in the reports and other documents we file with the SEC available at www.sec.gov, including without limitation the Company's Annual Report on Form 10-K for the fiscal year ended December 31, 2016 and the Company's Quarterly Reports on Form 10-Q.

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