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): December 7, 2015

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.)**

Lise-Meitner-Strasse 30
85354 Freising-Weihenstephan, Germany
(Address of principal executive offices, including zip code)

Registrant's telephone number, including area code: +49 81 6114 11400

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))

Item 7.01 Regulation FD Disclosure.

Attached hereto as Exhibit 99.1 and incorporated by reference herein is a press release of Pieris Pharmaceuticals, Inc.

Attached hereto as Exhibit 99.2 and incorporated by reference herein is a presentation of Pieris Pharmaceuticals, Inc. at the 57th Annual Meeting of the American Society of Hematology.

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 Press Release of Pieris Pharmaceuticals, Inc., dated December 7, 2015.

99.2 Presentation of Pieris Pharmaceuticals, Inc. at the 57th Annual Meeting of the American Society of Hematology, dated December 7, 2015.

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: December 7, 2015

PIERIS PHARMACEUTICALS, INC.

By: /s/ Darlene Deptula-Hicks
Name: Darlene Deptula-Hicks
Title: Chief Financial Officer

EXHIBIT INDEX

Exhibit No. Description

99.1 Press Release of Pieris Pharmaceuticals, Inc., dated December 7, 2015.

99.2 Presentation of Pieris Pharmaceuticals, Inc. at the 57th Annual Meeting of the American Society of Hematology, dated December 7, 2015.

Exhibit 99.1

PRESS RELEASE

PIERIS PHARMACEUTICALS PRESENTS CLINICAL DATA FOR ITS HEPCIDIN ANTAGONIST PROGRAM, PRS-080, AT THE 2015 AMERICAN SOCIETY OF HEMATOLOGY (ASH) ANNUAL MEETING

BOSTON, MA, December 7, 2015 – Pieris Pharmaceuticals, Inc. (NASDAQ: PIRS), a biotechnology company advancing its proprietary Anticalin® bio therapeutic technologies, announced today that it will present detailed data today summarizing the results from a Phase I clinical study in healthy male volunteers with its PRS-080 Anticalin hepcidin antagonist at the 57th Annual Meeting of the American Society of Hematology (ASH) taking place in Orlando, FL.

The oral presentation entitled, "A Phase I Study Investigating Safety, Tolerability, Pharmacokinetics and Pharmacodynamic Activity of the Hepcidin Antagonist PRS-080#022. Results from a Randomized, Placebo Controlled, Double-Blind Study Following Single Administration to Healthy Subjects," outlined the favorable safety profile of the drug, as well as demonstrable proof of mechanism shown by increased serum iron levels as well as transferrin saturation in treated subjects.

PRS-080 was well tolerated, with no serious adverse events (SAEs) observed in the single ascending dose (SAD) study at six dose levels administered by intravenous infusion in 48 healthy male subjects ranging from 0.08 to 16.0 mg/kg (clinicaltrials.gov identifier NCT02340572). Reported AEs were of mild to moderate severity with no apparent dose dependency or difference between active and placebo treatment groups. The plasma half-life of PRS-080 ranged between 71 and 81 hours among dose cohorts.

Within one hour of PRS-080 administration, a marked decrease in plasma hepcidin was observed, followed by dose-dependent elevations of both serum iron concentration and transferrin saturation. Moreover, the durations of serum iron elevation and transferrin saturation also increased in a dose-dependent manner. Among all subjects receiving PRS-080 doses of 1.2 mg/ml and higher, statistically significant increases in total serum iron mobilization were observed relative to placebo ($p = .005$).

Louis Matis, M.D., Pieris SVP and Chief Development Officer commented, "We are extremely pleased by the safety profile as well as the pharmacodynamic activity of our hepcidin antagonist in these healthy subjects. Hepcidin is the root cause of and therefore, an attractive target for treating the hypoferremia and iron-restricted reduction of erythropoiesis seen in anemias of chronic disease (ACD), which are often associated with poor prognosis and lower quality of life. Management of ACD using intravenous iron and erythropoiesis stimulating agents is ineffective for subsets of patients and may have adverse effects, driving the need for new alternative therapies. We expect to soon initiate the dosing of anemic patients with chronic kidney disease (CKD) undergoing hemodialysis, for whom elevated hepcidin is strongly associated with the severity of anemia."

About PRS-080

PRS-080 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. Funded in part by an EC FP7 health program grant, Pieris' hepcidin antagonist program was supported by the EUROCALIN consortium. Details of the consortium's charter can be found at www.eurocalin-fp7.eu. Patients with ESRD 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 Pharmaceuticals is a clinical stage biotechnology company that discovers and develops Anticalin-based drugs to target validated disease pathways in a unique and transformative way. Our pipeline includes immuno-oncology multi-specifics tailored for the tumor micro-environment, an inhaled Anticalin to treat uncontrolled asthma and a half-life-optimized Anticalin to treat anemia. Proprietary to Pieris, Anticalins are a novel class of protein therapeutics validated in the clinic and by partnerships with leading pharmaceutical companies. Anticalin®, Anticalins® are registered trademarks 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 of future clinical trials for our PRS-080 program, 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, 2014 and the Company's Quarterly Reports on Form 10-Q.

Company Contact:
Pieris Pharmaceuticals, Inc.
Darlene Deptula-Hicks
SVP & Chief Financial Officer
+1-603-553-5803
deptula@pieris.com

Investor Relations Contact:
The Trout Group
Thomas Hoffmann
+1-646-378-2931
thoffmann@troutgroup.com

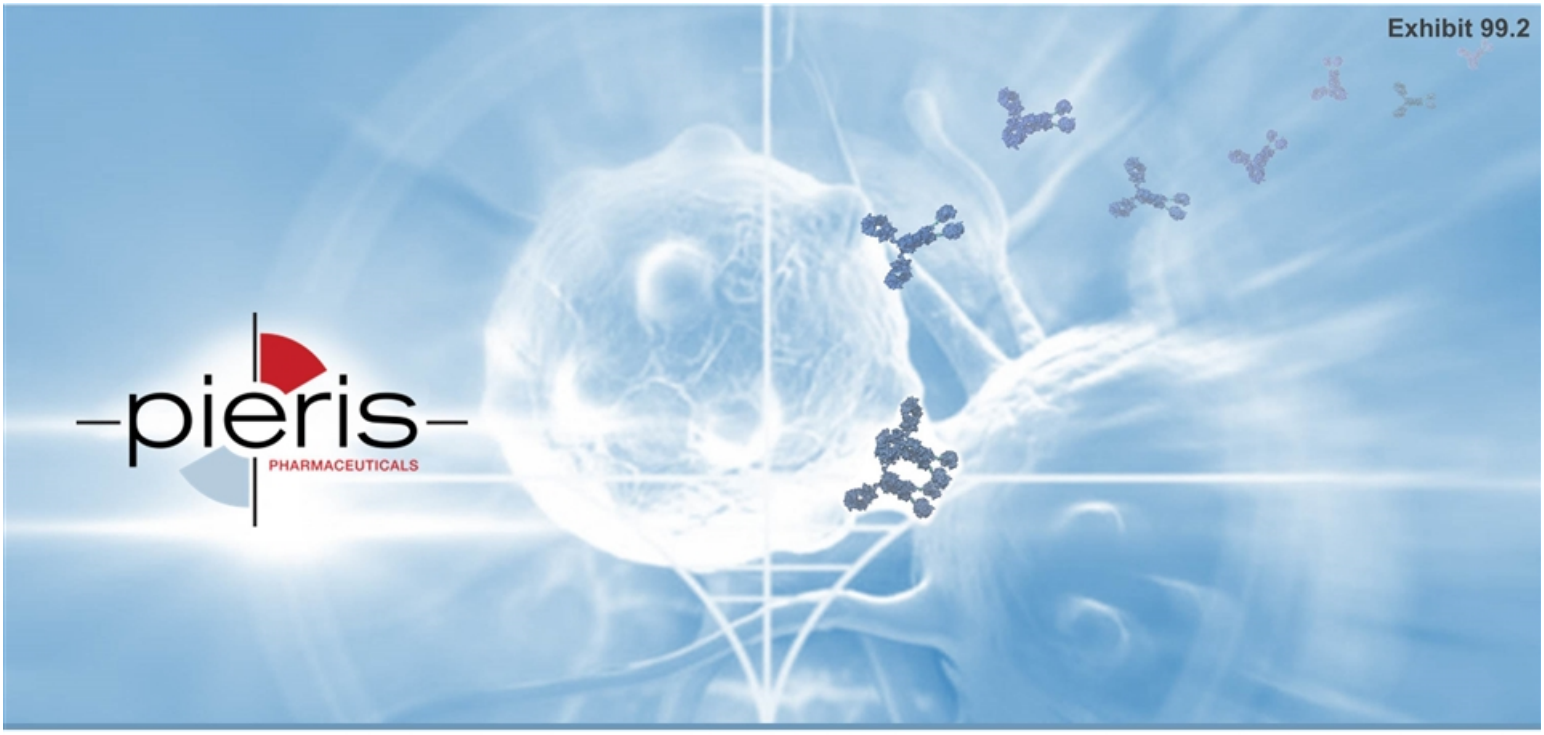
or

Media Inquiries:
Gretchen Schweitzer
+49 172 861 8540
gschweitzer@macbiocom.com

The Del Mar Consulting Group, Inc.
Robert Prag, President
+1-858-794-9500
bprag@delmarconsulting.com

##END##

Exhibit 99.2



A Phase I Study Investigating the Safety, Tolerability, Pharmacokinetics and Pharmacodynamic Activity of the Hepcidin Antagonist PRS-080#022. Results from a Randomized, Placebo Controlled, Double-Blind Study Following Single Administration to Healthy Subjects

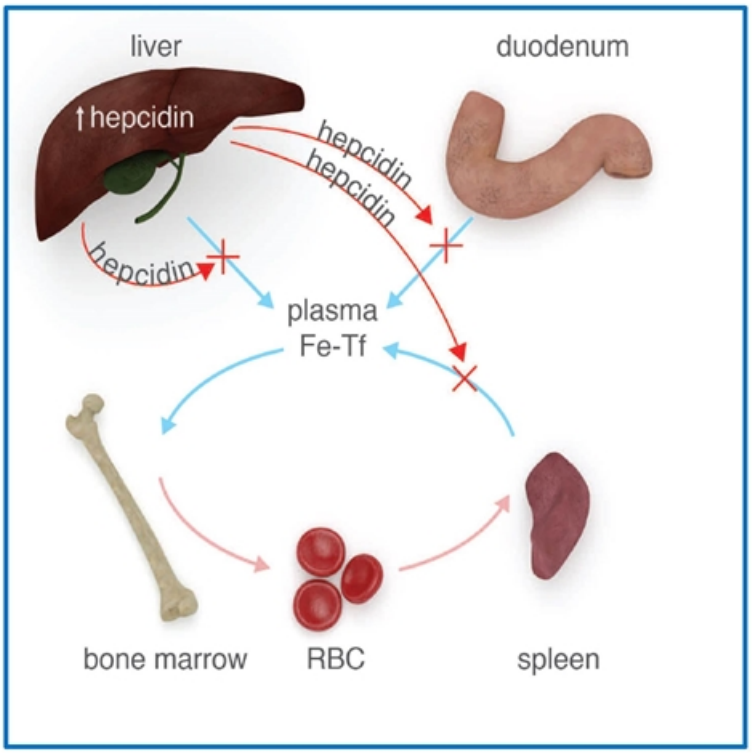
December 07, 2015

57th Annual Meeting of the American Society of Hematology, Orlando, FL

Hepcidin Plays a Central Role in Iron Metabolism

Hepcidin is a 25 amino acid peptide hormone that serves as a **key regulator of iron metabolism** by inhibiting iron entry into plasma from the three main sources of iron:

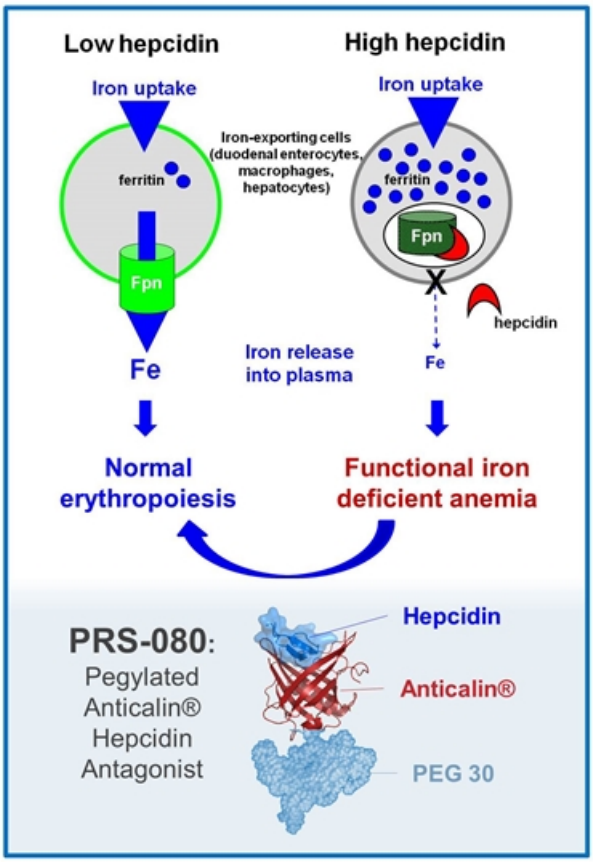
- Dietary absorption in the duodenum
- Release of recycled iron from macrophages
- Release of stored iron from hepatocytes



Haematologica 2013 98:11

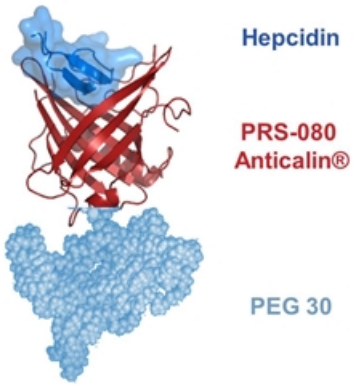
Antagonizing Elevated Hepcidin Levels in Anemias of Chronic Disease (ACD)

- **Hepcidin elevated in multiple chronic inflammatory conditions associated with anemia**
  - Infections, cancer, RA, chronic kidney disease (CKD)
- **Iron metabolism regulated by hepcidin/ferroportin**
  - Hepcidin inhibits iron export from cells by blocking ferroportin
  - Excess hepcidin is the root cause of hypoferremia and iron-restricted reduction of erythropoiesis seen in ACD
  - Hepcidin inhibits erythroid colony formation at reduced erythropoietin concentrations
- **Inhibition of hepcidin to treat functional iron deficient erythropoiesis and anemia expected to**
  - Increase availability of internal iron sources
  - Increase ESA responsiveness allowing reduction of ESA doses
  - Prevent iron overload from exogenous administration
  - Increase and stabilize Hb levels



PRS-080 is a Highly Potent Anticalin® Hepcidin Antagonist

- **Anticalins®** – derived from human lipocalins – are a **novel class of therapeutic binding proteins** (MW 16-20kD), that demonstrate high target affinity and exquisite specificity
- **PRS-080 is a pegylated Anticalin®** protein derived from the human lipocalin NGAL, that acts as a **potent hepcidin antagonist**
  - 50 pM affinity for hepcidin
  - Produced by bacterial expression in E. coli
  - Inhibits hepcidin-induced ferroportin internalization
  - Optimized plasma half life by conjugation to PEG 30
  - No adverse effects in non-human primate toxicity studies



PRS-080 Has Been Investigated in Phase I in Healthy Subjects

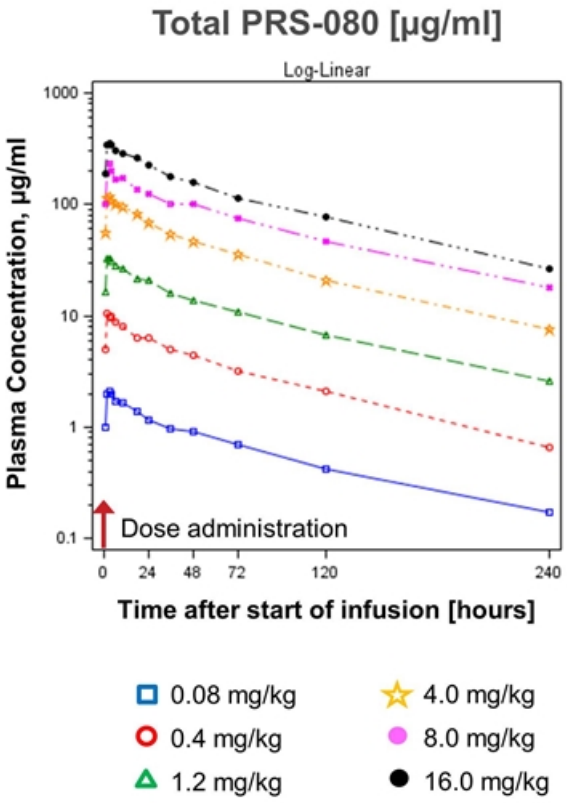
- Single dose escalating study in healthy male subjects, n=48
  - Randomized, double blinded, placebo controlled study
- 6 dose cohorts
  - 0.08, 0.4, 1.2, 4.0, 8.0, 16.0 mg/kg (based on API without PEG)
  - I.V. infusion over 2 hours
  - 6 subjects receiving PRS-080, 2 subjects receiving placebo per cohort
- Endpoints
  - Safety and maximal tolerated dose
  - Pharmacokinetics
  - Pharmacodynamics (serum iron, transferrin saturation)
  - Hepcidin plasma concentrations
  - Immunogenicity

PRS-080 Was Well Tolerated in Healthy Subjects

- No serious Adverse Events (AE)
- 39 treatment emergent AEs (TEAE) in 22 subjects
  - 30 mild TEAEs
  - 9 moderate TEAEs
- Headache was most common TEAE (10 subjects)
- Otherwise, no association of AEs to specific organs
- No apparent correlation between dose and number of TEAEs
- No hypersensitivity, no infusion reactions
- Vital signs and ECG without changes
- No cytokine release upon PRS-080 administration
  - IFN-γ, IL-1β, IL-6 and TNF-α

PRS-080 Shows Dose Proportional Pharmacokinetics

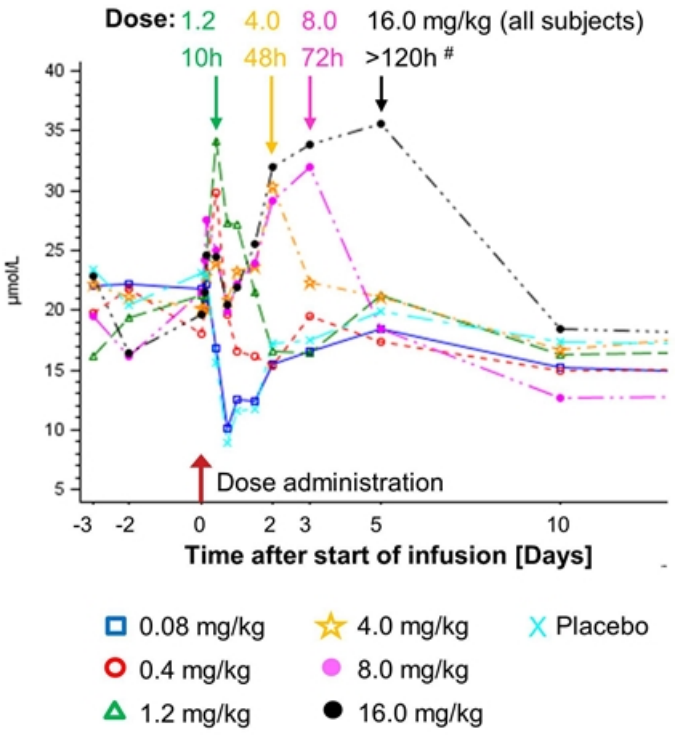
- **Total PRS-080**
  - Dose proportional C_{max} and AUC
  - T_{max} at around 1h after end of infusion
  - $T_{1/2}$ at 64 to 81 hours (geometric mean)
    - 71 to 81 hours, arithmetic mean
  - Small volume of distribution (48 - 65 ml/kg)
    - Consistent with distribution to blood volume
- **Free PRS-080**
  - C_{max} and AUC lower compared to total PRS-080
  - $T_{1/2}$ at 40 to 62 hours
    - Consistent with "consumption" of free PRS-080 by hepcidin binding



PRS-080 Shows Dose-Dependent Effects

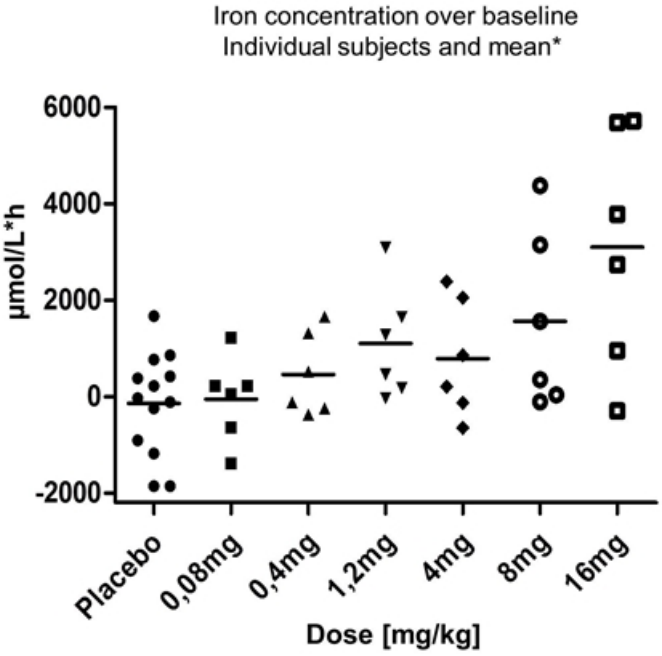
Increased Duration & AUC of Elevated Serum Iron Concentrations

Mean Iron Concentrations



Time point of peak iron conc. (hrs after start of infusion)

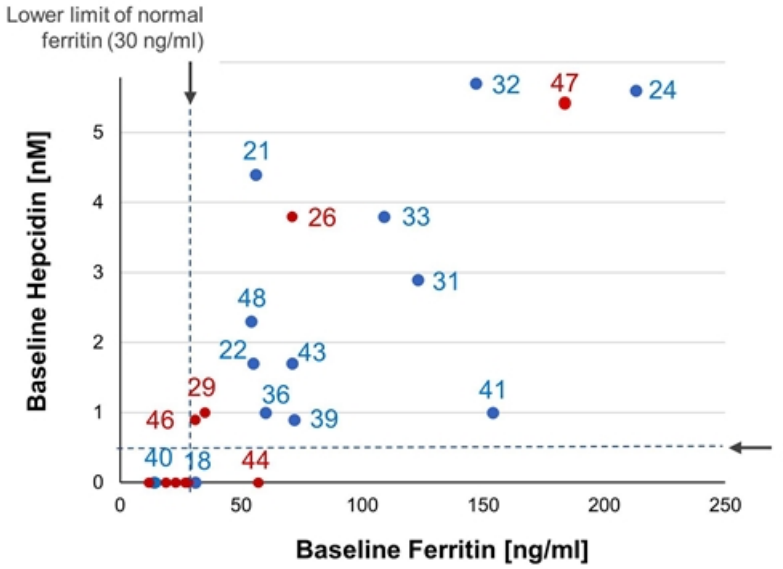
Area Under the Curve



* Mean placebo value subtracted

PRS-080 Induced Iron Response is Correlated to Baseline Ferritin & Hepcidin

- Serum iron response generally observed in subjects with normal ferritin (> 30 ng/ml) and detectable hepcidin (> 0.5 nM) at baseline
- Subjects of dose cohorts 1.2 to 16.0 mg/kg shown below
  - Subjects achieving iron response (> 34.5 µM = Fe ↑)
  - Subjects without iron response (< 34.5 µM = Fe →)



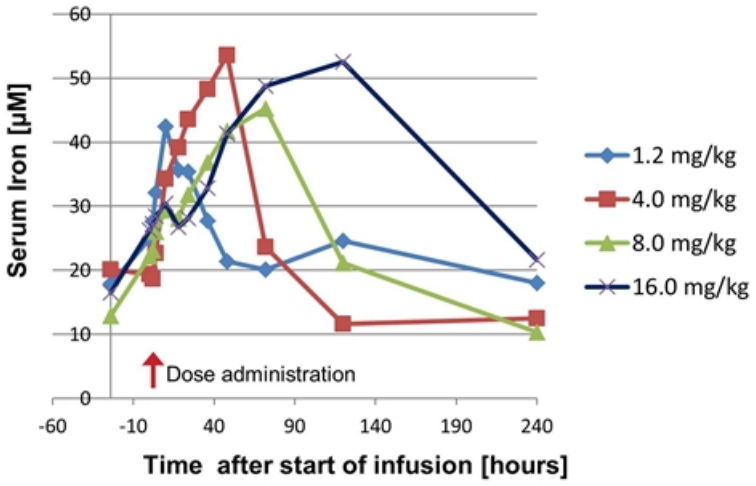
	Ferritin > 30 ng/ml Hepcidin >0.5 nM	No. of subjects	
		Fe ↑	Fe →
PRS-080 [1.2–16 mg/kg]	+	11	4
	-	2	7
Placebo	+	1	9
	-	1	1

Duration of Iron Response After PRS-080 Administration is Dose-Dependent

- Duration of response increases with dose
- Individual peak serum iron concentrations are independent of dose

Mean Iron Concentrations

Subjects achieving iron response > 34.5 µM



Time to Peak
Duration of Response
Peak Iron Concentration

Dose [mg/kg]	1.2	4.0	8.0	16.0
Time to peak iron concentration	10h	48h	72h	120h
Duration of iron response*	25h	64h	94h	185h
Peak serum iron concentration [µM]	42.5	53.6	45.2	52.2

* Estimated time point where serum iron falls <34.5 µM

PRS-080 Shows Favorable Safety Profile/ Confirms Mechanism of Action in Phase 1

- PRS-080 was **well tolerated** in healthy subjects
- **Pharmacokinetics as expected**: $T_{1/2}$ ~ 3 days
- Immediate **dose-dependent decrease in circulating hepcidin**
- **Dose-dependent duration of serum iron and TSAT responses**
  - From 24 hours up to >120 hours
  - Predominantly observed in subjects with normal ferritin (>30 ng/ml) and detectable hepcidin (>0.5 nM) at baseline
    - Sufficient tissue iron stores and target expression
  - Robust responses at doses of 1.2 mg/kg and above, with **statistically significant increase in total serum iron** relative to placebo (p = .005)
- **No risk of immunogenicity observed**
- **Data support further investigation of PRS-080 in patients with ACD**

Next Steps: Phase Ib/IIa Study to Investigate PRS-080 in Anemic CKD5 Patients

Planned Phase Ib/IIa in CKD5 hemodialysis patients

- Ib: Single Ascending dose; Safety, PK and pharmacodynamic activity (iron, TSAT, hepcidin)
- IIa: MAD, 4 week repeated dosing; anemia (Hb) as primary outcome measure

VALIDATED BIOLOGY

Elevated hepcidin levels in CKD patients as cause for anemia

- Restricted iron utilization
- Impaired erythropoiesis
- Anemia despite i.v. iron and high ESA doses

PROMISING INVESTIGATIONAL DRUG

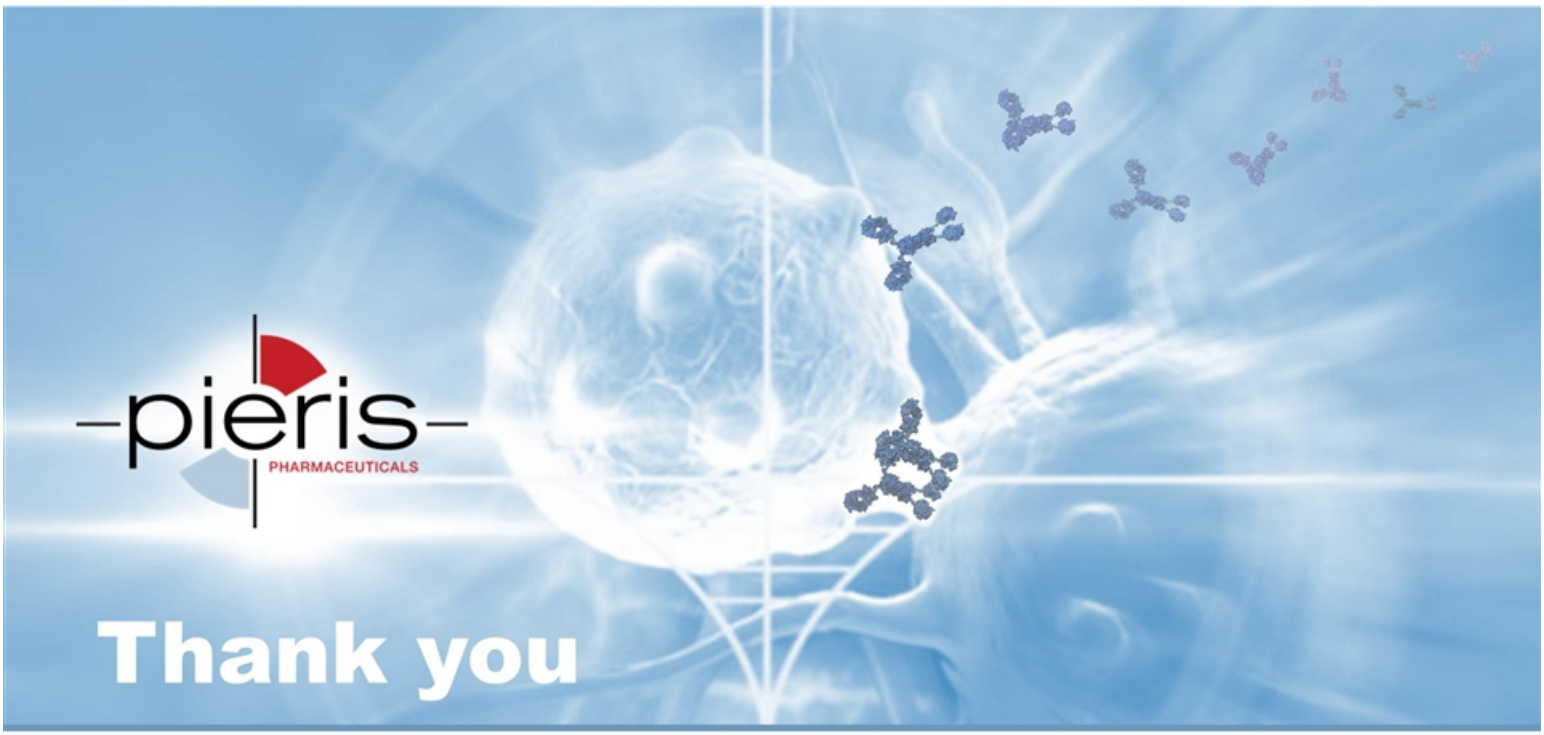
PRS-080 = hepcidin antagonist

- Increases iron mobilization
- Tailored half-life
- Aim to
  - Increase erythropoiesis
  - Reduce ESA and prevent iron overload
  - Reduce anemia

PROMISING CLINICAL ACTIVITY

Phase I study in healthy subjects

- ✓ Excellent safety
- ✓ Pharmacologic activity demonstrated



Pieris Pharmaceuticals, Inc.

Corporate Headquarters
255 State Street, 9th floor
Boston, MA 02109
USA

www.pieris.com

Pieris Pharmaceuticals GmbH

Munich Site
Lise-Meitner-Strasse 30
85354 Freising
Germany