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 16, 2019

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

Nevada	**001-37471**	**30-0784346**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**
255 State Street, 9th Floor Boston, MA		**02109**
(Address of principal executive offices)		**(Zip Code)**

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

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 (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.

Attached hereto as Exhibit 99.1 and incorporated by reference herein is the June 16, 2019 PRS-080 Presentation at the 24th Congress of the European Hematology Association.

The information set forth under this "Item 7.01. Regulation FD Disclosure," including Exhibit 99.1 attached hereto, shall not be deemed "filed" for any purpose, and shall not be deemed incorporated by reference into any filing under the Securities Act of 1933, as amended, or the Securities Exchange Act of 1934, as amended, regardless of any general incorporation language in any such filing. except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits

(d) *Exhibits.*

99.1 PRS-080 Presentation, 24th Congress of the European Hematology Association, Dated June 16, 2019.

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.

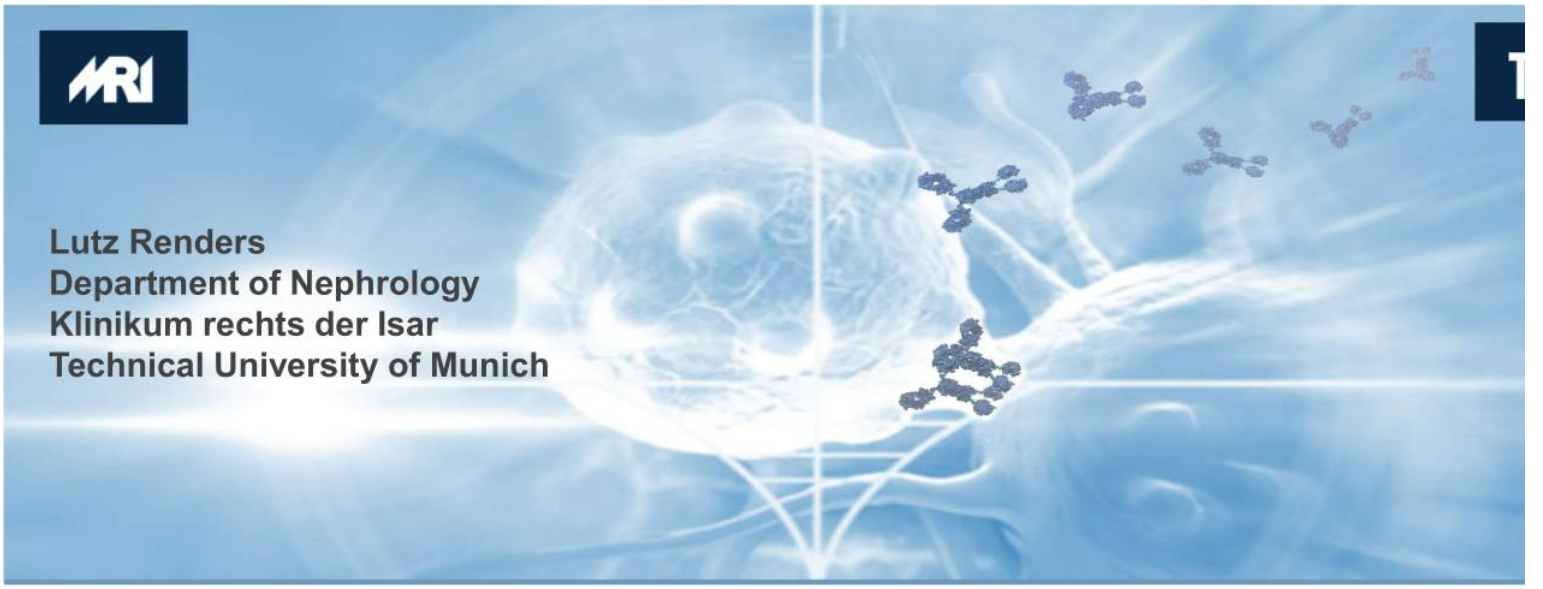
PIERIS PHARMACEUTICALS, INC.

Dated: June 17, 2019

/s/ Allan Reine

Allan Reine

Chief Financial Officer



Lutz Renders
Department of Nephrology
Klinikum rechts der Isar
Technical University of Munich

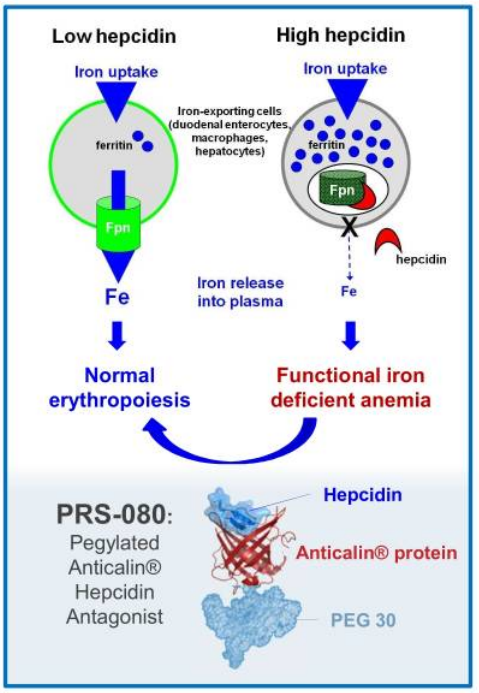
A PHASE 2A STUDY TO EVALUATE THE SAFETY, PHARMACOKINETICS, AND PHARMACODYNAMICS OF REPEATED ADMINISTRATIONS OF THE HEPCIDIN ANTAGONIST PRS-080 OVER 4 WEEKS IN ANEMIC CHRONIC KIDNEY DISEASE PATIENTS UNDERGOING HEMODIALYSIS

Lutz Renders, Frank Dellanna, František Švára, Jitka Řehořová, Ondřej Viklický, Ming Wen, Matthias Braunisch, Karoline Meurer, Anne Mas
Goran Martić, Kayti Aviano, Louis Matis, Ingmar Bruns

24th Congress of the European Hematology Association, June 13-16, 2019, Amsterdam

Antagonizing Elevated Hepcidin Levels in Anemias of Chronic Disease

- **Hepcidin is elevated in multiple chronic inflammatory conditions associated with anemia**
  - Infections, cancer, rheumatoid arthritis (RA), chronic kidney disease (CKD)
- **Iron metabolism is 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 anemia of chronic disease (ACD)
  - Hepcidin inhibits erythroid colony formation at reduced erythropoietin concentrations
- **Inhibition of hepcidin to treat functional iron deficient erythropoiesis and anemia is expected to**
  - Increase availability of internal iron sources
  - Increase erythropoietin stimulating agents (ESA) responsiveness allowing reduction of ESA doses
  - Prevent iron overload from exogenous administration
  - Increase and stabilize hemoglobin (Hb) levels



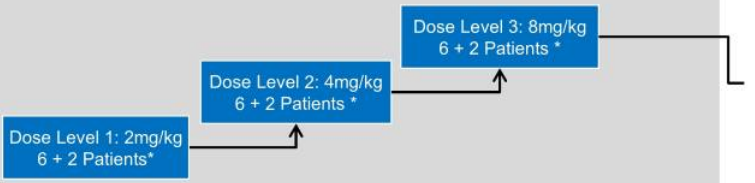
PRS-080: Phase 1b & Phase 2a Outline

Patient Population: ESRD/CKD patients on dialysis

- Ferritin ≥ 300 ng/ml
- Hepcidin 5-75 nM

Phase 1b: Single Administration

- Hb 9 – 12 g/dL
- TSAT ≤ 40%



- Safety
- Pharmacokinetics
- Hepcidin neutralization
- Iron / TSAT

*: 6 + 2 Patients: 6 active and 2 placebo

PRS-080: Phase 1b & Phase 2a (P2a) Outline

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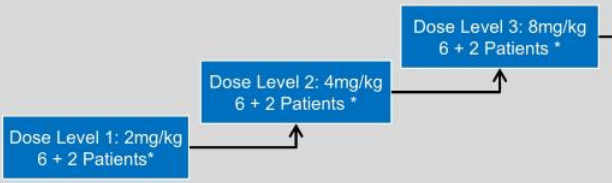
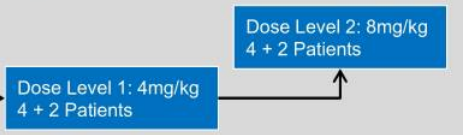


- Safety
- Pharmacokinetics
- Hepcidin neutralization
- Iron / TSAT

*: 6 + 2 Patients: 6 active and 2 placebo

Phase 2a: Repeated Administration

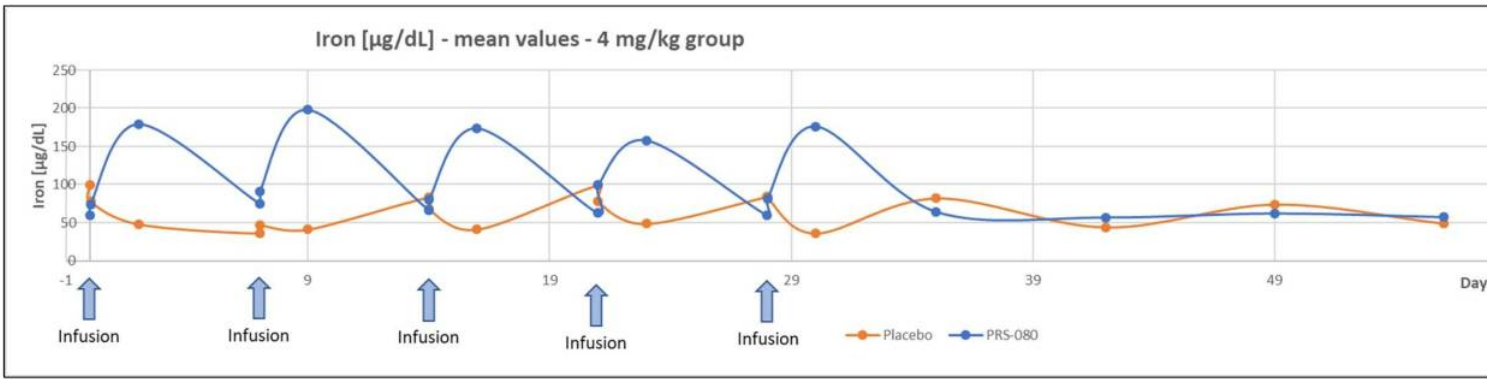
- Hb < 10.5 g/dL
- TSAT ≤ 30%



5 administrations (1x/week, for 4 weeks)

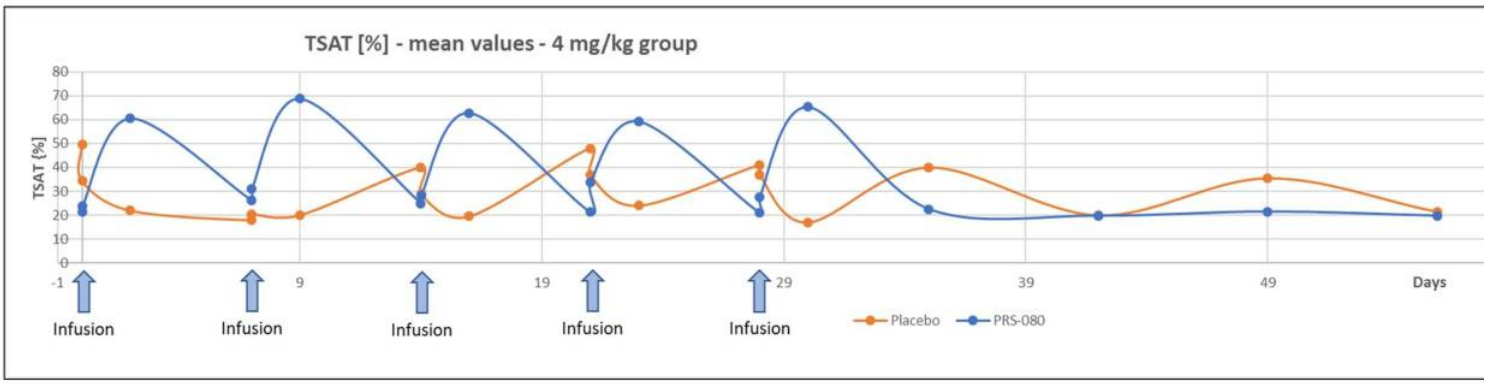
- Safety
- Pharmacokinetics
- Hepcidin neutralization
- Iron / TSAT
- **Hb**
- Reticulocyte Hb (CHr)

P2a: Mean Iron Values of 4 mg/kg Patient Group



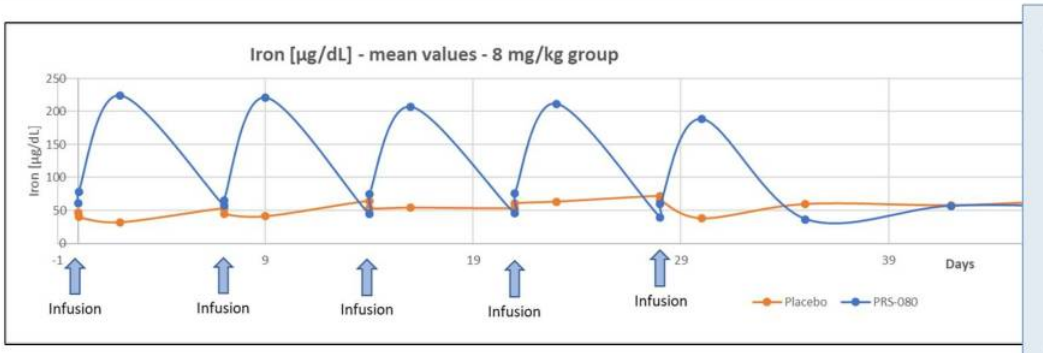
- Iron response and mobilization in serum iron after each dose of PRS-080 in drug-treated patients
- No iron response in placebo-treated patients

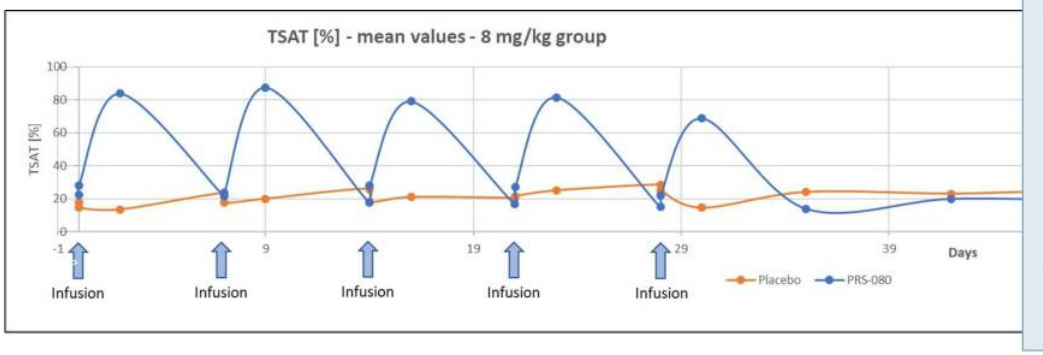
P2a: Mean TSAT% Values of 4 mg/kg Patient Group



- Mobilization of iron in TSAT after each dose of PRS-080 in drug-treated patients
- No iron response in placebo-treated patients

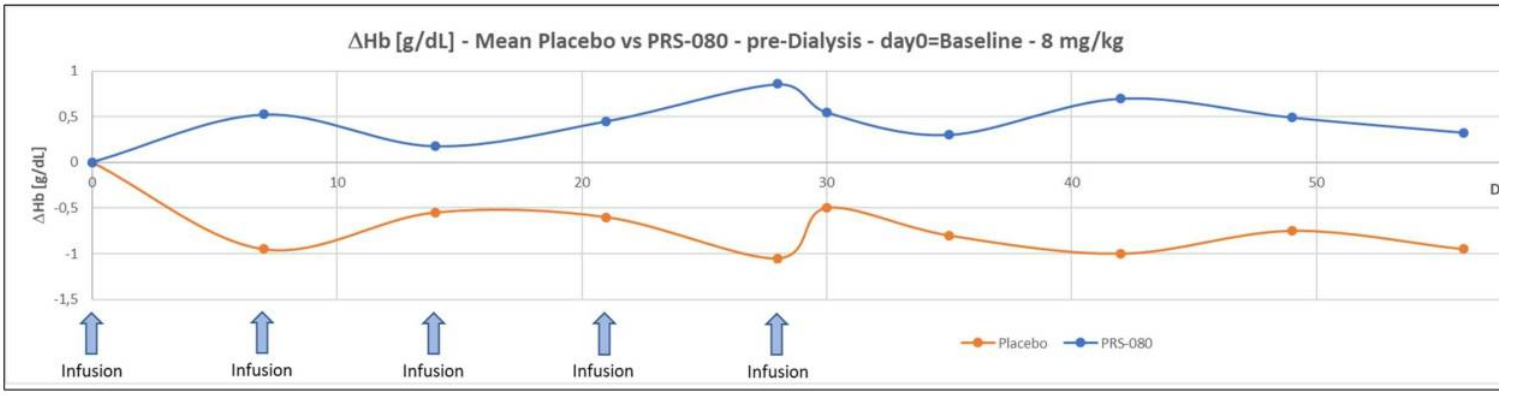
P2a: Mean Iron and TSAT% Values of 8 mg/kg Patient Group





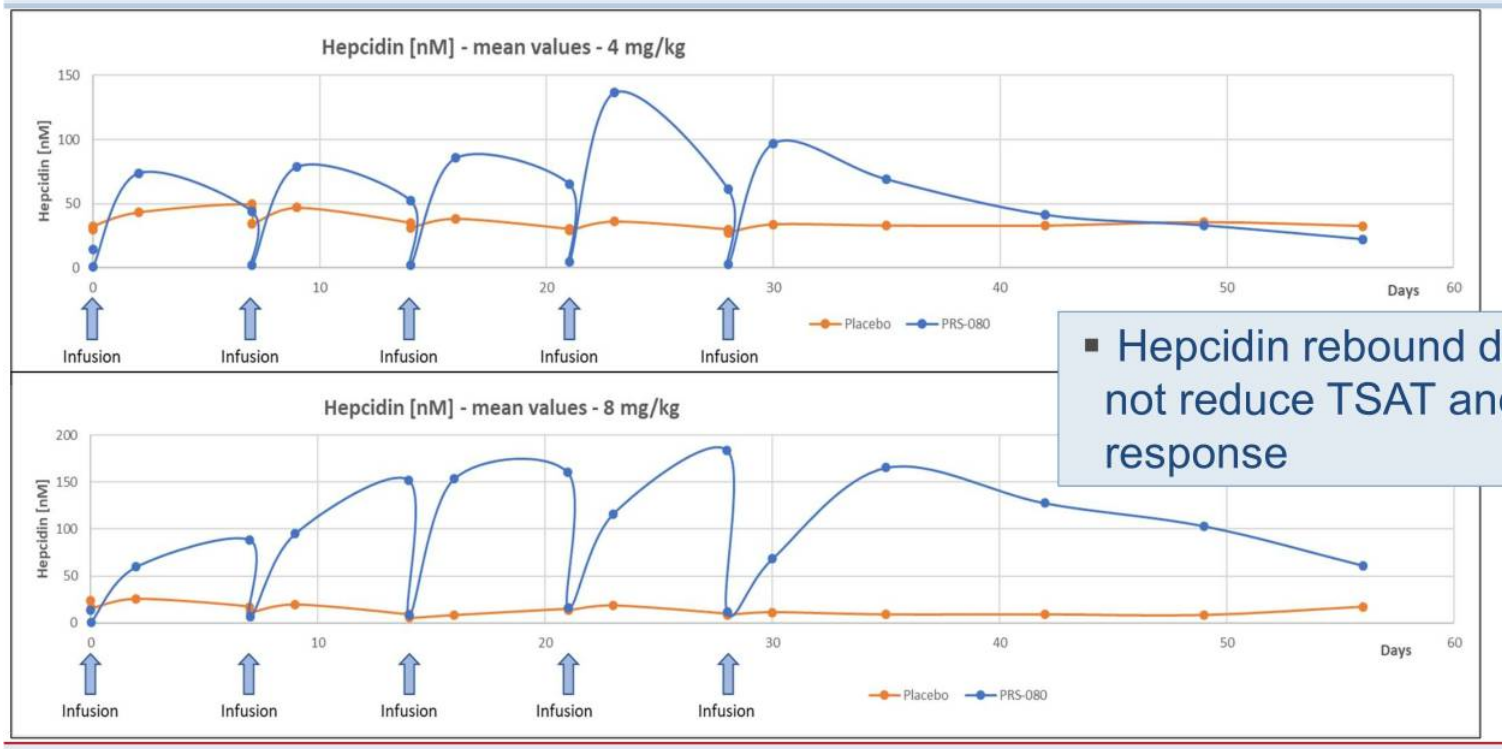
- Iron response and mobilization in both se iron and TSAT after ea dose of PRS-080 in dr treated patients
- Slightly higher peak ir response in the 8 mg/k treatment group vs 4 mg/kg group
- No iron response in placebo-treated patien

P2a: At 8mg/kg, Preliminary Evidence of an Increase in Hb With PRS-080 Treatment Compared to Placebo Group



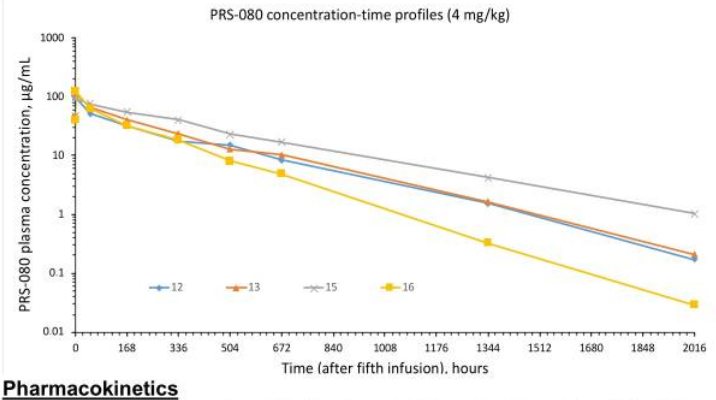
- Both Placebo and PRS-080 groups with no iron administration during stu
- Modest increase in Hb in the treated patient group
- Decline in the placebo group, possibly related to discontinuation of paren iron administration

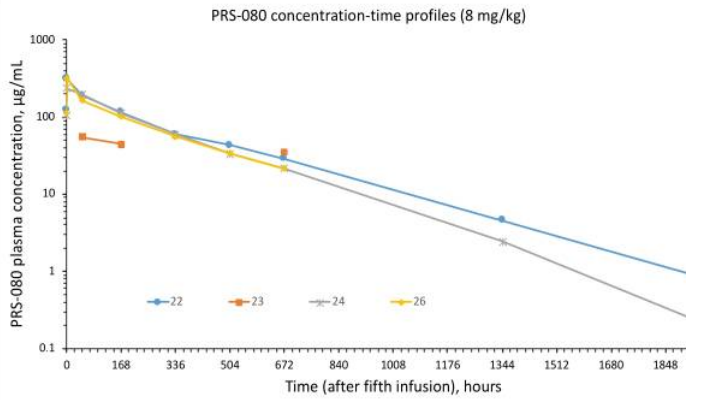
P2a: Hepcidin Analysis – Dose-Dependent Rebound



- Hepcidin rebound do not reduce TSAT and response

P2a: PRS-080 half-life in CKD patients





Pharmacokinetics

- 4 patients (Subject number 12, 13, 15 and 16) received 4 mg/kg PRS-080
- 4 patients (Subject number 22, 23, 24 and 26) received 8 mg/kg PRS-080
- Blood samples for PRS-080 determination were collected up to 84 days (approximately 2016 hours) after administration of fifth and last dose.
- PRS-080 terminal phase half-life was calculated by non-compartmental method using nominal (planned) time points and preliminary values are provided

PRS-080 half-life (n = 7) *

- Geometric mean (%CV) PRS-080 half-life was estimated to be 237 hours (2

* Subject 23 provided intermittent PK samples and is not included in half-life calculation

- PRS-080 half-life estimate was consistent with previously reported values in Phase 1b study
- PRS-080 with sufficient half-life and possible prolongation by renal insufficiency
- No accumulation of PRS-080

Phase 2a Multidose Study of the Hepcidin Inhibitor PRS-080 in Anemic Chronic Kidney Disease Patients Undergoing Hemodialysis: Summary

- PRS-080 was safe and well tolerated at both 4 mg/kg and 8 mg/kg treatment dose levels (da not shown)
- No treatment-related adverse events (AEs) or serious adverse events (SAEs) observed (dat not shown)
- Robust iron mobilization with increases in both serum iron and TSAT
- Peak iron concentrations were higher in the 8 mg/kg treatment group
- No clear difference in Hb values between placebo and PRS-080 in 4 mg/kg treatment group over the course of treatment (data not shown)
- Preliminary evidence of Hb response with separation of Hb values between placebo and PR 080 shown in the 8 mg/kg treatment group during the treatment period
  - Apparent Hb increase in drug-treated patients, even after discontinuation of iron treatme
  - Hb decline in placebo patients, potentially related to the withdrawal of iron treatment
- Half-life suggests adequate dosing schedule, reduced clearance possibly due to impaired re function but no accumulation effects