UNITED STATES
SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 6-K

REPORT OF FOREIGN ISSUER
PURSUANT TO RULE 13a-16 OR 15d-16
OF THE SECURITIES EXCHANGE ACT OF 1934

Date of Report: March 13, 2023

(Commission File No. 001-39308)

CALLIDITAS THERAPEUTICS AB

(Translation of registrant's name into English)

Kungsbron 1, D5
SE-111 22
Stockholm, Sweden
(Address of registrant's principal executive office)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.
Form 20-F ☒ Form 40-F ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1): ☐

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7): ☐

INFORMATION CONTAINED IN THIS REPORT ON FORM 6-K

On March 13, 2023, Calliditas Therapeutics AB (the "Registrant") gave a presentation to investors regarding the topline data from Part B of its NefIgArd trial for Nefecon. The investor presentation is attached hereto as Exhibit 99.1 and is incorporated by reference herein.

The information contained in this Form 6-K, including Exhibit 99.1, is intended to be furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933 or the Exchange Act, except as expressly set forth by specific reference in such filing.

EXHIBIT INDEX

Exhibit	Description
99.1	Investor Presentation

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, thereunto duly authorized.

CALLIDITAS THERAPEUTICS AB

Date: March 13, 2023

By: /s/ Fredrik Johansson
Fredrik Johansson
Chief Financial Officer

Exhibit 99.1


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Nef-301
Summary of Full Phase 3 Trial Results
March 13, 2023

Disclaimers

Important information

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including, without limitation, statements regarding Calliditas' strategy, planned regulatory submissions, anticipated regulatory approvals and clinical development plans, timing and data readouts. The words "may," "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "estimate," "predict," "project," "potential," "continue," "target," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements in this presentation are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties, and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this presentation, including, without limitation, any related to Calliditas' business, operations, continued and additional regulatory approvals for TARPEYO and Kinpeygo, market acceptance of TARPEYO and Kinpeygo, competitive products, clinical trials, supply chain, strategy, goals and anticipated timelines and other risks identified in the section entitled "Risk Factors" in Calliditas' reports filed with the Securities and Exchange Commission. Calliditas cautions you not to place undue reliance on any forward-looking statements, which speak only as of the date they are made. Calliditas disclaims any obligation to publicly update or revise any such statements to reflect any change in expectations or in events, conditions, or circumstances on which any such statements may be based, or that may affect the likelihood that actual results will differ from those set forth in the forward-looking statements. Any forward-looking statements contained in this presentation represent Calliditas' views only as of the date hereof and should not be relied upon as representing its views as of any subsequent date.

Primary endpoint successfully met in Phase 3 NefIgArd study

Beneficial eGFR treatment effect observed, irrespective of UPCR baseline. **Primary endpoint achieved - highly statistically significant with p-value <0.0001**

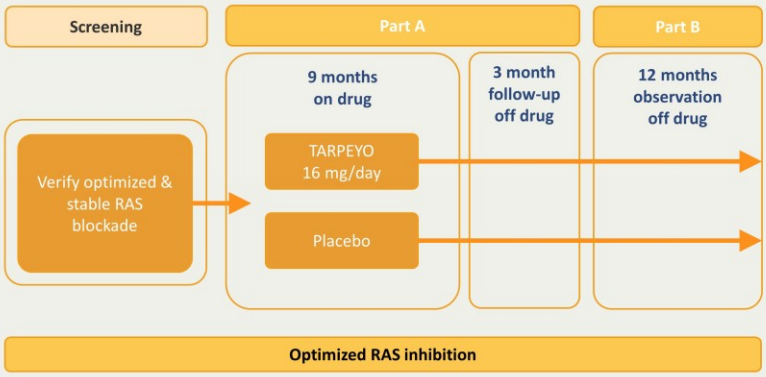
Supportive eGFR slope analyses over 2 years highly statistically significant. All estimates well in excess of the difference per year in 2 year eGFR total slope required to **predict clinically meaningful treatment effects** on the composite endpoint of ESRD, eGFR<15 mL/min/1.73 m^2 or sustained doubling of serum creatinine (Inker et al 2019)

A single treatment course of 9 months **slowed the loss of kidney function by 50%** compared to placebo at 24 months

UPCR effect of 30%+ reduction shown to be **durable for the entire 15 month follow up period**, with maximum effect observed at 12 months (three months after final dose)

The Company believes that the dataset is supportive of **filing for full regulatory approval for entire study population**

NefIgArd study design



Base inclusion criteria:

- Biopsy proven IgAN; > 1 gram of proteinuria; > 35 eGFR < 90 ml/min 36o patients, including 200 from Part A
- Patients were required to have well-controlled blood pressure of <140/90 mmHg to enter into the study, to ensure no BP confounding effects on proteinuria reduction.
- No immunosuppressive drugs were permitted during the study; changes to anti-hypertensive medications were discouraged.

Part A

- 200 patients in 19 countries with >145 sites
- Primary endpoint: proteinuria
  Key secondary endpoint: eGFR
- Read out positive data in November 2020

Part B

- Post approval follow up trial design
  - confirm the long-term renal benefit of observed proteinuria reduction
- 360 patients, including 200 from Part A
- Primary endpoint: difference in kidney function as measured by eGFR over the 2-year period
- Read out positive data in March 2023

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Disposition, Demographics and Baseline Characteristics

Disposition

	Nefecon 16 mg	Placebo	Total
All randomised	197	198	395
Safety Analysis Set [a]	195	194	389
Full Analysis Set [b]	182	182	364
Early discontinuation of study	24	19	43

[a] The Safety Analysis Set includes all randomized patients who received at least 1 dose of study treatment.
[b] The Part B Full Analysis Set excludes 29 patients enrolled for regulatory purposes in China after global recruitment was complete.

Demographic characteristics

- **Demographic characteristics are representative of the intended primary IgAN population. Disease characteristics describe a clinically relevant high-risk IgAN population.**
- **Treatment groups were balanced with regards to baseline characteristics.**
- **Blood pressure was well controlled at study entry.**

	Nef-301 Part B Full Analysis Set		
	Nefecon 16 mg (N=182)	**Placebo (N=182)**	**Total (N=364)**
Age (years) (Median [range])	43 [21, 69]	42 [34, 49]	43 [20, 73]
Sex (n, % male)	117 (64%)	123 (68%)	240 (66%)
Race (n, % White)	138 (76%)	137 (75%)	275 (76%)
(n, % Asian)	43 (24%)	40 (22%)	83 (23%)
Systolic BP/Diastolic BP (Median)	126/79	124/79	125/79
UPCR (g/gram) (Median)	1.28	1.25	1.26
eGFR CKD-EPI (mL/min/1.73 m^2) (Median)	56.1	55.1	55.5

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Efficacy Results

eGFR
Primary analysis of eGFR $AUC_{(0-2)}$
The Nef-301 Part B Primary Endpoint was met

- Over 2 years, eGFR was on average 5.05 mL/min/1.73 m^2 higher with Nefecon compared to placebo (p<0.0001)
  - Mean change in eGFR over the 2-year period was -2.47 mL/min/1.73 m^2 for Nefecon 16 mg versus -7.52 mL/min/1.73 m^2 for placebo

Nef-301 Primary analysis of eGFR $AUC_{(0-2)}$ (Full Analysis Set N=364)		
	Nefecon 16 mg (N=182)	**Placebo (N=182)**
eGFR $AUC_{(0-2)}$ (95% CI) [a]	-4.4% (-7.0% to -1.8%)	-13.5% (-15.8% to -11.1%)
Absolute change from baseline in eGFR over 2 years (mL/min/1.73 m^2)	-2.47 (-3.88 to -1.02)	-7.52 (-8.83 to -6.18)
Comparison: Nefecon 16 mg versus Placebo		
Percentage change in eGFR $AUC_{(0-2)}$ (95% CI); p-value	10% (6%, 15%); p<0.0001	
Absolute change (mL/min/1.73 m^2)	5.05	

[a] $AUC_{(0-2)}$ is a time-weighted average of eGFR observed at each time point over 2 years, with the treatment effect interpreted as the average effect of Nefecon over 2 years.

Supportive eGFR Analysis

eGFR 2-year slope analysis

- **Supportive analyses of eGFR 2-year slope were statistically significant and clinically relevant**
- **The improvement in total 2-year eGFR slope was estimated to be 1.8 to 3.0 mL/min/1.73 m^2 per year for Nefecon 16 mg once daily compared to placebo, depending on the analysis method used**
- **All estimates are well in excess of the difference per year in 2 year eGFR total slope required to predict clinically meaningful treatment effects on the composite endpoint of ESRD, eGFR<15 mL/min/1.73 m^2 or sustained doubling of serum creatinine (Inker et al 2019)**

Nef-301 Part B eGFR 2-year Analyses (Full Analysis Set N=364)		
Difference between Nefecon 16 mg and Placebo in 2-year eGFR total slope (mL/min/1.73 m^2 per year) 1-sided p-value	Absolute change in eGFR from baseline at 24 months	
	Nefecon 16 mg (N=182)	Placebo (N=182)
1.8 – 3.0 with p-values <0.0001 - 0.0035	-6mL/min/1.73 m^2	-12mL/min/1.73 m^2

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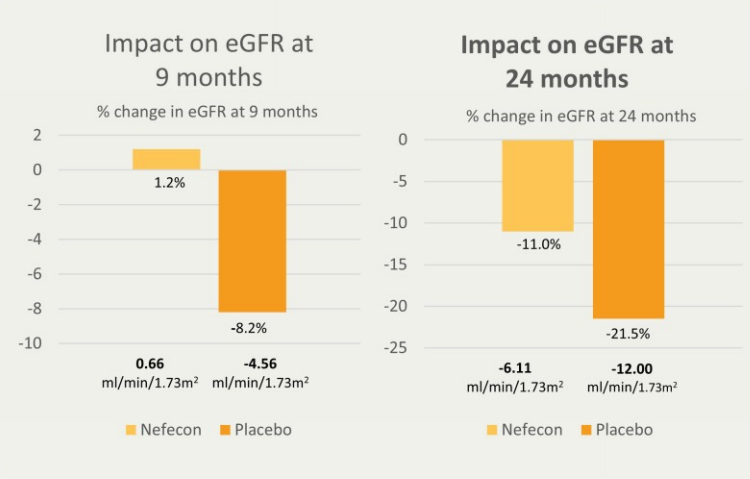
eGFR Phase 3 Data

Sustained eGFR effect observed at 24 months

9 months of dosing with 16mg Nefecon in 364 patients resulted in 50% less loss of kidney function vs placebo at 24 months.



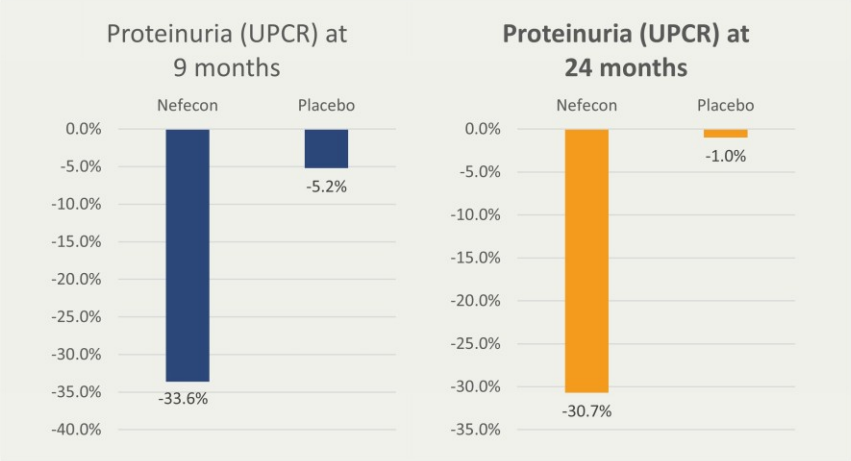
Efficacy Findings

- Statistically significant eGFR stabilization with Nefecon (16 mg) compared to placebo following 9 months treatment ($p < 0.0001$)
- After 9 months:
  - eGFR increase for Nefecon treated patients: 0.66 ml/min/1.73m^2
  - eGFR decline for placebo: 4.56ml/min/1.73m^2
- After 24 months:
  - eGFR decline for Nefecon treated patients: 6ml/min/1.73m^2
  - eGFR decline for placebo: 12ml/min/1.73m^2

UPCR Phase 3 Data

Effect on UPCR maintained at 9 month level, or lower, from the end of treatment through 24 months

- The percent reduction in UPCR for Nefecon 16 mg versus placebo increased over time from 3 to 12 months, and thereafter returned to end of treatment (9 month) levels at the end of the follow-up period (15 months).



Efficacy Summary

The Part B **Primary Endpoint of eGFR AUC(0-2) was met,** showing **statistical significance** of Nefecon (TARPEYO / Kinpeygo) compared to placebo ($p<0.0001$)

Supportive **analyses of 2-year eGFR total slope were statistically significant and clinically relevant**, showing a magnitude ranging from approximately 1.8 – 3.0mL/min/1.73 m^2 per year (active compared to placebo), with p-values ranging from <0.0001 to 0.0035

All estimates are **well in excess of the threshold required** to predict clinically meaningful treatment effects

A **treatment benefit** on eGFR was apparent **across baseline UPCR subgroups**

Sustained proteinuria effects and **long lasting eGFR treatment benefit** even after 15 months after discontinuation, **supporting disease modification**

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Safety Results

Phase 3 safety summary – Part B Full Safety Analysis Set

(≥5% Nefecon-treated patients and ≥2% higher than placebo)

	Nef-301 Part B	
Adverse event N (%)	**Nefecon 16 mg (N=195)**	**Placebo (N=194)**
Peripheral edema	33 (16.9)	10 (5.2)
Hypertension	23 (11.8)	6 (3.1)
Muscle spasms	23 (11.8)	8 (4.1)
Acne	22 (11.3)	2 (1.0)
URTI	16 (8.2)	12 (6.2)
Face edema	15 (7.7)	1 (0.5)
Weight increased	13 (6.7)	6 (3.1)
Dyspepsia	13 (6.7)	4 (2.1)
Arthralgia	12 (6.2)	4 (2.1)
WBC increased	11 (5.6)	1 (0.5)

Safety Summary

- **Nefecon was generally well tolerated**

- **The adverse event profile was similar to that reported in Part A:**
  - The most commonly reported TEAEs observed with an increased frequency compared to placebo were oedema peripheral, hypertension, muscle spasms, and acne**.**
  - **The majority of TEAEs were of mild or moderate severity.**
  - TEAEs led to discontinuation of study drug in <10% of Nefecon-treated patients.

Professor Jonathan Barratt

The Mayer Professor of Renal Medicine at the University of Leicester

Jonathan leads the Renal Research Group within the College of Life Sciences University of Leicester. His research is focussed on a bench to bedside approach to improving our understanding of the pathogenesis of IgA nephropathy a common global cause of kidney failure. Jonathan is the IgA nephropathy Rare Disease Group lead for the UK National Registry of Rare Kidney Diseases (RaDaR) and a member of the steering committee for the International IgA Nephropathy Network. He works closely with pharmaceutical companies interested in new treatments for IgA nephropathy and is Chief Investigator for a number of international randomised controlled Phase 2 and 3 clinical trials in IgA nephropathy and was a member of the FDA and American Society of Nephrology Kidney Health Initiative: Identifying Surrogate Endpoints for Clinical Trials in IgA Nephropathy Work group.


Questions?
Answers.