PRESS RELEASE

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Saniona Tesomet Phase 2a Prader-Willi syndrome trial update

- **Tesomet 0.5 mg/day achieved reductions in weight and hyperphagia in adult PWS patients**
- **Tesomet 0.125 mg/day was well tolerated in adolescent PWS patients, but did not achieve sufficient plasma levels known to be efficacious in previous Phase 2 and Phase 3 studies**
- **Ongoing dose-finding, open-label extension study in PWS adolescents continues at a higher dose of 0.25 mg/day**
- **Tesofensine (the active ingredient in Tesomet) was recently shown to be effective in the Phase 3 registration trial in adult obese patients**

Saniona (OMX: SANION), a biotech company focused on CNS and eating disorders, today reported an update on its ongoing dose-finding Phase 2a study of Tesomet in adolescent patients with Prader-Willi syndrome (PWS). The treatment was well tolerated in this younger group of patients, and eight of the nine patients are currently 7 weeks into an open-label continuation study at the 0.125 mg/day dose. Saniona has filed and received approval for the dose to be increased to 0.25 mg/day in the open-label extension study in the Czech Republic; approval in Hungary is pending.

"We know from the first part of this Phase 2a study that Tesomet is efficacious in adult patients at a dose of 0.5 mg per day. This dose results in plasma levels that are severalfold higher in PWS patients than in our previous trials with normal obese subjects, since Tesomet's half-life is much longer in PWS patients," says Jørgen Drejer, CEO of Saniona. "We are now continuing to seek to establish the optimal dosing regimen in the adolescent population. Because of their age, we started with a conservative, low dose of 0.125mg per day – four times lower than the dose tested in adults, which has not resulted in Tesomet plasma levels correlated with efficacy, and will now double the dose to 0.25 mg/day in the open-label extension.

Based on the strong efficacy on both hyperphagia and weight seen in the first part of the study in adult PWS patients, and the successful Phase 3 trial of tesofensine (the active ingredient in Tesomet) in adult obese subjects, we are confident that a higher dose of Tesomet holds the potential of treating debilitating hyperphagia and significantly reduce weight in this severely underserved population."

This Phase 2a study was an exploratory randomized, double-blind, placebo-controlled Phase 2a trial. The primary endpoint of the study was to examine the change in bodyweight over 12 weeks of treatment with Tesomet compared to placebo. Secondary objectives included eating behavior and food craving (hyperphagia), body composition, lipids and other metabolic parameters. The study was divided into two parts.

The first part included nine adult PWS patients and was successfully concluded in 2018. The results showed that Tesomet 0.5 mg/day may provide clinically meaningful weight loss and a significant reduction in hyperphagia in adult patients. The study results also suggested that the optimal dose in PWS may be 2-4 times lower than in other indications such as hypothalamic obesity, since Tesomet's half-life is much longer in PWS patients.

Therefore, the second part of the study included nine adolescent PWS patients who received Tesomet 0.125 mg/day, which is four times lower than the dose given to adult PWS patients during the first part of the study.

The half-life of tesofensine in the adolescent patient population was confirmed to be very long as also seen in the adult patient population. This means that the mean plasma levels of tesofensine built up slowly during the three-month treatment period, and at the end were only half (~5 ng/ml) of what is considered a therapeutically relevant plasma level (~10 ng/ml) from previous studies in obese patients. Reductions in hyperphagia score and an increase in body weight were observed in both treatment groups. The difference was not statistically significant between the two groups. The treatment was well tolerated and eight of the nine adolescent patients are now 7 weeks into a three-month open-label extension study.

Saniona has applied for regulatory approval to continue the study at a higher dose of 0.25 mg in another three-month open-label extension of the study. The objective is to achieve a similar plasma level of tesofensine in PWS patients as obtained in previous Phase 2 and Phase 3 studies in obese patients where tesofensine has proven to be well tolerated and highly effective in controlling appetite and reduce weight. The application has been approved in Czech Republic and is pending in Hungary. The first patients are expected to be switched to the 0.25 mg dose in March.

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About Saniona

Saniona is a research and development company focused on drugs for diseases of the central nervous system and eating disorders. The company has four programs in clinical development. Saniona intends to develop and commercialize treatments for orphan indications such as Prader-Willi syndrome and hypothalamic obesity on its own. The research is focused on ion channels and the company has a broad portfolio of research programs. Saniona has partnerships with Boehringer Ingelheim GmbH, Productos Medix, S.A de S.V and Cadent Therapeutics. Saniona is based in Copenhagen, Denmark, and the company's shares are listed at Nasdaq Stockholm Small Cap (OMX: SANION). Read more at www.saniona.com.

About Prader-Willi Syndrome (PWS)

Prader-Willi Syndrome (PWS) is recognized as the most common genetic cause of life-threatening obesity. The disease results from a deletion or loss of function of a cluster of genes on chromosome 15, which leads to dysfunctional signaling in the brain's appetite/satiety center (hypothalamus). Patients suffer from a constant, extreme, ravenous insatiable appetite which persists no matter how much the patients eat. As a result, many of those affected with PWS become morbidly obese and suffer significant mortality. Compulsive eating and obsession with food usually begin before age 6. The urge to eat is physiological, overwhelming and difficult to control. Caregivers need to strictly limit the patients' access to food, usually by installing locks on refrigerators and on all closets and cabinets where food is stored. Currently, there is no cure for this disease. Patients with PWS have a shortened life expectancy. Common causes of mortality in PWS include respiratory disease, cardiac disease, infection, choking, gastric rupture, and pulmonary embolism. However, if obesity is avoided and complications are well managed, life expectancy for individuals with PWS is normal or near normal and

most individuals can lead healthy lives[1]. PWS occurs in approximately one out of every 15,000 births[2]. Males and females are affected equally. The condition is named after Andrea Prader, Heinrich Willi, and Alexis Labhart who described it in detail in 1956. The common characteristics defined in the initial report included small hands and feet, abnormal growth and body composition (small stature, very low lean body mass, and early-onset childhood obesity), hypotonia (weak muscles) at birth, insatiable hunger, extreme obesity, and intellectual disability.

[1] Butler MG, Lee PDK, Whitman, BY. Management of Prader-Willi Syndrome. 3rd ed. New York, NY: Springer Verlag Inc.; 2006. 0387253971

[2] https://www.fpwr.org/about-prader-willi-syndrome/ Foundation for Prader-Willi Research retrieved October 2016