NEWS RELEASE

BioVie and Dr. Sheldon Jordan Jointly Announce Topline Results from an Investigator-Sponsored Exploratory Biomarker and Imaging Trial of NE3107 for the Treatment of Alzheimer's Disease

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- Vast majority of patients saw significant improvements in the Global Rating of Change (overall impression of patient's daily abilities) with NE3107 treatment ($p<0.0001$ to $p<0.05$).
- NE3107 is associated with significant improvements in cognition as evidenced by the ADAS-Cog12 scale. 82% of 17 patients with MMSE >=20 experienced a 2.6 point decrease in ADAS-Cog12 ($p=0.0046$).
- Reductions in TNF a (considered to be an initial factor driving inflammation) after NE3107 treatment are significantly correlated with improvement in cognition.
- NE3107 treatment associated with trending improvements in ratio of p-tau:A b . 60% of 10 patients with MMSE >=20 improved 0.002 in the ratio of p-tau / A b ($p=0.055$).
- Early analyses of imaging data suggest fundamental biological improvements in blood flow and reduced oxidative stress that are consistent with the mechanism for NE3107 and are unlikely to be accounted for by placebo effect.
- No drug-related adverse events (AEs) were reported.
- Detailed results will be presented at the CTAD 2022 Annual Conference.

CARSON CITY, Nev., Sept. 07, 2022 (GLOBE NEWSWIRE) -- BioVie Inc., (NASDAQ: BIVI) ("BioVie" or the "Company") a clinical-stage company developing innovative drug therapies for the treatment of advanced liver disease and neurological and neurodegenerative disorders, today announced topline results from an investigator-Sponsored Phase 2 clinical trial of NE3107 for the treatment of Alzheimer's Disease (AD). Updates from other trials underway are also provided.

AD research has largely focused on Amyloid Beta (Ab) and phospho-tau (p-tau) for decades and has resulted in a large number of trials targeting these mechanisms. [1] More recently, however, research focus has shifted towards

targeting neuroinflammation, as evidenced by the 23 disease-modifying agents listed in clinicaltrials.gov in 2021 investigating inflammation or the immune system. NE3107 is the only molecule in this group that is pursuing a two-pronged approach targeting both neuroinflammation and insulin resistance. Furthermore, NE3107 is the only molecule in the group that is conducting a potentially pivotal Phase 3 trial (NCT04669028) that is currently underway in mild- to moderate-AD patients with co-primary endpoints of cognition, as measured by the Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-Cog12), and function, as measured by Alzheimer's Disease Cooperative Study-Clinical Global Impression of Change (ADCS-CGIC), whereas 17 other agents are in Phase 2. This Phase 3 trial is expected to provide topline results in mid-2023.

Tumor Necrosis Factor Alpha (TNFa) is a cytokine identified as a major regulator of inflammation given that its excessive activation is associated with chronic inflammation. [2] It is considered to be the central mediator of inflammation due to its role at the top of the biochemical pathway that leads to the production of other inflammatory factors such as various cytokines (e.g., IFNg, IL-1b, IL23, IL-4, IL-17), neurotoxic Aβ peptide oligomers, activation of IKK and JNK (that leads to insulin resistance), and others. A large retrospective study analyzing the electronic medical records of 56 million unique patients demonstrated the linkage between TNFa and AD by showing that patients with rheumatoid arthritis and psoriasis taking TNF blocking agents had significantly lower AD risk. [3] Preclinical studies showed NE3107 is a modulator of TNFa production through its ability to modulate the activation of the Extracellular Regulated Kinase (ERK) and Nuclear Factor kappa B (NFkB). [4] By down regulating the activation of ERK and NFkB, NE3107 has been shown to reduce the production of TNFa. [5] The Company's newly generated data supporting a new patent application show that NE3107 inhibits inflammatory ERK activation and blocks the phosphorylation of TNF receptor 1 (TNFR1) in an IKK-MAP3K8-MEK dependent pathway to decrease forward-feeding TNF inflammatory cascades, thereby lowering the expression of other downstream inflammatory factors.

Since the NE3107 Phase 3 trial underway is focused on cognition and function and is not focused on collecting neuroinflammatory biomarkers, the Company supported a Phase 2 investigator-initiated trial (NCT05227820) to explore the link between NE3107's role in neuroinflammation and insulin resistance to the biomarkers historically used by the AD research community. It also was designed to provide, if possible, a glimpse into what can be expected regarding cognition and function when the Phase 3 trial reads out next year. This was done by using a modified ADAS-Cog12 scale for cognition and the Global Rating of Change (GRoC), which is an instrument that can be administered more easily in general clinical practices than the ADCS-CGIC.

The Phase 2 trial— A Phase II Open-Label Study for the Use of Anti-Inflammatory, Insulin-Sensitizing NE3107 for Treatment of Cognitive Decline Due to Degenerative Dementias (NCT05227820) — is an exploratory biomarker study conducted by Dr. Sheldon Jordan [6], who served as principal investigator for the trial. The trial was intended to explore NE3107's potential role in real-world clinical practice as an exploratory precursor informing the design of

subsequent placebo-controlled blinded studies.

Results from Phase 2 Exploratory Biomarker Trial

The trial enrolled a total of 23 patients – 17 patients with Mini-Mental State Examination (MMSE) scores greater than or equal to 20 (i.e., mild cognitive impairment [MCI] to mild AD) and 6 patients with MMSE <20 (i.e., moderate AD) – in an open-label, single arm study. The trial measured changes in cognition through verbal and visual test procedures, changes in biomarkers of Alzheimer's disease and inflammation that can be measured in cerebral spinal fluid (CSF) and serum samples, and with functional magnetic resonance imaging techniques in patients before and after treatment with 20 mg of NE3107 twice daily for 3 months.

Initial results showed that the measurements for most patients improved with NE3107 treatment, although MCI/mild AD patients showed greater change.

- The Global Rating of Change is a frequently used outcome measure that tracks improvements in a patient's conditions, abilities and overall sense of well-being as perceived by the clinician, the caretakers and the patients. A large proportion of MCI/mild AD patients in the trial showed improvements as evidenced by an increase of the GRoC scale after treatment with NE3107 compared to baseline as scored by the clinician (94% of 17 patients improved, p=0.0001), the study partner (65% of 17 patients improved, p=0.078), and the patient (88% of 17 patients improved, p=0.0039). A similar pattern of improvements was observed within the total pool of patients.
- Several tools were used to assess changes in cognition, including a modified ADAS-Cog12, the Quick Dementia Rating Scale (QDRS), Mini-Mental State Examination (MMSE), and the Montreal Cognitive Assessment (MoCA).
    - 82% of 17 MCI/mild AD patients improved with a mean ADAS-Cog12 change of -2.6 points (p=0.046) equating to a mean percentage change of -25.1% (p=0.0026). 61% of all 23 patients improved with a mean change of -1.04 (p=ns).
    - A similar pattern of improvement was observed using the QDRS. 71% of 17 MCI/mild AD patients improved with a mean change of –1.35 (p=.0051).
- Improvements in ADAS-Cog12 were correlated with improvements in the Global Rating of Change as reported by the clinician (R=0.52, p<0.05) and the caretaker (R=0.46, p<.05).
- 62% of 13 MCI/mild AD patients had decreased plasma TNF with a mean change of -0.55 pg/mL (p=0.22). Importantly, improvements in TNFa statistically correlated to improvements in ADAS-Cog12 (R=0.70, p=0.0077).
- The ratio of p-tau to Ab $_{42}$ has been shown by the Mayo Clinic and others to be an effective predictor of PET Ab status. [7] 63% of 16 patients improved with a mean change in the ratio of -0.0033 (p=0.069).

- While quantitative scoring of imaging data is currently underway, review of imaging scans and data by the study's Principal Investigator (who is an imaging expert) indicates that NE3107 treatment in patients with MMSE >=20 is associated with certain changes in the brain. Among patients with MMSE >=20:
    - 24% had increased blood flow in the brain while 6% declined. Blood flow is a marker for metabolism and brain activity.
    - 41% to 47% had reduced the hyperactivation associated with cognitive impairment that results when the hippocampus (part of the brain that plays a major role in learning and memory) is stressed while 6% declined. This is a marker for network connectivity in the brain
    - 41% had increased levels of glutathione (considered the master antioxidant and regulator of oxidative stress [8]) in the brain while 35% declined.
- No treatment related AEs were reported.

The Company will present the final data and statistical analyses, which may deviate from the initial result presented here, at the Clinical Trial in Alzheimer's Disease (CTAD) annual conference, to be held in San Francisco, CA November 29-December 2, 2022. Platform presentations and posters will provide detailed findings on changes on biomarker, neuropsychological assessments, and advanced MRI imaging. Research papers are being prepared to present the findings and will be submitted to peer-reviewed journals for publication.

"Through our work, we are seeking to deepen our understanding of brain degeneration and ultimately identify promising treatments that have the potential to counteract the degenerative process. Results from this trial provide encouraging signals that NE3107 may offer significant potential to reduce neuroinflammation and potentially improve metabolic parameters such as glutathione in the brain, and warrant further study in this patient population," said Dr. Jordan. "I was surprised to see imaging data changes and how so many patients had increased GRoC score, indicating they can perform more activities of daily living."

"We are delighted to support the work of Dr. Jordan and his team and are very pleased to have this initial data-driven validation of NE3107's potential role for the treatment of people suffering from cognitive impairment and dementia," stated Dr. Joseph Palumbo, BioVie's Chief Medical Officer. "Despite this being an open label study, the insights provided are instructional and directionally very interesting. While it is not possible to isolate the placebo effect in an open label study as a placebo-controlled double blinded trial can, results on TPO arterial spin label imaging, hippocampal blood oxygen level-dependent imaging, brain NMR spectroscopy measuring glutathione, and peripheral TNF reduction from this study provide helpful information to plan our next steps."

"This study provides us with an early opportunity to assess the effects of NE3107's mechanism of action in the brain, and these topline results represent the first detailed data linking NE3107's role in neuroinflammation and insulin resistance with the traditional biomarkers of interest to the AD research community," added Cuong V. Do,

President and CEO of BioVie. “I was excited about correlation of TNFa reduction with improvements in cognition and how treated patients saw an improvement in the ratio of p-tau:Ab. We supported Dr. Jordan’s Investigator-Sponsored trial as an exploratory biomarker study to guide our efforts to design future placebo-controlled trials to examine the potential impact of NE3107 on traditional AD biomarkers and measures of cognition and function. We are very grateful for the work of Dr. Jordan and his team on this important study and look forward to the team’s presentation at CTAD later this year.”

Commenting on the mixed results observed in AD research over the decades, Mr. Do added, “AD drug development at BioVie is based on the evidence that AD pathology is multifactorial in nature. While Ab and p-tau are important factors, decades of research on dozens of agents have not conclusively correlated improvements on these individual factors to improvements in cognition. We believe additional factors such as inflammation, insulin resistance, metabolic dyshomeostasis, apoptosis, oxidative stress, and glucose utilization also play critical roles in AD etiology. Data shows that NE3107’s ability to reduce TNFa (the major regulator of inflammation) is highly correlated to improvements in cognition. We are pleased that the Principal Investigator observed these changes in 23 patients in only 3 months. We hypothesize that the modulation of TNFa levels and its inflammatory activation via TNFR1 lead to a multitude of changes among the many factors downstream from this master regulator, which collectively lead to improvements in neuronal health and cognition.”

Update on other clinal trials

In addition to the Phase 2 exploratory biomarker trial, the Company has three other trials underway.

- The potentially pivotal Phase 3 trial for NE3107 in AD (NCT04669028) has enrolled one-half of the targeted 316 patients. In blinded data, no drug-related adverse events have been seen in daily medical reviews. The study pre-specified the potential to increase enrollment up to 400 patients as deemed appropriate through a review by the data safety monitoring board (DSMB) in a manner that is blinded to the Company. This DSMB review will take place later this year and will determine if a sample size adjustment is needed for the purpose of enhancing the probability of achieving statistical significance. If no sample size adjustments are needed, the Company expects full enrollment of the study by the end of the year, enabling topline data readout by mid-2023. The study may be delayed if additional patients need to be enrolled in the event of a DSMB recommendation.
- The Phase 2 trial (NCT05083260) is enrolling 40 patients to examine NE3107 in Parkinson’s disease (PD) with two design objectives. The primary objective is a drug-drug interaction study as requested by the FDA to demonstrate the absence of adverse interactions of NE3107 with levodopa in humans (no indications of adverse DDI were observed in prior animal studies). The secondary objective is to explore an efficacy signal and determine if preclinical indications of promotoric activity and apparent enhancement of levodopa activity

in MPTP rodents and marmosets can be observed in humans. In preclinical studies, NE3107 had promotoric activity equal to levodopa and the combination of NE3107 with levodopa was superior to either agent alone, providing the most “on time” without dyskinesia. The Company expects the trial to be fully enrolled in the next couple of months and data readout by the end of 2022. Similar to the AD Phase 3, no drug-related adverse events have been seen in daily medical reviews. We are detecting an efficacy signal among patients who have completed 28 days of treatment, but more data is needed to quantify the full therapeutic impact.

- The Phase 2b trial for BIV201 in refractory ascites (NCT04112199) is enrolling 30 patients with co-primary endpoints measuring the incidence of ascites-related complications over 180 days and change in ascites fluid removed over 90 days. The trial is taking much longer than expected. Patient recruitment has been hampered by Covid-19, which appears to have disproportionately affected this population of very sick patients. The pace of patient screening, however, appears to be picking up in recent weeks. At this pace, the Company anticipates data readout by mid-2023.

The updated corporate overview and additional information can be found at **https://bioviepharma.com/investors/** .

About BioVie

BioVie Inc. (NASDAQ: BIVI) is a clinical-stage company developing innovative therapies to overcome unmet medical needs in chronic debilitating conditions. In neurodegenerative disease, the Company’s drug candidate NE3107 inhibits inflammatory activation of ERK and NFkB (e.g., TNF signaling) that leads to neuroinflammation and insulin resistance, but not their homeostatic functions (e.g., insulin signaling and neuron growth and survival). Both are drivers of Alzheimer’s and Parkinson’s diseases. The Company is conducting a potentially pivotal Phase 3 randomized, double blind, placebo controlled, parallel group, multicenter study to evaluate NE3107 in patients who have mild to moderate Alzheimer's disease (NCT04669028) and is targeting primary completion in mid-2023. An estimated six million Americans suffer from Alzheimer’s. A Phase 2 study of NE3107 in Parkinson’s disease (NCT05083260) is enrolling patients and expects to have topline data readout by the end of 2022. In liver disease, the Company’s Orphan drug candidate BIV201 (continuous infusion terlipressin), with FDA Fast Track status, is being evaluated in a US Phase 2b study for the treatment of refractory ascites due to liver cirrhosis with top-line results anticipated in mid-2023. BIV201 is administered as a patent-pending liquid formulation. The active agent is approved in about 40 countries for related complications of advanced liver cirrhosis but is not available in the US or Japan. For more information, visit **http://www.bioviepharma.com/.**

Forward-Looking Statements

This press release contains forward-looking statements, which may be identified by words such as "expect," "look forward to," "anticipate" "intend," "plan," "believe," "seek," "estimate," "will," "project" or words of similar meaning. Although BioVie Inc. believes such forward-looking statements are based on reasonable assumptions, it can give no

assurance that its expectations will be attained. Actual results may vary materially from those expressed or implied by the statements herein due to the Company's ability to successfully raise sufficient capital on reasonable terms or at all, available cash on hand and contractual and statutory limitations that could impair our ability to pay future dividends, our ability to complete our pre-clinical or clinical studies and to obtain approval for our product candidates, to successfully defend potential future litigation, changes in local or national economic conditionsas well as various additional risks, many of which are now unknown and generally out of the Company's control, and which are detailed from time to time in reports filed by the Company with the SEC, including quarterly reports on Form 10-Q, reports on Form 8-K and annual reports on Form 10-K. BioVie Inc. does not undertake any duty to update any statements contained herein (including any forward-looking statements), except as required by law.

Abbreviations Used

Ab	Amyloid beta
AD	Alzheimer's disease
ADAS Cog 12	Alzheimer's Disease Assessment Scale-Cognitive subscale 12
ADCS-CGIC	Alzheimer's Disease Cooperative Study-Clinical Global Impression of Change Scale
AKI	Acute kidney injury
CSF	Cerebral spinal fluid
CTAD	Clinical Trials on Alzheimer's Disease (a medical conference)
DSMB	Data and Safety Monitoring Board
ERK	Extracellular signal regulated kinase 1 and 2
GRoC	Global Rating of Change
HRS	Hepatic renal syndrome
IKK	IkappaB kinase, an inflammatory signaling protein that activates NFkB
INFg	Interferon gamma
IL-1b	Interleukin one beta
IL- 4, 17, 23	Interleukin 4, 17, 23
JNK	c-Jun N-terminal kinases, an inflammatory signaling protein kinase
MAP3K8	Mitogen-activated protein kinase kinase kinase 8, an inflammatory signaling protein involved in pathological inflammation
MCI	Mild cognitive impairment
MEK	Mitogen and ERK kinase, an ERK activating protein kinase
MMSE	Mini Mental State Examination
MoCA	Montreal Cognitive Assessment
NE3107	17a-ethynyl-androst-5-ene-3b,7b,17b-triol
NFkB	Nuclear factor kappa-light-chain-enhancer of activated B cells
p38	One of several mitogen-activated protein kinases involved in inflammatory signaling
PD	Parkinson's disease
pg	picogram
p-tau	Hyperphosphorylated tau protein
QDRS	Quick Dementia Rating Scale
TNFa	Tumor necrosis factor alpha
TNFR1	Tumor necrosis factor receptor R1

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[1] Cummings et al. 2022 DOI: 10.1002/trc2.12295

[2] Jang et al. Int J Mol Sci. 2021 Mar; 22(5): 2719.

[3] Zhou et al. 2020 PLoS ONE 15(3): e0229819.

[4] Lu 2010 Am J Physiol Endocrinol Metab 298 E1036; Wang 2010 J Pharmacol Exp Ther 333 70

[5] Offner 2009 J Pharmacol Exp Ther 329 1100

[6] Dr. Jordan is Adjunct Clinical Associate Professor of Neurology at UCLA. Dr. Jordan is board certified by the American Board of Psychiatry & Neurology, American Board of Clinical Neurophysiology, American Board of Addiction Medicine, and American Board of Interventional Pain Medicine. He was elected as Fellow of the American Academy of Neurology and holds additional certifications from the American Society of Neuroimaging in Magnetic Resonance Imaging, Computed Tomography and Ultrasonography. Dr. Jordan is the senior author of a recent paper on quantitative MRI imaging in dementia (Kuhn et al., 2021 Quant Imaging Med Surg 11)

[7] Campbell et al. Alzheimers Dement (Amst). 2021; 13(1): e12190.

[8] Teskey et al. Advances in Clinical Chemistry Volume 87, 2018, Pages 141-159