A Multicenter, Randomized, Double-Blind, Placebo-Controlled, Parallel Design, Prospective, 52-Week, Phase 2 Clinical Study to Evaluate the Safety and Efficacy of GV1001 Administered Subcutaneously for the Treatment of Mild to Moderate Alzheimer*s Disease

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This study has been transitioned to CTIS with ID 2024-511610-20-00 check the CTIS register for the current data. Cognitive and functional abilities will be evaluated using psychometric scales (ie, cognitive subscale of the Alzheimer*s Disease...

Ethical review	Approved WMO
Status	Recruiting
Health condition type	Structural brain disorders
Study type	Interventional

Summary

ID

NL-OMON53512

Source
ToetsingOnline

Brief title
GV1001 SC for the Treatment of Mild to Moderate (Stage 4 and 5) AD

Condition

- Structural brain disorders

Synonym
Alzheimer's Disease

Research involving
Human

Sponsors and support

Primary sponsor: GemVax & KAEL Co., Ltd.
Source(s) of monetary or material Support: GemVax & KAEL Co.;Ltd

Intervention

Keyword: Alzheimer's Disease, Mild to Moderate (Stage 4 and 5) Alzheimer's Disease

Outcome measures

Primary outcome

Change from baseline in ADAS-Cog11 score at Week 52

Adverse events (AEs), laboratory test results (hematology, serum chemistry, and urinalysis), electrocardiogram (ECG) findings, and vital signs measurements (pulse rate, blood pressure, respiratory rate, body temperature). Suicidal ideation and behavior will be assessed using the Columbia-Suicide Severity Rating Scale (C SSRS)

Secondary outcome

o evaluate the efficacy of GV1001 (0.56 mg and 1.12 mg) relative to placebo on cognition and function in participants with mild to moderate AD, as measured by:

- A-IADL-Q
- CDR-SB
- ADAS-cog11
- NPI

- MMSE

- ADCS-CGIC/CIBIC-Plus

- QoL-AD

Secondary efficacy endpoints:

• Clinical worsening, defined as >=4 points change from baseline in the ADAS-cog11 score at Week 12, Week 26, Week 38, and Week 52

• Change from baseline in A-IADL-Q score at Week 12, Week 26, Week 38 and Week 52

• Change from baseline in NPI score at Week 12, Week 26, Week 38, and Week 52

• Change from baseline in MMSE score at Week 12, Week 26, Week 38, and Week 52

• Change from baseline in CDR-SB score at Week 12, Week 26, Week 38, and Week 52

• Change from baseline in ADCS-CGIC/CIBIC-Plus score at Week 12, Week 26, Week 38, and Week 52

• Change from baseline in QoL-AD score at Week 26 and Week 52

Study description

Background summary

1.1.1 Alzheimer*s Disease
Alzheimer*s disease (AD) is the most common cause of dementia and the sixth leading cause of death in adults in the United States (US) (Herbert et al, 2013; Xu et al, 2010; Burns and Iliffe, 2009). An estimated 6.07 million people in the US had AD in 2020, and the number is expected to rise to 13.85 million per year by 2060 (Rajan et al, 2021). Worldwide, approximately 47 million people are affected with dementia with AD being the most common cause and accounting for about 70% of cases (Emmady and Tadi, 2021). The average life expectancy after diagnosis is 8 years (Alzheimer*s Association, 2021). The economic burden of AD is substantial. Costs associated with AD ranged between

$159 and $215 billion in 2010, and are projected to be between $379 and more than $500 billion in 2040 (Hurd et al, 2013).
The exact mechanism of AD is unknown; therefore, treatment is difficult. According to a 2015 report by the American Pharmaceutical Association (PhRMA, 2018), between 1998 and 2014 about 123 drugs under development for AD failed. Only 4 drugs (donepezil, galantamine, rivastigmine, and memantine) have been approved until 2003 for use by the US Food and Drug Administration (FDA) (PhRMA, 2018). In addition, a combination product of memantine and donepezil is available (Alzheimer*s Association, 2021). Another product (tacrine) was approved prior to 1998, but was discontinued in May 2012 for safety reasons. Most recently, aducanumab (Aduhelm*) has been approved by the FDA using the accelerated approval pathway; however, some uncertainty about the drug*s clinical benefit remains (FDA, 2021). Available symptomatic treatments may alleviate cognitive and behavioral symptoms and improve quality of life, but do not impact disease progression.
Although the exact cause of AD is not known, several hypotheses are being explored including cholinergic hypothesis (Francis et al, 1999), genetic mutations, amyloid beta (Aβ) aggregation (Hardy and Allsop, 1991), and tau hyperphosphorylation (Mudher and Lovestone, 2002). Among these, amyloid hypothesis is believed to be most important. Truncated Aβ is pathologically increased in the AD brain and its monomers start to aggregate and form oligomers and finally plaques. The oligomers are thought to be toxic to surrounding neurons and glial cells. Aducanumab is the first novel therapy for AD approved since 2003 and is the first treatment directed at the underlying pathophysiology of AD, the presence of Aβ plaques in the brain (FDA, 2021). Tauopathy is also thought to play critical roles in the pathogenesis of AD and is strongly correlated with cognitive decline. Tau, a protein closely related to the microtubule associated with cell structure and transport of intracellular substances, is hyperphosphorylated by a phosphorylating enzyme to form a bundle and form a neurofibrillary tangle (NFT). As a result, the axoplasmic transport of nerve cells fails to function and brain cells die (Haass et al, 2012). Inflammation is one of the important pathogenic mechanisms in the progression of AD. Inflammation causes functional impairment and death of neuronal cells.
Therefore, an effective and well tolerated treatment for AD that not only manages symptoms but prevents or improves memory loss and decline in cognition continues to be an important unmet medical need in the long-term management of AD. New drugs in development aim to modify the disease process itself, by impacting one or more of the many wide-ranging brain changes that AD causes. Drugs in development include those that target Aβ and tau protein, β secretase, inflammation, and 5-HT2A receptor (Alzheimer*s Association, 2021).
1.1.2 GV1001
GV1001 (hTERT peptide; tertomotide hydrochloride) is a single peptide of 16 amino acids derived from telomerase reverse transcriptase (TERT). This peptide corresponds to a fragment from the catalytic site of the enzyme telomerase. Telomerase is a reverse transcriptase that maintains telomere function in dividing cells. GV1001 was first developed as an active immunotherapy in the

treatment of cancer forms expressing telomerase. After administration, GV1001 is processed and presented in the human leukocyte antigen (HLA) complex and gives rise to T-cells that recognize cells expressing telomerase.
Telomerase has been proposed to possess anti-aging properties. The catalytic subunit of telomerase, TERT, is expressed in neurons throughout the brain during development, but is absent from neurons in the adult brain (Mattson, 2000). Telomerase reverse transcriptase has been shown to exhibit neuroprotective properties in experimental models of neurodegenerative disorders suggesting that restoring TERT expression in neurons in the adult brain may protect against age related neurodegeneration (Zhu et al, 2000). The TERT protein may offer neuronal resistance against pathological tau by reducing production of oxidative species and improving mitochondrial function (Spilsbury et al, 2015). The neuroprotective properties of TERT are the basis for new treatments like GV1001 to slow or stop disease progression or to prevent neurodegenerative diseases (GemVax Study 20140000002332-1 and Study 20140000002332-2).
GV1001 and AD
Studies using in vivo and in vitro AD models have shown that GV1001 inhibits neurotoxicity, apoptosis, and the production of reactive oxygen species induced by Aβ in neural stem cells (NSCs). It effectively blocks Aβ toxicity by mimicking the extra-telomeric functions of hTERT. The findings from these in vivo and in vitro studies, summarized below, led to the development of GV1001 as a therapeutic agent for AD.
First, GV1001 was shown to protect neural stem cells (NSCs) against Aβ (Park et al, 2014; Park et al, 2016). Although Aβ decreased viability, proliferation, and mobilization of NSCs, treatment with GV1001 restored the cells to wild-type levels. These effects were mediated by mimicking the extra-telomeric functions of hTERT. In addition, GV1001 treatment was shown to increase the expression level of survival-related proteins, and decrease the levels of death and inflammation-related proteins.
Second, GV1001 has been shown to have direct effects against oxidative stress. Treatment with GV1001 rescued hydrogen peroxide (H_2O_2)-injured NSCs. GV1001 was also shown to have antioxidant and neuroprotective effects, which appear to be mediated by scavenging free radicals, increasing survival signals and decreasing death signals (Park et al, 2014; Park et al, 2016). Third, GV1001 significantly improved memory functions when evaluated in a transgenic (Tg) AD mice model. This was shown in both a passive avoidance test and Y-maze test. In the mouse model, GV1001 was effective from the mild to the moderate stage (9 month to 18 month old Tg mouse) and more effective at the moderate stage (internal report only, not published yet).
When it comes to mechanisms, GV1001 decreased the amount of Aβ, prevented formation of NFT resulting from pathologic changes of the tau protein, inhibited astrogliosis, and promoted neurogenesis. These results suggest that GV1001 may play a major role in reducing cytotoxicity and enhancing rehabilitation of brain function.
GV1001 reproduces the exact amino acid sequence from position 611 to position 626 of hTERT. Several in vitro studies have shown that GV1001 protects neural

cells against neurotoxicity, apoptosis, and reactive oxygen species (ROS) induced by Aβ and oxidative stress inducing cell, anti-apoptotic effects, mitochondrial stabilization, and anti-aging and antioxidant effects. Acting on several mechanisms related to AD pathology, GV1001 may be efficacious in these patients. In a completed Phase 2 study conducted in Korea, GV1001 showed significant improvement in change from baseline of Severe Impairment Battery score at Week 24 and demonstrated a c

Study objective

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Cognitive and functional abilities will be evaluated using psychometric scales (ie, cognitive subscale of the Alzheimer*s Disease Assessment Scale [ADAS cog11]), assessment of activities of daily living (ie, Amsterdam Instrumental Activities of Daily Living Questionnaire [A-IADL-Q]), and global ratings of dementia (ie, Clinical Dementia Rating-Sum of Boxes [CDR SB], Neuropsychiatric Inventory [NPI], Mini Mental State Examination [MMSE],Alzheimer*s Disease Cooperative Study-Clinical Global Impression of Change [ADCS-CGIC], Clinician*s Interview-Based Impression of Change - Plus Family Input [CIBIC-Plus], and Quality of Life in Alzheimer*s Disease [QoL-AD]). Efficacy evaluations will be performed at baseline, Week 12, Week 26, Week 38, and Week 52 (the primary endpoint [PE]). Structural changes in the brain will be evaluated using volumetric magnetic resonance imaging (vMRI) at baseline and Week 52. The safety of GV1001 will be assessed throughout the study.

Study design

This is a multicenter, randomized, double-blind, placebo-controlled, parallel group Phase 2 study in participants with mild to moderate AD. The study population will include male and female participants between 55 and 85 years of age (inclusive), with a diagnosis of probable AD as demonstrated by meeting National Institute of Neurological and Communicative Disorders and Stroke-Alzheimer*s Disease and Related Disorders Association (NINCDS ADRDA) criteria. Participants should have a magnetic resonance imaging (MRI) or computed tomography (CT) scan performed within the 2 years prior to screening with findings consistent with a diagnosis of AD. Results from Aβ positron emission tomography (PET) scan or cerebrospinal fluid (CSF) examination performed within the 2 years prior to screening will be used to confirm eligibility. If no Aβ PET scan or CSF examination results are available, participants will undergo an Aβ PET scan at screening. Participants or their legal representative, as well as the participant*s caregiver, must be able to provide written informed consent.
The study will consist of a screening period (up to 60 days prior to the first dose), a 52 week double blind treatment period, and an end-of-study (EOS)

visit. Eligible participants will be randomized in a 1:1:1 ratio to receive treatment with GV1001 0.56 mg, GV1001 1.12 mg, or placebo (normal saline) every week for 4 weeks beginning on Day 1 (of Week 1) followed by every 2 weeks through Week 50.
An independent Data and Safety Monitoring Board (DSMB) review will be planned when 90 participants (50%) have either completed Week 26 or have discontinued the study to evaluate safety data. The DSMB may recommend early stopping of the study for safety reasons.
If a participant discontinues treatment prematurely, the participant will be asked to continue with the scheduled study visits until the EOS visit. If a participant discontinues the study prematurely (except for those who withdraw consent), the participant will be asked to come for an early termination (ET) visit for efficacy scale and safety assessments. These assessments are the same as those scheduled at the PE visit at Week 52. If the ET visit takes place within 4 weeks after a completed protocol scheduled visit with efficacy assessments, efficacy scale assessments are not required at the ET visit.
Prior to randomization, eligibility of potential participants will be confirmed through an adjudication process in which screening data (eg, MMSE, MRI/CT scans) obtained to evaluate AD status are reviewed by a central independent adjudicator. The central independent adjudicator will review the scoring sheet completed by the Investigator prior to randomization and provide an independent assessment of the participant*s eligibility and may request exclusion of a participant from entry into the study. A central independent reader will review magnetic resonance imaging (MRI) and/or computed tomography (CT) scans to confirm eligibility. Investigators must not randomize a participant prior to receipt of this independent confirmation of the participant*s eligibility.
Efficacy evaluations will be performed at baseline, Week 12, Week 26, Week 38, and Week 52 using the ADAS-cog11, A-IADL-Q, CDR-SB, NPI, MMSE, CIBIC-Plus, and QoL AD scales. The ADAS-cog11 scale will be evaluated by a central independent reader for each visit. At the visits where several efficacy assessments are administered, every effort should be made to perform the efficacy evaluations in the same order at each visit (ADAS-cog11, A-IADL-Q, CDR SB, NPI, MMSE,ADCS-CGIC, CIBIC-Plus, and QoL AD). To ensure the objectivity and accuracy of the study results, efficacy evaluations must be performed by adequately trained and experienced raters. The raters must be certified for this study to administer the ADAS-cog11, A IADL Q, CDR-SB, NPI, MMSE, CIBIC-Plus, QoL-AD, and C-SSRS scales. Training, certification, and materials for rating will be provided by a rater training group. To mitigate the risk of breaking the blind, the efficacy evaluator is not to be involved in the participant*s treatment or have access to the record of reported AEs.
Safety will be assessed throughout the study by monitoring for AEs, laboratory evaluations, electrocardiogram (ECG) findings, vital signs measurements, physical examination, and suicidal ideation and behavior (C-SSRS). Blood and CSF samples will be collected to evaluate the effect of GV1001 on analysis of biomarkers of AD. Blood samples will also be collected for and immunogenicity analysis (antibodies to GV1001).

Intervention

The study will consist of a screening period (up to 60 days prior to the first dose), a 52-week double-blind treatment period, and an EOS visit. For an individual participant, the maximum duration of study participation is approximately 14.5 months, including an up to 60-day screening period. GV1001 (0.56 mg or 1.12 mg) or placebo (normal saline) will be administered by a SC injection once weekly for 4 weeks (4 times) beginning on Day 1 (of Week 1) followed by a SC injection every 2 weeks through Week 50 (23 times) for a total of 27 SC injections.

Study burden and risks

1.3 Benefit/Risk Assessment
Although there have been numerous clinical studies evaluating treatments for AD, most treatments currently approved for use are symptomatic treatments (ie, donepezil, galantamine, rivastigmine, memantine, or memantine/donepezil combination product). To date, treatments with a single mechanism of action have been developed based on the pathophysiology of AD. However, these are not disease modifying agents, and their effect is unsatisfactory. Recently, aducanumab has been approved as the first treatment directed at the underlying pathophysiology of AD, the presence of Aβ plaques in the brain (FDA, 2021). As AD is thought to be a complex disease caused by a variety of mechanisms, therapeutic agents with various mechanisms of actions may be more successful in clinical practice than a single mechanism-based therapy.
Unlike conventional therapeutic drugs with single mechanism of action, GV1001 with its multiple mechanisms of action is expected to be effective as a therapeutic agent for AD as it has anti inflammatory and anti oxidative properties and blocks the accumulation of Aβ and inhibits tau protein condensation. Therapeutic agents, such as GV1001, with various mechanisms rather than single mechanism-based agents may be successful in the treatment of AD patients in clinical practice. The current study is expected to further demonstrate the potential of GV1001 as a new class of medication for the treatment of AD.
In this study, participants may continue to take their prior medications for AD available per local regulations, as long as they were taking a stable dose for at least 12 weeks prior to the screening visit. Participants may continue to take over-the-counter (OTC) cognition supplement during the study if they were not exceeding the recommended dose for at least 12 weeks prior to the screening visit.
Treatment with GV1001 has been well tolerated in clinical studies in multiple cancer indications and BPH. Therefore, the potential adverse effects or risks associated with GV1001 in the current study are expected to be small and manageable, while the expected benefits may be substantial. However, the experience with GV1001 as treatment for AD is limited. Further information on the safety of GV1001 will be obtained in the current study.

The available information suggests that the present study may have a favorable risk-benefit ratio. However, the potential benefits of GV1001 as treatment for AD have not been fully explored. The GV1001 clinical development program will continue to evaluate the risk-benefit of GV1001 in specific clinical circumstances with high medical need, including AD.
Refer to Section 3 for details of study procedures, dose, and study design justification. Detailed information about the known and expected benefits and risks and reasonably expected AEs of GV1001 is available in the Investigator*s Brochure.

Contacts

Public
GemVax & KAEL Co., Ltd.

3fl, Unjung-ro 117
Bundang-gu 13461
KR
Scientific
GemVax & KAEL Co., Ltd.

3fl, Unjung-ro 117
Bundang-gu 13461
KR

Trial sites

Listed location countries

Netherlands

Eligibility criteria

Age
Adults (18-64 years)
Elderly (65 years and older)

Inclusion criteria

1. Male or female participants 55 to 85 years of age (both inclusive) at the

time of signing the informed consent.
2. Diagnosis of probable AD based on NINCDS-ADRDA criteria (a and b) as determined by a neurologist, geriatrician, psychiatrist, or clinician approved by the Sponsor or designee.
a. Presence of an early and significant episodic memory impairment that includes the following features:
i. Gradual and progressive change in memory function reported by patients or informants over >6 months.
ii. Objective evidence of significantly impaired episodic memory on testing: this generally consists of recall deficit that does not improve significantly or does not normalize with cueing or recognition testing and after effective encoding of information has been previously controlled.
iii. The episodic memory impairment can be isolated or associated with other cognitive changes at the onset of AD or as AD advances.
b. One or more findings for probable AD by either MRI, Aβ PET scan, historical CSF results, or a historical genetic test in the 2 years before screening, or an MRI or Aβ PET scan at screening. The MRI must have findings consistent with AD and without any other disease that may cause dementia. The Aβ PET scan and historical CSF results must be consistent with the presence of amyloid pathology.
3. Mild or moderate dementia as evidenced by MMSE score >=13 to <=24 at screening (Visit 1).
4. not applicable.
5. not applicable.
6. If receiving an approved medication for AD (ie, donepezil, galantamine, rivastigmine, memantine, or memantine/donepezil combination product), must be on the medication with a stable dose for at least 12 weeks before the screening visit (dosing should remain stable throughout the study).
7. If receiving an OTC supplement for cognition (eg, gingko biloba, omega-3 polyunsaturated fatty acid, vitamin E, curcumin), must not be exceeding the recommended dose for at least 12 weeks prior to screening visit.
8. Able to visit the study center and undergo cognitive, functional, and other tests specified in the protocol.
9. Has a caregiver who:
• Agrees to accompany the participant to all study visits and able to supervise the participant's compliance with the study procedures and provide detailed information about the participant.
• Either lives with the participant or sees the participant on average for >=1 hour/day >=3 days/week, or in the Investigator's opinion, the extent of contact is sufficient to provide meaningful assessment of changes in participant behavior and function over time and provide information on safety and tolerability.
• Is able to read, understand, and speak the designated language at the study center.
• Caregiver must be cognitively able to fulfill the requirements of the study.

For a full list of inclusion criteria, please refer to the protocol.

Exclusion criteria

1. Any other cause of dementia shown by MRI/CT findings within 2 years of screening (or at screening) and neurological examination at screening and Day 1. • Possible, probable, or definite vascular dementia according to the National Institute of Neurological Disorders and Stroke and Association Internationale pour la Recherche* et l*Enseignement en Neurosciences (NINDS-AIREN) criteria. • Evidence of significant abnormality that would suggest another potential etiology for dementia (eg, evidence of cerebral contusion, encephalomalacia, aneurysm, vascular malformation, >5 microhemorrhages, macrohemorrhage, single infarct >1 cm3). • Other central nervous system diseases that may cause cognitive impairment (eg, cerebrovascular disease including cerebrovascular dementia, Parkinsonism, Huntington*s disease, subdural hematoma, normal pressure hydrocephalus, brain tumor, Creutzfeldt-Jakob disease). 2. Concurrent or history of schizophrenia or bipolar affective disorder; OR any other clinically significant psychiatric condition that in the Investigator*s opinion prevents the participant from participating, or is likely to confound interpretation of drug effect or affect cognitive assessments or patients safety. OR the presence or history of suicidal attempts or suicidal ideation evidenced by endorsing Items 4 or 5 of the C-SSRS at screening or Day 1, endorsing any suicidal behavior item on the C-SSRS Since Last Visit form on Day 1, or any suicide attempt within 2 years prior to screening. 3. Vitamin B12, folic acid, syphilis serology, and thyroid stimulating hormone (TSH) results that are thought to contribute to the severity of dementia or cause dementia. Participants may be enrolled if in the Investigator*s medical judgment, the abnormal laboratory values are not the cause of the cognitive symptoms. 4. History of known or suspected seizures including febrile seizures (excluding self-limited childhood febrile seizures), a history of significant head trauma with loss of consciousness or recent unconsciousness that is not explained. 5. Acute or unstable cardiovascular disease, active peptic ulcer, uncontrolled hypertension, uncontrolled diabetes or insulin dependent patients or any medical condition that may interfere with the completion of the clinical study. 6. Known allergies, hypersensitivity, or intolerance to GV1001 or similar products or excipients. 7. History of alcohol, substance abuse or dependence as per DSM-V criteria (except nicotine dependence) within the last 2 years. 8. Concurrent malignancies or invasive cancers diagnosed within the past 5 years except for adequately treated non-metastatic basal cell carcinoma or squamous cell carcinoma of skin, in situ carcinoma of the uterine cervix or non-metastatic prostate cancer. 9. Sexually-active WOCBP or man capable of fathering a child who do not consent to using medicinally acceptable contraception (such as surgical sterilization, intrauterine contraceptive device, condom or diaphragm, an injectable or inserted contraceptive) during the study and for 3 months after the last dose

of study treatment. 10. Pregnant, breast feeding, or planning a pregnancy or fathering a child while enrolled in the study or for 3 months after the last dose of study treatment. 11. Use of anxiolytics, narcotics, or sleep aids in a manner that would interfere with cognitive testing, in the opinion of the Investigator. Atypical antipsychotics may be used at the discretion of the Investigator. Tricyclic antidepressants and monoamine oxidase (MAO) inhibitors are prohibited. 12. Previous treatment with GV1001. 13. Received an investigational product for AD within the last 6 months. 14. Participated in another clinical study within 4 weeks prior to this study. 15. Treated with aducanumab or participated in a clinical study with aducanumab. 16. Renal impairment (creatinine clearance [CrCL] <30 mL/min). 17. Severe liver dysfunction (alanine aminotransferase [ALT] or aspartate aminotransferase [AST] >2 times the upper limit of normal [ULN]). 18. Body weight <=35 kg. 19. Resides in a moderate to high dependency continuous care facility (residence in low grade assisted living facility where there is sufficient autonomy to permit valid evaluation of activities of daily living is allowed). 20. Any other reason that in the opinion of the Investigator would make the participant ineligible to participate or to complete this study.

Study design

Design

Study phase:	2
Study type:	Interventional
Intervention model:	Parallel
Allocation:	Randomized controlled trial
Masking:	Double blinded (masking used)
Control:	Placebo
Primary purpose:	Treatment

Recruitment

NL

Recruitment status:	Recruiting
Start date (anticipated):	30-05-2023
Enrollment:	15
Type:	Actual

Medical products/devices used

Product type:	Medicine
Brand name:	GV1001
Generic name:	GV1001

Ethics review

Approved WMO Date:	07-09-2022
Application type:	First submission
Review commission:	BEBO: Stichting Beoordeling Ethiek Bio-Medisch Onderzoek (Assen)
Approved WMO Date:	27-12-2022
Application type:	First submission
Review commission:	BEBO: Stichting Beoordeling Ethiek Bio-Medisch Onderzoek (Assen)
Approved WMO Date:	06-01-2023
Application type:	Amendment
Review commission:	BEBO: Stichting Beoordeling Ethiek Bio-Medisch Onderzoek (Assen)
Approved WMO Date:	30-03-2023
Application type:	Amendment
Review commission:	BEBO: Stichting Beoordeling Ethiek Bio-Medisch Onderzoek (Assen)
Approved WMO Date:	04-05-2023
Application type:	Amendment
Review commission:	BEBO: Stichting Beoordeling Ethiek Bio-Medisch Onderzoek (Assen)
Approved WMO Date:	05-05-2023
Application type:	Amendment
Review commission:	BEBO: Stichting Beoordeling Ethiek Bio-Medisch Onderzoek (Assen)

Approved WMO Date:	16-08-2023
Application type:	Amendment
Review commission:	BEBO: Stichting Beoordeling Ethiek Bio-Medisch Onderzoek (Assen)
Approved WMO Date:	21-08-2023
Application type:	Amendment
Review commission:	BEBO: Stichting Beoordeling Ethiek Bio-Medisch Onderzoek (Assen)

Study registrations

Followed up by the following (possibly more current) registration

No registrations found.

Other (possibly less up-to-date) registrations in this register

No registrations found.

In other registers

Register	ID
EU-CTR	CTIS2024-511610-20-00
EudraCT	EUCTR2021-004809-40-NL
ClinicalTrials.gov	NCT05189210
CCMO	NL80139.056.22