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Current State of Monoclonal Antibodies in Clinical Trials to Treat Parkinson's Disease

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ABSTRACT

Parkinson's disease (PD) is the fastest-growing neurological disorder globally, with nearly 90,000 new diagnoses annually in the U.S. alone. The disease is driven by the aggregation of α-synuclein (α-syn), which destroys dopaminergic neurons. Current therapies, such as levodopa, only provide symptomatic relief without targeting the root cause of neurodegeneration. Monoclonal antibodies (mAbs) that target α-syn aggregates have emerged as a potential disease-modifying treatment. This review evaluates the development and results of major clinical trials, including prasinezumab (Roche/Prothena), cinpanemab (Biogen), MEDI1341/TAK-341 (AstraZeneca/Takeda), and Lu AF82422 (Lundbeck), and discusses their mechanisms, efficacy, and limitations, and assesses their potential to advance disease-modifying therapy for PD.

INTRODUCTION

Parkinson's disease (PD) is a neurodegenerative disorder characterized by tremors and loss of muscle control, affecting approximately 10 million people globally, with projections reaching 13 million by 2040 (Ahanger & Dar, 2024). PD results from the progressive loss of dopamine-producing neurons in the brain, driven by the accumulation of misfolded α-synuclein (α-syn) proteins (Becerra-Calixto et al., 2023).

The use of monoclonal antibodies (mAbs) in the treatment of autoimmune diseases has grown substantially over the past few decades. It was not until the first mAb to treat Alzheimer's disease, aducanumab (Aduhelm), was approved in June of 2021 that there had ever been a mAb to treat neurodegenerative diseases. With many more mAbs to treat Alzheimer's coming to market, we are seeing attention turn to Parkinson's disease as a new mAb market. Here, we discuss the early results of monoclonal antibodies (mAbs) that have entered clinical trials for the treatment of Parkinson's disease (Luo et al., 2024).

METHODOLOGY

Three databases, PubMed, Google Scholar, ClinicalTrials.gov, and the reference lists of the most recent advances in published literature, were searched for literature about monoclonal antibody development and ongoing clinical trials in Parkinson's disease. Search restrictions such as research published between 2010 and 2025 ensure the review reflects the latest advances.

Selected articles provide a wide range of materials, including peer-reviewed clinical trial reports, extensive review articles, and original studies focused on α-synuclein or the mechanisms of monoclonal antibodies.

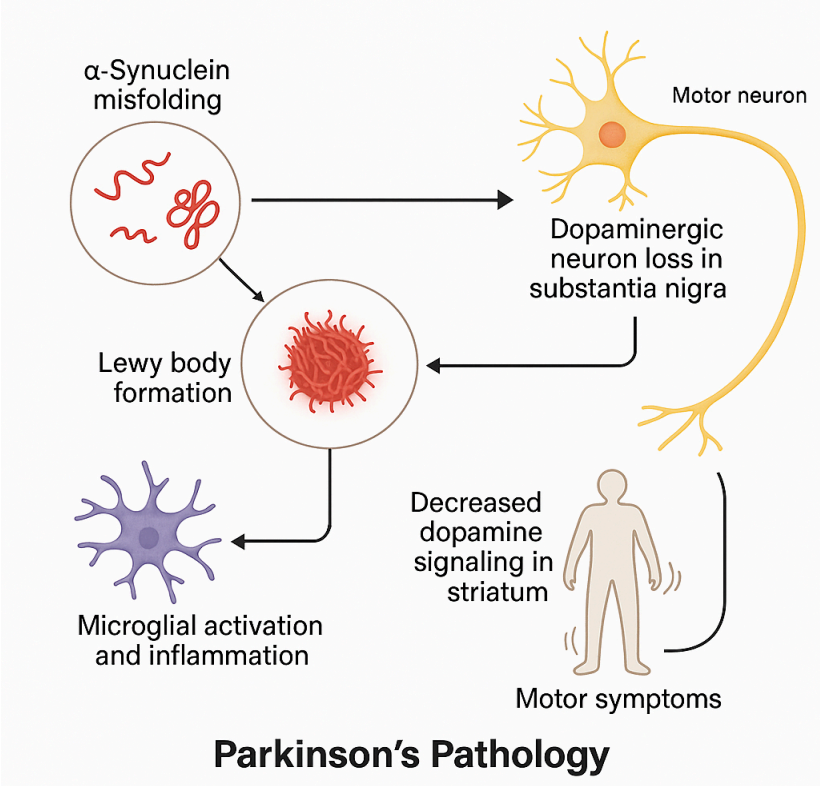

Figure 1. Pathological cascade of Parkinson's disease.-revision

Parkinson's Background

Parkinson's disease (PD) is the second most prevalent neurodegenerative disorder globally, affecting over 10 million people, with projections reaching 13 million by 2040 (Ahanger & Dar, 2024). PD results from the progressive loss of dopaminergic neurons in the substantia nigra, primarily caused by the misfolding and aggregation of α-synuclein (α-syn) into Lewy bodies. These intracellular inclusions disrupt neuronal communication and trigger inflammation, leading to hallmark motor symptoms—bradykinesia, rigidity,

resting tremor—and non-motor symptoms such as sleep disturbance and depression (Janssen Daalen et al., 2024; Parkinson's Symptoms, n.d.).

Although some forms of PD are linked to mutations in genes such as *SNCA*, *PRKN*, and *LRRK2*, environmental factors, pesticide exposure, air pollution, and trichloroethylene play significant roles in sporadic cases (Paul et al., 2023; Murata et al., 2022; De Miranda et al., 2021; Dorsey & Bloem, 2024). Misfolded α-syn activates microglia, producing inflammatory mediators (ROS, TNF-α, IL-1β) that amplify neurodegeneration (Glass et al., 2010; Ferreira & Romero-Ramos, 2018; Grozdanov et al., 2019). The resulting cascade of inflammation, mitochondrial dysfunction, and dopaminergic loss defines PD pathology (Figure 1).

Monoclonal Antibody Background

Monoclonal antibodies (mAbs) are engineered IgG1-class proteins designed to target specific antigens (Castelli et al., 2019). Produced by hybridoma technology and humanized to reduce immune rejection (Bixbite, 2025), mAbs have revolutionized treatments for autoimmune diseases and cancers, and are now being developed for neurodegenerative conditions (Frontzek & Aguzzi, 2020; De Genst et al., 2014).

For Parkinson's disease, mAbs offer a strategy to neutralize extracellular α-syn aggregates, preventing their cell-to-cell spread and reducing neuroinflammation. Passive immunization with α-syn antibodies has shown preclinical promise in reducing aggregation and preserving dopaminergic neurons (Masliah et al., 2011; Calabresi et al., 2023). However, safety challenges, such as amyloid-related imaging abnormalities (ARIA) observed with Alzheimer's mAb therapies, underscore the need for improved selectivity and delivery (Manoutcharian & Gevorkian, 2024). These advances have led to next-generation antibodies such as prasinezumab and TAK-341, which demonstrate high specificity and tolerability in early clinical trials.

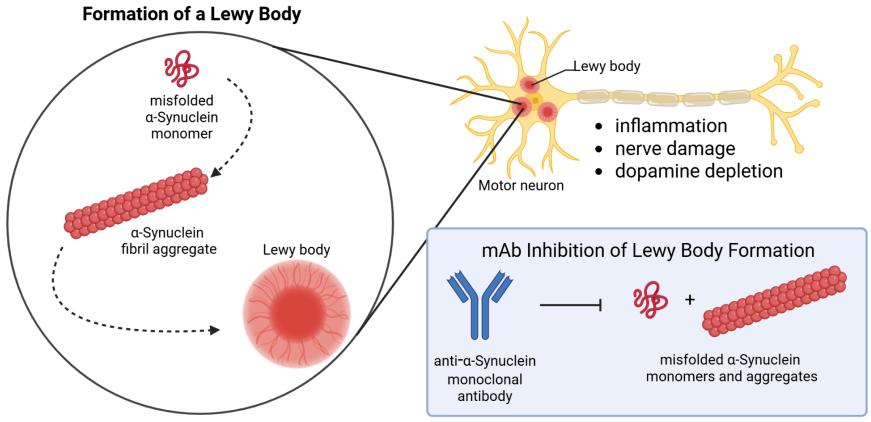

Figure 2. Formation of Lewy Bodies and mAb inhibition of α-syn aggregation-revision

Monoclonal Antibody Clinical Candidates for PD

Prasinezumab (Roche/Prothena)

Prasinezumab is a mAb being developed by Roche/Genentech that is in Phase III trials for the treatment of PD. The mAb has progressed through a Phase II PASADENA study and a Phase IIb PADOVA study. These studies showed that prasinezumab was effective in treating early symptoms, specifically motor progression in PD. The PADOVA study provided the first biomarker evidence that prasinezumab inhibits PD progression. Over 500 people with early-stage Parkinson's disease already on symptomatic treatment were treated for a minimum of 18 months with prasinezumab. Although this study lacked statistical significance, Padova reported that prasinezumab may be effective in slowing the progression of motor symptoms, with a Hazard Ratio (HR) of 0.84 (Manoutcharian & Gevorkian, 2024). The HR is measured to compare the risk of disease progression between two groups over time, and a score of less than one indicates that PADOVA's investigation of pasinezumab showed the treatment reduces the risk. At 104 weeks (2 years), reduced motor progression was demonstrated in 30-40% of patients when compared to the placebo group (*National Cancer Institute (.gov)*, 2011). These combined results prompted the progression of prasinezumab to Phase III trials in June 2025.

Cinpanemab - Biogen

Cinpanemab (BIIB054) is a mAb being developed by Biogen in Phase II trials for the treatment of PD. The mAb is designed to target the aggregated α-syn while having low affinity for the monomeric forms. In the Phase I study, a randomized, placebo-controlled dose-escalation trial involving healthy volunteers and individuals with PD assessed the drug's safety and target engagement (Brys et al., 2019). Due to the low drug concentration and low levels of α-syn, conventional measurement of drug-target interactions was impractical. The researchers developed a new method for quantifying drug-target interactions by using zero-length crosslinking to stabilize the drug-target complexes, followed by detection using the Meso Scale Discovery electrochemiluminescence assay. With this new detection method, Biogen demonstrated that cinpanemab behaved as expected, with low monomeric α-syn binding detected in the central nervous system through cerebrospinal fluid (CSF) (Liu et al., 2024).

In the Phase II SPARK trial, a group of early PD patients received intravenous infusions of cinpanemab or a placebo at doses of 250 mg, 1250 mg, or 3500 mg every 4 weeks. The change in the Movement Disorder Society-Unified Parkinson's Disease Rating Scale (MDS-UPDRS) baseline scores was the primary endpoint of the trial. The trial's secondary endpoint was assessed after week 72, as at week 52, DaT-SPECT imaging, CSF, and plasma biomarkers showed no difference between the placebo and cinpanemab groups (Hutchison et al., 2024). However, individuals taking cinpanemab experienced headaches, falls, and nasopharyngitis. (*Cinpanemab in Parkinson's Disease: Imaging and Fluid Biomarker Results from the Phase 2 SPARK Study*, 2021)**.**

MEDI1341/TAK-341 - AstraZeneca/MedImmune/Takeda

MEDI1341 is a monoclonal antibody (mAb) targeting alpha-synuclein, developed by AstraZeneca and Takeda Pharmaceutical Company Limited, which have agreed to collaborate on its development, with plans to commercialize it later. This antibody has high affinity (74 pM), a strong binding between the antibody and specific antigen, does not cross-react with other proteins, and has a decrease in the ability of the drug to trigger immune responses(*AstraZeneca and Takeda Establish Collaboration to Develop and Commercialise MEDI1341 for Parkinson's Disease*, 2017). MEDI1341 can bind both monomeric and aggregated forms of α-syn in hopes of addressing different stages of PD (Schofield et al., 2019). MEDI1341 has demonstrated the ability to cross the blood-brain barrier and seclude extracellular α-syn. Moreover, the antibody has been shown to block cell-to-cell pathology in vitro and inhibit the propagation of α-syn in vivo as it efficiently penetrates the central nervous system (Schofield et al., 2019).

In rat and monkey trials, the antibody showed a decrease in free α-syn in CSF and interstitial fluid. In mice specifically, MEDI1341 significantly reduced the accumulation and axonal propagation of α-syn in the brain(Schofield et al., 2019). In the Phase I clinical trial, MEDI1341 was administered intravenously (IV) at six different ascending dose levels (Alfaidi et al., 2024),(Alfaidi et al., 2024). Safety assessments from MEDI1341 and the placebo showed no AE that could be attributed to MEDI1341 across PD patients in all tested doses. Furthermore, penetration of the antibody into the CSF was demonstrated, confirming central nervous system exposure (Schofield et al., 2019),(Schofield et al., 2019). MEDI1341's predictable pharmacokinetics in early human testing provided a foundation for Phase II studies (Alfaidi et al., 2024).

Upon the acquisition of the treatment by the AstraZeneca and Takeda collaboration, MEDI1341 was renamed TAK-341. The company progressed the mAb to a Phase II trial to assess efficacy in a 52-week trial with multiple system atrophy patients. This is measured by the Unified Multiple System Atrophy Rating Scale Part I (UMSARS) (*ClinicalTrials.gov*, n.d.-a). The rating scale is composed of four subscales: functional disability, clinical examination, blood pressure and heart rate, and chorea-based disability (Krismer et al., 2022). Phase II trials for TAK-341 have concluded, but results are still pending publication.

Lu AF82422 - Lundbeck

Lu AF82422 is a humanized monoclonal antibody (mAb) developed by Lundbeck that targets pathological α-syn. In its first-in-human Phase I trial, ascending IV doses were administered to healthy subjects and early PD (*ClinicalTrials.gov*, n.d.-b). At all doses, the antibody was well tolerated with no serious AE. However, the treatment displayed had frequent minor AE, including lumbar puncture-related headaches and mild infections (Buur et al., 2024). Dose-proportional increases in plasma and CSF levels suggest prolonged systemic exposure with a plasma half-life of 700 h. The findings from this provide the basis for further clinical development of Lu AF82422 as a promising therapeutic for PD (Buur et al., 2024).

One of the significant challenges of immune therapies for neurodegenerative diseases has been overcoming the blood-brain barrier (BBB). For Lu Af822422, the CSF concentration was approximately 0.16-0.51% in humans, consistent with the typically reported CSF concentrations of therapeutic antibodies, which range from 0.1-0.4% (Kallunki et al., 2025).
The mAb has affinity for both the monomeric and fibrillar α-syn of 36 nM and 0.3 nM, respectively, demonstrating a preference for the aggregate form. Given that 100 mg/kg dosing results in a 7.6 μM plasma concentration, these affinities result in target engagement of 20% against the monomer and 90% against the aggregated α-syn in the CSF.

The half-life of prasinezumab is 10-18 days, while the half-lives of cinpanemab and Lu AF82422 are 28-35 days and 28-30 days, respectively. In Phase 2 investigations, prasinezumab and cinpanemab were administered every four weeks, with the highest doses being 4500 mg and 3500 mg, respectively (Pagano et al., 2021). There isn't much information on non-specific tissue binding for other α-synuclein antibodies. In histological staining studies with Lu AF82422, there was no sign of non-specific staining, which is in line with previous findings on safety and pharmacokinetics (Fjord-Larsen et al., 2021).

Lu AF82422 inhibits α-syn seeding initiated by truncated seeds and forms of α-syn from MSA. This prevents smaller seeds that are internalized in cells from aggregating.
Bind to multiple pathogenic forms of a-syn, including fibrillar, phosphorylated, and truncated species stemming from the human brain. In the Phase II clinical trial for MSA (NCT05104476), although the treatment showed a trend toward slowed disease progression, the primary endpoint was not achieved. The development of Lu AF82422 is expected to continue in enhancing the understanding of a-syn immunotherapies targeting the treatment of α-synucleinopathies.

mAb	**Company**	**Clinical Results**	**Current Status**
Prasinezumab	Roche/Prothena	Monoclonal antibody targeting aggregated α-synuclein	Phase II/III
Cinpanemab	Biogen	Monoclonal antibody against aggregated α-synuclein	Phase II (completed)

MEDI1341	AstraZeneca/MedImmune	Monoclonal antibody targeting α-synuclein	Phase I/II
Lu AF82422	Lundbeck	Monoclonal antibody against all α-synuclein with a preference for aggregates	Phase II

CONCLUSION

The development of mAbs shows a promising, significant disease-modifying approach for PD treatment. By binding to and reducing the aggregation of α-syn, the mAbs can directly address the disease pathology. Clinical studies of mAbs prasinezumab, cinpanemab, MEDI1341/TAK-341, and Lu AF82422 have demonstrated the progress and limitations of these efforts in treating PD. Prasinezumbab and MEDI1341/TAK-341 have both shown safety profiles and biomarkers that encourage researchers. Prasinezumbab, which has advanced furthest, also demonstrated reduced motor progression in 30-40% of patients compared to the placebo group at 104 weeks. Cinpanemab, despite successful target engagement, has shown limited efficacy, with no difference between the placebo and treatment groups. MEDI1341/TAK-341 has demonstrated blood-barrier penetration and reduced extracellular α-syn, and its ongoing Phase II study is promising. Lu AF82422 has demonstrated a long half-life and binds to multiple α-syn species relevant to PD. The phase II trial on MSA failed to meet its primary endpoint, but the trend toward slowed disease progression is promising for antibodies treating PD. While no mAb has been approved for the treatment of PD, the progression of MEDI1341/TAK-341, Lu AF82422, and prasinezumab marks promising steps towards reaching an approved PD treatment.

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