REVIEW

The Effect of GLP-1RA on the Motor Function of Patients With Parkinson Disease

A Systematic Review and Meta-Analysis

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Introduction

Parkinson disease (PD) is a neurodegenerative disorder of significant global relevance. It is the fastest-growing neurologic condition worldwide, surpassing all other neurologic disorders in terms of rising prevalence, mortality, and years lived with disability.[1] In 2021, more than 6 million individuals were estimated to be living with PD globally, and projections indicate that this number could exceed 25 million by 2050, driven primarily by population aging, alongside population growth and increased life expectancy.[2] The burden of PD is substantial not only because of its prevalence but also because of its profound socioeconomic impact, encompassing both direct health care costs and indirect costs associated with productivity loss and the need for long-term caregiving.[3]

The increasing incidence and prevalence of PD are observed across nearly all regions worldwide, with an even more pronounced rise in middle-income countries.[4] Furthermore, PD poses significant global challenges regarding equitable access to diagnosis, treatment, and comprehensive care. This is particularly concerning given that most affected individuals reside in low- and middle-income countries, where substantial disparities persist in access to neurologic care, specialist services, and essential medications.[5]

From a pathophysiologic standpoint, PD is primarily characterized by the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to striatal dopamine deficiency and the classical motor symptoms of bradykinesia, rigidity, and resting tremor, alongside a wide spectrum of nonmotor manifestations.[6] However, PD is increasingly recognized as a multifactorial disorder, involving complex mechanisms such as oxidative stress, mitochondrial dysfunction, chronic neuroinflammation, misfolded protein aggregation—particularly of α-synuclein—and, more recently, cerebral insulin resistance and impaired energy metabolism.[7]

Current therapeutic strategies, including monoamine oxidase type B inhibitors, dopamine agonists, levodopa combined with dopa-decarboxylase inhibitors, and deep brain stimulation, are primarily symptomatic and do not alter the underlying disease progression.[8] This underscores an urgent need for interventions capable of targeting the fundamental neurodegenerative mechanisms driving PD.

A growing body of evidence supports a strong association between PD and type 2 diabetes mellitus (T2DM), as both conditions share key pathophysiologic mechanisms, including insulin resistance, mitochondrial dysfunction, and chronic inflammation.[9] This overlap has spurred increasing interest in the potential neuroprotective properties of glucagon-like peptide-1 receptor agonists (GLP-1RAs), originally developed for glycemic control in T2DM.[10] These agents can cross the blood-brain barrier and exhibit pleiotropic effects

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relevant to PD pathogenesis, including enhancement of brain insulin signaling, attenuation of neuroinflammation, reduction of oxidative stress, stabilization of mitochondrial function, and promotion of neuronal survival and dopaminergic neurotransmission.[11]

Preclinical studies have demonstrated that GLP-1RAs can prevent dopaminergic neuronal loss, restore striatal dopamine levels, and improve both motor and cognitive outcomes in PD models.[12] Clinical trials involving agents such as exenatide, liraglutide, and lixisenatide have reported symptomatic improvements and suggest potential disease-modifying effects; however, larger and longer-term studies are required to validate these findings and to better delineate the patient subgroups most likely to benefit. Recent developments include the emergence of dual glucagon-like peptide-1 and glucose-dependent insulinotropic polypeptide receptor agonists and next-generation compounds engineered for enhanced CNS penetration, representing a promising frontier in PD therapeutics.[11]

Given the increasing interest in disease-modifying strategies for PD and the persistent limitations within the current body of evidence—particularly the paucity of placebo-controlled trials conducted under rigorous methodological standards[13]—this systematic review and meta-analysis aims to critically evaluate the effects of GLP-1 receptor agonists compared with placebo, with a primary focus on their impact on motor function in patients with PD.

Methods

The protocol of this review was registered in the International Prospective Register of Systematic Reviews under the protocol CRD42024569468 in DATA. The systematic review and meta-analysis were conducted and reported following the Cochrane Handbook for Systematic Reviews of Interventions and the Preferred Reporting Item for Systematic Reviews and Meta-Analyses guidelines.[14]

Eligibility Criteria

Studies were considered eligible if they met all the following criteria: (1) randomized controlled trials (RCTs), (2) comparing GLP-1RA with placebo, (3) patients with PD, and (4) reporting on the Movement Disorder Society-sponsored revision of the Unified Parkinson's (MDS-UPDRS) Part III.

MDS-UPDRS was used to assess the impact of the GLP-1RA on the motor function of patients with PD. The MDS-UPDRS comprises 4 parts: Part I evaluates nonmotor experiences of daily living, Part II focuses on motor experiences of daily living, Part III assesses motor aspects, and Part IV addresses motor complications.[15]

Exclusion criteria were (1) overlapping populations, (2) absence of outcomes of interest, and (3) conference abstracts. No restrictions were determined regarding publication date, status, or language.

Search Strategy and Data Extraction

A systematic search was conducted in Medline, Scopus, and Cochrane databases up to June 2024 using the following terms: "Parkinson Disease", "Parkinson's Disease", "Glucagon-Like Peptide-1 Receptor Agonists", "Dulagrutide", "Exenatide", "Liraglutide", "Lixisenatide", and "Semaglutide". The references from all included studies and previous systematic reviews and meta-analyses were also searched manually. Two authors (L.C. and S.B.) independently screened titles and abstracts and evaluated the articles in full for eligibility based on prespecified criteria. Any disagreements were resolved through a panel discussion between the authors.

End Points

The primary end point was the change from baseline in MDS-UPDRS Part III scores. Secondary end points included changes from baseline in MDS-UPDRS Parts I, II, and IV scores.

Quality Assessment

Two independent authors (L.C and S.B.) assessed the risk of bias in the included randomized trials using the revised Cochrane Risk of Bias Tool for Randomized Trials.

Statistical Analysis

A mean difference (MD) with 95% CIs was calculated to evaluate treatment effects for continuous outcomes. Heterogeneity was assessed using the Cochran Q test and I^2 statistics, with significance defined as p values < 0.10 and I^2 > 25%. For outcomes reported by at least 3 studies with substantial heterogeneity, a leave-one-out sensitivity analysis was conducted. Statistical analyses were performed using the Mantel-Haenszel method and a random-effects model, implemented in Review Manager Version 8.1.1.

Results

Study Selection and Characteristics

Our systematic search yielded 55 potential articles (Figure 1). After removing duplicates and excluding studies that did not meet the inclusion criteria, 4 studies underwent full-text review. The study by Aviles-Olmos et al.[6] was excluded because of the lack of a placebo group. Ultimately, 3 RCTs were included, encompassing 514 patients with PD. Of them, 299 patients received GLP-1RA, whereas 215 were assigned to the placebo group. The characteristics of the included studies are summarized in Table. One study evaluated the effects of exenatide, another used NLY01 (a longer-lasting version of exenatide), and a third used lixisenatide.

Pooled Analysis of All Studies

In the pooled analysis, no significant improvement in motor function was observed when comparing GLP-1RA with placebo (MDS-UPDRS Part III; MD −0.90; 95% CI −2.77 to 0.97; p = 0.34; I^2 = 70%; Figure 2). However, GLP-1RA was associated with a detrimental effect on the motor aspects of

Figure 1 PRISMA Flow Diagram

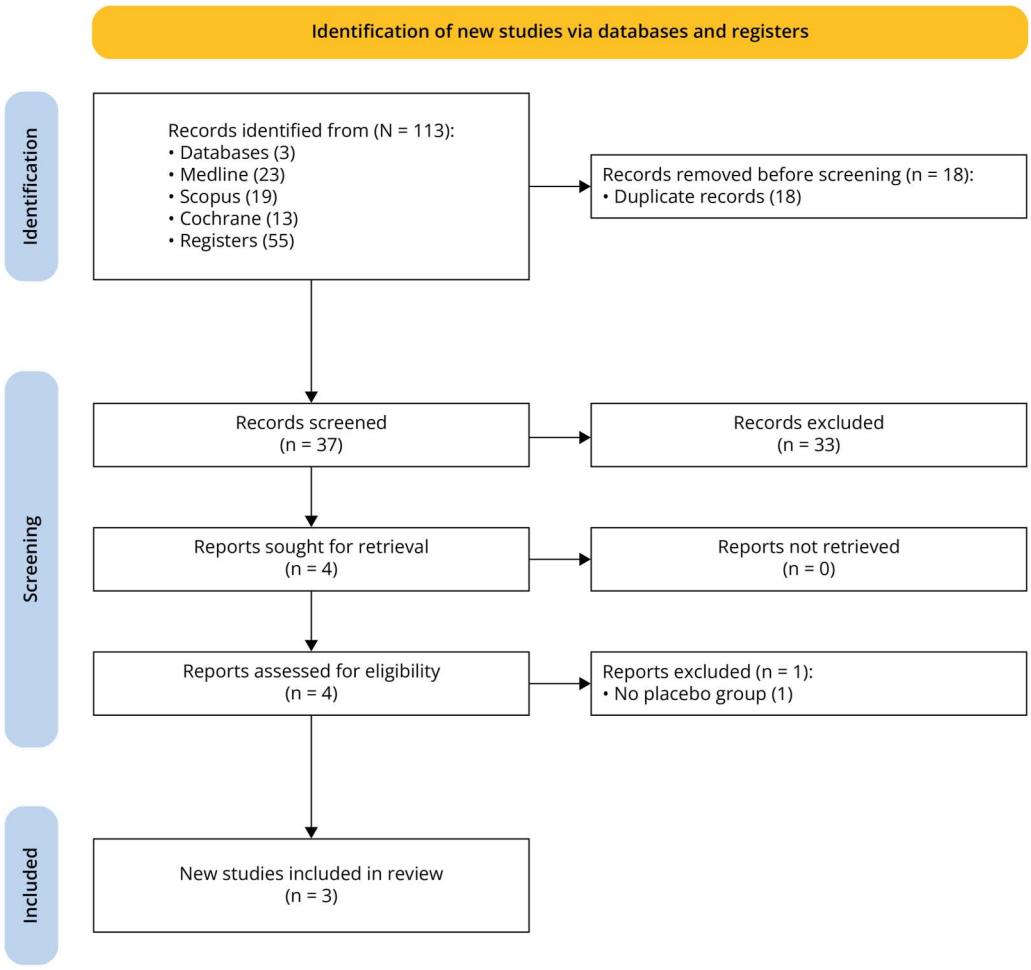
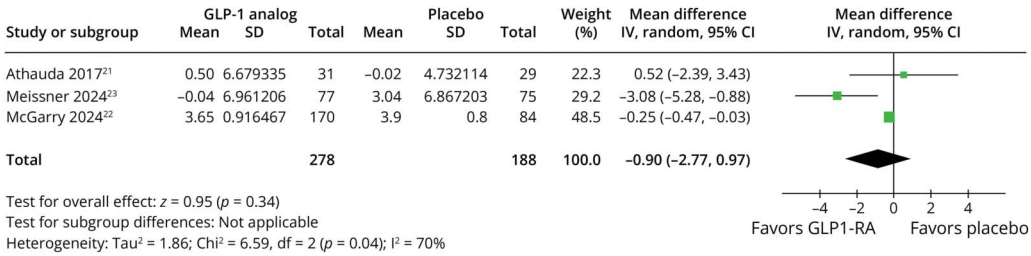


Table Baseline Characteristics of the Included Studies

Study	Intervention	Number of patients		Age (y)		Male sex (%)		MDS-UPDRS[b] part 3		Hoehn and Yahr stage ≤2 (%)		Hoehn and Yahr stage 2.5 (%)	
		GLP1-RA	Placebo	GLP1-RA	Placebo	GLP1-RA	Placebo	GLP1-RA	Placebo	GLP1-RA	Placebo	GLP1-RA	Placebo
Athauda 2017	Exenatide 2.0 mg	31	29	61.6 ± 8.2	57.8 ± 8.0	71	76	32.8 ± 9.8	27.1 ± 10.3	94	100	6	0
Meissner 2024	Lixisenatide 20 μg	78	78	59.5 ± 8.1	59.9 ± 8.4	56	62	14.8 ± 7.3	15.5 ± 7.8	NA	NA	NA	NA
McGarry 2024	NLY01[a] 2.5 mg	85	84	62.1 ± 9.0	61.8 ± 8.1	71	62	22.7 ± 8.1	22.3 ± 9.1	96	95	4	5
	NLY01[a] 5.0 mg	85		60.6 ± 10.0		64		22.0 ± 8.2		96		4	

Abbreviations: GLP-1RA = glucagon-like peptide-1 receptor agonist; MDS-UPDRS = Movement Disorder Society-sponsored revision of the Unified Parkinson's; NA = not available.
[a] Longer-lasting version of exenatide.
[b] Movement Disorder Society-sponsored revision of the Unified Parkinson's Disease Rating Scale.

Figure 2 No Improvement in Motor Function in Patients With PD Was Observed When Comparing GLP-1RA With Placebo (MDS-UPDRS Part III)



GLP-1RA = glucagon-like peptide-1 receptor agonist; MDS-UPDRS = Movement Disorder Society-sponsored revision of the Unified Parkinson's; PD = Parkinson disease.

experiences of daily living in patients with PD when compared with placebo (MDS-UPDRS Part II; MD 0.20; 95% CI 0.09–0.30; $p < 0.01$; $I^2 = 0\%$; Figure 3). Furthermore, no significant differences were found in the nonmotor aspects of experiences of daily living (MDS-UPDRS Part I; MD 0.03; 95% CI −0.40 to 0.45; $p = 0.91$; $I^2 = 16\%$; Figure 4) or motor complications (MDS-UPDRS Part IV; MD −0.01; 95% CI −0.40 to 0.38; $p = 0.96$; $I^2 = 0\%$; Figure 5).

Follow-up duration varied considerably across studies. Athauda et al.[16] reported a follow-up period of 60 weeks, whereas McGarry et al.[17] documented a follow-up of 44 weeks, and Meissner et al.[18] extended the follow-up duration to 14 months. This variability underscores differences in the longitudinal assessments employed in these studies.

Sensitivity Analysis and Quality Assessment

We conducted a leave-one-out sensitivity analysis for all outcomes with high heterogeneity. The analysis showed no improvement in motor function among patients with PD when excluding the studies by Athauda et al.[16] (MDS-UPDRS Part III; MD −1.44; 95% CI −4.18 to 1.29; $p = 0.30$; $I^2 = 84\%$; Figure 6) or McGarry et al.[17] (MDS-UPDRS Part III; MD −1.41; 95% CI −4.93 to 2.11; $p = 0.30$; $I^2 = 73\%$; Figure 6). However, an improvement in motor function was observed when excluding the study by Meissner et al.[18] (MDS-UPDRS Part III; MD −0.25; 95% CI −0.46 to 0.03; $p = 0.03$; $I^2 = 0\%$; Figure 6), suggesting a potential influence of this study on the pooled results. Regarding the quality assessment of the included studies, 2 RCTs were identified as having some concerns[16,17] while 1 RCT was determined to have a low risk,[18] as illustrated in Figure 7.

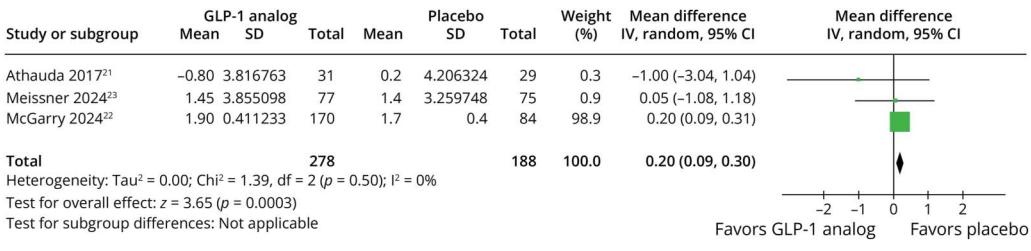
Discussion

This systematic review and meta-analysis, encompassing 3 RCTs with 514 patients, indicates that GLP-1RA administration in patients with PD does not effectively improve motor function.

Initially developed for diabetes management, the GLP-1RAs enhance glycemic control by stimulating insulin secretion and inhibiting glucagon secretion. Over time, their application has expanded to include potential benefits in cardiovascular disease, polycystic ovary syndrome, and neurodegenerative disorders, including PD.[19]

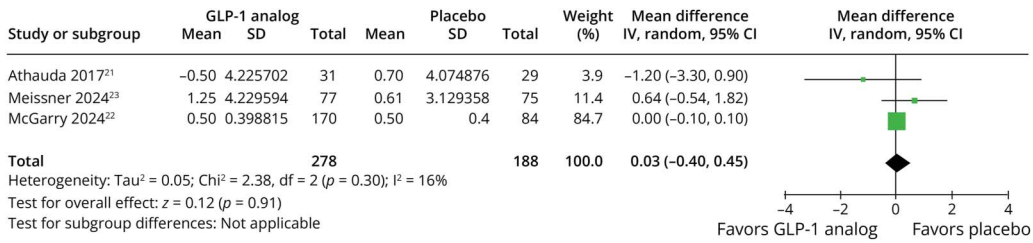
Neurodegenerative disorders often trigger a neuroinflammatory response, primarily due to the actions of microglia and astrocytes. In PD, the abnormal buildup of α-synuclein and the formation of Lewy bodies can act as triggers for chronic neuroinflammation. Given the anti-inflammatory properties of GLP-1RA, these drugs have been proposed as potential

Figure 3 GLP-1RA had a Detrimental Effect on Motor Aspects of Experiences of Daily Living in Patients With PD When Compared With Placebo (MDS-UPDRS Part II)



GLP-1RA = glucagon-like peptide-1 receptor agonist; MDS-UPDRS = Movement Disorder Society-sponsored revision of the Unified Parkinson's; PD = Parkinson disease.

Figure 4 No Improvement in Nonmotor Aspects of Experiences of Daily Living in Patients With PD Was Observed When Comparing GLP-1RA With Placebo (MDS-UPDRS Part I)



GLP-1RA = glucagon-like peptide-1 receptor agonist; MDS-UPDRS = Movement Disorder Society-sponsored revision of the Unified Parkinson's; PD = Parkinson disease.

treatments for reducing neuroinflammation in neurodegenerative conditions.[20]

However, not all studies provided data on off-medication status, which may have limited the analysis and contributed to some uncertainty in the results, as the analysis was confined to on-medication assessments.

The baseline characteristics of the studies varied. For instance, Athauda et al.[16] included patients with a more advanced disease stage compared with the other studies, which focused on patients with earlier stages of PD. These differences in disease progression may help explain the variation in outcomes.[17,18]

The pooled analysis of the motor function of patients with PD demonstrated high heterogeneity, allowing for a leave-one-out sensitivity analysis to assess the influency of each study. Although the leave-one-out analysis of Athauda et al.[16] and Meissner et al.[18] did not show any improvement in motor function, the leave-one-out analysis of McGarry et al.[17] resulted in different findings. McGarry et al.[17] had the largest sample size, used a novel longer-lasting version of exenatide, and had a shorter follow-up period, which may explain the contrasting results.

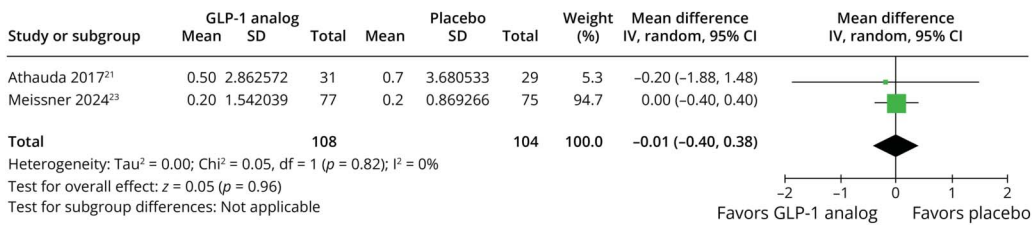
Despite the findings from Athalda et al.[16] and Meissner et al.[18] that GLP1-RA had a positive effect on disease progression, McGarry et al.,[17] the largest study in this meta-analysis, found no significant benefit. This discrepancy might be due to the formulation of NLY01, which was designed to enhance blood-brain barrier penetration. Subgroup analysis in both McGarry et al.[17] and Meissner et al.[18] indicated a potential benefit in younger patients, suggesting that further research in this subgroup may be warranted.

An important consideration in the included studies is the early stage of disease in many patients, where motor impairments may not yet be severe, potentially making the adverse effects of GLP-1RA more pronounced. This could explain the negative impact of GLP-1RA on motor experiences of daily living (MDS-UPDRS Part II) observed in certain cohorts. Although adverse events were reported across all studies, the lack of consistent classification methods prevented a quantitative synthesis of safety outcomes.

Another plausible rationale for the diversity of outcomes may be the potential variance in the anti-inflammatory attributes of GLP-1RA during the early phase of the disorder, in contrast to the advanced stages.

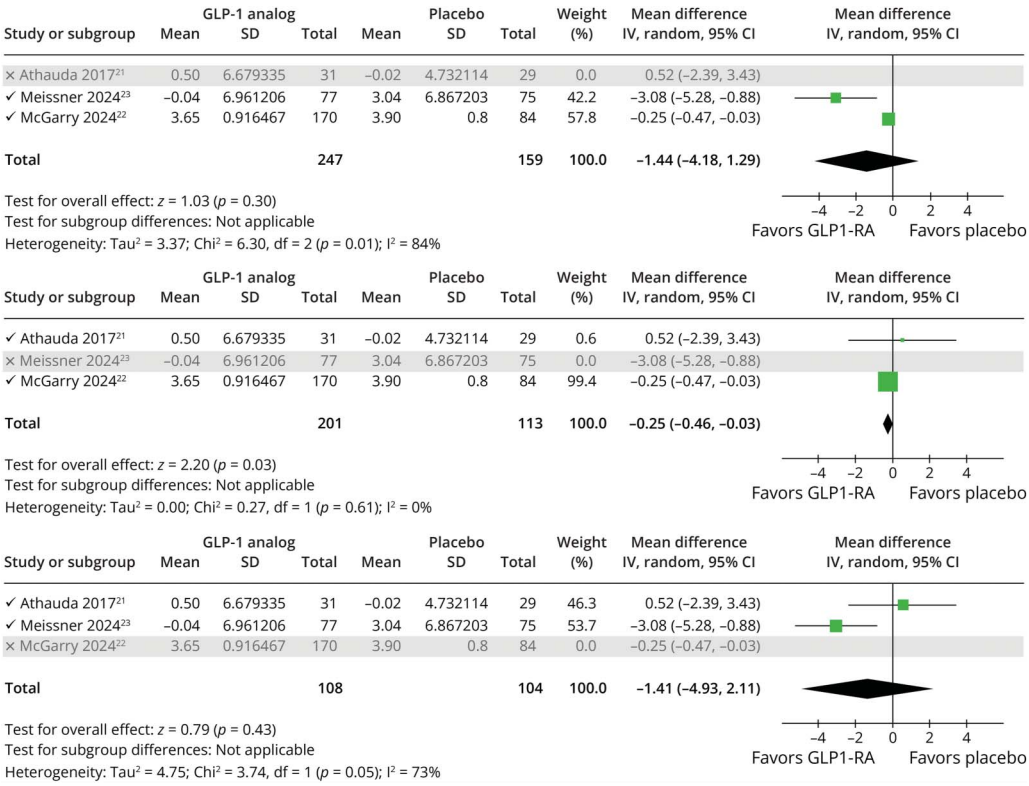
A recent meta-analysis[13] investigating the efficacy of hypoglycemic agents on clinical outcomes in patients with PD has evaluated not only GLP1-RA but also glitazones as part of the intervention group. The findings of this analysis indicate a positive impact on clinical outcomes for individuals with PD. It is essential to acknowledge, however, that the eligibility criteria for this meta-analysis were

Figure 5 No Improvement in Motor Complications in Patients With PD Was Observed When Comparing GLP-1 Analogs With Placebo (MDS-UPDRS Part IV)



GLP-1RA = glucagon-like peptide-1 receptor agonist; MDS-UPDRS = Movement Disorder Society-sponsored revision of the Unified Parkinson's; PD = Parkinson disease.

Figure 6 Sensitivity Analysis of Change From Baseline of the Motor Function in Patients With PD (MDS-UPDRS Part III)

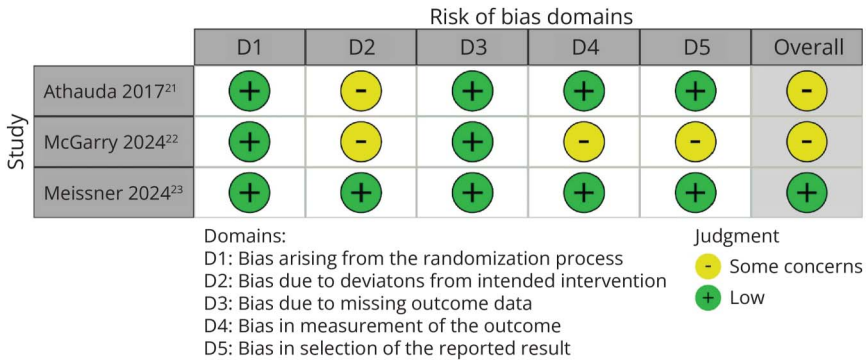


MDS-UPDRS = Movement Disorder Society-sponsored revision of the Unified Parkinson's.

supposed to be limited to placebo-controlled studies. Notably, the study by Aviles-Olmos et al.,[21] despite the absence of a placebo comparison, was included in the evaluation of MDS-UPDRS Part III, PDQ-39, as well as nausea and vomiting outcomes. The authors of the study by Aviles-Olmos et al.[21] explicitly cautioned against interpreting their data as evidence of symptomatic efficacy or neuroprotection. This inclusion raises concerns regarding potential confounding factors, evidenced by the substantial heterogeneity observed in Figure 2 of this recently published meta-analysis.[13]

A recent phase 3 RCT conducted by Vijiaratnam et al.,[22] published after the systematic search performed for this systematic review and meta-analysis, assessed the weekly administration of exenatide in patients with moderate PD over a 96-week period. The findings revealed no significant differences in motor outcomes between the placebo group

Figure 7 Risk of Bias Assessment of Randomized Controlled Trials



TAKE-HOME POINTS

- Patient selection for glucagon-like peptide-1 receptor agonists (GLP-1RAs) requires careful consideration.
- The potential benefits of GLP-1RA on the motor function in Parkinson disease (PD) remain unclear.
- There may be detrimental effects on the motor aspects of daily living in patients with PD undergoing GLP-1RA administration.
- Close monitoring of patients receiving GLP-1RAs is essential.

and those receiving exenatide. Although these results were not included in this meta-analysis, they align with both the general and subgroup analyses, suggesting that despite the promising data from preclinical trials, GLP-1 receptor agonists may not provide meaningful benefits for motor function in patients with PD.

Limitations

A more precise analysis would have benefited from including an off-medication state, but the limited data availability restricted the analysis to the on-medication state. Although each study used the latest measure of change from baseline, the varying follow-up periods across the studies may have affected the overall results.

Although the sample size in this analysis was substantial, several other RCTs had larger sample sizes, suggesting the need for further studies to evaluate the effects of this intervention more accurately.

In addition, GLP1-RA was initially licensed to patients with diabetes, but all 3 RCTs excluded individuals with diabetes. As a result, a subgroup analysis for diabetic patients was not possible because of a lack of relevant data, and it remains uncertain whether they could derive similar benefits from GLP-1RA.

Conclusion

The administration of GLP1-RA did not result in a statistically significant improvement in motor function in patients with PD and was associated with a detrimental effect on motor aspects of experiences of daily living in patients with PD when compared with placebo.

Author Contributions

G. Ristori Costa: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data. L.F. Ferreira Cavalcante: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data. S. Massafelli Battistuta: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data; analysis or interpretation of data. P.F. Makabe: drafting/revision of the manuscript for content, including medical writing for content. I.S. Fanucci Bueno: drafting/revision of the manuscript for content, including medical writing for content. B. Yuamoto: drafting/revision of the manuscript for content, including medical writing for content. F.E. Gonçalves Vilela: drafting/revision of the manuscript for content, including medical writing for content. L.G. Giacon Meloni: drafting/revision of the manuscript for content, including medical writing for content. D.D. de Faria: drafting/revision of the manuscript for content, including medical writing for content. R. Anghinah: drafting/revision of the manuscript for content, including medical writing for content. D. Haddad Santos: drafting/revision of the manuscript for content, including medical writing for content; study concept or design.

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