# The scientific justification for repurposing GLP-1 receptor agonists for Parkinson's

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**ABSTRACT:**

Glucagon-like peptide-1 (GLP-1) receptor agonists are a class of drugs that have been widely used in the treatment of type 2 diabetes. More recently, certain members of the GLP-1 receptor agonist family have been approved for use in weight loss therapy. From early in their development, researchers have reported on the neuroprotective properties of GLP-1 receptor agonists in models of neurological conditions such as stroke, traumatic brain injury and Parkinson's disease (PD). GLP-1 receptor agonists have been shown to rescue models of PD, and epidemiological research has demonstrated that their use in type 2 diabetes is associated with a reduced risk of developing PD. Since 2010, Cure Parkinson's has been championing the repurposing of specific GLP-1 receptor agonists for PD, supporting clinical trials assessing the disease-modifying potential of these agents. At present, two GLP-1 receptor agonists, exenatide and lixisenatide, have reported encouraging results in phase 2 double-blind clinical trials for PD and are currently in late stage clinical development. In this review, we will provide an overview of the current data supporting the justification for the repurposing efforts of certain GLP-1 receptor agonists as possible disease-modifying therapies for PD. Cure Parkinson's would welcome any feedback or thoughts regarding this review from the wider research and PD communities.

**THE HISTORY OF GLP-1 RECEPTOR AGONISTS:**

The story of GLP-1 receptor agonists begins with the Gila monster. This unassuming creature native to North America belongs to the only family of poisonous lizards, using its venom for defense against predators. It spends 95% of its life underground; however, an encounter with a Gila monster might result in an unpleasant bite, causing pain and weakness. The Gila monster became a topic of interest for the research community when Dr John Eng at the Veterans Administration Centre in the Bronx (NY) learned that these creatures were able to slow down their metabolism and maintain constant blood sugar levels during long periods of not eating. Dr Eng analysed the venom of the Gila monster and discovered a molecule that he named exendin-4 (Eng et al., 1992). This peptide was able to stimulate the synthesis and release of insulin from beta cells in the pancreas. To the surprise of the researchers involved in this work, exendin-4 was found to be both structurally and functionally similar to a hormone found in the human pancreas called glucagon-like peptide-1 (GLP-1). Like exendin-4, GLP-1 activates insulin production in the pancreas, but only when glucose production is high. However, Dr Eng and colleagues noted that GLP-1 only remains active in the body for a couple of minutes, while exendin-4 is active for hours. This observation indicated exendin-4-like agents could be useful long-acting treatments for conditions like type 2 diabetes.

Preclinical testing of exendin-4 for diabetes began in the early 1990s and was led by Dr Josephine Egan and colleagues at the National Institute of Aging in the US. In 1999, a once-daily injection of exendin-4 was shown to normalize blood glucose concentration in diabetic mice (Greig et al., 1999), paving the way for clinical development. With help from the National Institute of Aging, the biotech company Amylin Pharmaceuticals developed a synthetic version of exendin-4 called exenatide, which, after years of clinical testing, was approved in 2005 by the Food and Drug Administration (FDA) as a subcutaneous injection for the treatment of type 2 diabetes.

**GLUCAGON-LIKE PEPTIDE-1 (GLP-1):**

GLP-1 belongs to a family of incretin hormones, gut-derived peptides that are secreted after nutrient intake and in response to mechanical forces such as gastric distension (Nauck and Meier, 2018). In the gastrointestinal tract, it is produced by enteroendocrine cells, or L-cells, and by binding to the GLP-1 receptor in the pancreas it is able to stimulate insulin production from pancreatic B-cells. Moreover, GLP-1 is also endogenous to the central nervous system (CNS) as there are distinct populations of neurons in the caudal medulla of the hindbrain and the hypothalamus that also produce GLP-1 (Kreymann et al., 1989; Larsen et al., 1997), with the GLP-1 receptor expressed on a variety of cell types in addition to neurons, microglia and astrocytes (Alvarez et al., 2005). Within the CNS, hindbrain GLP-1 neurons project to numerous brain regions relevant to regulation of metabolism and appetite (Cabou and Burcelin, 2011).

**GLP-1 RECEPTOR AGONISTS**:

There are currently eight GLP-1 mimetic agents that have been approved for clinical use: exenatide, lixisenatide, liraglutide, semaglutide, beinaglutide, albiglutide, tirzepatide and dulaglutide (Boer and Holst, 2020). These agents have individual strengths and weaknesses, variable chemical structure and slightly different indications for use. Importantly, some of them have a prominent weight loss effect, which might be a contraindication for use in PD (see below). In addition, the agents report different levels of brain penetrance, which may be an important consideration for clinical testing in PD. A summary of the main GLP-1R agonists approved by international regulatory agencies can be found in Table 1.

| Owner | Compound | Trade Name | Description | Formulation | Administration | Frequency |
|---|---|---|---|---|---|---|
| AstraZeneca | Exenatide | Bydureon | Synthetic exendin-4 | Long-acting | SC | Once a week |
| | | Byetta | | Short-acting | SC | Twice a day |
| Sanofi | Lixisenatide | Adlyxin/Lyxumia | Synthetic exendin-4 | Short-acting | SC | Once a day |
| Novo Nordisk | Liraglutide | Victoza | Human GLP-1 analogue | Long-acting | SC | Once a day |
| | | Saxenda | | Long-acting | SC | Once a day |
| | Semaglutide | Ozempic | Human GLP-1 analogue | Long-acting | SC | Once a week |
| | | Wegovy | | Long-acting | SC | Once a week |
| | | Rybelsus | | Long-acting | Oral | Once a day |
| Benemae | Beinaglutide | Feisumei | Human GLP-1 analogue | Short-acting | SC | Once a day |
| GlaxoSmithKline | Albiglutide* | Tanzeum | GLP-1 fused with albumin | Long-acting | SC | Once a week |
| Eli Lilly | Tirzepatide | Mounjaro | Dual GLP-1/GIP agonist | Long-acting | SC | Once a week |
| | Dulaglutide | Trulicity | Human GLP-1 analogue | Long-acting | SC | Once a week |

**Table 1.** GLP-1 receptor agonists historically approved by the United States FDA and international regulatory agencies. SC = subcutaneous injection. * = withdrawn in 2017

**GLP-1 RECEPTOR AGONISM AND NEUROPROTECTION:**

Early in the process of developing these agents, researchers observed that both GLP-1 and exenatide exhibited neuroprotective properties in preclinical models. They were found to protect cultured rat

hippocampal neurons against glutamate-induced apoptosis (Perry et al., 2002), reduce the levels of Alzheimer's associated amyloid-β peptide (Aβ) in vivo (Perry et al., 2003), and rescue models of peripheral sensory neuropathy (Perry et al., 2007). These results prompted a deeper analysis of this group of compounds for the treatment of neurodegenerative disease, culminating in a large portfolio of preclinical data and completed clinical trials for both Alzheimer's and PD (for a review of both fields see Kopp et al., 2022).

**GLP-1 RECEPTOR AGONISTS IN MODELS OF PARKINSON'S:**

GLP-1 receptor agonists have been shown to impact a range of functions across a plethora of experimental models of PD (Athauda and Foltynie, 2016). In cell culture models of PD, researchers have demonstrated the neuroprotective and trophic effects of GLP-1 and related analogues (Perry et al., 2002; Li et al., 2009). Moreover, these responses could be reversed by pharmacological or genetic blockage of the GLP-1 receptor, demonstrating that activation of the GLP-1 receptor is necessary for this effect (Li et al., 2009).

The neuroprotective effect of exenatide/exendin-4 and other GLP-1 receptor agonists has been evaluated in multiple neurotoxin rodent models of PD (Bertilsson et al., 2008; Kim et al., 2009; Li et al., 2009; Liu et al., 2015; Hansen et al., 2016; Chen et al., 2018; Zhang et al., 2018; Yu et al., 2020; Khalaf et al., 2023). GLP-1 receptor agonism was found to markedly protect the dopaminergic system by preserving the number of dopamine neurons, as well as preventing motor complications in these models. These agents have also been tested in alpha-synuclein-based mouse models of PD, where they have improved behavioural outcomes and neuropathology (Yun et al., 2018; Zhang et al., 2023).

Researchers have speculated that the neuroprotective effects of GLP-1 receptor agonism may be due to its attenuation of neuroinflammation (Kopp et al., 2022). Previous studies found that mitochondrial dysfunction, oxidative stress, and reactive oxygen species (ROS) production are upstream priming signals for NLRP3 inflammasome activation (Ahmed et al., 2021; Jayaram and Krishnamurthy, 2021), implicating this pathway in the inflammatory response seen in PD. Additionally, not only is there evidence of neuroprotection, but GLP-1 receptor agonists may also augment the effects of L-dopa treatment and reduce dyskinetic movements in rodent models of PD (Badawi et al., 2019; Kuo et al., 2023).

**EPIDEMIOLOGICAL DATA:**

In addition to preclinical findings, epidemiological studies provide additional support for the repurposing of GLP-1 receptor agonists for PD. Past studies have shown that people with type 2 diabetes were at a higher risk of developing PD, and people with both PD and type 2 diabetes showed a greater severity of motor symptoms (Komici et al., 2021). Nonetheless, multiple studies have now reported, that people with type 2 diabetes being treated with GLP-1 receptor agonists are less likely to develop PD than those on other classes of diabetes treatment (Brauer et al., 2020; Tang et al 2023). It is also interesting to note that postprandial plasma levels of GLP-1 are reduced in people with PD compared to controls (Manfready et al., 2021).

**POTENTIAL MECHANISM OF ACTION:**

The exact group of pathways that might be involved in the beneficial effects seen in models of PD (or in diabetic patients) are currently unknown; however, the possible contenders are presented below in Figure 1. These include activation of mitogen-associated protein kinase/extracellular signal-regulated kinase (MAPK/ERK) and phosphoinositide 3-kinase/protein kinase B (PI3K/AKT), which acts as a major regulator of physiological responses to normal ageing (Athauda and Foltynie, 2016). AKT has the ability to phosphorylate over 50 substrate proteins, such as glycogen synthase kinase-3 beta (GSK-3B). Dysregulation of GSK-3B signalling has been shown to contribute to PD-like pathophysiology, including alpha-synuclein aggregation, oxidative stress and mitochondrial dysfunction (Golpich et al., 2014).

Recent research has pointed towards the MAPK pathway involvement as well. Models of synucleinopathy have been reported to exhibit neuronal insulin resistance and dysfunctional insulin signalling. This effect is associated with inhibition of the neuroprotective AKT pathways, and increased expression of the MAPK-associated stress pathways. *In vitro* administration of exenatide restored insulin and AKT signalling, and suppressed the MAPK pathways (Athauda et al., 2024). In the phase 2 exenatide clinical trial (Athauda et al., 2017), exenatide treatment was found to be associated with clinical improvement in individuals with higher baseline MAPK expression (Athauda et al., 2024). These, as well as other pathways not mentioned here, might contribute to the therapeutic effect of GLP-1 receptor agonists in PD; however, further research is needed to better elucidate the potential mechanisms of action of these agents.

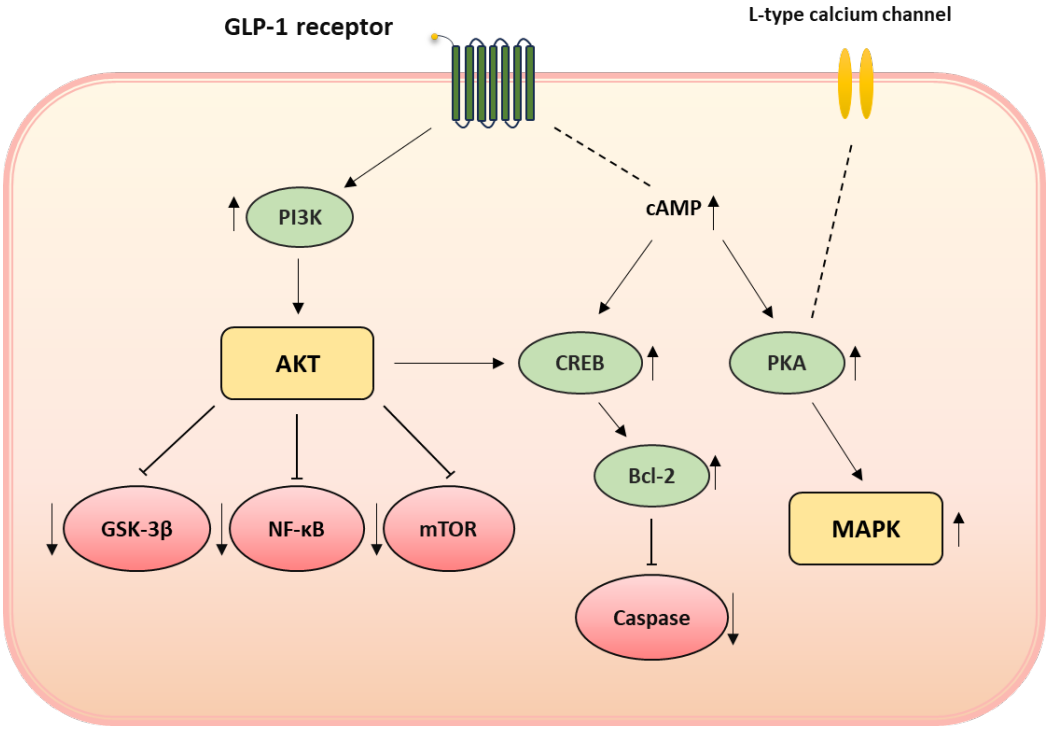


**Figure 1.** Biochemical changes associated with the activation of the GLP-1 receptor inside the cell.

**CLINICAL TRIALS OF GLP-1 RECEPTOR AGONISTS FOR PARKINSON'S:**

Since 2012, Cure Parkinson's has convened an annual meeting of PD experts, called the international Linked Clinical Trials (iLCT) committee. Their task is to evaluate and prioritise agents that have

demonstrated potential for disease modification in PD, in an effort to speed up the search for a therapy that can slow, stop or reverse the condition (Wyse et al., 2024). The iLCT committee is made up of more than 20 world-leading experts on PD, who are tasked with reviewing a collection of approximately 20 drug dossiers that Cure Parkinson's prepares each year. In the first year of this initiative, exenatide, lixisenatide and liraglutide were all prioritised by the iLCT committee, which gave Cure Parkinson's a mandate to move those agents into clinical trial. All three agents have now been clinically evaluated in Phase 2 trials for PD. Additional GLP-1 receptor agonists have also been undergoing clinical development for PD, some of which are mentioned below.

*Exenatide*

Exenatide has the longest and most robust clinical history of any GLP-1R agonist being tested in PD, with 5 clinical trials conducted to date, including a Phase 3 clinical trial (which is nearing completion).

The first clinical trial of exenatide for PD was conducted in 2008 as a randomised, open-label pilot study involving 45 participants over 12 months (NCT01174810), and was funded by Cure Parkinson's. Exenatide was self-administered twice-daily in the form of a subcutaneous injection using a pen device. At the time of the study, a placebo pen was unfortunately not available due to the prohibitive costs associated with their manufacture. As a result, the investigators configured the trial to be open-label, with 21 participants receiving exenatide and 24 staying on conventional PD medication for the duration of the trial. Assessments of PD severity using MDS-UPDRS part 3 were made after an overnight withdrawal of PD medication and were video recorded to allow objective rating by blinded observers. One participant receiving exenatide withdrew from the study due to severe weight loss, which was fully reversible on cessation of the drug. The pilot study achieved its primary outcome and exenatide was found to be safe and well-tolerated. After 12 months, participants in the exenatide group had a mean improvement of 2.7 points on the MDS-UPDRS part 3 motor subscale, compared with a mean decline of 2.2 points in the control group. The overall difference in MDS-UPDRS part 3 scores was 4.9 points between participant groups, which was statistically significant ($P$ = 0.037; Aviles-Olmos et al., 2013).

All participants who completed the trial (n=43) were invited for a further follow-up assessment 12 months after the study finished to investigate whether these possible advantages persisted in the prolonged absence of this medication. Motor severity of PD was compared after overnight withdrawal of conventional PD medication using blinded video assessments of the MDR-UPDRS, together with several non-motor tests. Compared to the control group, participants previously exposed to exenatide had an advantage of 5.6 points on the MDS-UPDRS part 3 using blinded video rating, which was statistically significant ($P$ = 0.002; Aviles-Olmos et al., 2014). These data provided encouragement for the further study of exenatide in a larger, placebo-controlled trial.

In 2014, a randomised, double-blind, placebo-controlled trial of exenatide (EXENATIDE-PD) was initiated at University College London (NCT01971242). It involved 60 people with mild to moderate PD who were randomised into either the exenatide (n=31) and placebo (n=29) groups. In this study, exenatide or placebo was self-administered once-weekly for 48 weeks in addition to regular PD medication, followed by a 12-week washout period. Participants were assessed with physical and neurological examinations (MDS-UPDRS) as well as mood and cognition tests over 12 weeks. Additionally, DaTscan imaging was performed at first and last visit to assess dopaminergic function of presynaptic terminals and as an exploratory biomarker of neuron loss in PD.

At the end of the washout period, participants in the placebo group had declined by 2.1 points in the MDS-UPDRS part 3 OFF medication state, while the exenatide group improved by 1.0 point. This overall

advantage of 3.5 points favouring the exenatide group was statistically significant (*P* = 0.0318). At the end of the drug exposure period of the study (48 weeks), the placebo group had declined by 1.7 points while the exenatide group improved by 2.3 points, resulting in a difference of 4.3 points between the two groups, which was again statistically significant (*P* = 0.0026). No statistically significant differences were detected in MDS-UPDRS part 1, 2 and 4, nor in the MDS-UPDRS part 3 in the ON medication state. Furthermore, DaTscan imaging showed significant declines in dopamine transporter binding between first and second scans for both groups; however, a reduced rate of decline of DaTscan binding in the exenatide group compared to the placebo group was observed in the right putamen, left putamen and right caudate nucleus (Athauda et al., 2017).

With the trial achieving its primary endpoint, the clinical development of exenatide for PD was progressed into Phase 3 testing. As this larger study was being set up, post-hoc analyses of the Phase 2 trial data were conducted to determine if exenatide-responders could be identified. The results of this analysis suggested that the trial participants who had the strongest benefit to exenatide treatment were younger at the time of diagnosis and more tremor-dominant (Athauda et al., 2019a). The deep dive analysis of the data also highlighted improvements in non-motor symptoms of PD (Athauda et al., 2018). At the same time, efforts were made to elucidate the mechanism of action involved in the positive effect that was observed in the exenatide-treated group. To do this, researchers analysed the contents of neuronal-derived exosomes extracted from blood samples collected from the trial participants. Analysis of the contents of these exosomes indicated that the exenatide treated cohort had elevated tyrosine phosphorylation of insulin receptor substrate 1, as well as increased expression of downstream substrates, such as total AKT and phosphorylated mechanistic target of rapamycin (mTOR). Interestingly, the improvements observed in MDS-UPDRS part 3 OFF medication scores were positively associated with levels of total mTOR and phosphorylated mTOR (Athauda et al., 2019b). The results may provide an objective measure of target engagement for future clinical trials of GLP-1 receptor agonists.

The Phase 3 clinical trial of exenatide (EXENATIDE-PD3; NCT04232969) commenced in 2020 as a randomised, double-blind, parallel-group, placebo-controlled study taking place across six NHS sites in the UK. 196 participants were randomised to receive exenatide or placebo injections once weekly for 96 weeks, and the trial completed in 2024 (Vijiaratnam et al., 2021).

In addition to the UK exenatide clinical trial programme, there have been additional studies conducted globally. A Phase 2 trial of exenatide was recently conducted in Sweden, recruiting 60 participants with PD who were randomly assigned to receive injections of exenatide or placebo for 18 months (NCT04305002). The aim of this study was to investigate the effect of exenatide on PD progression, represented by the change in FDG-PET, which is used to image regional cerebral glucose metabolism as a marker of neuronal activity and by extension neuronal survival (Brumberg et al., 2020). This trial also completed in 2024.

Additionally, a small imaging study of 5 people with PD was conducted at the University of Florida. Participants received exenatide once per week for 12 months, and multiple imaging methods were used to assess changes in the brain affected by PD (NCT03456687). Both functional and structural MRI scans of the brain were taken, and changes in free-water accumulation and blood oxygen level-dependent signal were measured in the substantia nigra and the posterior putamen at baseline and after 12 months.

*Lixisenatide*

Another GLP-1 receptor agonist that is being evaluated for its disease-modifying potential in PD is lixisenatide, which was approved for the treatment of type 2 diabetes by the European Commission in 2013. Similar to exenatide, its chemical structure is derived from the peptide exendin-4; however, lixisenatide comprises a longer amino acid sequence, with six lysine residues attached to its N-terminus (Christensen et al., 2009).

The clinical development of lixisenatide for the treatment of PD commenced with the LixiPark Phase 2 clinical trial, a multi-centre, parallel group, two-arm, randomised, double-blind and placebo-controlled study involving 21 research centres across France (NCT03439943). The study involved 156 people with early PD receiving once-daily subcutaneous injections of lixisenatide or placebo for 12 months, followed by a wash-out period of 2 months. The primary endpoint of this trial was the change in MDS-UPDRS part 3 motor score from baseline to end-point (12 months), evaluated in the ON condition.

The results of this trial showed that after 12 months of daily treatment, the mean MDS-UPDRS part 3 score (in the ON medication state) remained at baseline levels for lixisenatide recipients while the placebo group's motor symptoms had continued to deteriorate (0.0 vs 3.0; $P = 0.0068$). Moreover, at month 14 (after two months of the termination of lixisenatide or placebo treatment), the mean MDS-UPDRS part 3 score (in the OFF medication state), a prespecified secondary outcome, was significantly lower for lixisenatide versus placebo (17.7 vs 20.6; $P = 0.0445$). No between-group differences in other secondary outcomes such as changes in MDS-UPDRS parts 1, 2 and 4 were statistically significant (Meissner et al., 2024).

*Liraglutide*

Liraglutide belongs to a different class of GLP-1 receptor agonists, as its molecular structure is based on a modified version of the human GLP-1 peptide with an increased half-life (Knudsen and Lau, 2019). It was approved for the treatment of type 2 diabetes by the European Medicines Agency in 2015.

In 2017, a Phase 2 clinical trial of liraglutide for the treatment of PD was conducted at the Cedars-Sinai Medical Centre in the US. This was a single-centre, randomised, double-blind, placebo-controlled trial with 63 participants self-administering liraglutide injections once-daily (n=42) or placebo (n=21) for 52 weeks. Primary outcomes were the adjusted difference in the MDS-UPDRS part 3 score in the OFF medication state, Non-Motor Symptoms Scale (NMSS) and Mattis Dementia Rating Scale at week 54. The results of this trial indicated that NMSS scores improved by 6.6 in the liraglutide group and worsened by 6.5 points in the placebo group, with the 13.1 point adjusted mean difference trending towards significance ($P = 0.07$). None of the other primary outcomes of this trial were met. However, secondary outcome analysis revealed a significant improvement in the MDS-UPDRS part 2 scores, which represents the motor experiences of daily living (-4.1 points, $P = 0.001$; Malatt et al., 2022). This trial provided encouraging insights into the effects of liraglutide, particularly with non-motor symptoms and aspects of daily living.

*Semaglutide*

Semaglutide is a modification of liraglutide that is protease-resistant, and it was approved by the United States FDA as a once-weekly treatment for type 2 diabetes in 2017. This GLP-1 receptor agonist was developed by Novo Nordisk, and is currently in clinical development for Alzheimer's, with a Phase 3 clinical trial due to complete in 2024 (NCT05891496). There is also a clinical trial underway in Japan investigating oral semaglutide (or placebo) tablets for 99 individuals with PD for 48 weeks (jRCT2051230090 - https://rctportal.niph.go.jp/en/detail?trial_id=jRCT2051230090). The primary

outcome of that study is change in clinically defined off-state MDS-UPDRS part 3 score at 48 weeks. There were plans for a Phase 2 clinical trial in PD; however, the current status of that study is unclear (NCT03659682).

*Novel GLP-1 receptor agonists in clinical development*

In addition to the testing and repurposing of GLP-1 receptor agonists approved for type 2 diabetes treatment, novel molecules in this class are also being clinically investigated. An example of a novel GLP-1 receptor agonist is NLY01, developed by the biotech firm Neuraly. NLY01 is a pegylated exendin-4 analogue, structurally very similar to exenatide, that was prioritised by the iLCT committee in 2017. Neuraly announced topline results from their Phase 2 trial in patients with early, untreated PD in March 2023 (NCT04154072), where it was reported that the trial did not meet its primary outcome. However, post-hoc analysis of the results suggested a treatment effect in younger patients (<60 years), which may indicate a possible avenue for future research (McGarry et al., 2024).

Another GLP-1R agonist that is currently under clinical investigation for its disease-modifying potential in PD is PT320, a sustained-release two-week formulation of exenatide developed by a South Korean company called Peptron. A Phase 2 clinical trial (NCT04269642) was conducted to determine the efficacy of the drug, which did not meet its primary outcome. Peptron has communicated, however, that there was sufficient additional positive data to continue with their development of PT320 (https://www.koreabiomed.com/news/articleView.html?idxno=20071).

**POTENTIAL COMPLICATIONS WITH REPURPOSING GLP-1 RECEPTOR AGONISTS FOR PARKINSON'S:**

It is important to note that GLP-1 receptor agonists have a well characterised feature of appetite suppression, which can result in significant weight loss (Jensterle et al., 2022); in fact, semaglutide is also approved for the indication of weight management in adults with chronic obesity. A prominent weight loss effect could be a major contraindication for the use of certain GLP-1 receptor agonists in PD. In addition to this, GLP-1 receptor agonists exhibit variable levels of brain penetrance depending on the biochemical structure, which may be a significant factor to be taken into account in their clinical development for PD. At present, only exenatide, lixisenatide and liraglutide have demonstrated any impact on PD symptoms.

**SUMMARY:**

GLP-1 receptor agonists have had a long history as a treatment for type 2 diabetes, and their repurposing for neurodegenerative diseases like PD may represent an innovative use for this class of agents. The disease-modifying potential of GLP-1 receptor agonists has been evaluated in many PD models, which indicate that compounds like exenatide, lixisenatide and liraglutide exhibit neuroprotective, neurotropic and anti-inflammatory properties. The exact physiological mechanisms affected by these drugs to be able to exert these effects remain to be elucidated; however, activation of MAPK/ERK and AKT kinases might be partially responsible. The results of multiple clinical trials show that some GLP-1 receptor agonists (exenatide, lixisenatide and liraglutide) are safe and well-tolerated in people with PD and preliminary evidence suggests that exenatide & lixisenatide treatment slows motor symptom progression. The results of the Phase 3 clinical trial of exenatide will further inform our understanding of the potential disease-modifying effects of this class of drugs on PD progression in a larger cohort, which will then determine the next steps for clinical development.

Cure Parkinson's has championed the repurposing of GLP-1 receptor agonists for the treatment of PD since funding the first clinical trial of exenatide. We hope that, should sufficient evidence supporting their disease-modifying potential come to light, it will be possible to introduce suitable GLP-1 receptor agonists as an adjunct to standard of care treatment, or potentially as a preventative measure for prodromal cohorts, although this has yet to be clinically tested. Based on continued positive results, we remain committed to helping this class of drugs progress through clinical research and development to become available for people with PD.

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