

Review

Infectious Diseases as a Potential Risk Factor for Parkinson's Disease and Parkinsonism: A Scoping Review

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Abstract

Parkinsonism is a syndrome characterized by bradykinesia, rigidity, tremor, and postural instability, with Parkinson's disease (PD) being the most common cause. Increasing evidence suggests that infectious diseases may play a role in triggering or exacerbating Parkinsonian symptoms through direct neurotoxicity, neuroinflammation, or alterations in the gut-brain axis. **Methods:** A scoping review following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines was conducted. PubMed, Cochrane, and MEDLINE databases were searched for studies examining infections, PD, and Parkinsonism. Eligible studies assessed infection-related PD or Parkinsonism risk and symptom modulation. **Results:** Thirty-seven studies met the inclusion criteria. Several infections were linked to Parkinsonism, with chronic viral infections such as hepatitis C virus (HCV) and hepatitis B virus (HBV), and bacterial infections including *Helicobacter pylori* and *Clostridium difficile*, associated with an increased risk of PD, while some infections showed no significant effect. Acute infections, such as urinary tract and respiratory infections, were frequently linked to transient worsening of symptoms. Antiviral therapy and *H. pylori* eradication demonstrated potential benefits, although findings were inconsistent. **Conclusion:** The relationship between infections and PD is complex, with certain infections potentially triggering or worsening symptoms, while others appear neutral or protective. Understanding infection-related mechanisms of neurodegeneration is essential for developing preventive and therapeutic strategies. Further longitudinal studies are needed to clarify causal links and optimize infection management in PD.

Keywords: Parkinsonism; neuroinflammation; bradykinesia

1. Introduction

Parkinsonism is a clinical syndrome characterized by bradykinesia, rigidity, tremor, and postural instability [1]. It encompasses a broad range of conditions, including Parkinson's Disease (PD), multiple system atrophy (MSA), progressive supranuclear palsy (PSP), corticobasal degeneration (CBD), drug-induced Parkinsonism, vascular Parkinsonism, and post-infectious Parkinsonism [2]. While PD is the most common cause of Parkinsonism, increasing evidence suggests that infectious diseases may play a role in triggering or exacerbating Parkinsonian symptoms, either through direct neurotoxicity, neuroinflammation, or alterations in the gut-brain axis [3]. The potential link between infectious diseases and Parkinsonism has historical precedence. The 1918 influenza pandemic was related to a

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dramatic increase in cases of post-encephalitic Parkinsonism, also known as von Economo encephalitis, which presented with severe Parkinsonian symptoms in affected individuals [4]. This relationship raised questions about whether viral infections could contribute to long-term neurodegenerative processes leading to Parkinsonism [5]. More recently, research has examined the possible role of other viruses, including herpes simplex virus (HSV), Epstein-Barr virus (EBV) [6], varicella-zoster virus (VZV) [7], and the hepatitis C virus (HCV), in the development of Parkinsonian syndromes. Findings indicate that chronic viral infections, including HSV, HCV, and hepatitis B virus (HBV)[8,9], may be related to an increased risk of PD. At the same time, other studies suggest a possible protective effect of certain viral infections or their treatments [10]. Beyond viral infections, bacterial pathogens such as *Helicobacter pylori* (*H. pylori*) have also been implicated in Parkinsonism. *H. pylori* infection has been shown to contribute to the worsening of motor symptoms in individuals with PD, potentially through systemic inflammation, gut dysbiosis, and impaired absorption of Levodopa, the primary treatment for PD [11–13]. Some studies in this review also demonstrated that *H. pylori* eradication therapy was related to improvements in PD motor symptoms, supporting the role of gut microbiome disruptions in disease pathophysiology [14,15]. Furthermore, chronic infections, such as those caused by *Borrelia burgdorferi* (*B. burgdorferi*) (Lyme disease) and *Treponema pallidum* (*T. pallidum*) (syphilis) [16], have been reported to induce Parkinsonian symptoms, raising concerns about the role of persistent bacterial infections in neurodegeneration. Urinary tract infections (UTIs)[17] and respiratory infections [18–22] have also been related to transient worsening of PD symptoms, particularly in hospitalized individuals. The mechanisms underlying infection-related Parkinsonism remain poorly understood but may include neuroinflammation, molecular mimicry, and immune system dysregulation. Infections may induce chronic neuroinflammation by activating microglia and astrocytes, leading to the release of pro-inflammatory cytokines that damage dopaminergic neurons in the substantia nigra [23]. Additionally, some pathogens may trigger autoimmunity through molecular mimicry, where immune responses against microbial antigens mistakenly target neuronal proteins, leading to neurodegeneration [24]. The gut-brain axis, which links the gut microbiome to central nervous system function, has also emerged as a potential pathway through which infections might contribute to Parkinsonism [25]. Disruptions in the gut microbiome due to infections can alter intestinal permeability, allowing bacterial endotoxins and inflammatory mediators to reach the brain and promote neuroinflammation [26]. Additionally, studies suggest that systemic infections, particularly those occurring in early adulthood, may be related to a higher risk of later PD development, highlighting the need to explore infection timing as a potential factor in PD pathogenesis [27]. Despite these observations, the relationship between infections, PD, and Parkinsonism remains incompletely understood. While some infections appear to cause transient or reversible Parkinsonian symptoms, others may contribute to long-term neurodegeneration. This scoping review aims to systematically map the literature on the relationship between infectious diseases, PD, and Parkinsonism. Specifically, this scoping review seeks to identify the types of pathogens related to PD and Parkinsonism, summarize potential mechanisms linking infections to neurodegeneration and PD and Parkinsonian syndromes, and highlight gaps in the current literature to guide future research on infection-related PD and Parkinsonism.

2. Materials and Methods

2.1. Source of Data and Search Strategy

This scoping review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines [28]. A systematic literature search on infectious diseases as potential risk factors for PD and Parkinsonism

was performed using PubMed, Cochrane, and Medline electronic databases and manual searches. They were searched from inception to 18th March 2025. Searches were restricted to articles in the English language. Appendix 1 provides a detailed list of search terms utilized.

3. Inclusion and Exclusion Criteria

Studies were included with the following criteria: studies involving individuals diagnosed with PD or Parkinsonism, studies examining a relationship between infection or infectious diseases and PD or Parkinsonism, including but not limited to viral and bacterial infections, studies focusing on neurodegeneration, risk factors, etiology, association, relationship, or correlation between infections and PD or Parkinsonism, were cohort, case-control, case, cross-sectional, clinical, epidemiological, longitudinal, prospective, retrospective or experimental studies involving human participants, studies that reported on the risk of developing PD or Parkinsonism, worsening of symptoms, or relationships between infections and neurodegeneration, were peer-reviewed, and were published in the English language. Studies were excluded with the following criteria: studies involving animal models or in vitro studies, studies not examining a relationship between infection or infectious diseases and PD or Parkinsonism, studies focusing on neurodegeneration without investigating infections as a risk factor, or studies on infections without assessing their impact on PD or Parkinsonism, were reviews, meta-analyses, editorials, commentaries, conference abstracts, or opinion papers, had incomplete reporting of results, lack of clear diagnostic criteria for PD or Parkinsonism, or insufficient sample size, were not peer-reviewed, were not published in the English language, and were without full-text availability (Table 1).

4. Study Selection

Two reviewers (PA, DC) independently screened all titles and abstracts of the identified studies. Full texts were obtained for the studies deemed eligible from the initial screening. Two reviewers (PA, DC) independently reviewed full texts. Any discrepancies were discussed and resolved through discussion between reviewers.

5. Study Quality

No formal risk of bias assessment or study quality grading was conducted, consistent with the methodological framework for scoping reviews. However, to ensure the reliability of the included studies, two reviewers (PA, DC) independently evaluated descriptive study characteristics. Studies were evaluated based on study design (e.g., cohort, case-control, cross-sectional, randomized trials), sample size, data sources (e.g., hospital records, national health databases, self-reported surveys), and methodological rigor. Key aspects considered included inclusion and exclusion criteria, statistical adjustments for confounders, follow-up duration in cohort studies, and validation of diagnostic criteria for Parkinsonism and infectious diseases. Studies with small sample sizes, unclear case definitions, or high risk of selection bias were noted but were not excluded, given the exploratory nature of this review. Findings were interpreted in the context of study limitations, and inconsistencies between studies were examined to identify potential sources of heterogeneity. Any discrepancies were discussed and resolved through discussion between reviewers.

6. Results

6.1. Study Selection

The electronic search of databases yielded 223 articles. One hundred and fifty-two articles were excluded after reviewing titles and abstracts. Thirty-two articles were excluded

Table 1. Inclusion and Exclusion Criteria for the Scoping Review

Parameter	Inclusion Criteria	Exclusion Criteria
Population	Studies involving individuals diagnosed with PD or Parkinsonism.	Studies involving animal models or in vitro studies.
Exposure	Studies examining a relationship between infection or infectious diseases and PD or Parkinsonism, including but not limited to viral and bacterial infections.	Studies that do not examine a relationship between infection and PD or Parkinsonism.
Focus	Studies exploring neurodegeneration, risk factors, etiology, association, relationship, or correlation between infections and PD or Parkinsonism.	Studies focusing on neurodegeneration without investigating infections as a risk factor, or studies on infections without assessing their impact on PD or Parkinsonism.
Study Design	Cohort, case-control, case, cross-sectional, clinical, epidemiological, longitudinal, prospective, retrospective, or experimental studies involving human participants.	Reviews, meta-analyses, editorials, commentaries, conference abstracts, or opinion papers.
Outcome Measures	Studies reporting on the risk of developing PD or Parkinsonism, worsening of symptoms, or relationships between infections and neurodegeneration.	Studies with incomplete reporting of results, lack of clear diagnostic criteria for PD or Parkinsonism, or insufficient sample size.
Language & Publication	Studies that are peer-reviewed and published in the English language.	Studies that are not peer-reviewed, published in languages other than English, or without full-text availability.

because they were duplicates. The remaining 39 articles were retrieved and assessed for eligibility via full-text review. Two articles were excluded because they were not eligible. Thirty-seven remaining articles [29–47] were found eligible and included in the review (Figure 1).

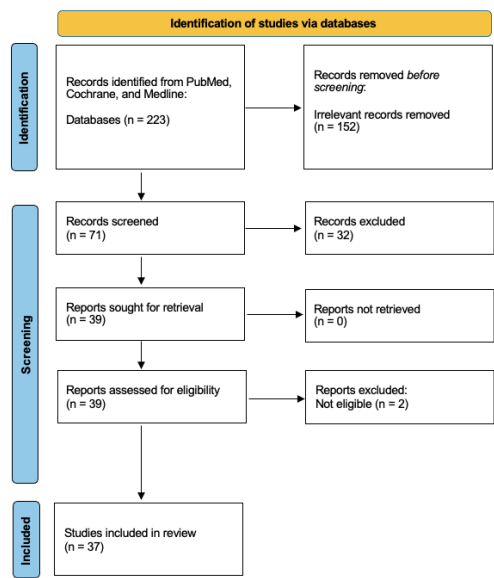


Figure 1. PRISMA Flow Diagram of Study Selection

6.2. Characteristics of Selected Studies

The selected studies employed diverse methodologies, including retrospective and prospective cohort studies, case-control studies, and population-based analyses, to examine the relationship between infections and PD or Parkinsonism. Many were large-scale epidemiological studies leveraging national health databases, hospital records, and electronic health records, while others used clinical cohorts, serological testing, and Mendelian randomization approaches. Study populations spanned North America, Europe, Asia, and the Middle East, with some studies assessing PD risk factors in the general population, while others focused on infection-related symptom exacerbation in diagnosed individuals with PD. Follow-up durations ranged from six months to over 15 years. Infection classification methods varied, with some studies relying on self-reported history or physician diagnoses, while others employed serological tests, microbiological cultures, or hospital admission records. Most studies investigated viral infections, particularly hepatitis viruses, herpesviruses, and COVID-19, while others explored bacterial infections such as *Clostridium difficile* (*C. difficile*) and *H. pylori* and parasitic infections like *Toxoplasma gondii* (*T. gondii*) and *Toxocara* species. Additionally, several studies examined the impact of infection treatment on PD risk and symptoms, including antiviral therapy for hepatitis C, *H. pylori* eradication, and antibiotic treatment for neurosyphilis-related Parkinsonism, highlighting the potential role of infection management in PD prevention and symptom modulation (Table 2).

6.3. Characteristics of Participants

The study populations included both diagnosed individuals with PD and broader population-based cohorts, with some studies focusing on older adults (over 60 years) due to the age-related nature of PD, while others examined younger individuals to explore early-life risk factors. Sample sizes ranged from small clinical case series with fewer than

Table 2. Summary of Included Studies

Authors	Number of Participants	Pathogen Studied
Wang et al. (1993)	279	Environmental and viral exposures
Pintar Hubble et al. (1995)	280	<i>Nocardia</i> species
Martyn et al. (1995)	515	Croup, diphtheria
Tsui et al. (1999)	7073	Viral respiratory infections
Hufschmidt et al. (2010)	10683	Urinary tract infections
Harris et al. (2012)	403	Influenza, zoonotic infections
Bu et al. (2015)	272	Multiple pathogens
Tan et al. (2015)	102	<i>Helicobacter pylori</i>
Kim et al. (2016)	3116	Hepatitis C virus
Tsai et al. (2016)	249835	Hepatitis C virus
Mahami Oskouei et al. (2016)	150	<i>Toxoplasma gondii</i>
Liu et al. (2017)	94	<i>Helicobacter pylori</i>
Pakpoor et al. (2017)	176000	Hepatitis B/C virus
Alvarado-Esquivel et al. (2017)	260	<i>Toxoplasma gondii</i>
Huang et al. (2018)	18210	<i>Helicobacter pylori</i>
Goldstein et al. (2019)	252273	Hepatitis B/C virus
Lin et al. (2019)	188152	Hepatitis C virus
Kang et al. (2020)	4670423	<i>Clostridium difficile</i>
Camacho-Soto et al. (2020)	207885	Herpesvirus
Hasan et al. (2020)	918	Hospital-treated infections
Tan et al. (2020)	80	<i>Helicobacter pylori</i>
Cheng et al. (2020)	65355	Herpes zoster
Choi et al. (2020)	1010317	Hepatitis B/C virus
Taquet et al. (2021)	236379	COVID-19
Cocoros et al. (2021)	61426	Influenza
Sun et al. (2022)	405841	Hospital-treated infections
Araújo et al. (2023)	600	COVID-19
Selim et al. (2023)	17199	Hepatitis C virus
Chang et al. (2024)	9350	<i>Helicobacter pylori</i>
Tunnicliffe et al. (2024)	1174759	Herpes zoster
Hernandez-Con et al. (2024)	48356	Hepatitis C virus
Zhang et al. (2024)	54	<i>Treponema pallidum</i>
Yin et al. (2024)	207	COVID-19
Vingeliene et al. (2024)	3698319	Gastrointestinal and respiratory infections
Zhao et al. (2024)	932	<i>Helicobacter pylori</i>
Wang et al. (2025)	69731	Hospital-treated infections
Khatir et al. (2025)	181	<i>Toxocara</i> infection

100 participants to large-scale epidemiological studies with millions of individuals. Studies assessing infection-related PD risk primarily included middle-aged and older adults with a history of viral, bacterial, or parasitic infections, particularly HBV and HCV, which were investigated in cohorts exceeding one million individuals with follow-ups of up to 15 years. Others focused on early-life respiratory and gastrointestinal infections (ages 21–30) to assess potential long-term contributions to PD development. Studies examining infection-related symptom worsening primarily included individuals with existing PD diagnoses, often recruited from movement disorder clinics or neurology departments.

6.4. Evidence Synthesis and Results

The findings across studies revealed a complex and heterogeneous relationship between infections and PD. Some infections or pathogens were related to increased PD risk, others had no significant relationship, and a few showed potential protective effects.

6.5. Risk of Developing Parkinsonism or Parkinson's Disease

Several studies have identified infections as potential risk factors for PD or Parkinsonism. Early research suggested that early-life respiratory infections, particularly diphtheria (OR: 2.3, 95% CI: 1.2–4.7) and croup (OR: 4.1, 95% CI: 1.1–16.1), were related to an increased risk of PD, though recall bias was noted as a limitation [21]. Similarly, individuals in occupations with high exposure to airborne infections, such as healthcare workers (OR: 2.07, 95% CI: 1.34–3.20) and teachers (OR: 2.50, 95% CI: 1.67–3.74), demonstrated a significantly elevated risk of developing PD [20]. These findings suggested that chronic exposure to pathogens might contribute to neurodegeneration. Further investigations into viral infections have yielded mixed results. Wang et al., 1993, did not find a significant relationship between PD and past infections such as measles, rubella, HSV-1, and cytomegalovirus (CMV) ($p > 0.05$). Instead, environmental exposures like drinking river water (OR: 2.12, 95% CI: 1.02–4.45) and proximity to rubber plants (OR: 3.02, 95% CI: 1.43–6.30) were linked to increased PD risk, highlighting the potential role of environmental toxins in PD pathogenesis [43]. However, more recent studies have focused on the role of chronic infections, particularly hepatitis viruses, in increasing susceptibility to PD. Individuals with HCV have been found to have a 29% higher risk of developing PD (HR: 1.29, 95% CI: 1.06–1.56), while HBV was inversely related to PD risk (HR: 0.66, 95% CI: 0.55–0.80) [46]. In contrast, a later study confirmed an increased PD risk for both HBV (RR: 1.76, 95% CI: 1.28–2.37) and HCV (RR: 1.51, 95% CI: 1.18–1.90) [47]. Additionally, antiviral therapy has been shown to significantly reduce PD incidence among HCV-infected individuals (HR: 0.71, 95% CI: 0.58–0.87), suggesting a neuroprotective effect in treating chronic viral infections. Recent studies have also explored the link between bacterial infections and PD risk. Kang et al., 2020, found an increased PD risk following *C. difficile* infection, though the relationship was only significant within two years post-infection (HR: 1.38, 95% CI: 1.12–1.69). Similarly, prior influenza infections were related to PD risk more than ten years post-infection (OR: 1.73, 95% CI: 1.11–2.71), while UTIs showed a persistent relationship with PD (OR: 1.19, 95% CI: 1.01–1.40). Viral infections, particularly herpesviruses and COVID-19, have also been examined for their role in PD. Herpes zoster has been related to an increased PD risk (HR: 1.80, 95% CI: 1.43–2.28), while a more recent study did not find a significant relationship (HR: 0.95, 95% CI: 0.90–1.01). Additionally, COVID-19 survivors requiring intensive care unit (ICU) admission had a higher incidence of Parkinsonism (HR: 1.45, 95% CI: 1.05–2.00), a finding further supported by Araújo et al., 2023, who reported a 45% increased risk of developing Parkinsonian symptoms among those with severe infections (HR: 1.45, 95% CI: 1.12–1.88).

6.6. Worsening of Parkinsonian Symptoms

Several studies have suggested that infections could exacerbate motor and non-motor symptoms in individuals with PD. Among individuals with PD who contracted COVID-19, 35.3% experienced worsening symptoms, with bradykinesia (33.3%) and tremor (29.2%) being the most affected motor symptoms. The study suggested that prolonged neuroinflammation following infection might contribute to transient symptom worsening. UTIs have also been identified as a potential aggravating factor for PD symptoms. Hufschmidt et al., 2020, reported that three hospitalized individuals with PD and UTIs experienced transient worsening of motor function, which improved following antibiotic therapy, suggesting a reversible para-infectious effect. Gastrointestinal infections also emerged as a potential contributor to PD progression. Individuals with *H. pylori* infection had worse motor performance, as indicated by higher Unified Parkinson's Disease Rating Scale (UPDRS) Part III scores ($p = 0.04$) [42]. Similarly, another study reported that *H. pylori* infection increased PD risk (HR: 2.29, 95% CI: 1.44–3.66), with the effect being more pronounced among those aged 60 or older. These findings suggest that chronic bacterial infections affecting the gastrointestinal system may play a role in neurodegenerative processes related to PD.

6.7. Improvement of Parkinsonian Symptoms

A few studies have reported symptom improvement following the treatment of infections. Zhang et al., 2024, found that individuals with neurosyphilis (*T. pallidum*)-related Parkinsonism showed notable symptom improvement after antibiotic therapy, particularly in tremor and orofacial dyskinesia, while bradykinesia remained mainly unchanged [16]. The findings suggest that neurosyphilis-induced Parkinsonism may be partially reversible with appropriate antimicrobial therapy. The effects of *H. pylori* eradication on PD symptoms have also been investigated. A study found significant improvements in motor function, with individuals who successfully eradicated *H. pylori* showing lower UPDRS Part III scores ($p = 0.007$), particularly in finger tapping ($p = 0.045$) and leg agility ($p = 0.011$). These findings suggest that *H. pylori* infection may impair Levodopa absorption or contribute to systemic inflammation, both of which could worsen PD symptoms.

6.8. No Significant Findings

Several studies have not found significant relationships between infections and PD. Upon examination of hospital-treated infections and sepsis, no significant relationship with α -synucleinopathies was found, including PD (OR: 1.05, 95% CI: 0.78–1.40). Similarly, another study observed higher HCV seropositivity in individuals with PD compared to controls, but the virus was not related to long-term PD risk. Other studies investigated parasitic infections and PD but did not find compelling evidence of a relationship. Khatir et al., 2025, reported no significant relationship between *Toxocara* infection and PD risk or severity, as seropositivity rates were nearly identical between PD and non-PD groups. Research into *H. pylori* yielded conflicting results. One study found that *H. pylori* eradication did not lead to significant improvements in PD symptoms, contradicting earlier findings that suggested a potential benefit [14]. This inconsistency highlights the need for further investigation into the role of *H. pylori* in PD pathology. Finally, Harris et al., 2012, found no relationship between rubella, HSV, or VZV and PD risk. However, they did identify a potential link between severe influenza and PD (OR: 2.01, 95% CI: 1.16–3.48), suggesting that only certain viral infections might contribute to neurodegenerative processes.

7. Discussion

7.1. Interpretation of Findings

This scoping review provides an overview of the relationship between infectious diseases, PD, and Parkinsonism. The findings highlight a complex and heterogeneous relationship between various pathogens and Parkinsonian syndromes. Several infections, including viral, bacterial, and parasitic pathogens, have been linked to an increased risk of developing PD or worsening existing Parkinsonian symptoms. The evidence suggests that chronic infections, systemic inflammation, and gut-brain axis disruptions may contribute to neurodegenerative processes, though inconsistencies remain across studies. While some infections appear to exacerbate motor symptoms, others may play a protective role or have no significant relationship with PD or Parkinsonism.

7.2. Pathogens in Neurodegeneration

The relationship between infections and neurodegeneration is not unique to PD. Similar patterns have been observed in other neurodegenerative disorders, such as Alzheimer's Disease (AD) and multiple sclerosis (MS), where chronic infections and systemic inflammation have been implicated in disease pathogenesis [48]. For example, *Chlamydia pneumoniae* (*C. pneumoniae*) and HSV have been linked to AD due to their ability to induce neuroinflammation and amyloid aggregation [49,50]. The hypothesis that infections act as environmental triggers for neurodegeneration aligns with the infectious theory of neurodegenerative diseases, which suggests that persistent infections can contribute to chronic immune activation and neuronal damage [51]. In PD, growing evidence supports the role of the gut-brain axis in disease pathogenesis [52]. The concept of gut-first versus brain-first PD has gained traction, with some researchers proposing that gut infections may serve as an initial trigger for α -synuclein misfolding and aggregation, which later spreads to the central nervous system (CNS) [53]. This aligns with Braak's hypothesis, which suggests that PD pathology may begin in the enteric nervous system (ENS) before reaching the substantia nigra via the vagus nerve [54]. Studies have shown that individuals with inflammatory bowel diseases, such as Crohn's disease and ulcerative colitis, have a higher risk of developing PD, further supporting the gut-brain connection in neurodegeneration [55].

7.3. Infections as Environmental Triggers for PD

The idea that infections serve as environmental triggers for PD is supported by epidemiological studies demonstrating that early-life infections, particularly respiratory and gastrointestinal infections, may increase long-term PD risk [56,57]. This is consistent with the broader field of neuroepidemiology, which has examined the role of prenatal and childhood infections in shaping neurodevelopmental trajectories and susceptibility to later neurodegenerative diseases [58]. The potential role of viral infections in triggering autoimmunity and molecular mimicry has also been explored in disorders such as Guillain-Barré syndrome and MS, raising questions about whether similar processes contribute to PD pathogenesis [59].

7.4. Neuroinflammation and Immunity in Parkinsonism

Chronic neuroinflammation has emerged as a central mechanism linking infections to PD. Microglial activation and prolonged immune responses have been implicated in neurodegeneration across multiple disorders [60]. Longitudinal studies have shown that elevated pro-inflammatory cytokines, such as tumor necrosis factor- α (TNF-) and interleukin-6 (IL-6), are related to an increased risk of PD [61]. These findings are consistent with studies on neurocognitive disorders related to the human immunodeficiency virus (HIV), where chronic viral infections induce sustained neuro inflammation, leading to

dopaminergic dysfunction and cognitive impairment [62]. The role of systemic infections in worsening PD symptoms aligns with existing literature on sickness behavior. This phenomenon occurs when infections trigger temporary cognitive and motor impairments [63]. For example, UTIs and pneumonia have been shown to cause transient declines in motor function in individuals with PD [64,65]. These findings support the idea that inflammation-driven symptom exacerbation is key to disease management.

7.5. Implications for Future Research and Clinical Practice

Given the evidence linking infections to PD risk and symptom exacerbation, future research should explore targeted infection prevention strategies as a potential means of mitigating PD onset and progression. Longitudinal cohort studies with detailed infection histories and biomarker analyses could help clarify causal relationships and identify high-risk populations. Additionally, the therapeutic potential of anti-inflammatory and antimicrobial treatments in PD warrants further investigation. Recent studies on the use of tetracyclines and gut microbiome-modulating therapies in PD suggest that targeting infection-related pathways could offer novel therapeutic options [66]. Clinically, physicians should consider infection screening and early intervention as part of PD management, particularly in individuals experiencing rapid symptom worsening. A multidisciplinary approach integrating neurology, infectious disease management, and microbiome research may provide a more comprehensive strategy for addressing infection-related PD risks.

8. Appeneix

Search Terms ("Parkinson's disease" OR "Parkinson disease" OR "Parkinsonism") AND ("infection" OR "infectious disease" OR "viral infection" OR "bacterial infection" OR "pathogen") AND ("neurodegeneration" OR "risk factor" OR "etiology" OR "association" OR "relationship" OR "correlation") AND ("cohort study" OR "case-control study" OR "cross-sectional study" OR "epidemiological study" OR "clinical study" OR "experimental study" OR "human study" OR "longitudinal study" OR "prospective study" OR "retrospective study")

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