Chapter 9
Neuroprotection in a Spice Jar: The Promise of Ginger for Parkinson's Disease

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ABSTRACT

Parkinson's disease (PD) is a progressive neurodegenerative disorder involving α-synuclein aggregation in the substantia nigra and the loss of dopaminergic neurons. Its pathophysiology involves oxidative stress, mitochondrial dysfunction, impaired neurotransmission, and neuroinflammation. The existing treatments, including levodopa, dopamine agonists, and monoamine oxidase B inhibitors, are symptomatic but fail to halt disease progression or side effects, and therefore, neuroprotective strategies are needed. Zingiber officinale (ginger) contains phenolic compounds (6-gingerol, 6-shogaol, zingerone, and paradol) with antioxidant, anti-inflammatory, and anti-apoptotic properties. Preclinical studies show the neuroprotection of ginger by

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scavenging reactive oxygen species, enhancing defenses, inhibiting mediators, and controlling signaling cascades. Although clinical trials in PD are lacking, cognitive improvement has been reported with minimal side effects. Overall, ginger's bioactives are promising adjuvant neuroprotectants that require rigorous clinical evaluation

1.0 INTRODUCTION

Parkinson's disease (PD) is the second most prevalent neurodegenerative disorder after Alzheimer's disease, affecting around 10 million individuals globally (Luo *et al*., 2025; Reddy *et al*., 2020). It is a neurodegenerative disorder that predominantly involves the central nervous system's motor control systems and is pathologically characterized by the loss of dopaminergic neurons within the substantia nigra pars compacta (SNpc) and intraneuronal Lewy bodies consisting of oligomerized α-synuclein (Gao *et al*., 2022; Tysnes and Storstein, 2017). Thus, these neuropathological changes cause a significant decrease in striatal dopamine content, which is accountable for the characteristic motor symptoms of PD (bradykinesia, resting tremor, muscular stiffness, and postural instability), as well as non-motor symptoms such as cognitive decline and autonomic dysfunction (Luo *et al*., 2025; Ball *et al*., 2019). Clinically, PD is diagnosed based on these typical motor features, dopaminergic response, and elimination of other neurological disorders because there is no diagnostic test. While diagnosis is typically reliable in advanced stages, early PD can be challenging to distinguish from other Parkinsonian syndromes since symptoms are overlapping (Pahwa and Lyons, 2010; Virameteekul *et al*., 2023).

PD comes in familial and sporadic forms, with age, genetics, environmental exposures, ethnicity, and gender determining onset and progression through multiple factors (Reddy *et al*., 2020). Amongst these, senility is particularly relevant, with PD affecting approximately 1% of the population above 60 years old (Tysnes and Storstein, 2017). Clinically, PD progresses through different stages, from unilateral motor signs to complete dependence and potential cognitive impairments. With worsening of the disease, the patients have increasing motor dysfunction, ranging from tremors and gait from mild to impaired balance, immobility, and hallucinations at more severe stages (DeMaagd and Philip, 2015). As a result of population shifts globally, social, economic, and healthcare costs of PD will rise dramatically and become an emerging burden for patients, caregivers, and healthcare systems (Luo *et al*., 2025; Bloem, 2019).

Despite decades of research, the definitive etiology of PD remains unclear. However, a number of interrelated molecular and cellular mechanisms have been found to be implicated in its pathology (Gao *et al*., 2022; Angelopoulou *et al*., 2022). These include oxidative stress, which results in dopaminergic neuronal