K : Parkinsons disease; Animal model

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Parkinsons disease (PD) is the second most common neurodegenerative disorder with the features of movement disorders and cognitive de cits. Over 1 2% of the general population over the age of 60 years is a ected and about 5 million peoples in total are interrelated all over the world [1]. e pivotal pathological characteristic of PD is the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNc), which results in the decrease of dopamine (DA) in striatum [2]. e accumulation of intracellular -synuclein is histopathologically observed in PD patients [3]. Recent studies suggest that the in ammatory response is involved in pathogenesis as well [4]. e DA de ciency causes the subsequently functional changes in the basal ganglia circuitry, which represent the externally parkinsonian motor dysfunction as well as cognitive disturbance [5,6]. Levodopa (L-DOPA) is considered as the current standard treatment for symptomatic Parkinsonism. e decreased DA content in the basal ganglia circuitry of PD leads to the logical use of L-DOPA for substrate supplement. Along with the concerns about the less e ectiveness and dose-dependent side e ect of the administration of L-DOPA, a number of studies are placed in exploring the e ective therapeutic strategies for PD patients [7,8].

Animal models which mimic the human Parkinsonism are required to enhance our understanding of the pathophysiology and optimize the treatment of PD. Rodents models are commonly used both for its experimental value and economic reasons. Neurotoxic agents are employed initially to destroy the DA neurons in SN, which induces the Parkinsonism in animal models. PD-mimetic toxins are frequently used, including 6-hydroxydopamine (6-OHDA), 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) and its active metabolite 1-methyl-phenylpyridinium (MPP+), pesticides rotenone, paraquat and pesticide manganese ethylene-bis-dithiocarbamate (Maneb) and in ammogen lipopolysaccharide (LPS) [9]. e toxic agents mimic partially the mechanisms of PD pathogenesis and lead to the loss of dopaminergic neuron, which eventually results in the motor dysfunctions as well as sensorial and memory de cits [10]. According to the application route, PD animal models are divided into unilateral and bilateral models, that is, the focal or the systemic administration of the neurotoxicity. e focal injection usually applies the stereotactic injection to introduce the neurotoxic agent into the unilateral side of the SN. is method results in hemiparkinsonism. e systemic models usually apply the subcutaneous (s.c) or intraperitoneal (i.p) administration to induce the bilateral Parkinsonism. In addition, the discoveries of gene de ciency in familial PD and the mutation of proteins/enzymes in PD patients provide the possibility to utilize the transgenic models in research of PD [11].

In this review, we examine the commonly used PD rodent models in past decades, including both unilateral and bilateral models. Each of the experimental models has own speci cities and limitations. erefore, Utilization of the PD rodent models should depend on the speci c aims for the variety of studies.

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