ARE ANIMAL MODELS USEFUL IN PARKINSON’S DISEASE?
Heinz Reichmann
Department of Neurology, University of Dresden, Dresden, Germany

We need animal models to investigate the aetiopathogenesis of PD, mimic the course of the disease to test regular medication or disease-modifying treatment. Thus, a good animal model should present with a gradual loss of dopaminergic neurons causing the typical motor signs in PD. It should lead to Lewy body pathology and also should be rapid enough to enable tests for new substances. The most often used model is the MPTP model which derived from the observation to some drug addicts made a mistake in synthesizing heroine and produced MPTP which lead rather rapidly to a PD syndrome. Other toxins which are often used are 6-hydroxydopamine and rotenone. The use of such toxins has several limitations such as a too rapid loss of dopaminergic neurons, missing Lewy body pathology and problems to predict similar results in the human. Genetic models seem to be rather more suited since they use knock-in or knock-out mice with the typical gene defects described in human (e.g. parkin, α-synuclein or LLRK2. Unfortunately, all these models not only don’t lead to typical Parkinsonian features in animals but are also lacking Lewy body pathology or even selective dopaminergic loss in the striato-nigral system in animals. In addition, these models lack to create the typical non-motor symptoms which become more and more important in PD. Thus, at present no model is perfect and the search for better models has to be continued.