Parkinson’s disease (PD) is the second most common neurological disorder worldwide, affecting approximately 6.3 million people. Age is an increasing risk for PD with an incidence of 1:1000 for people over 65 and 1:100 over 75 years. The average age of onset is 55-65 years of age [1]. There are currently approximately 70,000 Australians living with PD. With an aging population, this number is rising 4% annually and is expected to double by 2025. This will have huge socio-economic costs to our community.

PD is a progressive neurological condition characterised by both motor (movement) and non-motor symptoms. The four main symptoms are tremor, bradykinesia (stiffness of movement), muscle rigidity and postural instability. These symptoms are due to a deficit in the neurotransmitter dopamine resulting from the death of specific neurons within a brain region known as the substantia nigra. PD is incurable, with current drug therapies only alleviating symptoms. The main form of treatment is medication to restore the dopamine levels; either drugs that can cross the blood-brain barrier and be converted into dopamine by the brain (such as levodopa) or those that can stimulate the dopamine receptors of the brain (dopamine agonists). Long-term use of anti-Parkinsonian medication is associated with serious side effects and medication has a maximum benefit of 5 - 10 years. Deep Brain Stimulation (DBS) of the subthalamic nucleus region of the brain has now been successfully performed to support and reduce drug medication treatment for PD [2]. However, for future therapeutic intervention to be truly effective, the neuronal degeneration must be halted or at the very least slowed.

To identify new therapeutic targets for PD, current research must focus on understanding why the dopamine producing cells are dying. Similar to other neurological disorders, PD is considered a complex disorder caused by a combination of genetic (10% of all PD cases) and environmental factors (metals, industrial pollutants, herbicides and pesticides) [3]. Studies have suggested that many of these share common disease mechanisms [4]. Specifically, recent evidence from postmortem, epidemiological and animal studies has implicated neuroinflammation and oxidative stress in the disease progression [5]. Understanding their contribution to disease pathogenesis is crucial in the development of new therapies to slow the neuronal cell death and thus provide relief to PD sufferers.

References