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Review Article

The Epidemiology of Parkinson's Disease

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Abstract

Parkinson's disease (PD) is the second most common neurodegenerative disorder and manifests as bradykinesia, rigidity, resting tremor and posture instability. Although the disease symptomatology can be well controlled by levodopa, related medications and deep brain stimulation, the etiology of PD remains obscure. The epidemiological features have been discussed in depth in the literature, but the methodologies used to approach the issues have varied greatly, and the results cover a wide range of factors and are generally inconclusive. The crude prevalence rate of PD has been reported to range from 15 per 100,000 to 12,500 per 100,000, and the incidence of PD from 15 per 100,000 to 328 per 100,000, with the disease being less common in Asian countries. Risk factor studies have pinpointed cigarette smoking, coffee/tea consumption and alcohol drinking as being mostly related to a lower risk of PD. The relationship between a higher risk of PD and drinking well-water and being exposed to herbicides/pesticides is controversial. Systemic diseases including gout, hyperlipidemia and hypertension may be related to a reduced risk of PD. A family history of PD, tremor, depression and head injury are related to a higher risk of PD. Genetic studies of the glucocerebrosidase, parkin and LRRK2 genes have contributed to our understanding of familial PD but not of sporadic PD. The health-related quality of life of PD patients is related not only to their motor disability, but also to their non-motor symptoms of depression, sleep disturbance, bladder and sexual dysfunction. The economic burden of PD is enormous, and the annual cost of medical service per PD patient can reach €13,804 (NT$599,547). [*Tzu Chi Med J* 2010;22(2):73–81]

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1. Introduction

Parkinson's disease (PD) is the second most common neurodegenerative disease and manifests as bradykinesia, resting tremor, cogwheel rigidity and posture instability. The slowly progressive character of the disease means that development may last for 20 years. Although the motor symptoms of PD can be well controlled by levodopa and other adjunctive medications in the early stages of the disease, treatment-related complications will inevitably occur after 5–7 years. As the disease progresses, the cardinal motor symptoms of PD as well as cognitive decline, neuropsychological problems, autonomic failure and treatment-related

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complications associated with levodopa will enormously reduce the patient's activities of daily living (ADL) and health-related quality of life (HR-QoL) [1]. The World Health Organization has conducted numerous projects to raise awareness of the public health importance of PD since 1997. One of the completed projects is the Global Parkinson's Disease Survey (GPDS), a multinational survey across three continents (and including Canada, Italy, Japan, Spain, UK and USA), which showed that the QoL of PD patients was intimately associated not only with the severity of the disease and its treatment, but also with patients' satisfaction with the explanation of their diagnosis, their emotional state and the current level of their optimism about the disease. The burden and public health importance of neurological diseases such as PD has been underestimated [2]. The task of carrying out public health investigations into PD needs enthusiasm to carry on. In this context, multidisciplinary teamwork is able to improve the wellbeing of PD patients. In this review, we will focus our attention on the current understanding of the risk factors of PD, its comorbidities, the economic burden of PD, and the HR-QoL of PD patients.

2. Prevalence and incidence

Prevalence is defined as the total number of persons with a disorder within a given population at a fixed point in time. Incidence is defined as the number of new cases of a disorder diagnosed during a specific time period [3]. The crude prevalence rate of PD in European countries has been found to range from 65.6 per 100,000 to 12,500 per 100,000, and the incidence from 5 per 100,000 to 346 per 100,000 [3–12]. In Asian countries, the crude prevalence rates seem to be lower and range from 15 per 100,000 to 328 per 100,000 [6,13–16]. Interestingly, the wide ranges for the prevalence and incidence rates for PD from various research groups might be due to differences in their research methodologies; these include case finding protocols, diagnostic criteria and the age of the study population [5]. However, in this context, it seems likely that the ethnic difference may be attributed to different environmental exposure risks or interethnic differences in genetic susceptibility genes.

3. Risk factors

The unknown basis of the etiology of PD makes the disease incurable. It is now considered to be a multifactorial disease resulting from both environmental exposure to various factors and differences in genetic susceptibility. Multiple environmental factors that may be related to the etiology of PD include exposure to pesticides and herbicides, intake of various metals (copper, lead-copper, lead-iron, iron-copper), drinking well-water and exposure to a neurotoxin (1-methyl-1-4 phenyl-1,2,3,6-tetrahydropyridine), yet none of these has been identified as the sole causative agent of PD [17–21].

Although mutations in the parkin, LRRK2, and glucocerebrosidase genes are commonly found in multiethnic populations with familial early PD, such mutations are rare in sporadic early PD, which accounts for most patients with PD [22–24]. Thus, environmental factors may be more important than ethnicity and genetic factors in the etiology of PD [25].

In contrast to the high risk factors associated with PD, many epidemiological studies have shown that cigarette smoking is inversely associated with the occurrence of PD [19,20,26,27], even in a population characterized by a high prevalence of pesticide exposure [26], although pesticides or herbicides may not necessarily be associated with PD [19,21]. Coffee and tea drinking have also been suggested to be associated with a lower risk of PD [28]. Physical activities may also be an issue such that a higher level of activity may lower the risk of PD [29]. Also mentioned in one paper is the possibility that an increase in body mass index is positively associated with a higher risk of PD [30] (Tables 1–3 [19–24,26,28–38]).

4. Comorbidity

4.1. Neuropsychological events

PD as a neurodegenerative disease is characterized clinically by motor symptoms, which are related to dopamine deficiency; this occurs as a consequence of the degeneration of the substantia nigra pars compacta and has gained much attention in terms of treatment intervention.

It is estimated that neurologists overlook discussing crucial non-motor symptoms of PD (including depression, anxiety, fatigue, and sleep disturbance) with their patients more than 50% of the time; this estimate comes from a prospective study of 101 patients [39]. However, only 12% of the sample had no non-motor symptoms in a brief report describing 99 non-demented PD patients [40]. Indeed, associated non-motor comorbidity in PD patients is a significant source of disability and impaired QoL. The non-motor symptom complex of PD includes neuropsychiatric symptoms, sleep disorders, dysautonomia and sensory complaints [41]. These non-motor symptoms are usually correlated with advancing age and disease severity, while other non-motor symptoms such as olfactory dysfunction, REM sleep behavior disorder, depression and gastrointestinal symptoms can occur early in the disease and deteriorate in parallel with the motor symptoms [41–44].

Table 1 — Smoking, drinking, vitamins and the risk of Parkinson's disease

Risk factor	Risk	Study period	Study design	Study population	Country	Description	Reference
Cigarette smoking	Lower	1992–2001	Prospective	M=63,348; F=79,977 (no PD s/s at baseline)	U.S.	N=413 had definite or probable PD during follow-up period	31
1. Cigarette smoking 2. Coffee consumption 3. Alcohol consumption 4. High intake of vitamins A and C	Lower Higher	1981–1998	Prospective cohort case-controlled	N=13,979 (no PD s/s at baseline)	U.S.	No. of PD=395; control=2320; OR=0.42 (95% CI=0.22–0.80) for current smoker 1+pack/d; OR=0.71 (95% CI=0.52–0.95) for coffee drinker 2+cups/d; OR=0.77 (95% CI=0.58–1.03) for alcohol drinker 2+drinks/d	20
Coffee Tea	Lower Lower	1982–1987 1992–1997	Cross-sectional	N=29,335	Finland	HRs of PD associated with amount of coffee consumed daily (0, 1–4, and ≥5 cups) were 1.00, 0.53 and 0.40 (p for trend=0.005), HR=0.41 (≥3 cups of tea daily)	28
Cigarette smoking	Lower	1998–1999	Case-controlled	PD=247; control=676	France	In a population characterized by a high prevalence of pesticide exposure; inverse relationship between cigarette smoking and PD (OR=0.6) and also in patients with professional pesticide exposure (OR=0.5)	26
Cigarette smoking Coffee/tea	Lower —	1990–1995	Case-controlled retrospective	PD=190; control=190	Italy (Tuscany)	p=0.001, χ^2 test; p<0.0001 McNemar test No difference	19
Well-water use Smoking	Higher Lower	1998	Case-controlled	PD=136; control=272	Italy	OR=2.0; 95% CI=1.1–3.6; p=0.0308 OR=0.7; 95% CI=0.4–1.1; p<0.06	32
Well-water drinking	—	1990–1995	Case-controlled	PD=190; control=190	Italy (Tuscany)	No significant difference between case/control: OR=0.79–1.08 (well-water); OR=0.82–1.12 (herbicides/pesticides)	19
Cigarette smoking	Lower	1960–2004	8 case-controlled studies; cohort	Case=2328; control=4113; cohort: case=488; control=4880	U.S.	1. Inverse association between PD and smoker 2. Dose-dependent reduction *Pooled data from case-controlled studies, OR=0.53/0.76/0.70 (current smoker/ex-smoker/ever smoked)	33
Cigarette smoking	Lower	2002–2003	Case-controlled	PD=114; control=205	China (Beijing)	Reduced risk for PD among those who had ever smoked (OR=0.49), current smoker (OR=0.44), ex-smoker (OR=0.54)	34
Cigarette smoking Well-water drinking	Lower —	1994–1998	Case-controlled	PD=377; control=377	India (New Delhi)	Reduced risk for PD among smoker ≤20 yr (OR=0.19; p=0.000) ≤10 yr exposure, no significant effect (OR=0.065; p=0.49) >10 yr exposure, significant increased risk (OR=1.94; p=0.080)	35

M=male; F=female; PD=Parkinson's disease; s/s=symptom/sign; U.S.=United States of America; OR=odds ratio; CI=confidence interval; HR=hazard ratio; —=not related.

Table 2 — Systemic diseases and the risk of Parkinson's disease

Risk factor	Risk	Study period	Study design	Study population	Country	Description	Reference
Gout	Lower (in men only)	1995–2001	Prospective case-controlled	PD=1052; control=6634	U.S. (Minnesota)	Previous history of gout, lower risk of PD OR=0.69; 95% CI=0.48–0.99 In M, OR=0.60; 95% CI=0.40–0.9 In F, OR=1.26; 95% CI=0.57–2.81 (not reduced risk in women)	36
Initiation of medications	Lower					Initiation of anti-gout medicine, lower risk of PD: OR=0.57; 95% CI=0.19–1.70	
1. Hypertension 2. High cholesterol 3. Diabetes mellitus	Not associated	1976–2000	Prospective cohort	F=121,046 M=50,833 PD=530	U.S. (Boston)	Self-reported history RR=0.96; 95% CI=0.80–1.15 RR=0.98; 95% CI=0.82–1.19 RR=1.04; 95% CI=0.74–1.46	37
Increasing level of total cholesterol	Lower (modestly)			From NHS and HPFS		RR=0.86; 95% CI=0.78–0.95; p for trend=0.02 (use of cholesterol-lowering drugs was not associated with PD risk)	
Hypertension	Lower	1981–1998	Prospective cohort case-controlled	N=13,979 (no PD s/s at baseline)	U.S.	No. of PD=395; control=2320 (OR=0.62; 95% CI=0.48–0.80) for current users of antihypertensive drug	20
Increase in BMI	Higher	1972; 1977; 1982; 1987; 1992; 1997	Cohort cross-sectional	M=22,367; F=23,439 (no PD s/s at baseline)	Finland	HR of PD at different BMI (<23, 23–24.9, 25–26.9, 27–29.9 and ≥30 kg/m^2) in M=1.00, 1.97, 1.83, 2.34, 2.44; in F=1.00, 1.50, 1.65, 1.79, 1.77; in all=1.00, 1.70, 1.70, 2.02, 2.03	30

PD=Parkinson's disease; U.S.=United States of America; OR=odds ratio; CI=confidence interval; M=male; F=female; RR=relative risk; NHS=Nurses' Health Study; HPFS=Health Professionals Follow-up Study; s/s=symptom/sign; BMI=body mass index; HR=hazard ratio.

Neuropsychiatric and cognitive problems vary from anxiety, apathy and depression to dementia (45). In a Sydney multicenter study of PD with prospective and long-term (more than 15 years) follow-up, high rates of PD with dementia were reported (46). Another systematic review of prevalence studies of dementia in PD patients suggested that 24–31% of patients have dementia and that 3–4% of dementia in the whole population is due to PD with dementia (47). Multiple regression analyses in a large-scale, nationwide, cross-sectional, epidemiological study revealed that PD severity was the strongest predictor of dementia risk while other neuropsychiatric syndromes made only modest additional contributions (48).

Depression is among the most common non-motor features of PD along with cognitive impairment and autonomic dysfunction (49). The estimated prevalence of depression in PD patients is thought to vary from 10% to 45% depending on the criteria used (40,50,51). Irrespective of whether a neuropathological process or a reactive basis underlies depression in PD, the implication from both an epidemiological and neurobiological point of view is that this issue needs to be investigated further. Impaired cognitive function and the presence of thought disorders are significant predictors of major depression in PD patients (52). Impairment of serotoninergic neurotransmission as well as effects on limbic noradrenergic and dopaminergic mechanisms exist in depressed patients with PD (53). The severity of depression and impaired cognition accounted for 37% of the variance in disability using the Unified Parkinson's Disease Rating Scale ADL score (54). Thus, these psychiatric or non-motor symptoms contribute significantly to disability among PD patients over 15 years of follow-up (46).

The occurrences of anxiety, fatigue, sleep disturbance and sensory symptoms in PD patients were 33%, 40%, 47% and 63%, respectively, as reported by Shulman et al (40). The authors also demonstrated that as the number of non-motor symptoms increased, PD severity also increased. Bladder and sexual dysfunction in PD patients do not respond to dopaminergic treatment and may influence the QoL of PD patients despite that fact that they remain able to move well (55). Drooling is another non-motor problem that may cause significant difficulty in speaking (7.27% *vs.* 0%; $p<0.01$), eating (3.64% *vs.* 0%; $p=0.01$) and interacting socially (12.73% *vs.* 0%; $p<0.01$) as compared to PD non-droolers; this may cause a significant decline in QoL (56).

4.2. Systemic disease

Since PD is a neurodegenerative disease that occurs mostly in aged patients, it may be associated with other systemic diseases such as diabetes mellitus,

Table 3 — Environmental/genetic factors and the risk of Parkinson's disease

Environmental risk factor	Risk	Study period	Study design	Study population	Country	Description	Reference
Previous head injury Family history of PD Family history of tremor History of depression	Higher Higher Higher Higher	Cross-sectional	Case-controlled	N=140; control=147	U.S.	OR=6.23; CI=2.58–15.07 OR=6.08; CI=2.35–15.58 OR=3.97; CI=1.17–13.50 OR=3.01; CI=1.32–6.88	21
Higher levels of physical activity	Lower	1986–2000	Prospective cohort	M=48,574; F=77,254	U.S.	Greater baseline physical activity associated with a lower risk of PD In men: RR=0.7; p=0.007 In women: RR=0.5; p=0.005	29
Family history of PD Family history of ET Age of mother at patient's birth Exposure to general anesthesia Farmer	Higher Higher Higher Higher Higher	1998	Case-controlled	PD=136; control=272	Italy	OR=41.7; 95% CI=12.2–142.5; p<0.0001 OR=10.8; 95% CI=2.6–43.7; p<0.0001 OR=2.6; 95% CI=1.4–3.7; p=0.0013 OR=2.2; 95% CI=1.3–3.8; p=0.0024 OR=7.7; 95% CI=1.4–44.1; p=0.0212	32
Patient with ≥3 children	Higher	1981–1998	Prospective cohort case-controlled	N=13,979 (no PD s/s at baseline)	U.S.	No. of PD=395; control=2320 Risk increase with increased number of children (1.25 for 1, 1.34 for 2, 1.90 for ≥3; p for trend=0.0003)	20
Herbicide/pesticide exposure	—	1990–1995	Retrospective-control	PD=190; control=190	Italy (Tuscany)	No significant difference, OR=0.82–1.12 (herbicides/pesticides)	19
Residency in rural area	—	1990–1995	Case-controlled Retrospective-control	PD=190	Italy (Tuscany)	OR=0.80–1.05	19
Admixed population	Lower	Not mentioned	Cross-sectional Retrograde	N=493 (15 elderly homes in Bangalore)	India (Bangalore)	OR=3.9; 95% CI=1.3–12.9 (PD occurrence is 5 times higher among Indians compared to Anglo-Indians)	38
Pet exposure Male sex Family history of PD Prior depression history (≤10 yr)	Lower Higher Higher Higher	Not mentioned	Case-controlled	PD=377	India	>10 yr pet exposure, risk of PD reduced (OR=0.50; p=0.001) OR=1.98; p=0.001 OR=9.98; p=0.001 OR=1.54; p=0.024	35
Glucocerebrosidase gene	Higher		Case-controlled cross-sectional	PD=278; control=179	U.S.	OR=3.4; 95% CI=1.5–7.4 OR=2.7; 95% CI=1.3–5.3 No. of mutation: all PD case=38/278 (13.7%)	22
Parkin gene	—		Hospital-based case-controlled	PD=102 (familiar PD, n=20; sporadic PD, n=82; control, n=105)	India	PD=102; familiar PD: no. of mutation, N=2 PD=102; sporadic PD: no. of mutation, not found PD=102; control: no. of mutation, not found	24
LRRK2			Case-controlled	PD=800; control=212	India	LRRK2 may be a rare cause of PD among Indians	23

PD=Parkinson's disease; U.S.=United States of America; OR=odds ratio; CI=confidence interval; M=male; F=female; RR=relative risk; ET=essential tremor; s/s=symptom/sign; —=not related.

hypertension, coronary artery disease, cerebral vascular disease, spinal degenerative disease, orthopedic disorders and other neurodegenerative disorders. Accepting that the results are somewhat inconsistent due to methodological differences in the few case-controlled studies available, the prevalence of diabetes mellitus, hypertension, and coronary artery disease are similar between PD patients and the general population. Stroke is the exception and has a lower prevalence among PD patients [57]. Through a comprehensive understanding of the temporal relation and severity of these medical diseases in association with PD, we may be able to modulate the risk of PD to some degree and improve the QoL of patients [6] (Table 2 [20,30,36,37]).

5. Economic burden

According to the Ministry of the Interior of Taiwan, in the year 2009, the total population of Taiwan was 23.12 million, and the population aged above 65 years was 2.457 million, which is about 10% of the whole population. Since the crude prevalence rate of PD for those aged more than 60 years is 1%, in the year 2009, there would have been more than 20,000 persons who might have been affected by PD. PD is a slowly progressive disease, and the therapeutic period may last for more than 20 years. Treatment modalities become more complicated as the disease progresses and the direct costs of treatment will also increase significantly in proportion to the increase in Hoehn and Yahr stage [58]. A combination of different medical regimens, such as levodopa, dopamine agonists, COMT (catechol-O-methyl transferase) inhibitors, MAO (monoamine oxidase) inhibitors, amantadine and anticholinergics will be prescribed when the patient starts to experience symptoms of wearing off, on-off phenomenon, and treatment-related dyskinesia [59]. Although medications play a major role throughout all stages of the disease, the direct and indirect costs of the disease are enormous and it is inevitable that motor fluctuation, dyskinesia and neuropsychological problems will occur, which will result in the patient's QoL deteriorating [58,60–63]. In this situation, it is no wonder that more invasive interventions such as the apomorphine pump, continuous intestinal delivery of levodopa through a pumping device (DuoDopa; Solvay Pharmaceuticals GmbH, Rostock, Germany) and/or deep brain stimulation are needed as adjunct procedures to improve the patient's ADL [17]. To compare the different treatment modalities in terms of efficacy in order to obtain the PD patient's direct and indirect costs is thus very important [64,65].

Despite the high direct medical cost of PD noted in the literature, in Europe it has been found that the direct costs of the economic burden of PD are largely attributable to inhospital care and nursing home costs, and the cost of medications contributes only a small percentage to the total costs [58]. So, it is no wonder that Roland et al stated in their recent study that caregivers experience a far greater burden from the "mental stress" than from the "physical stress" due to the fact that they need to be continuously vigilant and tend to worry constantly about their spouse's safety, which to them is priceless [66]. From a thorough understanding of PD patients' distribution in society and their treatment course during hospital care, it should be possible for medical administrations to allocate health care costs in a more efficient way and slow the accelerating economic burden of PD (Table 4 [58,60–63]).

6. Health-related quality of life

Factors affecting the HR-QoL of PD patients include the following: treatment-related neurobehavioral changes, non-motor symptoms, motor complications, education, and surgical intervention. The motor symptoms are obvious and easy to detect, and the related research articles are straightforward and widely understood. In contrast, the underestimated non-motor symptoms, such as depression, are claimed to be the most important predictive factors of QoL among PD patients [67]. The ability to communicate and be mobile, as well as the associated HR-QoL of PD patients, can be improved through self-managed rehabilitation, which is positively related to rehabilitation hours [68]. In addition to the clinical setting, the direct and indirect economic burden of the disease are negatively associated with the QoL of both the PD patient and their caregiver [60]. Social support plays a more important role in the HR-QoL of PD patients compared to clinical parameters. This is especially true in Eastern countries, such as Russia. This result was demonstrated by the work of Winter et al, who concluded that social service support for patients with PD should be considered during the development of national health care programs in order to improve the HR-QoL of PD patients [69]. However, knowledge of the factors affecting the HR-QoL of PD patients and associated research remain quite limited [1]. Therefore, treatment strategies capable of improving QoL in PD in the future will rely on the identification of the factors that most influence the QoL of PD patients using large population-based studies of PD patients.

7. Conclusion

PD patients increase in number with age and are more prevalent in the aging populations of developed

Table 4 — Economic burdens of Parkinson's disease

Reference	Country	Study design	Inclusion criteria	Exclusion criteria	Study year and period	Cost	Description
63	U.K.	Community-based cohort and outside the community	U.K. PDSBB clinical diagnostic criteria for PD, N=176		2002/2003	Annual service cost=€13,804 (direct social care=€660, 5%; direct health care=€2056, 15%; informal care=€11,088, 80%)	Definition of economic cost in PD includes: 1. Direct health care cost 2. Other direct cost, e.g. home care workers 3. Informal care from family and friends 4. Productivity costs from lost employment due to illness
62	Germany	Prospective 6-mo observation	UK PDSBB criteria, N=145 (M=97, F=48)		Jan–Jun 2000	I. Total direct medical and non-medical cost=€3380±4230 1. Total direct medical cost=€1370±3240 (including: rehabilitation=€420±1630; hospitalization=€710±2520; outpatient treatment=€40±30; ancillary treatment=€190±280; ambulatory diagnostic procedures=€10±30; PD drug cost=€1520±1250) 2. Non-medical direct cost=€480±1710 (including: transportation=€10±20; special equipment=€420±1640; social/home-keep services=€10±110; sickness benefit=€40±540) II. Total indirect cost=€3180±6480	1. Cost=mean±SD 2. 6-mo period cost 3. Cost assessment via patient diary and questionnaire 4. Data from GKV
60	Finland	Hospital-based, consecutive, outpatient clinic	PD with all H&Y stages	Treatment with comtan	1999/12-mo period	Direct cost: mean, US$5500; median, US$2700 Total cost: a. All PD, mean=US$13,100; median=US$3900 b. PD without early retirement, mean=US$7600; median=US$3200	
61	Canada	Population-based case-controlled cohort, prospective	Administration database from the population of Ontario: PD=15,304; control=30,608		1993–1994; 1998–1999	PD=$1961.47; elderly PD drug cost/PYS, control=$661.08/yr; PD drugs=$1201/PYS	PD *vs.* Control 1. Physician cost=1.4 times 2. Hospital admission=1.44 times 3. Hospital duration=1.19 times long 4. Drug cost=3.0 times *Costs based on PYS
58	U.K.	Prospective	Samples from 36 regional health authorities: PD total=777; usable data=440	Presence of an alternative major diagnosis	1998	Direct cost: 1. All PD patients by age=€9554; N=432 H&Y stage I=€4736; N=110 H&Y stage II=€4886; N=89 H&Y stage III=€9857; N=120 H&Y stage IV=€16,155: N=87 H&Y stage V=€29,265; N=17	1. Direct cost=National Health Service cost 38% plus social services 34% (increase with age) 2. Drug cost=24% of total cost (<65 yr old); 10% (>85 yr old) 3. Move from home to residential care=500% cost increase 4. Increase H&Y staging, increase cost ($p<0.001$)

U.K.=United Kingdom; PDSBB=Parkinson's Disease Society Brain Bank; PD=Parkinson's disease; SD=standard deviation; GKV=Gesetzliche Krankenversicherung (Germany's statutory health insurance); H&Y=Hoehn and Yahr; $=Canadian dollar; PYS=person-years survivor.

countries. Several susceptible genes have been identified but these genetic factors are only important in a small number of patients. Among a variety of environmental factors, smoking seems to be the most important and is an inversely correlated risk factor for PD. The limited methodologies used and underrepresented comorbidities that accompany PD hamper the possibility of improving PD patients' QoL and thus hinder the amelioration of the economic burden of the disease. Although there have been a number of studies with strict methodology and long patient follow-up that have been conducted in Europe and America, good data from Asia is lacking. Studies across continents are needed in order to fully explore various areas such as risk factors, comorbidities and treatment trends, as well as to investigate the economic burden of PD. These studies are urgently needed in order to improve the QoL of PD patients.

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