Heliyon 12 (2026) e45499

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon

Research article

Association of serum vitamin D deficiency and inadequate sunlight exposure with Parkinson's disease risk: A pilot study in Bangladesh

Md. Rabiul Islam [a,*], Md Sahabuddin [a], Nusrat Hossain Sheba [b], Zenat Zebin Hossain [a]

[a] Department of Public Health, Independent University, Bangladesh
[b] Department of Public Health, Northern University, Bangladesh

ARTICLE INFO

Keywords:
Vitamin D
Parkinson's disease
Sunlight exposure
Deficiency
Inadequate
Bangladesh

ABSTRACT

A role for vitamin D deficiency and inadequate sunlight exposure in Parkinson's disease (PD) has been suggested. A cross-sectional study was conducted to estimate the association of vitamin D deficiency and inadequate sunlight exposure with PD. A total of 87 PD patients and 88 neurologically healthy general patients with normal liver and kidney functions were selected for the study using a systematic random sampling technique. Participants were recruited from a tertiary-level hospital in Dhaka, Bangladesh, following standard ethical guidelines and after obtaining informed consent. The neurologically healthy general patients group consisted of individuals with no history of neurological disorders, such as PD or Alzheimer's disease. Of vitamin D-deficient participants (plasma concentration <20 ng/mL), 66.7% had PD versus 33.3% who were neurologically healthy ($P < 0.01$); similarly, of participants with inadequate daily sunlight exposure, 63.4% had PD versus 36.6% who were neurologically healthy ($P < 0.01$). This study determined a significant association between vitamin D deficiency and PD (AOR 3.02, 95% CI 1.22, 7.48, $p = 0.017$). In addition, inadequate daily sunlight exposure was also significantly linked to PD status (AOR 2.31, 95% CI 1.16, 4.62, $p = 0.018$). The study concluded that low vitamin D levels and inadequate sunlight exposure may be related to PD. However, large-scale, multicenter longitudinal or experimental studies are needed to establish causal relationships.

1. Introduction

Parkinson's disease (PD) is a prevalent neurodegenerative disorder that becomes more common with advancing age [1]. The prevalence of PD has been reported between 51.3 and 176.9 per 100,000 in Asia [2]. According to the World Health Organization (WHO) data published in 2020, PD deaths in Bangladesh reached 3782 of the total deaths in the country. The age-adjusted death rate is 3.49 per 100,000 population, ranking Bangladesh number 125 in the world [3]. Modifiable risk factors such as vitamin D levels and sunlight exposure may be associated with PD and warrant further investigation. Vitamin D, synthesized through sunlight exposure, plays a vital neuroprotective function [4].

There are some other factors associated with PD, including exposure to pesticides, medications, smoking, alcohol consumption, and brain injuries [5]. Vitamin D exhibits antioxidant properties in the brain, and its deficiency may be associated with the onset of PD [6]. In addition, it has been implicated to be crucial in maintaining cognitive function in old age [7]. The role of vitamin D in modulating

* Corresponding author.
E-mail address: rabiulislamjuphi@gmail.com (Md.R. Islam).

https://doi.org/10.1016/j.heliyon.2026.e45499
Received 30 August 2024; Received in revised form 17 September 2026; Accepted 23 September 2026
Available online 25 September 2026

the production of neurotrophic factors, regulating calcium homeostasis, and protecting against excitotoxicity underscores its importance in maintaining neural health [8,9]. Furthermore, it has been shown to have protective effects against inflammation and oxidative stress, which are key mechanisms implicated in neurodegenerative diseases, notably PD [10]. However, a universal consensus has not yet been reached on the thresholds for vitamin D deficiency and the optimal level for physical and mental health [11].

Research indicates that serum vitamin D levels below 30 ng/mL may adversely affect cognitive performance and elevate the risk of neurodegenerative disorders, such as PD and Alzheimer's disease [12,13]. The threshold for vitamin D deficiency, often defined as a serum 25-hydroxyvitamin D [25(OH)D] level below 20 ng/mL, has been linked to adverse neurological outcomes [14]. The relationship between PD and vitamin D levels remains inconsistent in scientific literature. Several studies have suggested that lower vitamin D levels are linked to an increased risk of PD or more severe symptoms, potentially due to vitamin D's neuroprotective and anti-inflammatory properties [11,15,16]. Studies conducted in the United States of America, China, Finland, and France also reported an association between PD and reduced plasma vitamin D concentrations [17–21]. However, other research has failed to establish a clear causal relationship, raising questions about whether low vitamin D is a contributing factor or a consequence of PD, possibly due to reduced outdoor activity and sun exposure in individuals with PD [22]. These inconsistencies highlight the need for further study

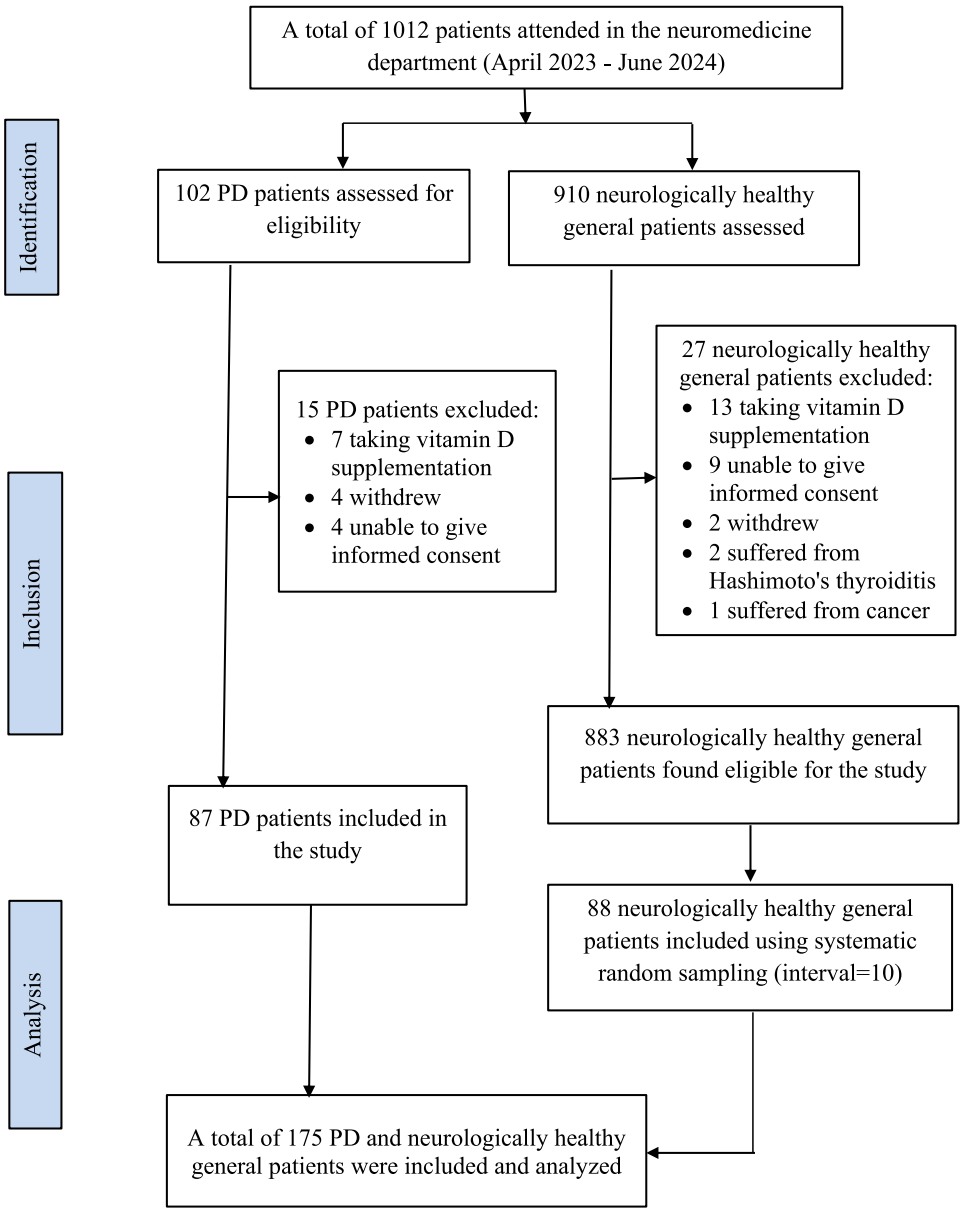

Fig. 1. STROBE flow diagram of participant selection for the study.

across diverse populations to better understand the direction and nature of this association.

Vitamin D obtained from sunlight or dietary sources is metabolized by 25-hydroxylase, predominantly in the liver, into 25-hydroxyvitamin D [25(OH)D], the principal form of vitamin D present in the bloodstream. It can serve as a serum marker for quantifying vitamin D levels in people with PD [23]. Several factors, such as latitude, seasonal changes, and local cultural practices, significantly influence sunlight exposure and vitamin D synthesis. Higher latitudes receive less ultraviolet B (UVB) radiation, especially during winter, limiting vitamin D production in the skin [24]. Seasonal variations also impact UVB availability, with lower levels during colder months reducing vitamin D synthesis [25]. Additionally, cultural practices such as clothing styles, outdoor duration, and nutrient intake affect vitamin D levels in the body [26]. These factors collectively contribute to global disparities in vitamin D status, emphasizing the need for conducting studies in different demographics. We found a dearth of research on the prevalence of PD and its associated risk factors in Southeast Asia, as well as in Bangladesh. To our knowledge, no study in Bangladesh has measured serum vitamin D levels and daily sunlight exposure among patients with PD. This study investigated the association of PD with vitamin D deficiency and daily sunlight exposure.

2. Methodology

2.1. Study design and study participant selection

A cross-sectional study was conducted among 175 patients (87 PD patients and 88 neurologically healthy general patients) who attended Bangladesh Specialized Hospital, a tertiary-level hospital in the capital of Bangladesh. A list of patients who attended the Department of Neuromedicine from April 2023 to June 2024 was recorded. A total of 88 neurologically healthy general patients were recruited in the study using a systematic random sampling technique with an interval of 10. We got a total of 910 neurologically healthy general patients; 27 did not meet our inclusion criteria, so we considered 883 eligible neurologically healthy general patients for the study. We found a scarcity of PD patients, and we got only 102 PD patients within this study period; 11 of them did not meet our inclusion criteria, and 4 withdrew from the study. Ultimately, we enrolled 87 PD patients in the study (Fig. 1). Participants were recruited on working days of the week, and samples were collected and analyzed in the hospital laboratory on the same day. Detailed medical and clinical histories were recorded on a pre-designed case record form.

2.2. Inclusion criteria, exclusion criteria

Adult patients who attended the Department of Neuromedicine of the hospital were included in the study. Patients with mental health problems, pregnancy, cancer, unconsciousness, taking vitamin D supplements, and known diseases like chronic kidney diseases or diseases that can influence vitamin D levels were excluded from the study. PD patients were confirmed by a doctor (neurologist) based on their clinical investigations and laboratory diagnosis. PD patients with diagnosed endocrine system diseases like hyperparathyroidism and other diseases of abnormal calcium and phosphorus metabolism, recent fractures or bone tumors ($\leq$6 months prior), known active ulcers, and active colitis were excluded from the study. The inclusion criterion for neurologically healthy general patients was the absence of any past or present diagnosis of neurological disease, such as PD or Alzheimer's disease. Before participant recruitment, we confirmed that participants had normal liver and kidney function. Following these inclusion and exclusion criteria, we got only 87 PD patients and 88 neurologically healthy general patients (Fig. 1).

2.3. Measurement of vitamin D level

Blood samples were taken from the forearm using an intravenous cannula, and immunoassay-based vitamin D quantification techniques were used to measure the 25(OH)D level, similar to the method described in Ref. [27]. The level of 25(OH)D was categorized as vitamin D deficiency [25(OH)D $<$ 20 ng/mL], insufficiency [25(OH)D = 20-29 ng/mL], and sufficiency [25(OH)D $\geq$ 30 ng/mL], respectively [15].

2.4. Measurement of daily sunlight exposure

Every participant's daily sunlight exposure was assessed during the data collection period at the hospital. A pretested, validated tool used by another study was used to measure participants' daily sun exposure [28]. Participants were exposed to sunlight for $>$1 h between 11.00 a.m. and 2.00 p.m. at least 3 days per week, and at least 18% of the body surface area without sunscreen was considered adequate; otherwise, it was inadequate [28].

2.5. Statistical analysis

The statistical programming language R was used for data analysis, and Microsoft Excel was used for data visualization. Both descriptive and inferential statistics were carried out in the study. Firstly, descriptive analyses, including mean with standard deviation (SD), frequency, and percentage, were performed for all variables. Secondly, Pearson's chi-square ($\chi 2$) test was used to measure the association between PD, vitamin D status, daily sunlight exposure, and other explanatory variables. Finally, binary logistic regression was used to estimate crude and adjusted odds ratios (AORs) to assess the association between PD and vitamin D levels and daily sunlight exposure among study participants. Potential confounding variables, including age, sex, smoking status, smokeless tobacco

use status, family history of PD, and income, have been adjusted in the regression analysis.

2.6. Ethics statement

Ethical approval for the study was obtained from the Institutional Review Board of Independent University, Bangladesh (Ref: IUB/IRB/Non-SR/2023/SPPH02). Informed consent was obtained prior to the data collection. Data were anonymous, and confidentiality was strictly maintained. Participants were informed of their full right to withdraw from the study or to refuse to answer any questions. No incentives were given to participate in the study. They were informed about the publication of the results in a scientific journal/conference. The study was conducted in accordance with the Declaration of Helsinki (2013 revision) issued by the World Medical Association.

3. Results

3.1. Background characteristics

Table 1 represents the background characteristics of the study participants. The mean age of the respondents was 63.18, and the majority (40.6%) of them were between 60 and 70 years old. Slightly more than half (52%) of them were female. Most of them (53.1%) were from the upper economic group, with family income above 80000 Bangladeshi taka (BDT) per month. Around 70% of respondents were from families with five or more members. Most respondents (60%) were nonsmokers. In addition, 97.1% respondents did not use any form of smokeless tobacco.

3.2. Daily sunlight exposure and clinical features of the respondents

This study showed that 59.4% of participants exposed themselves to sunlight for more than an hour between 11.00 a.m. and 2.00 p.m. at least three days per week, with at least 18% of their body surface area exposed without sunscreen (Table 2). Of the 87 PD patients and 88 neurologically healthy general patients, approximately one-fourth (24.6%) of the respondents had a family history of PD. The mean vitamin D level was 24.44 ng/mL, and most of the respondents had insufficient (20-29 ng/mL) levels of vitamin D. We found that only 29.1% of respondents had sufficient ($\geq$30 ng/mL) levels of vitamin D.

3.3. Distribution of background characteristics among neurologically healthy general patients and PD patients

Table 3 presents the distribution of background characteristics of the respondents according to their PD status. Of participants aged 70 years and above, 58.3% were neurologically healthy versus 41.7% who had PD; conversely, of those aged 50-60 years, 52.9% had PD versus 47.1% who were neurologically healthy. The study also found that among female participants, 52.7% had PD compared with

Table 1
Background characteristics of the study participants (n = 175).

Background characteristics	n	%
Age		
Mean ± SD	63.18 (±7.69)	
50-60	68	38.9
60-70	71	40.6
70 and above	36	20.5
Sex		
Male	84	48
Female	91	52
Monthly family income (Thousand BDT)		
Mean ± SD	97514.28 (±41549.97)	
Less than 70000	39	22.3
70000-80000	43	24.6
80000 and above	93	53.1
Occupation		
Job holder	59	33.7
Business	50	28.6
Housewife	66	37.7
Number of family members		
$\leq$ 4	51	29.1
$\geq$ 5	124	70.9
Smoking status		
No	105	60.0
Yes	70	40.0
Smokeless tobacco use		
No	170	97.1
Yes	5	2.9

Table 2
Overall distribution of sunlight exposure and clinical features of the study participants (n = 175).

Sunlight exposure and clinical features	n	%
Daily sunlight exposure		
Inadequate	71	40.6
Adequate	104	59.4
Having PD		
No	88	50.3
Yes	87	49.7
Family history of PD		
No	132	75.4
Yes	43	24.6
Vitamin D level (ng/mL)		
Mean ± SD	24.44(±9.37)	
Deficient < 20	54	30.9
Insufficient 20–29	70	40.0
Sufficient ≥ 30	51	29.1

Table 3
Distribution of background characteristics by neurologically healthy general patients (n = 88) and PD patients (n = 87).

Background characteristics	Neurologically healthy general patients (%)	PD patients (%)	P value
Age			
50-60	47.1	52.9	
60-70	49.3	50.7	0.530
70 and above	58.3	41.7	
Sex			
Male	53.6	46.4	
Female	47.3	52.7	0.401
Monthly family income (thousand BDT)			
Less than 70000 BDT	61.5	38.5	
70000-80000 BDT	48.8	51.2	0.270
80000 BDT and above	46.2	53.8	
Occupation			
Job holder	50.8	49.2	
Business	54.0	46.0	0.822
Housewife	50.0	50.0	
Smoking status			
No	49.5	50.5	
Yes	51.4	48.6	0.800
Smokeless tobacco use			
No	50.0	50.0	
Yes	60.0	40.0	0.653

Row percentages were calculated within each exposure category.

47.3% who were neurologically healthy; likewise, of participants with a monthly household income above 80000 BDT, 53.8% had PD versus 46.2% who were neurologically healthy. We found no significant association between PD and smoking or smokeless tobacco use among the respondents.

Table 4
Daily sunlight exposure and clinical features of the study participants by neurologically healthy general patients (n = 88) and PD patients (n = 87).

Daily sunlight exposure and clinical features	Neurologically healthy general patients (%)	PD patients (%)	P value
Daily sunlight exposure			
Inadequate	36.6	63.4	
Adequate	59.6	40.4	0.003
Family history of PD			
No	53.8	46.2	
Yes	39.5	60.5	0.104
Vitamin D level (ng/mL)			
Deficient < 20	33.3	66.7	
Insufficient 20–29	51.4	48.6	0.003
Sufficient ≥ 30	66.7	33.3	

Row percentages were calculated within each exposure category.

3.4. Distribution of clinical features by PD

The study found a significant difference between neurologically healthy general patients and PD patients in their daily sunlight exposure and vitamin D levels (Table 4). Of respondents with inadequate daily sunlight exposure, 63.4% had PD versus 36.6% who were neurologically healthy. Likewise, of participants with vitamin D deficiency, 66.7% had PD versus 33.3% who were neurologically healthy.

3.5. Association of PD with sunlight exposure and vitamin D level

After adjusting for age, sex, monthly household income, family history of PD, smoking, and smokeless tobacco use of the respondents in the binary logistic regression model, the study determined that daily sunlight exposure and vitamin D were associated with PD status of the participants (Fig. 2). The study found that inadequate daily sunlight exposure was significantly linked to PD (AOR 2.31, 95% CI 1.16, 4.62, p = 0.018). Additionally, vitamin D deficiency and PD status were positively correlated among the study participants (AOR 3.02, 95% CI 1.22, 7.48, p = 0.017). The study found no significant association between PD and the respondents' age. The AOR for 60-70 years old participants was 0.72 (95% CI 0.33, 1.56, p = 0.399), and for 70+ years, it was 0.39 (95% CI 0.15, 1.03, p = 0.057). Similarly, the study did not suggest any association of PD with sex (AOR 0.58, 95% CI 0.21, 1.59, p = 0.290), smoking (AOR 1.11, 95% CI 0.40, 3.06, p = 0.848), smokeless tobacco use (AOR 1.17, 95% CI 0.17, 10.08, p = 0.886), family history of PD (AOR 1.25, 95% CI 0.56, 2.82, p = 0.583) and monthly family income.

4. Discussion

This pilot study, conducted among 87 PD patients and 88 neurologically healthy general patients at a tertiary-level hospital in Bangladesh, provides compelling evidence that the chance of having PD is significantly associated with vitamin D deficiency and inadequate sunlight exposure, with odds of 3.02 and 2.31, respectively.

This study found that vitamin D deficiency and PD are positively correlated. To be more specific, individuals with a vitamin D level less than 20 ng/mL have approximately three times higher odds of having PD. This finding is supported by several previous studies, which have reported a significant association between reduced plasma vitamin D concentration and PD [17,18,29]. Another study by Wang et al. found a significant positive correlation of vitamin D deficiency and PD [30]. The prevalence of PD was found to be highest among subjects with the lowest quartile of 25(OH)D level, with an OR of 2.66 (95% CI 1.75, 4.03) compared to subjects with the highest quartile [30].

Similarly, another case-control study by Ming Xia and Qingjiu Zhou found a significantly higher prevalence of PD (OR 11.79, 95% CI 1.34, 10.35) among participants with the lowest plasma concentration of 25(OH)D [15]. In addition, our finding is consistent with the retrospective cross-sectional cohort study of Evatt et al., which reported that vitamin D level was lower among PD patients than the healthy controls [17]. Furthermore, the positive association between the probability of developing PD and lower serum vitamin D level was observed by the study of P. Knekt et al. [19]. Likewise, several other studies also found a significantly lower level of vitamin D among patients with PD, which corroborated the findings of this study [18,31–33].

The potential mechanism of the role of vitamin D deficiency and the chance of developing PD was examined in some studies. Previous studies on brain tissue showed that PD affects vitamin D receptors, which are widely distributed in the brain [34,35]. Thus,

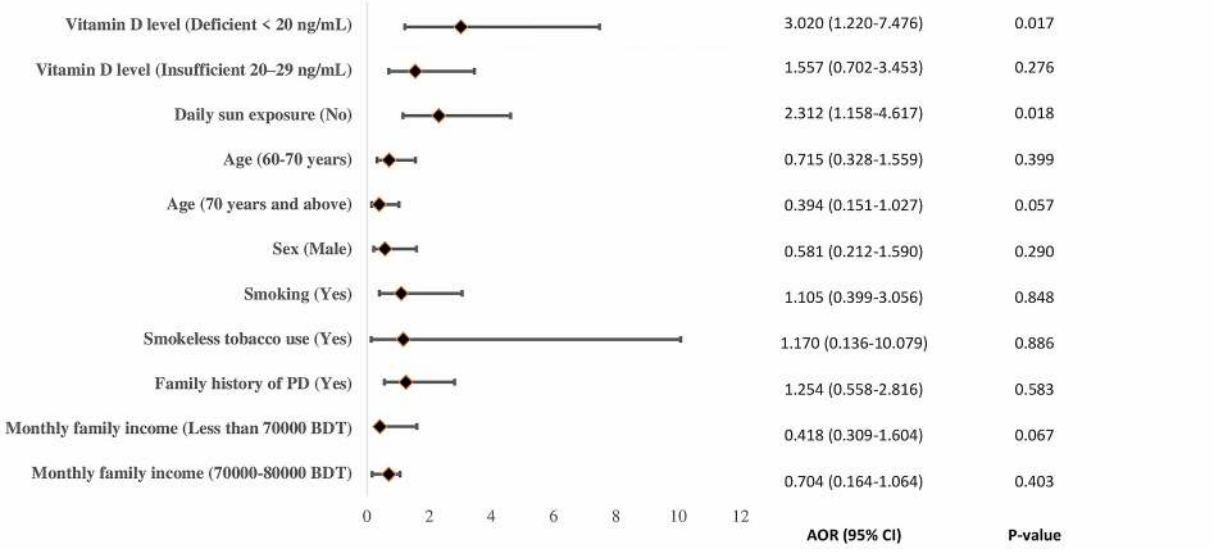

Fig. 2. Relationship between PD, sunlight exposure, and vitamin D levels.

PD plays a vital role in lowering the concentration of vitamin D in the body, suggesting reduced normal function of our brain.

The positive link between PD and inadequate sun exposure in this study has been supported by the study of Wang et al., in which they found a significant positive association between serum vitamin D level and sunlight exposure [36]. Lower levels of serum vitamin D and sunlight exposure are significantly associated with an increased risk of PD [15,36]. Vitamin D mainly comes from sunlight exposure [24]. Therefore, inadequate sunshine exposure may be related to vitamin D deficiency, which has been found to be significantly associated with PD status in this study, as well as several other studies mentioned above. Moreover, reduced sun exposure may exaggerate the problem by limiting the vitamin D mechanism in the body. However, sunlight exposure varies across different regions and races. Vitamin D production depends on both the intensity and duration of ultraviolet irradiation, which varies across different geographic regions [37].

On the other hand, this study did not find any significant association of PD with age, sex, monthly household income, family history of PD, smoking, or smokeless tobacco use status. This finding is supported by a study conducted at Isfahan Neurosciences Research Center in 2013, in which they reported that there are no sex differences in PD [38]. However, the study of Ming Xia and Qingjiu Zhou found an association between PD and age [15]. Their study was a case-control study, and moreover, their sample size and study setting were different from this study, which may be the possible explanation for such a difference. We did not find any significant association between PD and smoking and smokeless tobacco use. On the contrary, some studies found smokers have a substantially lower relative risk for PD than nonsmokers (pooled adjusted odds ratio of smokers vs. never-smokers, 0.2-0.5 [39–42]. In addition, the use of chewing tobacco has been reported to reduce PD risk, with hazard ratios of 0.4-0.5 [43,44]. One possible explanation is that nicotine may act as a stimulant to inhibit the action of the striatal dopamine transporter, increasing dopamine levels in the synaptic gap. This relationship of smoking with PD risk may be explained by the inverse modification of dopamine levels between PD and nicotine addiction [45]. Thus, these factors can be considered to further large-scale studies conducted in diverse settings.

The study examines the association of vitamin D deficiency and inadequate daily sunlight exposure with PD in a new geographic context, adding valuable insights to the existing body of research. This encourages region-specific longitudinal or experimental studies that could help understand how regional and cultural differences might influence the relationship between vitamin D levels, sunlight exposure, and PD. In addition, by highlighting vitamin D deficiency in a specific population with different geographic characteristics, this study can advocate region-specific strategies for the prevention and management of PD.

This study has some limitations. Firstly, the cross-sectional design of the study cannot establish a causal association of PD with vitamin D deficiency and inadequate sun exposure. It is difficult for the study to determine whether individuals with PD are more likely to have these deficiencies. However, the study found that PD is significantly associated with the level of vitamin D deficiency and inadequate sun exposure. Further longitudinal or experimental studies are needed to establish whether vitamin D deficiency plays a direct role in developing PD. Secondly, there is a small sample size. Due to the low incidence and prevalence of PD patients, we were unable to recruit a larger sample, limiting the study's ability to meet the required sample size for robust statistical power. This limitation should be considered in future research with a larger sample to enhance statistical reliability and generalizability. It limits the generalizability of the findings. In addition, we selected neurologically healthy general patients in 10 intervals to minimize the sampling bias. Further longitudinal or experimental studies with larger sample sizes may yield a stronger causal association between PD and vitamin D. Some key lifestyle and genetic factors beyond those listed in the study (e.g., dietary habits, physical activity) may also influence both vitamin D levels and PD risk. Future studies can consider these factors. Finally, sunlight duration fluctuates due to seasonal variation. Therefore, future studies should consider this confounder when predicting sunlight exposure as a risk factor for PD.

5. Conclusion

This cross-sectional pilot study provides evidence of an association between PD and vitamin D deficiency as well as inadequate sunlight exposure; however, it cannot establish causality. Further multicenter longitudinal or experimental studies with larger sample sizes considering dietary habits, physical activity, and cultural, seasonal, and regional variation in sunlight exposure may yield a stronger causal association between PD and vitamin D.

CRediT authorship contribution statement

Md. Rabiul Islam: Writing – review & editing, Writing – original draft, Visualization, Software, Project administration, Methodology, Formal analysis, Conceptualization. **Md Sahabuddin:** Writing – review & editing, Visualization, Project administration, Data curation. **Nusrat Hossain Sheba:** Writing – review & editing, Supervision, Methodology, Data curation. **Zenat Zebin Hossain:** Writing – review & editing, Validation, Project administration.

Ethics statement

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of Independent University, Bangladesh, with the approval number: IUB/IRB/Non-SR/2023/SPPH02, dated 27 March 2023. Informed consent was obtained prior to data collection.

Consent to participate and publication

All participants provided written informed consent to participate in the study and for their data to be published.

Data availability statement

Data will be made available on request. For requesting data, please write to the corresponding author.

Funding

This research did not receive any specific funding.

List of abbreviations

Abbreviation	Full Term
PD	Parkinson's disease
WHO	World Health Organization
25(OH)D	25-hydroxyvitamin D
UVB	Ultraviolet B
ng/mL	Nanograms per milliliter
SD	Standard Deviation
COR	Crude odds ratio
AOR	Adjusted odds ratio
CI	Confidence interval
BDT	Bangladeshi taka

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] F.R. Pérez-López, P. Chedraui, A.M. Fernández-Alonso, Vitamin D and aging: beyond calcium and bone metabolism, Maturitas 69 (2011) 27–36, https://doi.org/10.1016/j.maturitas.2011.02.014.
[2] W. Muangpaisan, H. Hori, C. Brayne, Systematic review of the prevalence and incidence of Parkinson's disease in Asia, J. Epidemiol. 19 (2009) 281–293, https://doi.org/10.2188/jea.JE20081034.
[3] Bangladesh: Parkinson's Disease, World life expect. https://www.worldlifeexpectancy.com/bangladesh-parkinson-disease, 2020. (Accessed 13 January 2025).
[4] Z. Zhou, R. Zhou, Z. Zhang, K. Li, The association between vitamin D status, vitamin D supplementation, sunlight exposure, and Parkinson's disease: a systematic review and meta-analysis, Med. Sci. Monit. Int. Med. J. Exp. Clin. Res. 25 (2019) 666–674, https://doi.org/10.12659/MSM.912840.
[5] A. Ascherio, M.A. Schwarzschild, The epidemiology of Parkinson's disease: risk factors and prevention, Lancet Neurol. 15 (2016) 1257–1272, https://doi.org/10.1016/S1474-4422(16)30230-7.
[6] M.J. Berridge, Vitamin D: a custodian of cell signalling stability in health and disease, Biochem. Soc. Trans. 43 (2015) 349–358, https://doi.org/10.1042/BST20140279.
[7] D.K. Simon, C.M. Tanner, P. Brundin, Parkinson disease epidemiology, pathology, genetics, and pathophysiology, Clin. Geriatr. Med. 36 (2020) 1–12, https://doi.org/10.1016/j.cger.2019.08.002.
[8] K. L Ng, L. Nguyễn, Role of vitamin d in Parkinson's disease, ISRN Neurol 2012 (2012) 134289, https://doi.org/10.5402/2012/134289.
[9] N.J. Groves, J.J. McGrath, T.H.J. Burne, Vitamin D as a neurosteroid affecting the developing and adult brain, Annu. Rev. Nutr. 34 (2014) 117–141, https://doi.org/10.1146/annurev-nutr-071813-105557.
[10] A. Mokhtari-Zaer, M. Hosseini, H. Salmani, Z. Arab, P. Zareian, Vitamin D3 attenuates lipopolysaccharide-induced cognitive impairment in rats by inhibiting inflammation and oxidative stress, Life Sci. 253 (2020) 117703, https://doi.org/10.1016/j.lfs.2020.117703.
[11] L. Lv, X. Tan, X. Peng, R. Bai, Q. Xiao, T. Zou, J. Tan, H. Zhang, C. Wang, The relationships of vitamin D, vitamin D receptor gene polymorphisms, and vitamin D supplementation with Parkinson's disease, Transl. Neurodegener. 9 (2020) 34, https://doi.org/10.1186/s40035-020-00213-2.
[12] T.J. Littlejohns, W.E. Henley, I.A. Lang, C. Annweiler, O. Beauchet, P.H.M. Chaves, L. Fried, B.R. Kestenbaum, L.H. Kuller, K.M. Langa, O.L. Lopez, K. Kos, M. Soni, D.J. Llewellyn, Vitamin D and the risk of dementia and Alzheimer disease, Neurology 83 (2014) 920–928, https://doi.org/10.1212/WNL.0000000000000755.
[13] C. Annweiler, A.M. Schott, G. Allali, S.A. Bridenbaugh, R.W. Kressig, P. Allain, F.R. Herrmann, O. Beauchet, Association of vitamin D deficiency with cognitive impairment in older women: cross-sectional study, Neurology 74 (2010) 27–32, https://doi.org/10.1212/WNL.0b013e3181beecd3.
[14] M.F. Holick, N.C. Binkley, H.A. Bischoff-Ferrari, C.M. Gordon, D.A. Hanley, R.P. Heaney, M.H. Murad, C.M. Weaver, Endocrine society, evaluation, treatment, and prevention of vitamin D deficiency: an endocrine society clinical practice guideline, J. Clin. Endocrinol. Metab. 96 (2011) 1911–1930, https://doi.org/10.1210/jc.2011-0385.
[15] M. Xia, Q. Zhou, Correlation between 25-hydroxy-vitamin D and Parkinson's disease, IBRO Neurosci. Rep. 16 (2024) 162–167, https://doi.org/10.1016/j.ibneur.2023.02.006.
[16] X. Luo, R. Ou, R. Dutta, Y. Tian, H. Xiong, H. Shang, Association between serum vitamin D levels and Parkinson's disease: a systematic review and meta-analysis, Front. Neurol. 9 (2018) 909, https://doi.org/10.3389/fneur.2018.00909.
[17] M.L. Evatt, M.R. Delong, N. Khazai, A. Rosen, S. Triche, V. Tangpricha, Prevalence of vitamin d insufficiency in patients with Parkinson disease and Alzheimer disease, Arch. Neurol. 65 (2008) 1348–1352, https://doi.org/10.1001/archneur.65.10.1348.
[18] H. Ding, K. Dhima, K.C. Lockhart, J.J. Locascio, A.N. Hoesing, K. Duong, A. Trisini-Lipsanopoulos, M.T. Hayes, U.S. Sohur, A.-M. Wills, B. Mollenhauer, A. W. Flaherty, A.Y. Hung, N. Mejia, V. Khurana, S.N. Gomperts, D.J. Selkoe, M.A. Schwarzschild, M.G. Schlossmacher, B.T. Hyman, L.R. Sudarsky, J.H. Growdon, C.R. Scherzer, Unrecognized vitamin D3 deficiency is common in Parkinson disease: Harvard biomarker study, Neurology 81 (2013) 1531–1537, https://doi.org/10.1212/WNL.0b013e3182a95818.
[19] P. Knekt, A. Kilkkinen, H. Rissanen, J. Marniemi, K. Sääksjärvi, M. Heliövaara, Serum vitamin D and the risk of Parkinson disease, Arch. Neurol. 67 (2010) 808–811, https://doi.org/10.1001/archneurol.2010.120.
[20] M.E. Fullard, J.E. Duda, A review of the relationship between vitamin D and parkinson disease symptoms, Front. Neurol. 11 (2020) 454, https://doi.org/10.3389/fneur.2020.00454.

[21] C. Annweiler, A.M. Schott, G. Berrut, V. Chauviré, D. Le Gall, M. Inzitari, O. Beauchet, Vitamin D and ageing: neurological issues, Neuropsychobiology 62 (2010) 139–150, https://doi.org/10.1159/000318570.
[22] M.E. Fullard, S.X. Xie, K. Marek, M. Stern, D. Jennings, A. Siderowf, A.W. Willis, A.S. Chen-Plotkin, Vitamin D in the Parkinson associated risk syndrome (PARS) study, Mov. Disord. Off. J. Mov. Disord. Soc. 32 (2017) 1636–1640, https://doi.org/10.1002/mds.27127.
[23] K. Yang, J. Chen, X. Li, Y. Zhou, Vitamin D concentration and risk of Alzheimer disease: a meta-analysis of prospective cohort studies, Medicine (Baltim.) 98 (2019) e16804, https://doi.org/10.1097/MD.0000000000016804.
[24] M.F. Holick, Vitamin D deficiency, N. Engl. J. Med. 357 (2007) 266–281, https://doi.org/10.1056/NEJMra070553.
[25] L. King, K. Dear, S.L. Harrison, I. van der Mei, A.M. Brodie, M.G. Kimlin, R.M. Lucas, Investigating the patterns and determinants of seasonal variation in vitamin D status in Australian adults: the Seasonal D cohort study, BMC Public Health 16 (2016) 892, https://doi.org/10.1186/s12889-016-3582-z.
[26] M. Pourhassan, R. Wirth, Seasonal variation in vitamin D status among frail older hospitalized patients, J. Frailty Aging 7 (2018) 95–99, https://doi.org/10.14283/jfa.2018.10.
[27] Z. Maunsell, D.J. Wright, S.J. Rainbow, Routine isotope-dilution liquid chromatography-tandem mass spectrometry assay for simultaneous measurement of the 25-hydroxy metabolites of vitamins D2 and D3, Clin. Chem. 51 (2005) 1683–1690, https://doi.org/10.1373/clinchem.2005.052936.
[28] V. Patwardhan, Z. Mughal, S. Chiplonkar, A. Webb, R. Kift, V. Khadilkar, R. Padidela, A. Khadilkar, Duration of casual sunlight exposure necessary for adequate vitamin D status in Indian men, Indian J. Endocrinol. Metab 22 (2018) 249, https://doi.org/10.4103/ijem.IJEM_473_17.
[29] M. Suzuki, M. Yoshioka, M. Hashimoto, M. Murakami, K. Kawasaki, M. Noya, D. Takahashi, M. Urashima, 25-hydroxyvitamin D, vitamin D receptor gene polymorphisms, and severity of Parkinson's disease, Mov. Disord. Off. J. Mov. Disord. Soc. 27 (2012) 264–271, https://doi.org/10.1002/mds.24016.
[30] L. Wang, M.L. Evatt, L.G. Maldonado, W.R. Perry, J.C. Ritchie, G.W. Beecham, E.R. Martin, J.L. Haines, M.A. Pericak-Vance, J.M. Vance, W.K. Scott, Vitamin D from different sources is inversely associated with Parkinson disease, Mov. Disord. Off. J. Mov. Disord. Soc. 30 (2015) 560–566, https://doi.org/10.1002/mds.26117.
[31] S. Shrestha, P.L. Lutsey, A. Alonso, X. Huang, T.H. Mosley, H. Chen, Serum 25-hydroxyvitamin D concentrations in mid-adulthood and Parkinson's disease risk, Mov. Disord. Off. J. Mov. Disord. Soc. 31 (2016) 972–978, https://doi.org/10.1002/mds.26573.
[32] I. Sleeman, T. Aspray, R. Lawson, S. Coleman, G. Duncan, T.K. Khoo, I. Schoenmakers, L. Rochester, D. Burn, A. Yarnall, The role of vitamin D in disease progression in early Parkinson's disease, J. Park. Dis 7 (2017) 669–675, https://doi.org/10.3233/JPD-171122.
[33] M. Moghaddasi, M. Mamarabadi, M. Aghaii, Serum 25-hydroxyvitamin D3 concentration in Iranian patients with Parkinson's disease, Iran, J. Neurol. 12 (2013) 56–59. https://pubmed.ncbi.nlm.nih.gov/24250903/.
[34] A. Pignolo, S. Mastrilli, C. Davì, V. Arnao, P. Aridon, F.A. Dos Santos Mendes, C. Gagliardo, M. D'Amelio, Vitamin D and Parkinson's disease, Nutrients 14 (2022) 1220, https://doi.org/10.3390/nu14061220.
[35] I. Ramasamy, Vitamin D metabolism and guidelines for vitamin D supplementation, Clin. Biochem. Rev. 41 (2020) 103–126, https://doi.org/10.33176/AACB-20-00006.
[36] J. Wang, D. Yang, Y. Yu, G. Shao, Q. Wang, Vitamin D and sunlight exposure in newly-diagnosed Parkinson's disease, Nutrients 8 (2016) 142, https://doi.org/10.3390/nu8030142.
[37] M.K.B. Bogh, A.V. Schmedes, P.A. Philipsen, E. Thieden, H.C. Wulf, Vitamin D production depends on ultraviolet-B dose but not on dose rate: a randomized controlled trial, Exp. Dermatol. 20 (2011) 14–18, https://doi.org/10.1111/j.1600-0625.2010.01201.x.
[38] R. Meamar, M. Maracy, A. Chitsaz, M.R.A. Ghazvini, M. Izadi, A.P. Tanhaei, Association between serum biochemical levels, related to bone metabolism and Parkinson's disease, J. Res. Med. Sci. Off. J. Isfahan Univ. Med. Sci. 18 (2013) S39–S42. https://pmc.ncbi.nlm.nih.gov/articles/PMC3743317/.
[39] M.A. Hernán, B. Takkouche, F. Caamaño-Isorna, J.J. Gestal-Otero, A meta-analysis of coffee drinking, cigarette smoking, and the risk of Parkinson's disease, Ann. Neurol. 52 (2002) 276–284, https://doi.org/10.1002/ana.10277.
[40] B. Ritz, A. Ascherio, H. Checkoway, K.S. Marder, L.M. Nelson, W.A. Rocca, G.W. Ross, D. Strickland, S.K. Van Den Eeden, J. Gorell, Pooled analysis of tobacco use and risk of Parkinson disease, Arch. Neurol. 64 (2007) 990–997, https://doi.org/10.1001/archneur.64.7.990.
[41] V. Gallo, P. Vineis, M. Cancellieri, P. Chiodini, R.A. Barker, C. Brayne, N. Pearce, R. Vermeulen, S. Panico, B. Bueno-de-Mesquita, N. Vanacore, L. Forsgren, S. Ramat, E. Ardanaz, L. Arriola, J. Peterson, O. Hansson, D. Gavrila, C. Sacerdote, S. Sieri, T. Kühn, V.A. Katzke, Y.T. van der Schouw, A. Kyrozis, G. Masala, A. Mattiello, R. Perneczky, L. Middleton, R. Saracci, E. Riboli, Exploring causality of the association between smoking and Parkinson's disease, Int. J. Epidemiol. 48 (2019) 912–925, https://doi.org/10.1093/ije/dyy230.
[42] W.K. Scott, F. Zhang, J.M. Stajich, B.L. Scott, M.A. Stacy, J.M. Vance, Family-based case-control study of cigarette smoking and Parkinson disease, Neurology 64 (2005) 442–447, https://doi.org/10.1212/01.WNL.0000150905.93241.B2.
[43] Z. Liu, A. Roosaar, T. Axéll, W. Ye, Tobacco use, oral health, and risk of Parkinson's disease, Am. J. Epidemiol. 185 (2017) 538–545, https://doi.org/10.1093/aje/kww146.
[44] F. Yang, N.L. Pedersen, W. Ye, Z. Liu, M. Norberg, L. Forsgren, Y. Trolle Lagerros, R. Bellocco, L. Alfredsson, A. Knutsson, J.-H. Jansson, P. Wennberg, M. R. Galanti, A.C.J. Lager, M. Araghi, M. Lundberg, C. Magnusson, K. Wirdefeldt, Moist smokeless tobacco (Snus) use and risk of Parkinson's disease, Int. J. Epidemiol. 46 (2017) 872–880, https://doi.org/10.1093/ije/dyw294.
[45] C. Wang, C. Zhou, T. Guo, P. Huang, X. Xu, M. Zhang, Association between cigarette smoking and Parkinson's disease: a neuroimaging study, Ther. Adv. Neurol. Disord. 15 (2022) 17562864221092566, https://doi.org/10.1177/17562864221092566.