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Research Article

Association of *EIF4G1* Gene Variants with Sporadic Parkinson's disease in a Chinese Han Population

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Abstract

Eukaryotic translation initiation factor 4G1 (*eIF4G1*) has been implicated in Parkinson's disease (PD) pathogenesis. However, the contribution of *EIF4G1* genetic variation to PD susceptibility remains unclear. To investigate the association between the *EIF4G1* variant rs2178403 and PD risk. We analyzed *EIF4G1* expression in PD and control samples using public GEO datasets (GSE54536). Additionally, we conducted a hospital-based case-control study with 541 sporadic PD patients and 401 age-/sex-matched healthy controls of Han Chinese ancestry. Genotyping of rs2178403 was performed using Sequenom MassARRAY iPLEX. GEO data revealed a non-significant trend toward elevated *EIF4G1* expression in PD samples ($p < 0.1$). Genetic analysis identified a significant association between the rs2178403 GG genotype and increased PD risk under a recessive model (OR = 1.31, 95% CI = 1.010–1.703, $p = 0.042$). Stratified analysis showed a stronger effect in females. These findings suggest rs2178403 may contribute to PD susceptibility in the Han Chinese population. This study supports an association between the *EIF4G1* variant rs2178403 and PD risk. Further investigation into *EIF4G1* inhibition as a potential therapeutic strategy for PD is warranted.

More Information

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Keywords: Eukaryotic translation initiation factor 4G1(*EIF4G1*); Parkinson's disease; rs2178403; mTOR signaling pathway; α-synuclein aggregation; Proteostasis dysregulation

Introduction

Parkinson's disease (PD) is the second most prevalent neurodegenerative disorder, affecting approximately 1% of individuals over 60 years and 4% - 5% of those over 85 [1,2]. PD is pathologically characterized by the progressive loss of dopaminergic neurons in the substantia nigra and the presence of Lewy bodies containing aggregated α-synuclein [3,4]. Clinically, PD presents with motor symptoms such as tremors, rigidity, bradykinesia, and postural instability, alongside non-motor symptoms like depression, sleep disturbances, and cognitive decline [5]. The etiology of PD is multifactorial, involving a complex interplay of genetic predispositions, environmental exposures, and aging-related cellular changes [6,7].

Recent genetic studies have identified both familial and sporadic forms of Parkinson's disease (PD), with numerous risk loci implicated through genome-wide association studies (GWAS) [8]. Among molecular pathways linked to PD pathogenesis, the mammalian target of rapamycin (mTOR) signaling cascade has garnered significant attention due to its central role in regulating autophagy, protein synthesis, and cellular metabolism [9,10]. mTOR functions through two multiprotein complexes, mTORC1 and mTORC2, with well-characterized upstream regulators and downstream effectors [11].

A key mTORC1 substrate is the eukaryotic translation initiation factor 4E-binding protein family (EIF4E-BP1/2/3) [12]. When hypophosphorylated, EIF4E-BPs sequester eIF4E and prevent assembly of the eIF4F complex, thereby suppressing cap-dependent translation initiation. The scaffolding protein *eIF4G1* is essential for eIF4F complex formation, which includes eIF4E and eIF4A [13]. The eukaryotic translation initiation factor 4G1 (*eIF4G1*) is a scaffolding protein essential for assembling the eIF4F complex, which includes eIF4E and eIF4A [14,15]. Under normal physiological conditions, *eIF4G1* interacts with eIF4E to recruit ribosomes to the 5'-cap of mRNAs, thus initiating protein synthesis [16,17]. However, alterations in *eIF4G1* levels or function can disturb proteostasis and have been associated with various neurodegenerative diseases [18]. Dysregulation of *eIF4G1* expression or function disrupts proteostasis and is implicated in neurodegenerative disorders. In PD, impaired mTORC1-eIF4F signaling contributes to the accumulation of misfolded α-synuclein by compromising both its clearance and the translation of proteostatic machinery [19].

Although *EIF4G1* mutations have been reported in familial PD, primarily in European cohorts, their role in sporadic PD across diverse ethnic populations remains underexplored [20]. To address this gap, we investigated the association between the *EIF4G1* variant rs2178403 and PD risk in a Han Chinese population. Complementarily, we analyzed *eIF4G1* expression patterns in public transcriptomic datasets to assess functional relevance. This integrated genetic-transcriptomic approach aims to clarify the contribution of *EIF4G1* to PD susceptibility and provide a mechanistic foundation for future therapeutic targeting.

Methods

Gene expression analysis

Gene expression data from Parkinson's disease (PD) patients and healthy controls were retrieved from the Gene Expression Omnibus (GEO) database (dataset GSE54536, designated as the training cohort). Following standardization of all samples, differential expression analysis was performed using thresholds of $|\log_2 \text{fold change}| > 1.5$ and false discovery rate (FDR) < 0.1.

Study population

Our case–control study recruited a total of 942 Han Chinese subjects, including 541 sporadic PD patients and 401 healthy subjects matched by age, sex, and ethnicity. Participants were recruited from the Parkinson Clinic Center of the First Affiliated Hospital of Sun Yat-sen University from January 2014 to June 2016. Parkinson's disease was diagnosed in accordance with the UK PD Society Brain Bank clinical diagnostic criteria [21]. The study was approved by the Ethics Committee of the First Affiliated Hospital of Sun Yat-Sen University, and all study subjects provided their informed written consent.

DNA isolation and single-nucleotide polymorphism genotyping

DNA extraction used the standard phenol-chloroform method [22]. Rs2178403 genotyping employed Sequenom MassARRAY iPLEX (San Diego, CA).

Statistical analysis

Our data comes from Gene Expression Omnibus (GEO) datasets (GSE54536). A volcano plot was used to identify differentially expressed genes (using n-fold ≥1.5 and *p - value* <0.1 as the threshold of statistical significance).

The haplotype block was analyzed with Haploview and used Beagle 4.1 for further haplotype analysis. For all other statistical analyses, we used SPSS, version 27.0 (IBM Corp., Armonk, NY, USA) [23]. Genotypes and allele frequencies were determined by direct counting. Sex and age differences between patients and controls were examined using Student's t-test. The Hardy–Weinberg equilibrium (HWE) of the genotype distributions across patients and controls was calculated using a chi-squared test. To test for differences in genotype and allele distributions between patients and controls, we used chi-squared tests or Fisher's exact tests. The odds ratio (OR) and the 95% confidence interval (CI) were used to describe the association between SNPs and the PD risk using logistic regression adjusted for age and sex. $p < 0.05$ (two-tailed) was considered significant.

Results

eIF4G1 showed a more significant increase in the PD group than control

We screened 874 differentially expressed genes (DEGs) between the PD group and the control group in GSE54536 using the R package "limma" ($p < 0.1$). The analysis data show that *eIF4G1* has a higher increase in the PD group than control. The green arrow in group 1 points to the position of *eIF4G1.*

Characteristics of the study population

Our case–control study enrolled a total of 942 Han Chinese participants. The PD group included 541 subjects (320 men and 221 women). The control group included 401 age-, sex-, and ethnicity-matched subjects (229 males and 172 females). The details are outlined in Table 1.

Distributions of the rs2178403 polymorphism in PD and control groups

The genotype distribution of rs2178403 in both the PD and healthy control groups was determined to identify any

Table 1: Demographic characteristics.

	PD group (n = 541)	Control group (n = 401)	p
Sex (male/female)	320/221	229/172	0.530
Age (mean ± SD)	63.36 ± 10.74	62.88 ± 10.13	0.938

deviation from HWE. Genotype and allele frequencies for rs2178403 are summarized in Table 2.

rs2178403 polymorphism and PD susceptibility

For rs2178403, GG genotype frequency in the PD group was significantly higher than in the control group, when compared by logistic regression analysis using recessive model and allelic models(AA +AG versus GG; OR =1.31, 95% CI = 1.010–1.703, p = 0.042; G versus A, OR =1.263, 95% CI = 1.041–1.532, p = 0.018). By contrast, no significant difference was identified using either the dominant (AA versus AG+GG, OR =1.46, 95% CI = 0.969–2.209, p = 0.069) (Table 3).

Next, we compared both groups by subgroup analysis based on sex. We found that female patients with the recessive model and allelic models were associated with an increased risk of developing PD compared to female controls (AA +AG versus GG; OR = 1.84, 95% CI = 1.23–2.78, p = 0.003; G versus A, OR = 1.50, 95% CI = 1.11–2.02, p = 0.008). In addition, male patients with the allelic models were associated with an increased risk of developing PD compared to male controls (G versus A, OR = 1.45, 95% CI = 1.11–1.89, p = 0.005). Furthermore, the dominant models detected no difference in the analysis (Figure 1). The results of different comparisons are presented in Table 3.

Discussion

EIF4G1 was confirmed as a candidate PD gene by Chartier-Harlin, et al. [24] This case-control study investigated the association between the *EIF4G1* variant rs2178403 and PD susceptibility in a Han Chinese cohort (541 patients and 401 controls). We concurrently analyzed *EIF4G1* expression patterns in public datasets to assess functional relevance. Our results support growing evidence implicating dysregulated mRNA translation initiation via the mTORC1-eIF4F axis in PD pathogenesis.

Progressive dopaminergic neuron loss and α-synuclein aggregation are core hallmarks of Parkinson's disease (PD), which reflect underlying proteostasis failure [4]. Parkinson's disease is considered to have multiple causes, resulting from both genetic and non-genetic factors. Genetic variants with large effect sizes have been identified in approximately 20% of persons with Parkinson's disease (monogenic Parkinson's disease). Autosomal dominant Parkinson's disease with incomplete penetrance includes variants in LRRK2, GBA1, VPS35, and SNCA [25,26]. mTORC1 signaling coordinates key cellular processes, including metabolism, autophagy, and cap-dependent translation through effectors such as EIF4E-BPs and the eIF4F complex [27]. Within eIF4F, *eIF4G1* acts as a critical scaffolding protein essential for complex assembly [28]. *EIF4G1* expression is increased in different types of

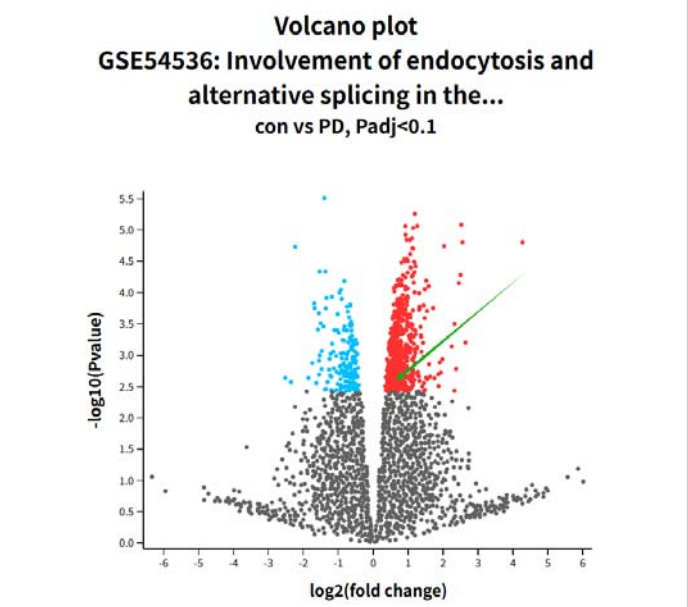

Figure 1: GROUP 1 volcano plot of *eIF4G1*.

Table 2: The genotype and allele frequencies for the SNP.

	Group	Genotype			Allele		HWE
		GG(%)	AG(%)	AA(%)	G(%)	A(%)	*p*
rs2178403	PD(n = 541)	256(47.32)	235(43.44)	50(9.24)	747(69.04)	335(30.96)	0.708
	Control(n = 401)	163(40.65)	186(46.38)	52(12.97)	512(63.84)	290(36.16)	0.926
female	PD(n = 221)	114(51.59)	82(37.10)	25(11.31)	310(70.14)	132(29.86)	
	Control(n = 179)	63(35.20)	84(46.93)	25(13.97)	210(61.05)	134(38.95)	
male	PD(n = 320)	142(44.38)	153(47.81)	25(7.81)	437(68.28)	156(24.37)	
	Control(n = 229)	100(43.67)	102(44.54)	27(11.79)	302(65.94)	156(34.06)	

Table 3: Summary of comparisons stratified by sex and age.

	Models	PD	Control	OR (95% CI)	*p*
rs2178403	Dominant (AA/(AG+GG)	50/491	52/349	1.46 (0.969-2.209)	0.069
	Recessive (AA+AG)/GG)	285/256	238/163	1.31 (1.010-1.703)	0.042
	Allele (G/A)	747/335	512/290	1.263 (1.041-1.532)	0.018
female	Dominant (AA/(AG+GG)	25/196	25/147	1.33(0.73-2.42)	0.343
	Recessive (AA+AG)/GG)	107/114	109/63	1.84(1.23-2.78)	0.003
	Allele (G/A)	310/132	210/134	1.50(1.11-2.02)	0.008
male	Dominant (AA/(AG+GG)	25/295	27/202	1.58(0.89-2.81)	0.119
	Recessive (AA+AG)/GG)	178/142	129/100	1.03(0.73-1.45)	0.869
	Allele (G/A)	437/156	302/156	1.45(1.11-1.89)	0.005

cancers [29,30]. Besides, increased *EIF4G1* could promote the formation of tumor emboli by facilitating the translation of IRES-containing p120 mRNAs [30]. Research has shown that Parkinson's Disease Genes VPS35 and *EIF4G1* Interact Genetically and Converge on α-Synuclein [31]. Our study focused on *EIF4G1*, although prior research in Han Chinese populations has shown no association between the *EIF4G1* variant rs2178403 and sporadic PD [32].

rs2178403 is an exonic variant in *EIF4G1* in which methionine (ATG) is substituted with valine (GTG) [33]. Under a recessive model, genetic analysis identified a significant association between rs2178403 and increased PD risk (OR = 1.31, p = 0.042). Notably, this effect was significantly stronger in females (OR = 1.84, p = 0.003) than in males. This pronounced sexual dimorphism may reflect neuroprotective interactions between estrogen and mTOR signaling. Further transcriptomic analysis revealed altered *EIF4G1* expression in PD peripheral blood samples compared to controls. These findings support the biological plausibility of *EIF4G1* involvement in PD pathogenesis, where dysregulated expression might disrupt eIF4F stoichiometry and drive aberrant translation. Such disturbances could ultimately compromise proteostasis, potentially by impairing clearance of misfolded α-synuclein or promoting synthesis of aggregation-prone proteins, contributing to nigrostriatal vulnerability [34].

Several limitations should be noted. The study's focus on a single variant (rs2178403) highlights the need for broader analysis across the *EIF4G1* locus. Public expression datasets may be limited by tissue specificity and cohort heterogeneity. While the observed gender disparity is statistically robust, its mechanism requires validation in neuronal models that account for hormonal modulation of mTOR-*EIF4G1* crosstalk. Future work must determine how the rs2178403 genotype or altered *EIF4G1* expression affects eIF4F complex dynamics, translation fidelity, and α-synuclein metabolism.

In conclusion, this study identifies rs2178403 within *EIF4G1* as a risk variant for Parkinson's disease (PD) susceptibility in the Han Chinese population, revealing novel sexual dimorphism in its effect. The female-predominant risk pattern suggests potential modulation of PD pathogenesis through endocrine-mTOR pathway interactions, highlighting the need for sex-specific therapeutic exploration. Our findings reinforce dysregulated mTORC1-mediated translation as a contributor to proteostatic failure in PD. Future research should expand genetic analyses across *EIF4G1* regulatory networks in diverse populations, establish functional consequences using neuron-specific models, and explore therapeutic modulation of this pathway to mitigate α-synuclein pathology.

Author contribution

QFX and ZLZ contributed to the study concept and design, and critical revision of the manuscript for intellectual content. JL, YC, and SZ contributed to the study concept and design, acquisition of the data, and analysis and interpretation of data. JL, YC, and LL recruited the subjects. SZ and LL obtained CSF samples. JL, YC, and ZLZ extracted DNA samples and performed the experiments. JL and YC analyzed the data. JL wrote the manuscript. The final manuscript was read and approved by all authors.

Data availability statement

The datasets analyzed during the current study are available from the corresponding authors upon reasonable request.

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