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Interaction between *Dectin-1* gene polymorphisms and low weight on the risk of invasive pulmonary aspergillosis in patients with lung cancer undergoing surgery

Wenfang Jin[1] · **Yu Yao**[1] · **Yanling Lv**[1]

Received: 17 July 2024 / Accepted: 5 May 2025
Published online: 20 May 2025

Abstract
The aim of this study was to investigate the impact of four single nucleotide polymorphisms (SNPs) within the *Dectin-1* gene, the interaction between SNPs of the *Dectin-1* gene and the low weight on susceptibility to invasive pulmonary aspergillosis (IPA) in patients with lung cancer undergoing surgery. Logistic regression was used to test the relationship between four SNPs of the *Dectin-1* gene and IPA susceptibility. The generalized multifactor dimensionality reduction (GMDR) model was used to assess the interaction between SNPs of *Dectin-1* gene and low weight. We found that both the rs3901533-TT and the rs3901533-TT or GT genotype were associated with an increased risk of IPA, the adjusted ORs (95% CI) were 1.98 (1.37–2.62) (TT vs. GG) and 1.43 (1.10–1.81) (GT+TT vs. GG), respectively. We also found that rs7309123-GG and rs7309123-GG+CG genotypes were associated with an increased risk of IPA, adjusted OR (95% CI) were 2.06 (1.43–2.71) (GG vs. CC), 1.63 (1.15–2.12) (CG+GG vs. CC), respectively. GMDR model found a statistically significant two-dimensional model combination (including rs3901533 and low weight). The participants with rs3901533-GT or TT genotype and low weight had the highest risk of IPA, compared to participants with rs3901533-GG genotype and without low weight, OR (95% CI) was 3.24 (1.68–4.92) ($p < 0.001$). In conclusion, rs3901533 and rs7309123 of *Dectin-1* gene, the interaction between rs3901533 and low weight were correlated with increased risk of IPA in patients with lung cancer undergoing surgery.

Keywords Invasive pulmonary aspergillosis · Lung cancer · Single nucleotide polymorphisms · Low weight · Interaction

Abbreviations

BMI	Body mass index
IPA	Invasive pulmonary aspergillosis
SNP	Single nucleotide polymorphisms
HWE	Hardy–Weinberg equilibrium
GMDR	Generalized multi-factor dimensionality reduction

✉ Yanling Lv, lvyanling226@163.com | [1]Department of Respiratory and Critical Care Medicine, The Second Hospital of Nanjing, Nanjing University of Chinese Medicine, 1-1 Zhongfu Road, Gulou District, Nanjing 210003, Jiangsu, China.

1 Introduction

Invasive pulmonary aspergillosis (IPA) is a serious infection caused by Aspergillus fumigatus and is a common fungal infection in clinical practice, mainly occurring in immune-suppressed patients with hematologic malignancies, hematopoietic stem cell or solid organ transplantation surgery [1, 2]. IPA was also found in patients with chronic obstructive pulmonary disease (COPD), diabetes and lung cancer after surgery [3, 4]. IPA is a common infectious disease clinically, with a rate of incidence of 5–7% [5], and the incidence of IPA in lung cancer patients is approximately 2.6% [3].

Although clinical management can significantly reduce the probability of developing IPA, especially for high-risk populations [6]. Effective clinical management can markedly diminish the chance of invasive pulmonary aspergillosis onset, particularly in high-risk groups, achieving a substantial risk reduction [7]. It suggested that there are other factors that influence IPA infection in addition to fungal exposure factors, such as genetic factors, such as single nucleotide polymorphisms (SNPs) [8]. Previous studies [9–11] have suggested that IPA development is associated with SNPs related to host innate immunity, which can significantly affect susceptibility to invasive fungal infections.

Dendritic cell-associated C-type lectine-1 (*Dectin-1*, also known as *CLEC7A*) is the first identified C-type lectin-like receptors (CLRs), which can recognize the presence of β-1,3-glucan, a polysaccharide found in the cells walls of various fungi [12, 13]. Previously, several studies [14–16] focused on the relationship between *Dectin-1* gene SNPs and fungal infections in different populations. In addition, several environmental factors have been reported as potential IA risk factors [17, 18]. However, to our knowledge, no study was performed to investigate the interaction between the *Dectin-1* gene and the environment on the risk of IPA. Therefore, this study aimed to evaluate the influence of SNPs of the *Dectin-1* gene and additional interaction with the environmental factors on IPA susceptibility in patients with lung cancer undergoing surgery.

2 Methods

2.1 Participants

In this study, all lung cancer free-IPA patients ($n = 2106$) who underwent surgical resection at the Second Hospital of Nanjing were selected, and then all selected patients were followed for 5 years, about 10.4% of patients who underwent surgical resection were diagnosed as IPA ($n = 210$). We exclude the patients who were lost to follow-up ($n = 48$) and patients with other comorbid diseases ($n = 30$) including T2DM, hypertension, and other types of cancer from the cohort. At last, a total of 210 lung cancer patients were diagnosed with IPA. The controls were selected from other patients who were not diagnosed with IPA, matched to cases (1:2) according to sex and age (± 3 years) (Fig. 1).

All IPA patients enrolled in our study were diagnosed by our hospital's clinician following the clinical guidelines suggested by the consensus group (EORTC/MSG) [19]: (1) Clinical manifestations: persistent fever and no response to broad-spectrum antibiotic treatment, with respiratory distress, persistent cough, chest pain and bloody phlegm; (2) Imaging examination: Chest CT shows a "crescent sign", a "cavity" formation, and a "satellite lesion"; (3) Microbiological evidence: The branching mycelium of Aspergillus can be observed directly from respiratory secretions or lung tissue, and Aspergillus can be isolated from lung tissue or bronchoalveolar lavage fluid; (4) Organizational pathological evidence: Lung tissue biopsy revealed invasive growth of Aspergillus, such as fungal hyphae that penetrate the alveolar wall and tissue necrosis. All participants have signed an informed consent form.

2.2 Genotyping methods

In this study, a total of four *Dectin-1* gene SNPs were selected, including rs3901533, rs7309123, rs2078178 and rs16910631. A total of 3 ml of whole blood was obtained from all participants after 8 h of fasting. The DNA of whole blood samples was extracted according to the instructions of the DNA blood micro-Kit (Qiagen, Hilden, Germany). PCR-based restriction fragment length polymorphism was used for genotyping the four SNPs selected. Table 1 shows the primers used for genotyping and a detailed description of the four SNPs.

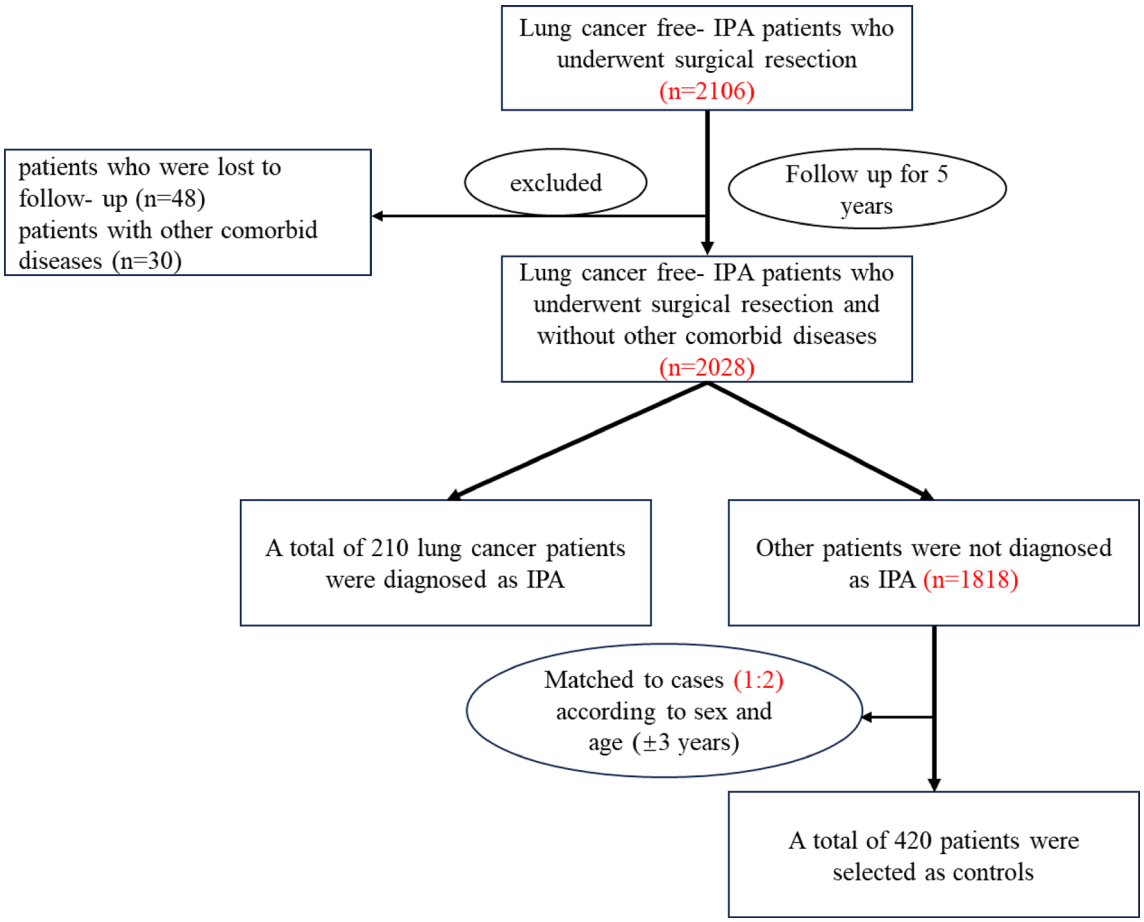

Fig. 1 The flow sheet for cases and controls selection

Table 1 Description of 4 SNPs within *Dectin-1* gene and the primers designed

SNPs	Location	Primer (5′→3′)	Nucleotide substitution	Chromosome
rs3901533	Intron	Forward: 5′-CTGGGAAAGGCAAAGAGGGTT-3′ Reverse: 5′-TTAGCCATTGTCTTCTCCTCCAA-3′	G/T	12:10,124,484 (GRCh38) 12:10,277,083 (GRCh37)
rs7309123	Intron	Forward: 5′-TTATGCTCAGCAGTTTGTACTG-3′ Reverse: 5′-AGCTTTTTGCTCCAATCTATGT-3′	C/G	12:10,119,994 (GRCh38) 12:10,272,593 (GRCh37)
rs2078178	Intron	Forward (G): 5′-AAACTGCCTAGGGGGACTGC-3′ Forward (A): 5′-AAACTGCCTAGGGGGACTGT-3′ Reverse: 5′-ACCTGACATCAACCTAGAGAGAAG-3′	G/A	12:10,123,963 (GRCh38) 12:10,276,562 (GRCh37)
rs16910631	Intron	Forward: 5′-TCTCAAAGGATTATTGCGGGAATTAAAC-3′ Reverse: 5′-TCTCAAAGGATTATTGCGGGAATTAAAT-3′	C/T	12:10,128,656 (GRCh38) 12:10,281,255 (GRCh37)

2.3 Statistical analysis

In this study, SPSS software (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. For continuous variables that follow a normal distribution, we calculated the means and standard deviations (SD), and an independent sample student's *t* test was used for comparison between the control and case group. For categorical variables, the percentages were calculated and compared between the case and control groups using χ^2 test. Online software (SNPStats: https://www.snpstats.net/) was used for Hardy–Weinberg equilibrium (HWE) testing. Logistic regression was used to test the relationship between four SNPs of the *Dectin-1* gene and the risk of IPA, and to test the interaction between four SNPs

and the low weight on susceptibility to IPA. Generalized multifactor dimensionality reduction (GMDR) [19] model was used to screen the combinations of high-order gene-environment interaction. All statistical significance were two-tailed, and the *p* value less than 0.05 was considered as the difference was significant.

3 Results

A total of 630 lung cancer patients were enrolled in this study, including 210 lung cancer patients with IPA and 420 lung cancer patients without IPA. In Table 2, we show the general and clinical characteristics of two groups. The mean age of all participants was 67.18 ± 9.87 years (69.21 ± 11.30 for lung cancer patients with IPA and 66.32 ± 10.26 for lung cancer patients without IPA). No significant differences were found for age, sex, alcohol drinkers, smoking status, tumor location and histopathology between two groups (all *p* values were higher than 0.05). Compared to controls, the mean age and low weight rates for controls were significantly lower than those of lung patients with IPA.

In this study, the HWE test was performed using the SNPStats online software, and we found that the genotypes frequencies of all SNPs were consistent with HWE in controls (all *p* values >0.05). Logistic regression analysis indicated that both the rs3901533-TT and the rs3901533-TT or GT genotype were associated with an increased risk of IPA, the adjusted ORs (95% CI) were 1.98 (1.37–2.62) (TT vs. GG) and 1.43 (1.10–1.81) (GT+TT vs. GG), respectively. We also found that the genotype rs7309123-GG and rs7309123-GG+CG was associated with an increased risk of IPA, adjusted OR (95% CI) were 2.06 (1.43–2.71) (GG vs. CC), 1.63 (1.15–2.12) (CG+GG vs. CC), respectively. However, no statistically significant relationship was found between rs2057482 or rs1957757 and the risk of IPA (Table 3).

A total of five variables including rs3901533, rs7309123, rs2078178, rs16910631 and low weight were included in the GMDR model. We found a statistically significant two-dimensional model combination (including rs3901533 and low weight). Training balanced accuracy, testing balanced accuracy, cross-validation consistency of the best selected model was 0.618, 0.601 and 10/10 ($p = 0.011$), which indicated that the *Dectin-1* gene—rs3901533 exhibited interaction effects with low weight on susceptibility to IPA in patients with lung cancer (Table 4).

Table 2 General and clinical characteristics for IPA patients and control participants

Variables	Lung cancer patients undergoing surgery		*p* values
	Patients with IPA (*n* = 210)	Patients with non-IPA (*n* = 420)	
Age (year) (means ± SD)	69.21 ± 11.30	68.32 ± 10.26	0.322
Males, *N* (%)	130 (61.90)	266 (63.3)	0.726
BMI (kg/m^2)			<0.001
Low weight	75 (35.71)	85 (20.24)	
Normal	115 (54.76)	288 (68.57)	
Overweight and obesity	20 (9.52)	47 (11.19)	
Smoking status			0.389
Never smoking	115 (54.76)	254 (60.48)	
Ex-smoking	92 (43.81)	161 (38.33)	
Current smoking	3 (1.43)	5 (1.19)	
Alcohol drinkers, *N* (%)	98 (46.67)	204 (48.57)	0.652
Tumor location, *N* (%)			0.213
Upper lobe	110 (52.38)	231 (55.00)	
Middle lobe	82 (39.05)	168 (40.00)	
Lower lobe	18 (8.57)	21 (5.00)	
Histopathology			0.900
SCC	87 (41.43)	166 (39.52)	
ADC	109 (51.90)	225 (53.57)	
Others[a]	14 (6.67)	29 (6.91)	

[a]Others carcinoma including ASC, LCC, CS and MEC; ASC: adenosquamous carcinoma; LCC: large cell carcinoma; MEC: mucoepidermoid carcinoma; CS: carcinosarcoma

Table 3 Logistic regression analysis on relationship between *Dectin-1* gene SNPs and IPA risk

SNPs	Genotypes and alleles	Frequencies *N* (%)		OR (95% CI)[a]	*p* values
		Patients with IPA (*n* = 210)	Patients without IPA (*n* = 420)		
rs3901533					
	GG	112 (53.33)	264 (62.86)	1.00	
	GT	79 (37.62)	136 (32.38)	1.28 (0.89–1.73)	
	TT	19 (9.05)	20 (4.76)	1.98 (1.37–2.62)	
	GT+TT	98 (46.67)	156 (37.14)	1.43 (1.10–1.81)	
	G	303 (72.14)	664 (79.05)		
	T	117 (27.86)	176 (20.95)		
HWE test for controls					0.645
rs7309123					
	CC	104 (49.52)	256 (60.95)	1.00	
	CG	80 (38.10)	139 (33.10)	1.42 (0.96–1.90)	
	GG	26 (12.38)	25 (5.95)	2.06 (1.43–2.71)	
	CG+GG	106 (50.48)	164 (39.05)	1.63 (1.15–2.12)	
	C	288 (68.57)	651 (77.50)		
	G	132 (31.43)	189 (22.50)		
HWE test for controls					0.296
rs2078178					
	GG	113 (53.81)	248 (59.05)	1.00	
	GA	81 (38.57)	153 (36.43)	1.24 (0.73–1.73)	
	AA	16 (7.62)	19 (4.52)	1.41 (0.66–2.17)	
	GA+AA	97 (46.19)	172 (40.95)	1.29 (0.71–1.89)	
	G	307 (73.10)	649 (77.26)		
	A	113 (26.90)	191 (22.74)		
HWE test for controls					0.451
rs16910631					
	CC	128 (60.95)	270 (64.29)	1.00	
	CT	73 (34.76)	138 (32.86)	1.10 (0.71–1.56)	
	TT	9 (4.29)	12 (2.86)	1.28 (0.58–1.96)	
	CT+TT	82 (39.05)	150 (35.71)	1.12 (0.69–1.57)	
	C	329 (78.33)	678 (80.71)		
	T	91 (21.67)	162 (19.29)		
HWE test for controls					0.256

[a]Adjusted for age, gender, BMI, smoking status, and alcohol drinking

Table 4 GMDR analysis on interaction between *Dectin-1* gene SNPs and low weight on IPA risk

Locus no	Best combination	Cross-validation consistency	Training balanced accuracy	Testing balanced accuracy	*p* values[a]
2	rs3901533, low weight	10/10	0.618	0.601	0.011
3	rs3901533, rs2078178, low weight	8/10	0.541	0.532	0.182
4	rs3901533, rs2078178, rs7309123, low weight	6/10	0.586	0.524	0.425
5	rs3901533, rs2078178, rs7309123, rs16910631, low weight	6/10	0.478	0.512	0.532

[a]Adjusted for age, gender, smoking status, and alcohol use

Ang then, all participants were grouped into four groups, including participants with the rs3901533-GG genotype and without low weight, participants with the rs3901533-GG genotype and low weight, participants with the rs3901533-GT or TT genotype and without low weight, and participants with the rs3901533-GT or TT genotype and low weight. Logistic regression was used to test the interaction effects between rs3901533 and low weight (Fig. 2). Compared to participants with the rs3901533-GG genotype and without low weight, participants with the rs3901533-GT or TT genotype and low weight had the highest risk of IPA, OR (95% CI) was 3.24 (1.68–4.92) ($p < 0.001$), after adjustment for age, gender, smoking status, and alcohol use.

4 Discussion

In this study, we found that both the rs3901533-TT, the rs3901533-TT or GT genotype, rs7309123-GG, and rs7309123-GG+CG genotypes were all associated with increased risk of IPA. However, no significant association was found between rs2057482 or rs1957757 and IPA risk. Previous studies [14, 20] have suggested that genetic defects in *Dectin-1* are associated with increased susceptibility to fungal infections. Cunha et al. [15] suggested that the rs16910526 polymorphism of the *Dectin-1* gene can increase susceptibility to invasive aspergillosis in patients undergoing hematopoietic stem cell transplantation (HSCT). Sainz et al. [16] found that the two intron SNPs of the *Dectin-1* gene (rs3901533 and rs7309123) can significantly increase the risk of IPA infection in patients with hematological malignancies. Chen et al. [11] found that two SNPs of the *Dectin-1* gene (rs3901533 and rs7309123) are associated with increased susceptibility to pulmonary IFD infection in the Han Chinese population. Chai et al. [21] suggested that the heterozygosity of *Dectin-1* gene rs16910526 has a limited impact on IA susceptibility and may be important in susceptible non HSCT patients. Zhou et al. [22] performed a meta-analysis and suggested that *Dectin-1* gene SNPs may play an important role in fungal infection (FI) susceptibility. Kalkanci et al. [23] suggested that *Dectin-1* gene rs7309123 was not associated with IA susceptibility, but *Dectin-1* gene rs16910526 polymorphism was associated with a lower IA risk. Fischer et al. [24] demonstrated that the *Dectin-1* gene rs7309123 may be an independent risk factor for IFD developing in patients with AML undergoing induction chemotherapy. Furthermore, SNPs of the *Dectin-1* gene have also been reported to have an association with other fungal infections, such as fungal keratitis [25]. A case–control study [25] performed for the Chinese population suggested that *Dectin-1* gene SNPs were not associated with fungal keratitis susceptibility in the Han Chinese northern population.

The detailed pathological mechanism of IPA is complicated. *Dectin-1* is mainly expressed primarily on the surface of dendritic cells and macrophages [26]. The β-Polysaccharides are the main components of fungal cell walls, and dectin-1 recognizes these components through its carbohydrate recognition domain in the extracellular region. Then, signal transduction is carried out through the cytoplasmic domain of the tyrosine activation motif of the dectin-1 immune receptor (ITAM). When ITAM binds to its ligand, spleen tyrosine kinase is recruited, activating member 9 of the cysteine protease recruitment domain family (CARD9)—nuclear factor κB (NF-κB) Axis, and then activates various genes, especially genes that encode pro-inflammatory cytokines [27]. Dectin-1 can also affect the regulation of Th1 and Th17 of the adaptive immune system, including both innate and adaptive immune systems [28].

The risk of IPA could be influenced by the synergistic effects of SNP–SNP, but to date, no study was performed to investigate the impact of gene–environment interaction on IPA risk [16]. And low weight has been reported to be an established risk factor for pulmonary aspergillosis [29–31]. And in this study, we also found that the distribution of low

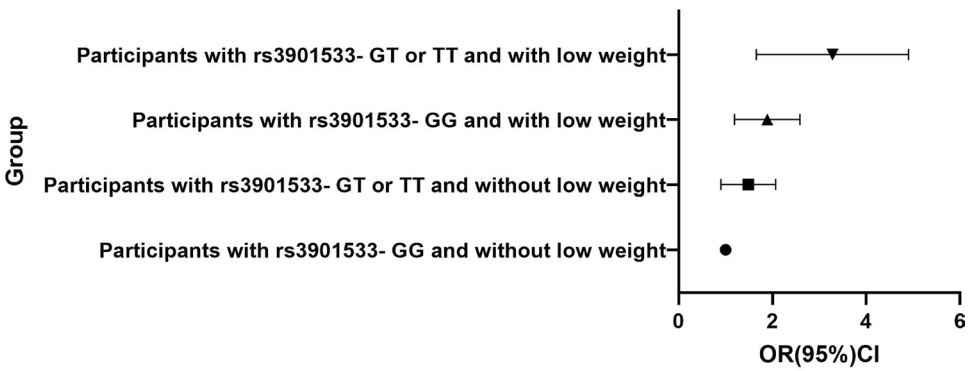

Fig. 2 Logistic regression analysis for interaction between rs3901533 and low weight on IPA risk. Adjusted for age, gender, smoking status, and alcohol use

weight was significantly different in cases and controls, and the prevalence of low weight was higher in IPA cases than in controls. Previously, to our knowledge, no study was performed to examine the role of interaction between the *Dectin-1* gene and low weight on susceptibility to IPA in patients with lung cancer undergoing surgery. And in this study, we used the GMDR model to investigate the high-dimensionality effect among 4 SNPs of the *Dectin-1* gene and low weight. We found a statistically significant interaction between rs3901533 and low weight on susceptibility to IPA in patients with lung cancer. Compared to participants with the rs3901533-GG genotype and with normal weight, participants with the rs3901533-GT or TT genotype and low weight had the highest risk of IPA.

There were several limitations in this study. First, the four selected SNPs could not represent the characteristics of the entire *Dectin-1* gene. Second, the results obtained from this study should be verified in different populations in the future.

In summary, the results indicated that the *Dectin-1* genes rs3901533 and rs7309123, the interaction between rs3901533 and low weight were correlated with increased risk of IPA in patients with lung cancer undergoing surgery.

Acknowledgements We appreciate the cooperation of the partners and staff who cooperated in this study.

Author contributions All authors have read and approved the manuscript. Wenfang Jin: manuscript writing, editing, and review. Yu Yao: statistical analysis, literature research, data acquisition. Yanling Lv: data verification.

Funding National Natural Science Foundation Youth Program (No: 82203561).

Data availability The data and figures generated and analyzed during this study are available from the corresponding authors upon reasonable request.

Declarations

Ethics approval and consent to participate Each participant understood the study process and signed a written informed consent before the start of the study. The protocol was approved by committee of the Second Hospital of Nanjing (2022-LY-kt042) in accordance with the Declaration of Helsinki.

Competing interests The authors declare no competing interests.

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