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Original Article

Association of *PGLYRP* and *HMOX-1* polymorphism with Parkinson's disease in the northern Han Chinese population

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Abstract: The association between Parkinson's disease (PD) and genetic factor has been established by some candidate genes, but limited knowledge is available related to SNPs of *PGLYRP* and *HMOX-1* genes newly identified as PD susceptible loci in Caucasian. In the current study, we investigated three single nucleotide polymorphisms (SNPs), *PGLYRP2* rs3813135, *PGLYRP4* rs10888557 and *HMOX-1* rs2071746, in the north Han Chinese population and analyzed their association with PD. Mismatched PCR and restriction fragment length polymorphism (RFLP) techniques were used to examine the genetic polymorphisms of the three SNPs in 336 PD patients and 364 control samples. However, we did not find that the distributions of genotype frequency exhibited significant differences between the PD and control groups ($P>0.05$) in the three SNPs loci. The similar distributions of genotypic frequency between case and control groups indicate that the three SNPs investigated in this study are unlikely to play roles as common risk factors for PD in the northern Han Chinese population.

Keywords: Parkinson's disease, peptidoglycan, *PGLYRP*, heme oxygenase-1, *HMOX-1*

Introduction

Parkinson's disease (PD) is one of the most common neurodegenerative diseases, resulting in movement disorders mainly manifested as slow motion, postural instability and characteristic tremor [1]. PD is mainly characterized by rest tremors, rigidity, slowness of movement and postural imbalance, but sometimes sense of smell, constipation and other symptoms may precede motor symptoms. In the autonomic and enteric nervous system, relevant α-synuclein pathological development may precede the brain [2-7]. Previous studies reported that infrequent bowel movements were associated with an increased risk of PD [8] and colon biopsy of some PD patients manifested increased levels of inflammatory cytokines and α-synuclein [4, 7]. Normally, α-synuclein is an abundant presynaptic brain protein. But colonic flora imbalance may result in the α-synuclein misfolding, aggregation and fibrillation of which is implicated as critical factors in the mechanism of PD pathological changes [9-12]. Peptidoglycan is a major structural component of the bacterial cell wall that serves to protect the plasma membrane. Peptidoglycan recognition proteins (PGRPs) are natural immune proteins. Because of PGRPs' uniqueness to bacteria, it is recognized as foreign and binding pattern recognition receptors to potently trigger an innate immune response. There are four highly conserved PGRPs encoded by *PGLYRPs 1-4* genes in human. Four PGRPs are selectively expressed in a range of tissues and secreted into the gut [13]. Genetic polymorphisms of *PGLYRP* genes have been associated with the risk of inflammatory bowel disease [10, 11, 14]. From recent case-control reports, some polymorphic sites in *PGLYRP 2, 3* and *4* genes were also related to PD in Caucasian population [15].

Oxidative stress is an early event which may cause death of dopaminergic neurons [16]. Oxidative stress and iron deposition are considered as risk factors for degeneration of the central nervous system, and many neurodegenerative diseases show increased accumulation of

Table 1. The frequency distribution of select characteristics in case and control groups

Variable	Cases (N = 336)		Controls (N = 364)	
	No.	%	No.	%
Sex				
Male	165	49.1	200	54.9
Female	171	50.9	164	45.1
Age				
50-60	128	38.1	125	34.3
60-70	107	31.8	111	30.5
≥70	101	30.1	128	35.2

iron at the nerve degeneration site [17-19]. In PD patients, increased iron levels are thought to cause the production of reactive oxygen species and accumulation of α-synuclein in substantia nigra (SN), leading to oxidative neuronal destruction in the brain region. Heme oxygenase-1 (HMOX-1) is an effective antioxidant and is highly expressed in the SN. The published data showed that overexpression of HMOX-1 in PD might contribute to significant iron deposition [20, 21]. The SNP rs2071746 in the *HMOX-1* gene was found to be associated with the risk of PD in a case-control study [22].

Therefore, in this study, we selected three SNPs, two of *PGLYRP2, 4* genes and one of *HMOX-1* gene to investigate the genetic polymorphism in the northern Han Chinese population and explored possible associations with the pathogenesis of PD.

Materials and methods

Subjects

Blood samples were recruited from 336 PD patients (mean age ± SD 64.07 ± 8.63, ranging from 50 to 89 years) and 364 unrelated control subjects (mean age ± SD 66.44 ± 10.29, ranging from 50 to 89 years) in the northern Han Chinese population (**Table 1**).

PD group: all patients were diagnosed by the Department of Neurology, the First Affiliated Hospital of China Medical University, according to the clinical diagnostic criteria for idiopathic PD. All patients showed the basic characteristics of at least two to three cardinal signs (tremor, rigidity, bradykinesia) and effect was evident after levodopa treatment.

Control subjects: all healthy individuals matching almost same age, race and gender with PD group were from the local community. Control participants had not been diagnosed of neurodegenerative diseases.

The study was approved by the Human Research Ethics Committee of China Medical University. All participants signed the informed consent form. Genomic DNA was extracted using Sodium dodecyl sulfate-protease K-phenol-chloroform method.

Mismatched primer design

To reveal the genotypes of rs3813135 located in *PGLYRP2* (NT_011295.12: g.15476534T>C) and rs2071746 located in *HMOX-1* (c.-495A>T), mismatched PCR primers were designed to generated *Stu* I and *Hin*d III restriction endonuclease sites in the amplified products of SNPs rs3813135 and rs2071746. While the allele C of the rs10888557 in *PGLYRP4* (NT_004487.20: g.153352574G>C) is recognized by restriction endonuclease *Bme*T110 I. The primers used in this study are shown in **Table 2**.

PCR-RFLP analysis

All three SNPs were amplified separately (System 1-3) and PCR products were subsequently sequenced. A 20 μL reaction volume was used which includes 10 μL of 2 × Power Taq PCR Master Mix (Bioteke, Beijing, China), 2 μL genomic DNA (about 20-40 ng/μL), and primer concentrations were shown in **Table 2**. Amplification conditions for system 1-3 are 94°C denaturation 1 min; 94°C denaturation 30 s, 65°C (System 1, 3) or 60°C (System 2) annealing 30 s, 72°C extension 10 s, a total of 32 cycles (System 1, 2) or 35 cycles (System 3); the last 72°C extension 1 min. Then PCR products were digested with the relevant restriction enzymes given in **Table 2** at 37°C for 2 h. The digested products were separated by 6% polyacrylamide gel, the fragments were visualized by UV gel imaging after ethidium bromide staining (**Figure 1**).

Statistical analysis

Power Marker V3.0 was used to calculate the frequency distribution and examine Hardy-Weinberg equilibrium (HWE). Genotype frequency deviations were calculated by a mutant vs. wild-type model between patients and controls. The Chi-square test was used to find the association of polymorphisms with PD, and the

Table 2. Three SNPs and mismatched PCR amplification parameters

SNP	PCR system	Primer direction	Sequence 5'→3'	Primer concentration	Restriction enzyme	Fragment size (bp)
rs3813135	System 1	Forward	ACGCAGAAGCTGTGTGTCaGG*	0.6 μM	*Stu* I	C: 188 + 22
		Reverse	TGGGGGTCATGGGGTTATCA	0.6 μM		T: 210
rs10888557	System 2	Forward	CATCTAATCCACAAAAGCTCTT	0.8 μM	*Bme*T110 I	C: 318 + 84
		Reverse	GCATATGAGTGACACCTGACAA	0.8 μM		G: 402
rs2071746	System 3	Forward	CCTGATGTTGCCCACCAaGC*	1.5 μM	*Hin*d III	T: 142 + 17
		Reverse	TGAGAAGCTGCAGGCTCTGGGT	1.5 μM		A: 159

*Lower case letters represent mismatched bases for the introduction of endonuclease sites.

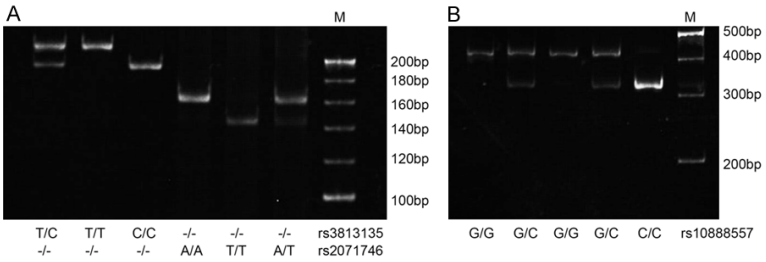

Figure 1. Electrophoresis patterns of the mismatched PCR-RFLP in 3 SNPs. Genotypes are indicated under each lane. A. Electrophoretic pattern for SNPs rs3813135 and rs2071746 is exhibited. M lane shows a 20 bp DNA molecular size ladder ranging from 100 to 200 bp. B. Electrophoretic pattern for SNP rs10888557 is exhibited. M lane shows a 100 bp DNA molecular size ladder ranging from 200 to 500 bp. bp = base pairs.

Table 3. The MAF of three SNPs in cases and controls

Gene	SNP	MAF		Minor allele
		Cases	Controls	
PGLYRP2	rs3813135	0.3601	0.3242	C
PGLYRP4	rs10888557	0.2292	0.2088	C
HMOX1	rs2071746	0.4182	0.4560	A

Odds ratio (OR) value as well as 95% confidence interval (CI) were used to evaluate the strength of association by SPSS V20.0 software. All analyses were performed by a two-tailed Chi-square test and P value < 0.05 was considered statistically significant.

Results

Genotyping of the three SNPs

The use of mismatched primers can solve the problem that there is no restriction site in the DNA sequence, and a restriction enzyme site created artificially in the PCR amplification product can equally accomplish the goals of the experiment. In this study, mismatched sequences containing the *Stu* I and *Hin*d III recognition sites were correctly generated for *PGLYRP2* and *HMOX-1* loci after confirmed by DNA sequencing. The amplified PCR products are of 210 bp (rs3813135), 402 bp (rs10888557), and 159 bp (rs2071746), respectively (as shown in **Table 2** and **Figure 1**). The undigested fragments represent alleles g.15476534T, g.153352574G, and c.-495A, while the fragments of 188 bp, 318 bp, and 142 bp represent alleles g.15476534C, g.153352574C, and c.-495T, respectively. Genotyping patterns of rs3813135 in system 1 and rs2071746 in system 3 were shown in **Figure 1A**, and rs10888557 in system 2 was shown in **Figure 1B**. Fragments shorter than 80 bp disappeared from the gel. Above results indicated that the mismatched PCR-RFLP assay can be applied to genotype the three SNPs.

Genetic parameters and association analysis

The distribution of minor allele frequency (MAF) in PD and control groups in the current study was shown in **Table 3**. Genetic parameters such as genotypic frequency and odds ratio were shown in **Table 4**. The observed frequencies of genotypes were all in Hardy-Weinberg equilibrium. Genotypic frequencies in each SNP locus were respectively similar through comparison of PD patients and controls. We subsequently performed association analyses on the three SNPs (**Table 4**) and found that the P values from these SNPs showed all no association with PD in the northern Han Chinese population. After gender stratification, there was no

Table 4. Genotypes of three SNPs and genetic parameters in cases and controls

Genotype	Controls (N = 364)	Cases (N = 336)	HWE	Association with PD
	No. (%)	No. (%)		
rs3813135 (C/T)				
CC	36 (9.8)	39 (11.6)	0.5301	*OR* = 0.804
CT	164 (45.1)	164 (48.8)		95% *CI*: 0.595~1.086
TT	164 (45.1)	133 (39.6)		*P* = 0.448
rs10888557 (C/G)				
CC	12 (3.3)	15 (4.5)	0.2679	*OR* = 0.879
CG	128 (35.2)	124 (36.9)		95% *CI*: 0.649~1.191
GG	224 (61.5)	197 (58.6)		*P* = 0.589
rs2071746 (A/T)				
AA	81 (22.3)	54 (16.1)	0.3026	*OR* = 1.090
AT	170 (46.7)	173 (51.5)		95% *CI*: 0.792~1.500
TT	113 (31.0)	109 (32.4)		*P* = 0.147

All SNP genotypes were in Hardy-Weinberg equilibrium. *CI* = confidence interval, *OR* = odds ratio. HWE = Hardy-Weinberg equilibrium in controls.

significant differences in the genotypes distributions of the investigated loci either (*P*>0.05) (shown in Tables S1 and S2). These results suggested that rs3813135, rs10888557, and rs2071746 loci showed a lack of correlation with PD in the northern Han Chinese population.

Discussion

In recent studies, the relationship between PD and SNPs has been proved in many susceptible genes [23]. The effects of race on SNP frequency and the founder effects have been recorded for several PD genes. The role of PGRPs is to maintain beneficial intestinal tract, and the loss of any PGRPs will result in the increased mucosal permeability and tissue damage [24]. The data showed that the intestinal tract was affected early in the PD process, which might be considered as a potential factor for PD [25]. Several variations of *PGLYRP* genes were associated with Crohn's disease [14]. Another meaningful phenomena, the deficiency of leucine-rich repeat kinase 2 (LRRK2), could be observed under experimental colitis increase susceptibility in mice. The reports suggested that LRRK2 might play an important role in the pathogenesis of human Crohn disease [26]. Because mutations in the *LRRK2* gene have been identified in families with autosomal dominant PD and in sporadic cases, So Goldman *et al.* hypothesized that the genetic variation of *PGLYRP*s might be associated with PD. Subsequently, they reported that some variations of *PGLYRP*s might affect the incidence of PD in Caucasians and found a significant associations of rs1813135, rs733731, and rs892145 of *PGLYRP2* gene; rs1987763 of *PGLYRP3* gene; rs10888557, rs12063091, rs3006440, rs3006448, rs3006458, and rs3014864 of *PGLYRP4* gene with PD. Interestingly, the minor alleles of above SNPs were related to a reduced risk of PD [15]. Additionally, the identified SNPs in *PGLYRP2* had a linkage disequilibrium relationship with each other. The significantly associated SNPs rs2987763 (*PGLYRP3*), rs10888557 and rs3014864 (*PGLYRP4*) located near the transcription factor binding sites, and the rs10888557 was strongly associated among them [15]. However, no research was relevant to *PGLYRP* genes and PD prevalence in Chinese population. Thus, we selected *PGLYRP2* rs3813135 located in the coding region and *PGLYRP4* rs10888557 located in the 5' flanking region to investigate their polymorphisms for the first time in Chinese population. Based on the data, however, we didn't observe any significant differences on rs3813135 and rs10888557 loci between the PD and control groups (*P*>0.05), which indicated that these SNPs might not be a genetic susceptible factor for PD in the northern China. According to the findings from previous and present studies, the genetic heterogeneity on the rs3813135 and rs1088857 was observed between populations of Asian and American descent. The

differences of race and region may be responsible for this phenomenon. Further increasing studies on genetic polymorphisms of PGLYRP genes in more ethnic groups and regions will be necessary for systemic association demonstration.

Heme oxygenase (HMOX) consists of 2 major isoenzymes, namely inducible heme oxygenase-1 (HMOX-1) and constitutive heme oxygenase-2 (HMOX-2). A study reported that rs2071746 on *HMOX-1* gene had a significant association with PD [22]. Because essential tremor (ET) and PD shared phenotypes and similarities of etiology, they subsequently found that the rs2071746 of *HMOX-1* and rs1051308 of *HMOX-2* were weak associated with a risk of ET in the Spanish population [27]. A report showed that the increase of serum HMOX-1 levels may be associated with chronic oxidative stress state in PD [28], but another study in the Germans mentioned that correlation analysis between the *HMOX-1* gene and PD exhibited negative association [29]. In the present study, we only selected SNP rs2071746 of *HMOX* gene to explore association with PD in the northern Chinese population. However, our data indicated that the rs2071746 of *HMOX* gene was not supported as a genetic susceptible factor for PD, similar to the results in the German population. Hence, these reports implied the differences of ethnicity and geographical position might affect association of *HMOX-1* rs2071746 with the incidence of PD.

In conclusion, the genetic polymorphisms of rs2071746, rs3813135 and rs10888557 were successfully detected by mismatched PCR-RFLP. In the northern Han Chinese population, the three SNPs didn't show any association with PD. For the first time, the relationship between these three SNPs and PD was studied in the Chinese community. This study will provide useful data for further exploring the association about *PGLYRP* and *HMOX-1* genes with PD.

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Disclosure of conflict of interest

None.

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Table S1. Three SNPs genotype frequencies stratified by gender

Gene	Genotypes	Cases/Controls		*P* value	
		Male	Female	Male	Female
rs3813135					
	CC	16/11	23/25	0.176	0.795
	CT	86/96	78/68		
	TT	63/93	70/71		
rs10888557					
	CC	6/8	9/4	0.165	0.269
	CG	63/76	61/52		
	GG	96/116	101/108		
rs2071746					
	AA	28/50	26/31	0.184	0.607
	AT	81/91	92/79		
	TT	56/59	53/54		

Table S2. The original data for individual participants

ID	Gender	Age	Nationality	PGLYRP2 (T/C)	PGLYRP4 (G/C)	HMOX1 (A/T)
CT1	M	82	Han	T/T	C/G	T/T
CT2	F	61	Han	T/T	G/G	A/A
CT3	M	80	Han	T/T	G/G	T/T
CT4	M	77	Han	T/T	C/G	A/T
CT5	F	71	Han	C/C	G/G	A/T
CT6	M	79	Han	T/T	G/G	T/T
CT7	F	78	Han	T/T	G/G	A/T
CT8	M	78	Han	T/T	G/G	A/A
CT9	M	80	Han	C/T	C/G	A/T
CT10	M	82	Han	C/T	G/G	A/A
CT11	F	59	Han	T/T	G/G	T/T
CT12	F	70	Han	C/C	C/G	A/T
CT13	F	75	Han	C/T	C/G	A/T
CT14	M	81	Han	C/T	G/G	A/T
CT15	M	80	Han	C/T	G/G	A/T
CT16	F	81	Han	C/T	G/G	T/T
CT17	M	60	Han	T/T	C/C	A/T
CT18	F	69	Han	T/T	G/G	A/A
CT19	F	81	Han	T/T	G/G	T/T
CT20	F	70	Han	C/T	C/G	A/T
CT21	M	80	Han	C/T	G/G	A/A
CT22	M	84	Han	T/T	G/G	A/T
CT23	M	80	Han	C/T	C/G	A/A
CT24	M	83	Han	C/T	C/C	A/T
CT25	M	64	Han	C/T	G/G	A/T
CT26	M	84	Han	T/T	C/G	A/A
CT27	F	72	Han	C/C	C/G	T/T
CT28	M	85	Han	C/T	C/G	A/T
CT29	M	77	Han	C/C	G/G	T/T
CT30	M	57	Han	C/C	G/G	A/A

CT31	M	84	Han	C/T	C/G	A/A
CT32	M	78	Han	C/T	G/G	A/T
CT33	M	87	Han	C/T	C/G	A/T
CT34	F	83	Han	C/T	G/G	T/T
CT35	M	80	Han	C/T	G/G	A/T
CT36	F	65	Han	T/T	C/C	A/T
CT37	F	53	Han	C/T	G/G	A/T
CT38	M	76	Han	C/T	G/G	A/T
CT39	M	80	Han	T/T	G/G	T/T
CT40	F	68	Han	C/T	G/G	T/T
CT41	M	81	Han	T/T	C/G	A/T
CT42	M	86	Han	T/T	G/G	A/T
CT43	F	53	Han	C/T	G/G	A/T
CT44	F	66	Han	C/T	G/G	A/A
CT45	M	79	Han	T/T	G/G	A/A
CT46	M	56	Han	C/T	G/G	A/A
CT47	F	88	Han	C/C	G/G	T/T
CT48	M	81	Han	T/T	G/G	A/T
CT49	M	77	Han	C/T	G/G	A/T
CT50	M	87	Han	T/T	G/G	A/A
CT51	M	78	Han	T/T	G/G	A/A
CT52	M	82	Han	C/C	G/G	T/T
CT53	M	82	Han	T/T	C/G	T/T
CT54	M	84	Han	C/T	G/G	A/T
CT55	M	86	Han	C/T	C/G	A/T
CT56	F	72	Han	C/T	G/G	A/T
CT57	M	80	Han	C/T	C/G	T/T
CT58	M	82	Han	T/T	G/G	A/A
CT59	M	75	Han	T/T	G/G	A/A
CT60	M	56	Han	C/T	G/G	A/T
CT61	M	87	Han	C/T	G/G	A/A
CT62	M	88	Han	C/T	G/G	A/T
CT63	M	78	Han	T/T	C/G	A/A
CT64	M	84	Han	C/T	C/G	A/T
CT65	M	56	Han	C/T	G/G	A/T
CT66	M	80	Han	T/T	G/G	A/T
CT67	M	80	Han	C/T	G/G	T/T
CT68	F	80	Han	C/C	G/G	A/A
CT69	M	58	Han	C/T	G/G	A/A
CT70	F	68	Han	C/T	G/G	A/A
CT71	M	89	Han	C/T	C/G	A/A
CT72	F	58	Han	C/T	C/G	A/A
CT73	F	68	Han	C/T	G/G	A/T
CT74	M	82	Han	T/T	G/G	A/A
CT75	F	67	Han	C/T	G/G	T/T
CT76	M	79	Han	C/T	C/G	A/A
CT77	F	83	Han	C/T	G/G	T/T
CT78	F	58	Han	T/T	G/G	A/T
CT79	F	82	Han	T/T	C/G	T/T

CT80	F	77	Han	T/T	G/G	A/A
CT81	M	57	Han	T/T	C/G	A/T
CT82	F	83	Han	C/C	G/G	A/A
CT83	M	85	Han	C/T	C/G	A/T
CT84	M	86	Han	C/T	G/G	A/T
CT85	M	83	Han	C/T	G/G	A/A
CT86	M	76	Han	C/T	G/G	A/A
CT87	F	63	Han	T/T	G/G	A/T
CT88	M	73	Han	C/T	G/G	T/T
CT89	M	77	Han	T/T	G/G	A/T
CT90	F	78	Han	C/T	G/G	A/A
CT91	F	66	Han	T/T	C/G	T/T
CT92	M	75	Han	T/T	C/G	T/T
CT93	F	78	Han	C/T	G/G	T/T
CT94	F	80	Han	C/T	G/G	T/T
CT95	M	65	Han	T/T	G/G	A/T
CT96	F	73	Han	T/T	G/G	A/A
CT97	M	78	Han	C/T	G/G	A/A
CT98	F	64	Han	C/C	G/G	A/T
CT99	M	77	Han	C/T	G/G	T/T
CT100	F	61	Han	T/T	C/G	T/T
CT101	F	72	Han	T/T	G/G	A/A
CT102	F	88	Han	T/T	C/G	A/T
CT103	F	64	Han	C/T	G/G	A/T
CT104	F	69	Han	T/T	G/G	T/T
CT105	F	54	Han	T/T	G/G	A/A
CT106	F	55	Han	C/T	G/G	A/A
CT107	F	79	Han	C/T	G/G	A/T
CT108	M	75	Han	C/C	C/G	A/T
CT109	M	50	Han	C/T	C/G	A/T
CT110	M	69	Han	T/T	C/G	A/A
CT111	F	82	Han	C/T	C/G	T/T
CT112	M	65	Han	C/T	G/G	T/T
CT113	M	71	Han	T/T	C/G	A/T
CT114	M	72	Han	C/T	C/G	A/T
CT115	M	69	Han	T/T	C/G	T/T
CT116	F	67	Han	T/T	G/G	A/T
CT117	M	78	Han	C/T	G/G	T/T
CT118	M	68	Han	T/T	G/G	T/T
CT119	F	50	Han	C/T	G/G	A/A
CT120	M	81	Han	T/T	C/G	T/T
CT121	M	56	Han	C/T	G/G	A/T
CT122	F	83	Han	C/T	C/G	A/T
CT123	F	54	Han	T/T	C/G	T/T
CT124	M	83	Han	C/T	G/G	A/T
CT125	M	79	Han	T/T	G/G	T/T
CT126	F	77	Han	T/T	C/G	T/T
CT127	F	55	Han	T/T	G/G	A/T
CT128	F	68	Han	T/T	G/G	A/T

CT129	F	68	Han	C/T	G/G	A/T
CT130	F	75	Han	C/T	G/G	A/T
CT131	F	79	Han	C/T	G/G	A/T
CT132	F	73	Han	C/T	G/G	A/A
CT133	M	78	Han	T/T	C/G	A/T
CT134	F	64	Han	T/T	G/G	A/A
CT135	M	74	Han	C/T	C/G	A/T
CT136	M	77	Han	C/T	G/G	A/A
CT137	F	73	Han	T/T	G/G	A/T
CT138	M	77	Han	T/T	C/G	A/T
CT139	M	79	Han	C/T	G/G	A/T
CT140	M	73	Han	C/T	C/G	A/T
CT141	F	66	Han	C/C	G/G	T/T
CT142	F	61	Han	T/T	C/G	A/T
CT143	F	81	Han	T/T	C/G	T/T
CT144	M	66	Han	T/T	G/G	T/T
CT145	F	74	Han	C/C	G/G	T/T
CT146	M	57	Han	C/T	C/G	A/A
CT147	F	50	Han	T/T	G/G	T/T
CT148	F	62	Han	C/T	C/G	A/T
CT149	F	56	Han	C/T	G/G	A/A
CT150	M	64	Han	C/T	C/G	A/A
CT151	F	61	Han	T/T	G/G	A/T
CT152	M	54	Han	C/T	G/G	A/A
CT153	M	59	Han	C/T	G/G	A/T
CT154	F	59	Han	C/C	C/G	A/T
CT155	F	67	Han	C/C	C/G	T/T
CT156	M	74	Han	T/T	G/G	A/T
CT157	F	64	Han	T/T	G/G	A/T
CT158	M	58	Han	T/T	G/G	A/T
CT159	F	85	Han	C/C	C/G	T/T
CT160	M	56	Han	C/T	G/G	A/A
CT161	F	59	Han	T/T	G/G	T/T
CT162	M	52	Han	T/T	C/G	T/T
CT163	F	59	Han	T/T	C/G	T/T
CT164	F	60	Han	T/T	G/G	A/T
CT165	M	57	Han	C/C	G/G	T/T
CT166	M	54	Han	C/T	G/G	A/T
CT167	F	65	Han	C/C	G/G	A/T
CT168	M	66	Han	C/T	C/G	T/T
CT169	F	70	Han	C/C	C/C	A/T
CT170	M	56	Han	T/T	C/G	A/T
CT171	F	53	Han	C/C	G/G	A/T
CT172	M	61	Han	C/C	G/G	A/T
CT173	M	65	Han	C/T	C/G	A/T
CT174	M	53	Han	C/T	G/G	T/T
CT175	F	61	Han	T/T	G/G	A/A
CT176	M	59	Han	T/T	G/G	A/T
CT177	M	55	Han	T/T	C/G	A/T

CT178	F	61	Han	T/T	G/G	A/T
CT179	M	58	Han	C/C	G/G	T/T
CT180	F	72	Han	T/T	G/G	A/T
CT181	F	56	Han	T/T	G/G	A/T
CT182	F	58	Han	T/T	G/G	A/T
CT183	F	76	Han	T/T	G/G	T/T
CT184	M	52	Han	T/T	G/G	A/T
CT185	M	66	Han	T/T	C/G	A/A
CT186	M	52	Han	T/T	G/G	A/T
CT187	M	72	Han	T/T	C/C	A/T
CT188	F	71	Han	C/T	G/G	T/T
CT189	M	53	Han	T/T	G/G	T/T
CT190	F	63	Han	T/T	C/G	A/T
CT191	F	62	Han	C/C	G/G	A/T
CT192	M	68	Han	T/T	G/G	T/T
CT193	M	61	Han	T/T	G/G	A/T
CT194	M	57	Han	T/T	G/G	A/A
CT195	F	73	Han	T/T	G/G	A/T
CT196	F	63	Han	C/T	G/G	T/T
CT197	F	54	Han	C/T	C/G	A/T
CT198	F	62	Han	C/T	G/G	A/T
CT199	F	58	Han	T/T	C/G	A/T
CT200	M	57	Han	T/T	C/G	A/T
CT201	M	64	Han	C/T	G/G	A/A
CT202	F	56	Han	T/T	C/G	T/T
CT203	F	55	Han	C/C	G/G	A/T
CT204	M	55	Han	C/T	C/G	T/T
CT205	M	60	Han	C/T	C/G	T/T
CT206	F	83	Han	C/T	C/G	A/A
CT207	F	55	Han	C/C	G/G	A/T
CT208	M	63	Han	C/T	C/G	T/T
CT209	F	54	Han	C/T	G/G	T/T
CT210	M	73	Han	C/T	G/G	T/T
CT211	M	60	Han	T/T	G/G	A/T
CT212	M	50	Han	T/T	C/G	T/T
CT213	F	66	Han	T/T	G/G	A/T
CT214	M	56	Han	T/T	G/G	T/T
CT215	M	64	Han	C/T	C/G	T/T
CT216	F	69	Han	T/T	G/G	T/T
CT217	F	77	Han	C/T	G/G	T/T
CT218	F	85	Han	C/C	C/G	A/A
CT219	F	66	Han	C/C	G/G	A/T
CT220	F	76	Han	C/C	C/G	A/T
CT221	F	74	Han	T/T	G/G	A/A
CT222	M	65	Han	T/T	C/G	A/T
CT223	F	68	Han	T/T	C/G	A/T
CT224	F	57	Han	C/T	G/G	T/T
CT225	M	54	Han	T/T	G/G	A/T
CT226	F	58	Han	C/T	G/G	A/A

CT227	M	53	Han	T/T	G/G	A/T
CT228	F	69	Han	T/T	C/G	A/T
CT229	F	66	Han	T/T	C/G	A/A
CT230	M	55	Han	C/T	G/G	A/T
CT231	F	65	Han	T/T	C/G	A/T
CT232	F	51	Han	C/T	G/G	T/T
CT233	M	79	Han	C/T	C/G	A/A
CT234	F	65	Han	C/T	G/G	T/T
CT235	M	59	Han	T/T	C/G	T/T
CT236	F	62	Han	T/T	C/G	A/T
CT237	F	62	Han	C/T	C/G	A/A
CT238	F	64	Han	T/T	G/G	A/T
CT239	M	66	Han	T/T	G/G	A/T
CT240	M	61	Han	T/T	G/G	A/T
CT241	F	80	Han	T/T	G/G	A/T
CT242	F	53	Han	C/T	G/G	T/T
CT243	F	80	Han	C/T	C/G	A/T
CT244	M	59	Han	C/T	C/G	A/A
CT245	F	78	Han	C/T	C/G	A/T
CT246	F	52	Han	T/T	C/G	A/T
CT247	M	68	Han	C/T	C/G	A/T
CT248	F	74	Han	C/T	G/G	A/T
CT249	M	54	Han	T/T	G/G	A/T
CT250	F	69	Han	C/T	C/G	A/T
CT251	F	57	Han	T/T	G/G	A/T
CT252	F	66	Han	C/T	G/G	A/T
CT253	F	51	Han	C/C	C/G	A/T
CT254	M	67	Han	C/C	G/G	A/T
CT255	F	52	Han	T/T	G/G	A/T
CT256	F	68	Han	C/T	C/G	A/T
CT257	M	69	Han	C/T	G/G	A/A
CT258	M	68	Han	T/T	C/G	A/T
CT259	F	57	Han	T/T	C/G	T/T
CT260	M	62	Han	T/T	G/G	A/T
CT261	F	76	Han	C/T	C/G	T/T
CT262	F	61	Han	C/T	G/G	A/T
CT263	M	62	Han	T/T	C/G	A/T
CT264	F	56	Han	C/T	G/G	T/T
CT265	F	81	Han	T/T	C/G	A/T
CT266	F	69	Han	C/T	G/G	A/T
CT267	M	63	Han	C/T	C/G	T/T
CT268	M	51	Han	T/T	G/G	T/T
CT269	M	61	Han	C/T	G/G	A/T
CT270	M	61	Han	C/T	G/G	A/A
CT271	M	60	Han	C/T	G/G	T/T
CT272	M	62	Han	C/T	G/G	A/T
CT273	F	71	Han	C/T	G/G	A/T
CT274	F	63	Han	C/T	G/G	A/A
CT275	M	53	Han	T/T	G/G	A/A

CT276	F	70	Han	T/T	C/G	A/A
CT277	M	53	Han	T/T	G/G	A/T
CT278	F	63	Han	C/T	G/G	A/T
CT279	F	82	Han	T/T	G/G	A/T
CT280	M	61	Han	T/T	C/G	A/A
CT281	M	53	Han	C/T	C/G	A/T
CT282	M	66	Han	T/T	C/C	T/T
CT283	F	61	Han	T/T	G/G	A/T
CT284	M	66	Han	C/T	C/C	A/T
CT285	M	59	Han	C/T	C/G	A/T
CT286	F	56	Han	C/T	G/G	A/A
CT287	F	85	Han	T/T	G/G	T/T
CT288	F	69	Han	T/T	C/C	T/T
CT289	M	57	Han	T/T	G/G	A/T
CT290	M	57.4	Han	C/T	G/G	A/A
CT291	F	54.7	Han	C/T	C/G	T/T
CT292	F	68.5	Han	T/T	G/G	A/T
CT293	F	78.2	Han	T/T	C/G	A/T
CT294	M	65.1	Han	T/T	G/G	T/T
CT295	M	55.9	Han	C/T	G/G	A/A
CT296	M	61	Han	T/T	C/G	A/A
CT297	M	64.7	Han	T/T	G/G	A/T
CT298	F	59.8	Han	T/T	G/G	A/T
CT299	M	57.9	Han	C/T	G/G	A/T
CT300	M	59.9	Han	T/T	C/C	A/A
CT301	M	55.8	Han	T/T	C/G	T/T
CT302	M	63.5	Han	T/T	C/G	A/T
CT303	F	55.9	Han	T/T	C/G	A/A
CT304	F	70.9	Han	T/T	C/G	T/T
CT305	M	55.8	Han	T/T	G/G	T/T
CT306	M	56.4	Han	C/C	G/G	A/A
CT307	M	62.8	Han	C/T	G/G	T/T
CT308	M	52	Han	T/T	C/G	T/T
CT309	M	51	Han	T/T	G/G	A/T
CT310	M	56.7	Han	C/T	C/G	T/T
CT311	F	53	Han	C/T	G/G	T/T
CT312	F	74.5	Han	C/T	C/G	T/T
CT313	M	60.6	Han	C/T	C/C	A/T
CT314	M	55.5	Han	C/T	G/G	A/T
CT315	M	58.1	Han	T/T	G/G	A/A
CT316	F	57.3	Han	T/T	C/G	T/T
CT317	M	59.8	Han	T/T	C/G	T/T
CT318	M	66.8	Han	C/T	C/G	T/T
CT319	M	58.8	Han	T/T	G/G	T/T
CT320	F	58.8	Han	C/T	G/G	T/T
CT321	F	70.4	Han	C/T	G/G	T/T
CT322	M	60.6	Han	C/T	G/G	T/T
CT323	F	55.1	Han	C/T	G/G	T/T
CT324	M	56.2	Han	T/T	C/G	A/A

CT325	F	64.3	Han	C/T	G/G	A/T
CT326	M	57.6	Han	T/T	G/G	T/T
CT327	M	62.2	Han	T/T	G/G	A/A
CT328	M	60.3	Han	T/T	C/G	A/A
CT329	M	56.5	Han	C/T	G/G	T/T
CT330	F	56.1	Han	T/T	G/G	A/A
CT331	M	55.5	Han	T/T	G/G	A/T
CT332	M	60.2	Han	T/T	G/G	A/T
CT333	F	57.1	Han	C/C	C/G	A/T
CT334	M	57.4	Han	T/T	C/G	A/A
CT335	M	66.1	Han	C/T	G/G	A/T
CT336	F	60.5	Han	C/T	C/G	T/T
CT337	F	58.4	Han	T/T	G/G	A/T
CT338	M	60.9	Han	C/T	C/G	A/T
CT339	F	54.8	Han	C/C	G/G	A/A
CT340	M	53.1	Han	C/T	G/G	T/T
CT341	F	67.2	Han	T/T	G/G	A/T
CT342	M	61.9	Han	C/C	C/C	A/T
CT343	F	58.6	Han	C/T	G/G	T/T
CT344	M	54.6	Han	C/T	C/G	A/T
CT345	M	54.3	Han	C/T	C/G	A/T
CT346	M	64.7	Han	C/T	G/G	T/T
CT347	F	56.1	Han	C/T	C/G	T/T
CT348	M	55.9	Han	C/T	C/G	A/A
CT349	M	56.9	Han	T/T	C/G	T/T
CT350	F	59.4	Han	C/T	C/C	A/T
CT351	M	60.1	Han	C/T	G/G	T/T
CT352	M	58	Han	T/T	G/G	A/A
CT353	M	56.1	Han	C/T	C/G	A/T
CT354	M	58	Han	T/T	C/G	T/T
CT355	M	55.6	Han	C/T	G/G	T/T
CT356	M	62	Han	T/T	C/G	A/T
CT357	M	59.8	Han	C/T	C/G	T/T
CT358	M	56.3	Han	C/C	G/G	T/T
CT359	M	65.4	Han	T/T	G/G	A/T
CT360	M	57.6	Han	C/T	C/G	T/T
CT361	M	59.7	Han	C/T	G/G	A/T
CT362	F	56.3	Han	C/T	G/G	A/A
CT363	M	58.3	Han	T/T	G/G	A/T
CT364	F	55.8	Han	C/C	G/G	T/T
PD1	M	78	Han	C/T	G/G	T/T
PD2	M	81	Han	C/T	G/G	A/T
PD3	M	54	Han	C/T	G/G	A/T
PD4	F	74	Han	T/T	G/G	A/T
PD5	M	52	Han	C/C	G/G	A/T
PD6	M	62	Han	T/T	C/G	T/T
PD7	M	81	Han	C/T	G/G	A/T
PD8	F	65	Han	C/C	C/G	A/T
PD9	F	61	Han	C/C	G/G	A/T
PD10	M	79	Han	T/T	G/G	T/T

PD11	F	57	Han	C/C	G/G	T/T
PD12	F	75	Han	T/T	G/G	T/T
PD13	F	72	Han	T/T	G/G	A/T
PD14	F	76	Han	C/C	G/G	A/T
PD15	F	70	Han	C/T	G/G	A/T
PD16	F	64	Han	C/T	G/G	A/T
PD17	F	56	Han	C/T	G/G	T/T
PD18	M	55	Han	C/T	C/G	T/T
PD19	F	61	Han	C/T	G/G	A/T
PD20	M	70	Han	C/T	G/G	T/T
PD21	M	71	Han	C/C	G/G	T/T
PD22	M	69	Han	C/T	C/G	T/T
PD23	F	53	Han	C/T	G/G	A/T
PD24	M	56	Han	T/T	C/G	T/T
PD25	M	67	Han	T/T	G/G	A/T
PD26	M	69	Han	T/T	C/G	A/A
PD27	M	76	Han	C/T	G/G	A/T
PD28	M	63	Han	C/T	G/G	A/A
PD29	M	68	Han	C/T	G/G	A/A
PD30	M	58	Han	T/T	G/G	A/T
PD31	M	76	Han	T/T	G/G	A/A
PD32	M	51	Han	T/T	G/G	T/T
PD33	M	72	Han	T/T	G/G	A/T
PD34	M	77	Han	C/T	G/G	T/T
PD35	M	51	Han	T/T	C/C	T/T
PD36	M	52	Han	C/T	C/C	T/T
PD37	F	65	Han	C/T	C/G	T/T
PD38	M	71	Han	C/T	G/G	T/T
PD39	F	54	Han	C/C	C/G	A/A
PD40	M	69	Han	C/C	G/G	A/T
PD41	F	68	Han	C/T	G/G	A/A
PD42	M	54	Han	T/T	C/G	A/A
PD43	F	69	Han	T/T	C/G	A/T
PD44	M	71	Han	C/T	G/G	A/T
PD45	F	75	Han	T/T	G/G	A/T
PD46	M	60	Han	T/T	C/G	A/A
PD47	F	78	Han	T/T	C/C	A/T
PD48	M	82	Han	C/T	G/G	T/T
PD49	M	78	Han	T/T	C/G	T/T
PD50	M	79	Han	C/T	C/G	T/T
PD51	F	53	Han	T/T	G/G	T/T
PD52	F	58	Han	T/T	C/G	A/A
PD53	F	55	Han	C/T	G/G	A/T
PD54	M	57	Han	C/T	C/G	T/T
PD55	M	61	Han	C/C	G/G	A/T
PD56	M	65	Han	C/T	C/G	A/A
PD57	M	61	Han	T/T	G/G	A/T
PD58	M	71	Han	T/T	G/G	A/A
PD59	M	75	Han	C/T	C/G	T/T
PD60	F	54	Han	C/T	G/G	T/T

PD61	F	74	Han	T/T	G/G	A/A
PD62	F	59	Han	C/T	G/G	A/T
PD63	F	66	Han	T/T	C/G	A/T
PD64	F	60	Han	C/T	C/G	T/T
PD65	F	57	Han	C/T	C/G	A/T
PD66	M	53	Han	T/T	G/G	T/T
PD67	F	71	Han	T/T	C/G	A/T
PD68	F	58	Han	C/T	C/G	A/T
PD69	F	77	Han	C/C	C/G	A/T
PD70	F	59	Han	T/T	G/G	A/A
PD71	F	60	Han	C/C	C/G	A/T
PD72	M	68	Han	C/T	G/G	A/A
PD73	M	69	Han	C/T	G/G	T/T
PD74	F	62	Han	T/T	C/G	A/T
PD75	M	60	Han	T/T	C/G	A/T
PD76	F	69	Han	T/T	G/G	A/T
PD77	M	79	Han	T/T	C/G	A/T
PD78	M	71	Han	T/T	G/G	A/T
PD79	F	66	Han	C/T	G/G	A/T
PD80	F	61	Han	C/T	C/G	A/T
PD81	F	58	Han	T/T	G/G	T/T
PD82	F	70	Han	T/T	G/G	A/T
PD83	M	62	Han	T/T	C/G	T/T
PD84	F	61	Han	C/T	C/G	T/T
PD85	M	77	Han	C/T	C/G	A/T
PD86	F	76	Han	T/T	G/G	A/T
PD87	F	69	Han	C/T	G/G	A/T
PD88	F	57	Han	C/T	G/G	T/T
PD89	M	74	Han	C/T	C/G	A/A
PD90	F	66	Han	C/T	G/G	T/T
PD91	M	50	Han	C/C	C/G	T/T
PD92	M	64	Han	C/T	C/G	T/T
PD93	F	67	Han	C/T	C/G	A/T
PD94	F	70	Han	T/T	C/G	T/T
PD95	M	63	Han	C/C	C/G	A/T
PD96	F	52	Han	T/T	C/G	A/T
PD97	M	64	Han	C/T	G/G	T/T
PD98	F	57	Han	T/T	G/G	A/T
PD99	F	59	Han	T/T	C/C	A/T
PD100	F	74	Han	C/T	G/G	A/T
PD101	M	72	Han	C/T	G/G	A/A
PD102	F	50	Han	C/T	G/G	A/A
PD103	M	51	Han	C/T	G/G	T/T
PD104	F	63	Han	T/T	C/C	A/T
PD105	M	66	Han	C/T	C/G	T/T
PD106	F	60	Han	T/T	G/G	A/T
PD107	M	75	Han	T/T	G/G	T/T
PD108	F	73	Han	C/T	C/G	T/T
PD109	F	68	Han	T/T	G/G	T/T

PD110	M	73	Han	C/T	C/G	T/T
PD111	F	68	Han	T/T	G/G	T/T
PD112	F	54	Han	C/T	G/G	A/T
PD113	M	60	Han	T/T	G/G	A/T
PD114	M	74	Han	T/T	C/G	T/T
PD115	M	68	Han	T/T	C/C	A/T
PD116	M	60	Han	T/T	C/G	A/T
PD117	F	55	Han	C/C	C/G	A/T
PD118	M	73	Han	C/C	G/G	A/A
PD119	F	68	Han	T/T	G/G	A/A
PD120	M	66	Han	T/T	G/G	A/T
PD121	M	63	Han	T/T	C/G	A/T
PD122	M	56	Han	T/T	C/G	A/T
PD123	M	67	Han	C/T	G/G	A/T
PD124	F	57	Han	C/C	C/G	A/T
PD125	M	56	Han	C/T	C/G	A/T
PD126	F	78	Han	C/C	G/G	A/T
PD127	M	54	Han	T/T	G/G	A/T
PD128	F	59	Han	C/C	C/G	A/T
PD129	M	72	Han	C/T	C/G	A/T
PD130	F	72	Han	T/T	G/G	T/T
PD131	F	59	Han	C/T	G/G	A/A
PD132	F	54	Han	T/T	C/C	T/T
PD133	M	75	Han	C/T	C/G	A/T
PD134	M	78	Han	T/T	G/G	A/A
PD135	M	72	Han	T/T	C/G	A/T
PD136	F	68	Han	C/T	C/G	A/T
PD137	M	56	Han	T/T	C/G	A/A
PD138	F	71	Han	C/T	G/G	A/A
PD139	M	81	Han	C/T	C/G	T/T
PD140	M	55	Han	C/T	C/G	A/A
PD141	M	50	Han	T/T	C/G	A/T
PD142	M	54	Han	C/C	G/G	A/A
PD143	M	51	Han	C/T	G/G	T/T
PD144	F	59	Han	C/C	C/G	T/T
PD145	F	57	Han	C/T	G/G	A/T
PD146	F	67	Han	C/T	G/G	T/T
PD147	F	72	Han	C/T	G/G	A/T
PD148	M	72	Han	T/T	G/G	T/T
PD149	F	57	Han	C/T	C/G	T/T
PD150	M	58	Han	C/T	C/G	A/A
PD151	M	72	Han	T/T	C/G	T/T
PD152	M	72	Han	T/T	C/G	T/T
PD153	F	65	Han	C/T	G/G	A/A
PD154	M	52	Han	C/T	C/G	A/T
PD155	F	66	Han	C/T	C/G	T/T
PD156	F	71	Han	C/C	G/G	T/T
PD157	F	62	Han	T/T	G/G	A/T
PD158	F	55	Han	T/T	G/G	T/T

PD159	M	61	Han	C/T	C/G	T/T
PD160	F	58	Han	C/C	G/G	A/A
PD161	M	57	Han	C/T	G/G	T/T
PD162	M	85	Han	C/C	G/G	A/A
PD163	F	59	Han	C/C	G/G	A/T
PD164	F	59	Han	T/T	C/G	A/T
PD165	F	53	Han	C/C	G/G	A/T
PD166	M	75	Han	C/T	G/G	A/T
PD167	M	54	Han	C/T	G/G	A/T
PD168	F	74	Han	C/T	G/G	A/T
PD169	F	53	Han	C/T	C/G	A/T
PD170	M	50	Han	T/T	C/G	A/T
PD171	F	62	Han	T/T	G/G	T/T
PD172	M	70	Han	T/T	G/G	A/T
PD173	F	81	Han	T/T	G/G	T/T
PD174	M	77	Han	T/T	C/G	A/T
PD175	M	56	Han	T/T	G/G	A/T
PD176	M	58	Han	C/T	C/G	A/A
PD177	M	53	Han	C/T	G/G	T/T
PD178	M	56	Han	C/T	G/G	A/A
PD179	F	66	Han	C/C	G/G	A/T
PD180	M	51	Han	C/T	G/G	A/T
PD181	F	55	Han	C/C	C/C	A/T
PD182	M	69	Han	C/T	C/G	T/T
PD183	F	78	Han	C/T	G/G	T/T
PD184	M	79	Han	C/C	G/G	A/A
PD185	F	72	Han	T/T	G/G	T/T
PD186	F	58	Han	C/T	C/G	A/T
PD187	M	73	Han	C/T	G/G	A/A
PD188	M	73	Han	C/T	C/G	A/A
PD189	F	77	Han	T/T	G/G	A/T
PD190	M	77	Han	C/T	G/G	A/T
PD191	F	77	Han	T/T	C/G	A/A
PD192	M	52	Han	C/T	G/G	A/A
PD193	M	58	Han	C/T	G/G	A/T
PD194	F	58	Han	C/T	C/G	A/T
PD195	F	79	Han	T/T	G/G	A/T
PD196	M	67	Han	C/T	G/G	A/T
PD197	F	67	Han	T/T	C/G	A/T
PD198	M	61	Han	T/T	G/G	T/T
PD199	M	66	Han	C/T	G/G	A/T
PD200	M	57	Han	T/T	G/G	A/T
PD201	F	79	Han	T/T	G/G	T/T
PD202	M	61	Han	C/T	G/G	A/T
PD203	F	58	Han	T/T	G/G	T/T
PD204	F	69	Han	C/T	C/G	A/A
PD205	M	73	Han	C/T	G/G	A/T
PD206	M	56	Han	T/T	G/G	A/T
PD207	M	70	Han	C/T	C/G	A/T
PD208	M	56	Han	C/T	C/G	T/T

PD209	F	57	Han	T/T	G/G	A/T
PD210	F	56	Han	T/T	C/C	A/T
PD211	F	68	Han	T/T	G/G	T/T
PD212	F	51	Han	C/T	C/C	A/T
PD213	M	76	Han	C/T	G/G	A/T
PD214	F	50	Han	C/T	G/G	A/A
PD215	F	55	Han	C/T	C/G	A/A
PD216	M	53	Han	C/C	G/G	A/T
PD217	M	70	Han	T/T	G/G	A/A
PD218	F	52	Han	C/T	C/G	T/T
PD219	F	52	Han	C/T	G/G	A/T
PD220	F	56	Han	T/T	G/G	A/T
PD221	F	54	Han	T/T	G/G	A/T
PD222	F	65	Han	C/T	G/G	T/T
PD223	F	61	Han	T/T	G/G	T/T
PD224	M	65	Han	T/T	G/G	A/T
PD225	F	54	Han	T/T	G/G	T/T
PD226	M	71	Han	C/T	G/G	T/T
PD227	M	60	Han	T/T	G/G	A/T
PD228	F	77	Han	C/T	C/C	A/T
PD229	F	50	Han	C/T	C/G	A/T
PD230	M	56	Han	T/T	G/G	A/T
PD231	M	71	Han	C/C	C/C	A/T
PD232	M	66	Han	C/T	G/G	A/T
PD233	F	59	Han	C/T	C/G	T/T
PD234	M	62	Han	T/T	G/G	T/T
PD235	M	65	Han	C/T	G/G	A/T
PD236	F	58	Han	C/C	C/G	T/T
PD237	M	55	Han	C/T	G/G	A/T
PD238	M	53	Han	T/T	G/G	A/T
PD239	F	73	Han	C/T	C/G	T/T
PD240	F	67	Han	C/T	C/G	A/T
PD241	F	54	Han	T/T	G/G	A/T
PD242	M	77	Han	C/C	G/G	A/T
PD243	M	53	Han	T/T	C/G	T/T
PD244	F	66	Han	C/T	C/G	T/T
PD245	F	69	Han	C/T	G/G	A/T
PD246	F	71	Han	C/T	C/G	A/T
PD247	F	68	Han	C/T	C/G	A/A
PD248	M	59	Han	C/T	G/G	A/T
PD249	F	58	Han	T/T	G/G	T/T
PD250	M	51	Han	C/T	G/G	A/A
PD251	M	54	Han	C/T	C/G	A/T
PD252	M	54	Han	C/T	C/C	T/T
PD253	F	63	Han	T/T	G/G	T/T
PD254	M	57	Han	T/T	G/G	A/T
PD255	M	58	Han	C/T	G/G	T/T
PD256	M	69	Han	C/T	G/G	T/T
PD257	F	55	Han	C/T	G/G	A/T
PD258	M	61	Han	T/T	C/G	A/A

PD259	F	67	Han	T/T	G/G	T/T
PD260	F	51	Han	T/T	G/G	A/T
PD261	M	53	Han	T/T	C/G	T/T
PD262	F	54	Han	C/T	G/G	T/T
PD263	M	66	Han	T/T	G/G	A/T
PD264	F	59	Han	C/T	G/G	A/T
PD265	M	52	Han	C/T	G/G	T/T
PD266	F	62	Han	T/T	G/G	A/T
PD267	M	58	Han	C/T	G/G	A/T
PD268	M	72	Han	T/T	G/G	T/T
PD269	M	69	Han	C/T	G/G	A/T
PD270	F	62	Han	C/T	G/G	A/T
PD271	M	51	Han	T/T	G/G	A/T
PD272	F	59	Han	C/C	G/G	A/T
PD273	F	66	Han	C/T	C/G	A/A
PD274	M	78	Han	C/T	G/G	A/T
PD275	F	63	Han	C/T	C/G	A/T
PD276	M	73	Han	C/T	C/C	A/T
PD277	F	70	Han	C/T	G/G	T/T
PD278	F	74	Han	T/T	C/G	A/T
PD279	F	74	Han	C/C	C/G	T/T
PD280	F	74	Han	C/T	G/G	T/T
PD281	M	71	Han	C/T	G/G	T/T
PD282	F	76	Han	T/T	G/G	A/A
PD283	M	53	Han	T/T	C/G	T/T
PD284	F	63	Han	T/T	C/C	A/A
PD285	F	50	Han	C/T	C/G	A/T
PD286	F	62	Han	C/T	C/G	A/T
PD287	F	68	Han	C/T	C/G	A/T
PD288	M	53	Han	C/T	C/G	A/T
PD289	F	76	Han	C/T	G/G	A/A
PD290	F	77	Han	T/T	G/G	A/A
PD291	F	62	Han	C/C	C/G	A/T
PD292	F	68	Han	C/T	C/G	A/T
PD293	M	77	Han	T/T	C/G	A/T
PD294	M	59	Han	C/T	G/G	A/T
PD295	M	69	Han	C/T	C/G	A/T
PD296	F	64	Han	C/T	G/G	T/T
PD297	M	55	Han	C/T	G/G	A/T
PD298	F	67	Han	C/T	G/G	A/T
PD299	F	57	Han	C/T	G/G	A/T
PD300	F	59	Han	T/T	G/G	T/T
PD301	M	55	Han	T/T	G/G	A/A
PD302	M	68	Han	T/T	C/G	A/T
PD303	M	56	Han	C/T	C/G	A/T
PD304	F	63	Han	C/T	C/G	A/A
PD305	F	72	Han	T/T	G/G	T/T
PD306	F	58	Han	C/T	C/G	A/A
PD307	M	62	Han	C/T	C/G	T/T
PD308	F	64	Han	T/T	C/G	A/A

PD309	M	52	Han	C/T	G/G	A/T
PD310	F	62	Han	T/T	G/G	A/T
PD311	M	65	Han	C/C	G/G	A/T
PD312	M	68	Han	T/T	C/G	T/T
PD313	M	58	Han	T/T	G/G	A/T
PD314	F	52	Han	C/T	G/G	A/T
PD315	F	75	Han	T/T	C/G	T/T
PD316	F	73	Han	T/T	G/G	A/T
PD317	F	82	Han	T/T	G/G	T/T
PD318	F	66	Han	T/T	C/G	A/T
PD319	M	72	Han	C/C	C/G	T/T
PD320	M	66	Han	C/C	C/G	A/T
PD321	M	60	Han	C/T	C/G	T/T
PD322	M	79	Han	C/T	G/G	T/T
PD323	F	62	Han	T/T	C/G	A/A
PD324	F	59	Han	C/T	G/G	A/T
PD325	F	65	Han	C/T	C/G	A/T
PD326	F	63	Han	C/C	G/G	A/T
PD327	F	57	Han	C/T	G/G	T/T
PD328	F	57	Han	C/T	C/G	A/T
PD329	F	78	Han	T/T	G/G	A/A
PD330	F	50	Han	C/T	G/G	A/T
PD331	M	59	Han	T/T	G/G	A/T
PD332	M	82	Han	C/T	C/G	A/T
PD333	M	70	Han	C/T	C/G	A/T
PD334	F	54	Han	T/T	C/G	T/T
PD335	F	55	Han	T/T	G/G	T/T
PD336	F	61	Han	T/T	G/G	A/T

CT: control; PD: PD patient.