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ORIGINAL ARTICLE

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Gene frequencies of the HPA-1 to HPA-8w platelet antigen alleles in Taiwanese, Indonesian, and Thai

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Abstract Human platelet antigen (HPA) systems consist of more than eight biallelic antigen polymorphisms in which a base pair substitution leads to change in an amino acid of a glycoprotein expressed on the platelet. HPA typing is essential in the diagnosis and treatment for a variety of diseases. We developed a polymerase chain reaction (PCR)-based method to detect HPA-1 through HPA-8w. In this method, the amplified PCR products were used to recognize the polymorphism after restriction enzyme digestions. Among 295 Taiwanese, 107 Indonesian, and 137 Thai subjects studied, HPA-1a, 2a, 4a, 5a, 6a, 7aw, and 8aw genes were present in every sample tested. HPA-1b, 2b, 4b, 5b, and 6b were rarely found among subjects. Only monomorphic HPA-7aw and 8aw alleles were noted in the samples. HPA-3a and 3b alleles showed frequencies of 0.595/0.405, 0.504/0.496, and 0.507/0.493 in Taiwanese, Indonesian, and Thai subjects, respectively. Our report is the first PCR-based method to detect most of the HPA antigen variants in Taiwanese, Indonesian, and Thai. The genomic typing results were also confirmed by direct sequencing for uncertain and some representative cases. The prevalence rates of HPA-1, 2, 3, 4, and 5 in this study were also consistent with other previous reports using different methods.

Keywords Human platelet antigen (HPA) · PCR-RFLP · Taiwanese · Indonesian · Thai

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Introduction

Human platelet membrane glycoprotein (GP) bears genetically determined alloantigens. All human platelet antigens (HPA) are important in neonatal alloimmune thrombocytopenia (NAIT), post-transfusion purpura (PTP), refractoriness to platelet transfusion therapy, coronary thrombosis, coronary stent thrombosis, and population genetics [16, 17, 19, 33, 34, 35]. So far, more than eight HPA systems have been described with the notation "a" for the common and "b" for the rare alleles [18, 32]. The molecular basis of these polymorphisms was identified as single-base changes, which led to single amino acid differences on the four major platelet membrane GP: GPIIb (HPA-3), GPIIIa (HPA-1, 4, 6, 7w, and 8w), GP1b (HPA-2), and GP1a (HPA-5) [18, 20]. Frequencies of platelet antigen vary between different populations [5, 9, 10, 11, 14, 23, 25, 29, 31]. Most studies only analyzed frequencies of part of HPAs [5, 9, 10, 11, 14, 22, 23, 25, 29, 31], and no data have been reported for HPA-1 to HPA-8w alleles in Taiwanese or Indonesian, HPA-7w and HPA-8w in Thai. We analyzed these alleles by polymerase chain reaction (PCR) and allele-specific restriction enzyme digestion (PCR-RFLP).

Materials and methods

Subjects

Included in the study were 295 nonrelated healthy Taiwanese, 107 Indonesian, and 137 Thai from whom 3 ml of EDTA-anticoagulated blood was collected as previously described [3].

DNA amplification and restriction enzyme analysis

Total genomic DNA was isolated from peripheral blood leukocytes of the subjects as described previously [2]. The strategy of oligonucleotide primer design and restriction enzyme analyses was the same as that presented in previous studies [2, 3] and is shown in Table 1. Some polymorphisms do not have any available restriction site in the gene; in addition, the restriction enzyme for some original authentic restriction sites is too expensive. Conse-

quently, one aim of this study was to design an accurate and affordable method to identify the subtypes of HPA by creating new restriction sites to differentiate various polymorphisms. For the HPA-1 polymorphism, there is a natural *Msp*I site for HPA-1b. The HPA-2a polymorphism, in which the polymorphism base C of codon 145 (ACG) of GP1b gene with the mutagenic base G at codon 146 (CCC→CGC) introduced to the third base from the 3′ end of the downstream primer created a *BstU*I restriction site (CGCG) after PCR reaction. There is an authentic *Fok*I site in the HPA-3a polymorphism. For the HPA-4a polymorphism, the mutagenic base C at codon 142 (ATG→ACG) of GPIIIa gene introduced by an upstream primer with mutagenic base C at the second base from the 3′ end with the polymorphism base G of codon 143 created a *BstU*I site. The HPA-5a polymorphism base G of codon 505 (GAG) of GPIa gene, with the mutagenic base T at codon 504 (AAA→TAA) introduced by an upstream primer developed a *Dde*I site (CTNAG). For the analysis of HPA-6b, the polymorphism base A at codon 489 of GPIIIa gene with the mutagenic base GG at codon 492 (CAG→GGG) introduced by a downstream primer had a restriction site CCANNNNNNTGG of *BstX*I. The HPA-7aw polymorphism C at codon 407 of GPIIIa gene with the mutagenic base G at codon 406 (TGT-GGT) introduced by an upstream primer with the mutant base at third base from the 3′ end created an *Ava*II site and an internal authentic *Ava*II site in the amplified region. For analyzing HPA-8aw, the polymorphism C at the first base of codon 636 of GPIIIa gene with the mutagenic base C at the third base of the same codon introduced by a downstream primer, hence developing the *BstU*I site. Several cases of different types of HPA-1 to HPA-8w were further checked by direct sequencing to confirm the results.

The DNA amplification was performed as described previously [2, 3], with some modification. Briefly, 500 ng of genomic DNA was mixed with 100 ng of each primer and 200 μmol/l of each dNTP in 100 μl reaction buffer containing 0.01% gelatin, 1.5 mmol/l $MgCl_2$, 50 mmol/l KCl, 10 mmol/l Tris-HCl (pH 8.3), and 2.5 units of Taq polymerase. PCR amplification was carried out using a DNA thermal cycler; the procedure was denaturation at 94°C for 2 min, the annealing temperature was modified based on the Tm (melting temperature) of the primers for 2 min (Table 1), and extension at 72°C for 3 min. The amplified products were digested with an appropriate restriction enzyme for overnight (Table 1), followed by electrophoreses in 2.5–3.5% agarose gels.

For uncertain cases, the PCR products were further analyzed using direct sequencing method to confirm the results. The positive and negative controls from the PCR reactions were also confirmed by direct sequencing. Direct sequencing was done as previously described [36].

Results

The results of the patterning change of different type of the HPA-1 to HPA-8w after restriction enzyme digestion are shown in Fig. 1 and Table 1. In the analysis of the

Table 1 Primer sequences for the HPA system. *U* upstream primer, *D* downstream primer. Mutagenic bases are underlined

	Primer sequence	Annealing temperature	Restriction enzyme	Size changes after digestion
HPA-1 GPIIIa	U: 5′-TCTTTGGGCTCCTGACTTAC-3'	56°C	*Msp*I	193 bp for 1a
Codon 33 CTG→CCG	D: 5′-CTGGGGACTGACTTGAGTGA-3′			161 bp, 32 bp for 1b
HPA-2 GPIb	U: 5′-CCACAATCAGCTGCAAAGCC-3′	60°C	*BstU*I	181 bp, 19 bp for 2a
Codon 145 ACG→ATG	D: 5′-TCTCCAGCTTGGGTGTGCGC-3′			200 bp for 2b
HPA-3 GPIIb	U: 5′-TGGAAGAAAGACCTGGGAAGG-3′	60°C	*Fok*I	115 bp, 112 bp for 3a
Codon 843 ATC→AGC	D: 5′-GAGAACTGGATCCTGAAGCC-3′			227 bp for 3b
HPA-4 GPIIIa	U: 5′-CAAGCTGGCCACCCAGACG-3′	56°C	*BstU*I	150 bp, 19 bp for 4a
Codon 143 CGA→CAA	D: 5′-GGCAAAGGATGCTGTCCTG-3′			169 bp for 4b
HPA-5 GPIa	U: 5′-AGGAAGAGTCTACCTGTTTACTATCTAA-3′	60°C	*Dde*I	231 bp, 25 bp for 5a
Codon 505 GAG→AAG	D: 5′-CTCTCATGGAAAATGGCAGTA-3′			256 bp for 5b
HPA-6 GPIIIa	U: 5'-AGGCCAAGGTGCGAGGCGGT-3′	66°C	*BstX*I	295 bp for 6a
Codon 489 CGA→CAA	D: 5′-CGCTGGCTGCAGACGGGCCCACCCTC–3′			273 bp, 22 bp for 6b
HPA-7w GPIIIa	U: identical to HPA-6	66°C	*Ava*II	142 bp, 135 bp, 18 bp for 7aw
Codon 407 CCC→GCC	D: identical to HPA-6			160 bp, 135 bp for 7bw
HPA-8w GPIIIa	U: 5′-GTCCTCTCTCCTTCAGAGAATG-3′	58°C	*BstU*I	88 bp,19 bp for 8aw
Codon 636 CGT→TGT	D: 5′-CACTGACTCAATCTCGTCGC-3′			107 bp for 8bw

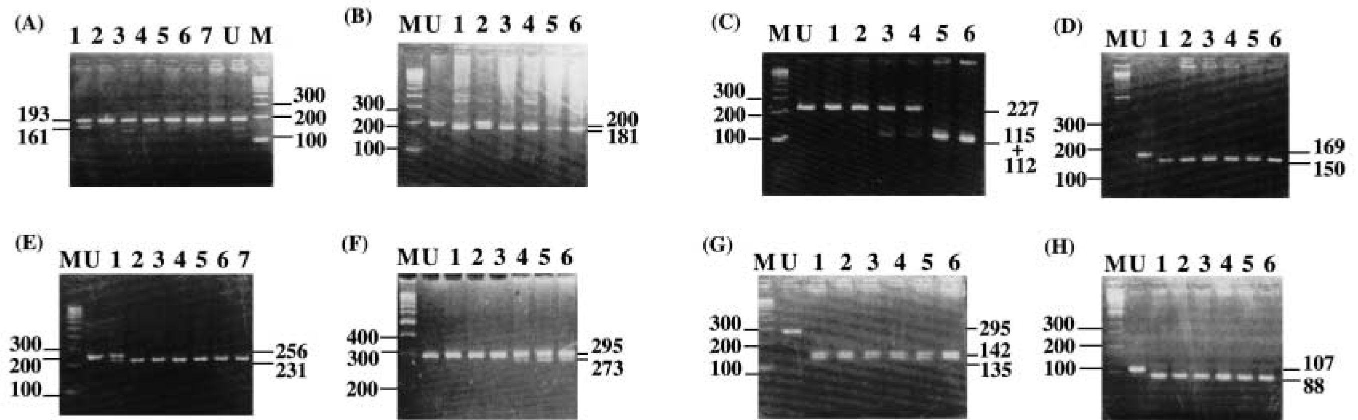

Fig. 1A–H The results of size change after appropriated restriction enzyme digestion for HPA-1 to HPA-8w are shown. **A** The HPA-1 PCR products after *Msp*I digestion. Lane 1 is HPA-1a/1b, and lanes 2–7 are HPA-1a/1a. **B** The HPA-2 PCR products after *BstU*I digestion. Lane 2 is HPA-2a/2b, and lanes 1 and 3–6 are HPA-2a/2a. **C** *Fok*I digestion of HPA-3 products. Lanes 1–2 are HPA-3b/3b, lanes 3–4 are HPA3a/3b, and lanes 5–6 are HPA-3a/3a. **D** *BstU*I digestion of HPA-4 products. Lanes 2–6 are HPA-4a/4a **E** *Dde*I digestion of HPA-5 products. Lane 1 is HPA-5a/5b, and lanes 2–6 are HPA-5a/5a. **F** *BstX*I digestion of HPA-6 products. Lanes 1–3 are HPA-6a/6a, lanes 4–6 are HPA-6a/6b. **G** *Ava*II digestion of HPA-7w products. Lanes 1–6 are HPA-7aw/7aw. **H** *BstU*I digestion of HPA-8w products. Lanes 1–6 are HPA-8aw/8aw. *M* 100 bp ladder marker. *U* uncut control

Table 2 The gene frequencies of HPA-1 to HPA-8w in Taiwanese, Indonesian, and Thai

HPA polymorphism/Ethnic	Taiwanese (*n*=295)	Indonesian (*n*=107)	Thai (*n*=137)
HPA-1	a 589/590[a] (99.83%) b 1/590 (0.17%)	a 212/214[a] (99.1%) b 2/214 (0.9%)	a 270/274[a] (98.5%) b 4/274 (1.5%)
HPA-2	a 567/590 (96.1%) b 23/590 (3.9%)	a 201/214 (93.9%) b 13/214 (6.1%)	a 257/274 (93.8%) b 17/274 (6.2%)
HPA-3	a 351/590 (59.5%) b 239/590 (40.5%)	a 108/214 (50.5%) b 106/214 (49.5%)	a 139/274 (50.7%) b 135/274 (49.3%)
HPA-4	a 589/590 (99.83%) b 1/590 (0.17%)	a 214/214 (100%) b 0/214 (0%)	a 274/274 (100%) b 0/274 (0%)
HPA-5	a 579/590 (98.14%) b 11/590 (1.86%)	a 213/214 (99.5%) b 1/214 (0.5%)	a 264/274 (96.3%) b 10/274 (3.7%)
HPA-6	a 578/590 (98%) b 12/590 (2%)	a 207/214 (96.7%) b 7/214 (3.3%)	a 270/274 (98.5%) b 4/274 (1.5%)
HPA-7w	a 590/590 (100%)	a 214/214 (100%)	a 274/274 (100%)
HPA-8w	a 590/590 (100%)	a 214/214 (100%)	a 274/274 (100%)

[a] Chromosome

HPA-1 polymorphism, a 193 base pair (bp) fragment was amplified and an *Msp*I restriction site was found in the HPA-1b. An undigested 193 bp band was noted in the HPA-1a (Fig. 1A, lanes 2–7, homozygote), whereas the HPA-1b product was digested into 161 and 32 bp fragments (Fig. 1A, lane 1, heterozygote). The band less than 35 bp could not be visualized well in a 3.5% agarose gel. The amplified product was 200 bp for HPA-2. Two fragments (181 and 19 bp) were generated for HPA-2a (Fig. 1B, lanes 1 and 3–6, homozygote) and one fragment (200 bp) for HPA-2b (Fig. 1B, lane 2, heterozygote) after *BstU*I digestion. The PCR product of HPA-3 was 227 bp. The 227 bp fragment remained undigested after *Fok*I digestion in the HPA-3b (Fig. 1C, lanes 1and 2, homozygote). For the HPA-3a, it was digested to 115 bp and 112 bp, which were unable to be separated under 3.5% agarose gel electrophoresis (Fig. 1C, lanes 5 and 6, homozygote), and three fragments (227 bp, 115 bp, and 112 bp) for heterozygote (Fig. 1C, lanes 3 and 4). For HPA-4, the PCR product was 169 bp. The HPA-4b had an undigested 169 bp fragment after *BstUI* digestion (data not shown), but the HPA-4a was cleaved to 150 bp and 19 bp fragments (Fig. 1D, lanes 2–6, homozygote). The amplified product of HPA-5 was 256 bp. After *Dde*I digestion, an undigested 256 bp fragment was noted in the HPA-5b (Fig. 1E, lane 1, heterozygote), but HPA-5a was digested to 231 bp and 25 bp bands (Fig. 1E, lanes 2–7, homozygote). For HPA-6, the PCR product was 295 bp. After *BstX*I digestion, two fragments (273 bp and 22 bp) were found in the HPA-6b (Fig. 1F, lanes 4–6, heterozygote), but HPA-6a had an undigested 295 bp fragment (Fig. 1F, lanes 1–3, homozygote). In analyzing the HPA-7w, the amplified product was 295 bp, which is identical to the PCR product of HPA-6. After digestion by restriction enzyme *Ava*II, three fragments (142 bp, 135 bp, and 18 bp) were found in the HPA-7aw (Fig. 1G, lanes 1–6, homozygote), but it was digested to two fragments (160 bp and 135 bp) for

the HPA-7bw (data not shown). In order to detect the HPA-8w polymorphism, *BstU*I digestion of the PCR products from the HPA-8aw yielded two fragments (88 bp and 19 bp) (Fig. 1H, lanes 1–6, homozygote). However, the HPA-8bw showed an undigested 107 bp fragment (data not shown).

Among the 295 Taiwanese, 107 Indonesian, and 137 Thai studied, the gene frequencies are shown in Table 2. HPA-1a, 2a, 4a, 5a, 6a, 7aw, and 8aw genes were present in all samples tested. However, HPA-1b, 2b, 4b, 5b, and 6b were rarely found among subjects. HPA-3a and 3b alleles showed frequencies of 0.595/0.405, 0.504/0.496, and 0.507/0.493 in Taiwanese, Indonesian, and Thai populations. All the samples from the three populations were monomorphic for HPA-7aw and 8aw.

Discussion

To complement the basis of the DNA sequence polymorphism corresponding to platelet-specific antigens, various DNA-based HPA typing techniques have been developed [1, 3, 5, 6, 7, 8, 11, 12, 13, 15, 21, 24, 26, 29]. The HPA genotyping could be performed by PCR followed by restriction enzyme digestion (PCR-RFLP) [24], by the PCR and sequence-specific oligonucleotide probing [15] or by the PCR and reverse dot-blot hybridization [1], by the PCR and single-strand conformation polymorphism analysis [8, 21], by the sequence-specific PCR amplification [12, 26], by the PCR with preferential homoduplex formation assay [7], and by the multiplex PCR and ligation-based typing [13]. In this study, we modified the PCR-RFLP method and used it to analyze the gene frequencies for HPA-1 through HPA-8w in Taiwanese, Indonesian, and Thai. We found the HPA-1a, 2a, 4a, 5a, 6a, 7aw, and 8aw were the most common alleles, and the HPA-3a/3b genes showed nearly equally gene frequencies in these three populations. The prevalence rates of HPA-1, 2, 3, 4, and 5 in this study were also consistent with reports in the literature using different methods [4, 14]. Our method provides an alternative approach to complete typing most of the known HPAs.

The frequencies of platelet antigens vary between different populations. In Caucasians, there are higher gene frequencies for HPA-1b, 2b, and 5b than there are other populations [27]. In addition Blacks also have higher gene frequencies of HPA-1b and 2b [5]. There are higher frequencies for HPA-6b in Japanese and Korean [24, 28, 29, 30]; however, we found the same higher frequencies of HPA-6b in Taiwanese, Indonesian, and Thai as in Japanese and Korean populations. Our results add the information that Orientals have higher frequencies of HPA-6b. The frequencies of HPA-3a and 3b of the three populations in this study are consistent with other populations [4, 14] and indicates that both alleles are almost equally distributed in all populations. Furthermore, the gene frequencies of HPA-7bw and 8bw are very rare for all populations [18].

Studies of platelet-specific antigens and their genes in different populations provide valuable information when facing specific clinical conditions such as NAIT and PTP. This study is the first complete analysis of HPA-1 to HPA-8w gene frequencies in Taiwanese, Indonesian, and Thai populations. Our results would have some impact on the capabilities to diagnose alloimmune thrombocytopenia, the prevention of intracranial bleedings of the fetus, and providing more accurate treatment with platelet transfusions.

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