Glucose - 6 - Phosphate Dehydrogenase (G6PD) variants in East Sepik Province of Papua New Guinea: G6PD Jammu, G6PD Vanua Lava, and a novel variant (G6PD Dagua)

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Abstract:
A community-based survey was conducted to assess the prevalence of G6PD deficiency and malaria in two villages (St. Martins, Dagua) of Wewak district, East Sepik Province, Papua New Guinea. A total of 551 individuals were recruited for the study. Malaria was diagnosed in 96 cases (17.4%), and 28 subjects with G6PD deficiency (5.1%) were identified by the WST-8/1-methoxy PMS method. The sequence of the entire G6PD gene was determined in 19 of the 28 G6PD-deficient cases. We found ten cases with the G6PD Jammu variant (871 G>A, 1311 C) and eight cases with the G6PD Vanua Lava variant (383 T>C). A novel mutation (595 A>G) predicting an amino acid change from isoleucine to valine was identified. This mutation has not been reported to date and was named G6PD Dagua. The detection of three G6PD variants indicates the heterogeneity of the studied population, and these variants could be important genetic markers for clarifying the gene flow among the population of Southeast Asian and Pacific Island countries.

Keywords: G6PD deficiency, Papua New Guinea, Dagua, Jammu, Vanua Lava, Malaria.

INTRODUCTION
Glucose-6-phosphate dehydrogenase (G6PD) deficiency is one of the most common enzymopathies in humans, affecting over 400 million people worldwide. This disorder may cause many clinical manifestations, including neonatal jaundice, acute or chronic hemolytic anemia, and favism. In most cases, G6PD deficiency is caused by mutation(s) of the G6PD gene, which is located on the X染色体. The G6PD gene is 15,864 base-pair long, with 13 exons, of which exon 1 is a non-coding region for amino acids. To date, at least 140 G6PD variants have been reported worldwide from various populations [5]. In Southeast Asia and Melanesia, more than 20 G6PD variants have been documented, and, interestingly, the ancestral origin of the population can be traced by the analysis of G6PD variants [1, 8, 11, 12, 13, 15, 16, 17, 18, 20, 21, and 23].

Papua New Guinea (PNG) has the highest morbidity and mortality rates related to malaria among the Pacific Islands. Safe use of anti-malaria drugs is the most important strategy to prevent and control malaria transmission in a community. Primaquine, an anti-malaria drug, can be used to treat not only gametocytes (sexual stage) to Plasmodium falciparum (P. falciparum) but also anti-relapse (hypnozoite-cidal stage) to Plasmodium vivax (P. vivax). However, primaquine is a strong oxidant which may cause hemolysis in G6PD-deficient people taking the drug. Thus, primaquine should not be used for malaria patients before the G6PD enzyme activity status is first confirmed.

G6PD deficiency is widespread in PNG and its frequency among males ranges from 0% to more than 50% in Morobe Province [27]. Biochemical studies of G6PD deficiency in PNG have revealed a high degree of heterogeneity, with 24 phenotypes described to date [6, 28]. However, only one molecular study on G6PD has been conducted in PNG. This reported the detection of G6PD Kaiping and G6PD Union variants in two samples [28].

Therefore, we carried out a community-based survey...
to investigate the prevalence of G6PD deficiency and the sequence of the entire G6PD gene in East Sepik Province, PNG.

**MATERIALS AND METHODS**

**Study sites**

The survey was carried out in St. Martins and Dagua villages of the Wewak district, East Sepik Province which is located in the northwestern part of Papua New Guinea, in July and August 2003. The Dagua population speaks a Pap-

![Fig 1: Location of St. Martins and Dagua in Wewak District, East Sepik Province, Papua New Guinea](image)

### Table 1: Oligonucleotide primers for PCR and sequencing assays

<table>
<thead>
<tr>
<th>Exon</th>
<th>Primers</th>
<th>Amplicon length</th>
<th>Temperature cycling parameters</th>
<th>Cycles</th>
</tr>
</thead>
</table>
| Exon 2     | Ex2F: CAAGGAGTGATTGTGGAATG
Ex2R: CAGGGATTCCCGAGGAGTAG
SEQ/Ex2: CAGGGATTCCCGAGGAGTAG** | 420 bp          | 94 °C; 95 °C; 66 °C; 72 °C; 72 °C; 4 °C | 35     |
| Exon 3 & 4 | Ex3&4F: CCTTGTGGCCAGTATGTAG
Ex3&4R: AGGAGGAGGAGGAGGAGCATCC
SEQ/Ex3, 4: GCTTGTGGCCAGTATGTAG** | 466 bp          | 94 °C; 95 °C; 66 °C; 72 °C; 72 °C; 4 °C | 35     |
| Exon 5     | B1: AGGATGATGTATGATGATG
B4: CCCTGACGCCTATAG
SEQ/Ex 5: GGGGACACTGACTTCTGAG** | 1,155 bp        | 94 °C; 95 °C; 56 °C; 72 °C; 72 °C; 4 °C | 35     |
| Exon 6 to 8| B5a: AGGAGGATCTGCGCTCTACTCC
B8: TGCCCTGTCACAGATGGGCC
SEQ/Ex6, 7: AGGAGGATCTGCGCTCTACTCC
SEQ/Ex8: TTAATGATCGAGATGAC** | 1,093 bp        | 94 °C; 95 °C; 72 °C; 72 °C; 72 °C; 4 °C | 40     |
| Exon 9 to 11| B9: TCCCTGCACCCCAACTCAAC
B12: ATGGAGATCTCCTCCACCTCA
SEQ/Ex9: GCCCGCTGACACCCAGCTCT
SEQ/Ex10, 11: ACTGGAGATCTCCTCCACCTGAG** | 1,069 bp        | 94 °C; 95 °C; 68 °C; 72 °C; 72 °C; 4 °C | 40     |
| Exon 12 & 13| RY: GCACATCTGGGCTATAG
21: AGAATGATGCTGAGGAGCTCAAT
SEQ/Ex12, 13: GCACATCTGGGCTATAG** | 454 bp          | 94 °C; 95 °C; 60 °C; 72 °C; 72 °C; 4 °C | 40     |

**Note**

(*) Primers published [3, 10]
(**) Primers used for sequencing
uan language and lives in the mainland along the coast, while the St. Martins population speaks an Austronesian language and lives on islands (Fig 1).

Field survey

A total of 551 male individuals (256 in St. Martins and 295 in Dagua) aged from 1 to 73 years old were recruited for the study. Blood was taken by finger prick for microscopic examination of the malaria parasite and for measurement of hemoglobin concentration and G6PD enzyme activity. Malaria parasites were identified in blood smears by experienced microscopists, and the hemoglobin concentration was measured with a HemoCue B-Haemoglobin analyzer (Angelhorm, Sweden). G6PD deficiency was detected by the WST-8/1-methoxy PMS method \[25\].

G6PD mutational analysis

A total of 28 blood-blotted filter papers from G6PD-deficient individuals were used to analyze the enzyme at the molecular level. However, only 19 samples (13 from St. Martins and 6 from Dagua) had enough DNA for the sequencing process. Genomic DNAs were extracted from the collected samples using a QIAamp DNA blood mini kit in accordance with the manufacturer’s instructions (QIAGEN, Germany). Since the genomic G6PD DNA consists of 13 exons with the coding regions located in exons 2-13 and is 15,864 base-pair long, we used the polymerase chain reaction (PCR) method to amplify six segments. The PCR primers and temperature cycling parameters for each reaction are shown in Table 1. The typical PCR reaction was done in a 25-µl reaction mixture containing: 1 µl of template DNA, 500 nM of each primer, 2 mM MgCl₂, 1 x PCR buffer, 200 µM of each dNTPs, and 0.75 U of Taq polymerase. Polymerase chain reaction was performed on the DNA thermal cycler (Eppendorf EP gradient S, Germany). Then, the reactive products were precipitated with ethanol to remove the unincorporated dye-labeled terminators. Samples were finally dissolved in a loading solution for separation and detection using the MegaBACE 1000 sequencing machine (GE Healthcare UK Ltd). Sequence analysis was performed using the MegaBACE sequencing analysis software, Ver 3.0.

Ethical considerations

This study was approved by the National Department of Health Medical Research Advisory Committee of Papua New Guinea and the Ethics Committee of Tokyo Women’s Medical University (approved number: 109). Written informed consent was obtained from each adult individual; while in the case of children, it was obtained from the guardian.

Statistical analysis

Student’s t-test and Fisher’s exact test were used in this study, P values of less than 0.05 were considered to be statistically significant.

RESULTS

The prevalence of malaria and G6PD deficiency in the study population are summarized in Table 2. Malaria infection was diagnosed in 96 cases (17.4%) and the malaria prevalence rate was 15.6% and 18.9% in the St. Martins and Dagua villages, respectively, with no significant difference between the two groups. 

Table 2: Distribution of malaria and G6PD deficiency in two studied villages

<table>
<thead>
<tr>
<th>Villages</th>
<th>Numbers</th>
<th>Malaria positive (%)</th>
<th>G6PD deficiency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pf (%)</td>
<td>Pf+m (%)</td>
</tr>
<tr>
<td>St. Martins</td>
<td>256</td>
<td>28 (10.9)</td>
<td>1 (0.4)</td>
</tr>
<tr>
<td>Dagua</td>
<td>295</td>
<td>34 (11.5)</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Total</td>
<td>551</td>
<td>62 (11.3)</td>
<td>2 (0.4)</td>
</tr>
</tbody>
</table>

Note: 
Pf = P. falciparum 
P.m = P. malariae 
P.f+m = P. falciparum + P. malariae 
P.v = P. vivax

cycler (Eppendorf EP gradient S, Germany). Then, the reactive products were precipitated with ethanol to remove the unincorporated dye-labeled terminators. Samples were finally dissolved in a loading solution for separation and detection using the MegaBACE 1000 sequencing machine (GE Healthcare UK Ltd). Sequence analysis was performed using the MegaBACE sequencing analysis software, Ver 3.0.

Mixed infection with Plasmodium malariae (P. malariae) and P. falciparum was also observed, but at a low prevalence. A total of 28 out of the 551 subjects were found to be suffering from G6PD deficiency (5.1%). The G6PD deficient prevalence was 5.5% and 4.8% in the St. Martins and Dagua villages, respectively, with no significant difference between the two groups. Infection of P. falciparum was detected in three out of the 28 G6PD-deficient individuals and 68 out of the 523 individuals with normal G6PD activity. The prevalence of P. falciparum did not differ significantly between the
G6PD deficiency and normal G6PD activity groups (P > 0.05). The hemoglobin concentration measured in 426 out of 551 study individuals ranged from 5.9 to 17.7 g/dl (mean ± SD: 10.7 ± 1.7 g/dl). Mean hemoglobin levels in the G6PD deficient and normal G6PD groups were 11.5 ± 2.1 and 10.7 ± 1.7 g/dl, respectively, with no significant difference observed between the two groups (P>0.05). Anemia (hemoglobin < 11g/dl) was detected in 253 cases (59.4%), and the prevalence of anemia in normal G6PD and G6PD deficient groups was 60.1% (242/403) and 47.8% (11/23), respectively. This finding shows no significant association between anemia and G6PD deficiency (P>0.05).

Table 3 presents a summary of the entire G6PD gene in St. Martins and Dagua.

<table>
<thead>
<tr>
<th>Sample No.</th>
<th>Villages</th>
<th>Exon 2</th>
<th>Exon 3</th>
<th>Exon 4</th>
<th>Exon 5</th>
<th>Exon 6</th>
<th>Exon 7</th>
<th>Exon 8</th>
<th>Exon 9</th>
<th>Exon 10</th>
<th>Exon 11</th>
<th>Exon 12</th>
<th>Exon 13</th>
<th>G6PD variant</th>
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<td>-</td>
<td>-</td>
<td>871 G&gt;A</td>
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<td>-</td>
<td>-</td>
<td>Jammu</td>
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<td>871 G&gt;A</td>
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<td>871 G&gt;A</td>
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<td>Dagua</td>
</tr>
</tbody>
</table>

Table 3: Summary of exon mutations of the G6PD gene in St. Martins and Dagua.

Note: - : No mutation
383 T>C: Mutation T>C at nucleotide 383
595 A>G: Mutation A>G at nucleotide 595
871 G>A: Mutation G>A at nucleotide 871

G6PD deficiency and normal G6PD activity groups (P > 0.05). The hemoglobin concentration measured in 426 out of 551 study individuals ranged from 5.9 to 17.7 g/dl (mean ± SD: 10.7 ± 1.7 g/dl). Mean hemoglobin levels in the G6PD deficient and normal G6PD groups were 11.5 ± 2.1 and 10.7 ± 1.7 g/dl, respectively, with no significant difference observed between the two groups (P>0.05). Anemia (hemoglobin < 11g/dl) was detected in 253 cases (59.4%), and the prevalence of anemia in normal G6PD and G6PD deficient groups was 60.1% (242/403) and 47.8% (11/23), respectively. This finding shows no significant association between anemia and G6PD deficiency (P>0.05).

Table 3 presents a summary of the entire G6PD gene in the 19 samples examined (13 in St. Martins and 6 in Dagua). Three G6PD variants were detected: G6PD Vanua Lava (383 T>C), G6PD Jammu (871 G>A, 1311 C), and a new variant (595 A>G). Nine cases with the G6PD Jammu variant and four cases with the G6PD Vanua Lava variant were evident among the St. Martins subjects. In Dagua, one case with the G6PD Jammu and four cases with the G6PD Vanua Lava variant were detected. A novel mutation was found in Dagua. This mutant showed a nucleotide change from A to G at position 595 of exon 6 (Fig 2), with no alterations in the remaining exons, which was predicted to result in an amino-acid change from isoleucine to valine. The subject was a 29-year-old male without anemia (hemoglobin concentration: 14.0g/dl) and was infected by *P. malariae*. We named this new mutation G6PD Dagua (DDBJ database accession number: AB 376963).

**DISCUSSION**

The prevalence of G6PD deficiency in our study population was 5.1%, which was within the range of 0 to 50% in a previous report from PNG [27] and quite similar to the reported rate of 3% in the Madang Province that shares a border with East Sepik [6]. A novel variant (G6PD Dagua) and two other variants (G6PD Vanua Lava and G6PD Jammu) have been detected in the 19 G6PD-deficient samples.

We detected four cases with the G6PD Vanua Lava variant in St. Martins. G6PD Vanua Lava has been reported from Malaysia [1], the Buru, Halmahera and Flores islands of Indonesia [12, 13], and Vanuatu [8]. The populations in all of the above studies, as well as St. Martins, are Austronesian speakers. The populations in PNG have been classified into two phyla according to the linguistic characteristics: non-Austronesian speakers (Papuan speakers) and Autronesian speakers [7]. The Austronesian speakers are thought to have moved from Southeast Asia to the coast and islands of PNG some 3,500 years ago [2] and expanded to Vanuatu 3,000 years ago [24]. Thus, G6PD Vanua Lava variant may have originated in Southeast Asia and spread via PNG to Vanuatu. Similarly, a 27-bp deletion in the erythrocyte band 3 gene was suggested to have come from Austronesian speakers and expanded to the population of PNG [14, 26].
G6PD Vanua Lava was also seen in Dagua. Gene flow between St. Martins (Austronesian speakers) and Dagua (Papuan speakers) has already been indicated by a report of a 9-bp deletion from mitochondrial DNA, a genetic marker of Austronesian speakers [26]. This would explain our finding of G6PD Vanua Lava in both the villages of St. Martins and Dagua.

We found 10 cases with the G6PD Jammu variant in St. Martins and Dagua villages. At present, only two cases of G6PD Jammu have been reported, one in India [4] and the other in Myanmar [21]. The appearance of G6PD Jammu was not coincident with the distribution of Austronesian speakers. Therefore, G6PD Jammu people in St. Martins and Dagua villages might originate from migrations of non-Austronesian speakers who settled in PNG 40,000 to 10,000 years ago [2, 9, 29]. Further microsatellite analysis is necessary to validate the origin of G6PD Jammu among people in PNG and India/Myanmar.

G6PD Dagua, a novel variant, is characterized by a change of nucleotide A>G at position 595 in exon 6 of the

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**Fig. 2: Location of the point mutations on exon 5, exon 6 and exon 9**

- A1. Wild-type (exon 5)
- A2. G6PD Vanua Lava (exon 5)
- B1. Wild-type (exon 6)
- B2. G6PD Dagua (exon 6)
- C1. Wild-type (exon 9)
- C2. G6PD Jammu (exon 9)
G6PD gene. Among the 19 samples in which we conducted total sequencing of the G6PD gene, one case with G6PD Dagua were detected.

Some studies have been carried out to demonstrate the protective effect of G6PD deficiency against malaria. A field study conducted in Gambia and Kenya suggested that the G6PD A variant is associated with substantial resistance to severe malaria [22]. However, there is no evidence that G6PD deficient males are resistant to *P. falciparum* infection [19], which is consistent with our study result.

G6PD Vanua Lava and G6PD Jammu are classified into class II (severe enzyme activity) [5], and G6PD deficient individuals are at risk for hemolytic anemia. However, most of the G6PD deficient people are clinically asymptomatic and have never had a hemolytic stage. Acute hemolytic anemia is precipitated by a number of strong oxidative agents such as infections, drugs, and nutrients. Our results showed a high prevalence of anemia in the study population, although G6PD deficiency was not the cause of the condition.

In conclusion, the detection of three G6PD variants, that is, G6PD Jammu, G6PD Vanua Lava, and G6PD Dagua indicates the heterogeneity of the studied population and suggests that these G6PD variants could be important genetic markers for clarifying the gene flow among the population of Southeast Asian and Pacific Island countries.

ACKNOWLEDGMENTS

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