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REPORT

# Report of the Third International Workshop on Molecular Blood Group Genotyping

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Vox Sanguinis

The Third International Society of Blood Transfusion Workshop on Molecular Blood Group Genotyping was held in 2008, with a feedback meeting at the International Society of Blood Transfusion Congress in Macao SAR, China. Thirty-three laboratories participated, eight less than in 2006. Six samples were distributed: sample 1 representing DNA from a sample referred because of abnormal serological results in D testing; samples 2 and 3 from transfusion-dependent patients for testing for all clinically important polymorphisms; sample 4 a mixture of two DNA samples designed to simulate a chimera, referred because of abnormal serological results in donor testing; and samples 5 and 6 plasma samples from RhD-negative pregnant women, for fetal RhD testing (only tested by 17 laboratories). For samples 1–3, 24 of 33 laboratories obtained completely correct results. For sample 4, the ability to detect the minority DNA population was partly dependent on method. Of the 17 laboratories that received samples 5 and 6, 13 reported correct results on both samples. Overall a small improvement from previous workshops was noted, but there is still room for improvement. The main conclusion for the 2006 workshop can be reiterated: with greater care and attention to detail, very high standards could be set for molecular blood group genotyping.

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## Introduction

Phenotypes for most blood group polymorphisms can be predicted from genotypes determined by molecular genetic methods. There are many applications of this technology, the main ones being determination of fetal *RHD* genotype in pregnant women with anti-D and determination of clinically significant blood groups in multiply transfused patients or those whose red blood cells give a positive direct antiglobulin reaction [1–4]. Molecular blood group genotyping is becoming routine in many reference laboratories worldwide and external quality assurance schemes are required. Participation in such an exercise is a requirement by the diagnostic regulatory authorities in some countries. International workshops were organized on behalf of the International Society of Blood Transfusion (ISBT) in 2004 and 2006, involving 30 and 41 laboratories, respectively. Each workshop had a feedback meeting at the ISBT Congresses in Edinburgh and Cape Town and reports were published [5,6]. An interim exercise, involving 29 laboratories took place in 2005. Details of these workshops can be found on http://blood.co.uk/ibgrl. This is the report of the 2008 workshop and the associated feedback meeting that took place in Macao SAR, China, in June 2008.

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## The workshop

### Purpose of the workshop

The main purpose for holding the workshop were: (i) to function as an external quality assurance scheme; (ii) to improve communication between laboratories performing molecular blood group genotyping; (iii) to identify and discuss the methods being used; (iv) to ascertain the accuracy and reliability of those methods and the laboratories using them; and (v) to provide, in addition to normal DNA samples, interesting and challenging DNA samples associated with unusual serological phenotypes.

### Laboratories participating

Thirty-three laboratories participated, from the following countries: Australia; Austria; Brazil (three laboratories); Canada (three); China; Czech Republic (two); Denmark (two); France (two); Germany (four); Italy; The Netherlands; Poland; Portugal; Slovenia; Spain; Sweden; Switzerland; UK (two); USA (four) (see Appendix). All participating laboratories submitted results.

### Samples distributed

Blood samples were obtained with informed consent. For samples 1–3, DNA was prepared from anticoagulated blood with Nucleon BACC2 reagents (GE Healthcare UK, Amersham, Buckinghamshire, UK), and for sample 4 with Qiagen Blood mini-kits. Six samples were sent out together, on dry-ice by courier, and were delivered within 48 h to the participants' laboratories in most cases.

Sample 1 (50 μl of 200 ng/μl) represented a DNA sample that was referred because of abnormal serological results in D testing. Participants were asked to test for *RHD* and to perform any other tests they felt to be appropriate.

Samples 2 (50 μl of 200 ng/μl) and 3 (50 μl of 150 ng/μl) represented DNA samples from multi-transfused, transfusion-dependent patients. Participants were asked to perform all red cell blood group tests that were available to them, including *RHD* zygosity. They were also asked to state which of the tests they would have performed if this had been a real clinical case.

Sample 4 (100 μl of 50 ng/μl) represented DNA from a sample that was referred because of abnormal serological results on donor testing. Participants were asked to perform any tests they considered appropriate.

Samples 5 and 6 were ethylenediaminetetraacetic acid-anticoagulated plasma samples from RhD-negative pregnant women in 16–18 weeks of gestation (2 ml of each), and these were only for RhD testing. For logistic reasons, it was not possible for all laboratories to receive the same samples (5 and 4 different women were used for samples 5 and 6, respectively). These samples were only sent to those laboratories that specifically requested them. Each plasma DNA as well as amniotic fluid DNA for all samples was tested by the organizing laboratory.

At least some testing was carried out on samples 1 to 4 by 33 laboratories and on samples 5 and 6 by 16 laboratories. Unfortunately, owing to a sample mix-up at distribution, the sample originally intended to be sent out as sample 1 was designated sample 2. Consequently, sample 1 had a normal D phenotype and *RHD* gene, despite being sent out as giving abnormal serological results in D testing.

## Methods and results

More complete details of the results obtained can be found on the workshop website: http://www.blood.co.uk/ibgrl.

All but one of the participants were punctual, submitting results by the deadline.

### Methods

#### *Samples 1 to 4*

A wide variety of technologies were employed for testing these DNA samples. The most commonly used was polymerase chain reaction with allele-specific primers (PCR-ASP), with the products visualized by gel electrophoresis. Of those laboratories employing this method, some used a commercial kit (BAGene, Lich, Germany or Inno-train Diagnostik GmbH, Frankfurt, Germany) and others used published or in-house protocols. Other methods employed were restriction-enzyme digestion of PCR products (PCR-restriction fragment length polymorphism) followed by gel electrophoresis, real-time quantitative PCR or allelic discrimination, pyrosequencing, DNA sequencing and microarray technology (BLOODchip, Progenika, Vizcaya, Spain or BeadChip, BioArray Solutions, Warren, NJ, USA).

Previous workshops resulted in concrete recommendations regarding the correct controls to be used. Almost all of the laboratories used at least most of the recommended controls: (i) a no-template (water) control, at least as a batch control; (ii) at least one in-tube positive control; and (iii) at least one negative control (less common than positive control). The exceptions were mostly those participants using commercial kits, which are validated batch-wise commercially.

In ABO testing, some laboratories (8 of 16) tested for a few subgroup alleles with a commercially available kit, while one laboratory used a method able to detect a whole panel of A and B subtypes and infrequent *O* alleles [7]. The latter method also allows routine testing for the relatively common hybrid *O* alleles that otherwise constitute a risk of being mistyped as $A^2$ or *B*. Most laboratories (14 of 17) tested for the main non-deletional *O* allele ($O^2$ or *O03*) and were able to distinguish $A^1$ from $A^2$.

In Rh a variety of different *RHD* exons were tested or sequenced. Of 31 laboratories, 23 routinely tested for *RHDΨ*,

 

seven occasionally tested for *RHDΨ*, often based on the ethnicity of the subject, and one did not test for *RHDΨ*. *RHDΨ* was a major cause of error in the 2004 and 2006 workshops [5,6], and would have caused errors again in this workshop had it been included. For *C*-testing, all of the 22 laboratories that reported their results tested for the *RHCE* intron 2 polymorphism associated with C/c.

When questioned about the molecular basis for the methods being employed, some laboratories stated that they did not know as they were using commercial kits.

## Results

### *Samples 1 to 3*

Sample 1 was submitted as a D variant but, because of an administrative error upon sample dispatch, was an apparently normal D-positive. This was not intended to be a trick. Of the 33 laboratories that tested this sample, two reported it to have a DVII gene and two to have a weak D type 1 gene, but this was not the consensus among the other laboratories. Sample 2 was weak D type 3: 19 identified a weak D type 3 gene; 13 just reported a D gene; and one reported it as weak D type 1. Sample 3 was DVI type 1: 23 identified DVI type 1 (or *RHD* with exons 4 and 5 missing); five as DVI; four reported *RHD*; and one was inconclusive.

There were very few errors in the remainder of the testing on samples 1–3: one mistyping in *MN*; one incorrect *RHD* zygosity determination; two mistypings for *Ee*; one incorrect Dombrock phenotype prediction, with the genotype not shown; and one typographical error in reporting S positive instead of s positive.

Table 1 shows serological phenotypes and phenotypes predicted from molecular tests for samples 1–3. This does not necessarily reflect the full range of errors as some laboratories did not report predicted phenotypes and some might have submitted a correct predicted phenotype despite reporting an incorrect genotype. Serological phenotypes shown in Table 1 are those obtained in the laboratories of the organizers and were not known by the participants.

Regarding *ABO* typing, three laboratories submitted genotyping results for sample 1 and 19 laboratories for samples 2 and 3, but, as seen in Table 1, not all interpreted their results to a predicted phenotype. It is also worth noting that the reported *ABO* genotypes were written in three (samples 1 and 3) or 18 (sample 2) different ways. The ABO phenotype prediction for sample 2 was reported in four different ways: A, A+, A1, and ABO:1,–2,3,4.

To put the results of samples 1–3 into another context, of the 33 laboratories that tested these samples, 24 had no errors, six had one error, two had two errors and one had three errors (all on *RHD* zygosity). This is probably a slight improvement compared to previous workshops, but that is difficult to judge as *RHDΨ* and the silent *FY* allele, genes that have caused difficulties in the past, were intentionally not present in these samples.

Additional tests carried out by a minority of laboratories on some samples included tests for the following blood groups: $Co^a/Co^b$ (11 laboratories); $Di^a/Di^b$ (10); $Lu^a/Lu^b$ (nine); $Kp^a/Kp^b$ (eight); $Kn^a/Kn^b$ (seven); $Wr^a/Wr^b$ (five); $Yt^a/Yt^b$ (five), Vw, $M^g$ (three); Sc1/Sc2 (three); $Js^a/Js^b$ (two); Hy, $Jo^a$ (two); $LW^a/LW^b$ (two) and $McC^a/McC^b$ (one). In addition, a few laboratories tested for a variety of *RHCE* variants. It was not possible to confirm the accuracy of these tests as the appropriate serological tests were not done, but one laboratory reported a Wr phenotype that did not concur with their reported genotype.

### *Sample 4*

Sample 4 was a 95%:5% mixture of DNA from two individuals, designed to simulate DNA from a chimera. Red cell phenotypes obtained from serological tests on each component and the phenotypes predicted from molecular testing of the DNA mixture are shown in Table 2.

Of the 32 laboratories that tested sample 4, seven suggested the presence of a chimera or mixture, eight reported abnormal reactions and 17 reported no abnormalities. Of the 14 laboratories that tested for ABO, seven failed to detect the minority *A* gene, whereas in D typing only two of 32 laboratories failed to detect the minority *RHD* gene. Only four of 24 laboratories detected the minority *K* allele. Nine laboratories reported sample 4 as *c/c* (majority) and 11 as *C/c* (minority); 10 laboratories reported sample 4 as *e/e* (majority) and 14 as *E/e* (minority). These differences probably reflect the different technologies being applied. In general, PCR-ASP methods appeared to be better at picking up a signal from the minor cell (DNA) population compared to PCR-restriction fragment length polymorphism and allelic discrimination/TaqMan-based methods. Both samples came from Fy(a–b+) individuals, yet six laboratories reported the presence of an $Fy^r$ allele, suggesting that $Fy^r$ might have been present, probably in the minority population. Both samples came from Jk(a+b+) individuals, yet one laboratory only detected weak signals for $Jk^b$.

## Samples 5 and 6

These were plasma samples from D-negative pregnant women for determination of fetal D type. Eighteen laboratories were sent samples: for one laboratory the samples were delayed in transit and were not tested, one laboratory did not report results on samples 5 and 6 and one laboratory only reported results on sample 6. Results are shown in Table 3. Overall, of the 17 participating laboratories, 13 reported correct results on both samples.

Sample 5 was made up from five plasma samples, all from 16–18 weeks' gestation. All were D-negative male fetuses,

 

**Table 1** Predicted phenotypes for samples 1–3. Numbers of laboratories obtaining each result are shown in parentheses

| | | Workshop results – predicted phenotypes | |
|---|---|---|---|
| **Polymorphism** | **Serology** | **Correct** | **Incorrect** |
| Sample 1 | | | |
| ABO | $A_1$ | A (2) | |
| MN | M+ N+ | M+ N+ (7) | |
| Ss | S– s+ | S– s+ (6) | |
| D | D+ | D+ (25) | Weak D (2), possible variant (1) |
| Cc | C+ c+ | C+ c+ (20) | |
| Ee | E+ e+ | E+ e+ (21) | |
| Kk | K+ k– | K+ k– (8) | |
| Duffy | Fy(a+b+) | Fy(a+b+) (8) | |
| Kidd | Jk(a+b+) | Jk(a+b+) (8) | |
| Dombrock | Not tested | Do(a–b+) (2) | |
| Sample 2 | | | |
| ABO | $A_1$ | $A_1$ (6); A (11) | |
| MN | M– N+ | M– N+ (17); N+ (3) | M+ N+ (1) |
| Ss | S– s+ | S– s+ (21); s+ (2) | |
| D | D weak | D+ (13); D+ weak (15[a]) | |
| Cc | C+ c+ | C+ c+ (29) | |
| Ee | E– e+ | E– e+ (29); e+ (1) | |
| Kk | K– k+ | K– k+ (25); K– (1); k+ (2) | |
| Duffy | Fy(a+b+) | Fy(a+b+) (28) | |
| Kidd | Jk(a+b+) | Jk(a+b+) (28) | |
| Dombrock | Not tested | Do(a+b+) (13) | Do(a+b–) (1) |
| Sample 3 | | | |
| ABO | O | O (17) | |
| MN | M+ N– | M+ N– (17); M+ (4) | |
| Ss | S– s+ | S– s+ (20); s+ (2) | S– S+ (1) |
| D | DVI | DVI (12[b]); D+ weak (15); partial D (5); variant D (5); D+ (5) | |
| Cc | C– c+ | C– c+ (27); c+ (1) | |
| Ee | E+ e+ | E+ e+ (26); E+ e inconclusive (1); E+ (1) | E– e+ (2) |
| Kk | K– k+ | K– k+ (26); K– (1); k+ (2) | |
| Duffy | Fy(a+b+) | Fy(a+b+) (28) | |
| Kidd | Jk(a+b+) | Jk(a+b+) (28) | |
| Dombrock | Not tested | Do(a+b+) (14) | |

[a]Including five that stated weak D type 3.

[b]Including five that stated DVI type 1.

confirmed by genetic testing on amniotic fluid, with a range of fetal DNA concentration of 28–73 pg/ml. From the 15 laboratories that tested these samples, 14 reported D-negative and one reported it as inconclusive owing to a positive result in exon 5 and a negative result in exon 7. Five of those laboratories obtained a positive result for a fetal identifier, five claimed to use a fetal identifier, but did not report their results, four did not test for a fetal marker and one obtained an inconclusive result for *SRY*.

Four plasma samples were used for constituting sample 6: all were D-positive. The range of fetal DNA concentration was 26–81 pg/ml. Thirteen laboratories reported a D-positive fetus, one D inconclusive because of insufficient positive replicates, and two D-negative. Both samples that gave rise to the incorrect and inconclusive results were tested and found positive by each of four other laboratories and had a fetal DNA concentration of 30 pg/ml. Twelve of 15 laboratories used at least 800 μl or more of plasma, although one laboratory only required 200 μl. The highest centrifugation speeds for separating cellular components was < 3000 ***g*** in seven laboratories, 3000–10 000 ***g*** in one laboratory and > 10 000 in seven laboratories. The DNA isolation kits utilized were Qiagen Blood kit (six laboratories), Qiagen MiniElute (five), MagnaPure (three) and two others. A recent international workshop showed that the best results for extraction of fetal DNA from maternal plasma were obtained with the QIAamp

 

**Table 2** Predicted phenotypes for sample 4. Numbers of laboratories obtaining each result are shown in parentheses

| Polymorphism | Serology | | Workshop results – predicted phenotypes |
|---|---|---|---|
| | 95% | 5% | |
| ABO | O | A | O (5); A (3); O/A (1); suspected A (1); O inconclusive (1) |
| MN | M+ N– | M+ N+ | M+ N– (6); M+ N+ (1); M+ (1); M+ N(+) (4) |
| Ss | S– s+ | S– s+ | S– s+ (15) |
| D | D– | D+ | D+ (17); D(+) (4); D– (2); D+/D– (1); D– inconclusive (2); $R_o^{Har}$ (1) |
| Cc | C– c+ | C+ c+ | C+ c+ (8); C– c+ (8); C(+) c+ (6); c+ (1) |
| Ee | E– e+ | E+ e+ | E– e+ (11); E+ e+ (9); E(+) e+ (4); e+ (1) |
| Kk | K– k+ | K+ k+ | K– k+ (17); K+ k+ (1); K(+) k+ (2) |
| Duffy | Fy(a–b+) | Fy(a+b+) | Fy(a–b+) (13); Fy(a–b+) (Fyx) (1); Fy(a–b weak) (1); Fy(a–b+) or Fy(a–b weak) or Fy(a–b–) (3) |
| Kidd | Jk(a+b+) | Jk(a+b+) | Jk(a+b+) (17); Jk(a+b weak) (1) |
| Dombrock | Not tested | | Do(a+b+) (6) |

**Table 3** Plasma fetal *RHD* typing results (17 laboratories received samples)

| | Correct | Incorrect | Inconclusive | No results reported |
|---|---|---|---|---|
| Sample 5 (D– male fetus) | 14 | 0 | 1 | 2 |
| Sample 6 (D+ fetus) | 13 | 2 | 1 | 1 |

**Table 4** Number of participating laboratories that test for fetal blood groups by genotyping on fetal DNA in maternal plasma, either as a routine service for research purposes

| | Fetal blood groups – number of laboratories | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | D | C | c | E | e | K | k | Ss |
| Routine | 11 | 3 | 5 | 3 | 1 | 3 | 1 | 0 |
| Research | 5 | 3 | 4 | 4 | 1 | 2 | 0 | 1 |

DSP Virus Kit, with the QIAamp DNA Blood Mini Kit showing comparable results when 500 ml or more of plasma was used [8]. All participating laboratories employed real-time quantitative PCR for fetal testing, with six laboratories using an ABI7000, 7300 or 7500, six an ABI7700 or 7900, three a Light Cycler and one a Step One Plus. Fourteen of 15 laboratories tested three or four replicates and all tested for between two and four different regions of *RHD*. The regions tested were exon 3 (one laboratory), exon 4 (six), exon 5 (11), exon 6 (one), exon 7 (12), exon 10 (12) and intron 4 (one). For controls, of the 16 laboratories 13 tested for a Y-chromosome marker, five tested for some other fetal identifier and 13 tested for a non-fetal reference gene.

Participants were also asked whether they performed fetal genotyping in a research or diagnostic setting, and for which blood groups. Results are shown in the Table 4.

## Conclusions and recommendations

As in previous workshops, the problem of terminology remained. No template was distributed for reporting results, although many participants used the template from previous workshops. An array of different types of terminology was used for reporting genotypes and phenotypes. Often genotypes were reported in the format of a phenotype and *vice versa*, making interpretation of the results difficult. There is still no defined terminology for molecularly defined blood group alleles and genotypes; the ISBT Terminology Committee is working on this. It should not be difficult, however, to report genotyping results in a clear and unambiguous way. In contrast, there is a well-defined terminology for blood group phenotypes [9], and this should be followed either in the numerical format (e.g. KEL:1,2, JK:–1,2) or in the alternative traditional format [e.g. K+ k+, Jk(a–b+)].

One issue that was discussed at the feedback meeting was intellectual property rights. One commercial company had contacted the workshop organizers to advise them that the determination of weak D and DEL types, as well as the detection of *RHD* zygosity using the *Rhesus boxes*, is covered by patents. The company had been informed by the German patent holder that not only commercial kits, but also in-house methods, were affected by the patents. They also requested that those alleles should be excluded from the workshop. The workshop organizers ignored this request as they considered that the patents did not cover a workshop or external quality assurance exercise. This demonstrates some of the difficulties that might arise in the future from commercial interests conflicting with the need to obtain high diagnostic standards in blood group genotyping.

 

Levels of accuracy in the workshop are reasonably high, but there is still a lot of room for improvement. The conclusion for the 2006 workshop [6] still appears to be applicable: with greater care and attention to detail, very high standards could be set for molecular blood group genotyping.

## Future workshops

The next workshop will be held in 2010 with the feedback meeting in Berlin, Germany. The format of the workshop will be worked out by the organizers. Once again at least part of the workshop will function as an external quality assurance exercise. In addition, samples with more complex and unusual genotypes will be sent out for the interest of those laboratories that are more research orientated.

Currently, there are no plans for an interim exercise in 2009. The website www.blood.co.uk/ibgrl will be maintained to provide information about previous and upcoming workshops.

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## Appendix

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