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# Prevalence of Red Blood Cell Major Blood Group Antigens and Phenotypes among Omani Blood Donors

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**ABSTRACT**

***Objectives:*** Most literature on the frequencies of red blood cell (RBC) phenotypes are published in Europeans and Africans countries, with the frequencies in the Omani population unknown. We sought to determine the prevalence of RBC blood group phenotypes among Omani blood donors. ***Methods***: Blood group ABO, RhD type, and phenotyping were performed for 21 blood group antigens on enrolled blood donors. The following antigens were assessed serologically: Rh (C, c, E, e), Kell (K, k, $Kp^a$, $Kp^b$), Kidd ($Jk^a$, $Jk^b$), Duffy ($Fy^a$, $Fy^b$), Lewis ($Le^a$, $Le^b$), Lutheran ($Lu^a$, $Lu^b$), MNS (M, N, S, s), and P1. ***Results:*** A total of 337 Omani blood donors were tested. The most common blood group was O+ (44.9%). Among the tested blood donors studied, 89.3% were RhD positive with R1r being the most common Rh phenotype. The k antigen was found at a frequency of 99.4%, while 4.5% of the blood donors studied were K+. The most common phenotype in the Duffy blood group system was Fy(a-b-), while the most common phenotypes in the Kidd and MNS blood group systems were Jk(a+b+) and M+N-S+s+ at 47.0% and 22.6%, respectively. The Le(a+) and Le(b+) antigens were found in 21.7% and 67.3% of the blood donors, respectively. One Jk(a-b-), one Le(a+b+), and two Lu(a-b-) individuals were identified. ***Conclusion:*** This is the first study to examine the frequencies of RBC phenotypes among the Omani blood donors. The study's results show Duffy blood group frequencies that resemble what has been reported in the African population, and higher frequencies of the rare null phenotypes compared to European populations.

The International Society of Blood Transfusion recognizes up to 39 genetically discrete blood group systems with more than 300 blood group antigens identified to date.[1,2] The reported frequencies and phenotypes of red blood cell (RBC) antigens differ in different populations and ethnic groups.[3,4] Most data in the literature report frequencies of common RBC antigens in Europeans, Africans, and some Asian countries. There is scanty of data in Arabs and the Middle East, including Saudi Arabians, Kuwait, India, and Iran.[5–8] While the study among Saudi Arabians showed similar Rh blood group phenotype frequencies to those described in the Europeans, the distribution of the MNS blood group system phenotypes was reported to be distinct. In addition, the K+k+ phenotype was found to be at much higher frequencies in Saudi Arabians than in Europeans or Africans.[5] Moreover, increased frequencies of the Duffy Fy(a-b-) and Lewis Le(a-b-) null phenotypes was reported.[5] Similar finding was observed with regard to the frequencies of the Duffy null phenotypes among the Northeastern Iranian blood donors, while K+k- and Kp(a+b-) phenotypes were found at higher frequencies.[8]

Oman has a high prevalence of hereditary blood disorders.[9,10] There are several papers that investigated the rates of alloimmunization in Omani sickle cell and thalassemia patients.[11–13] However, there is no data on the prevalence of RBC phenotypes in the Omani population to enable prediction of the risk of alloimmunization to certain blood group antigens. Herein, we investigated the prevalence of 21 blood group antigens in the Omani blood donors and compared it to European, African, and Asian populations and to what has been published in other

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populations in the region (Indians and Iranians). To the best of our knowledge, this is the first study of its kind in Oman.

## METHODS

The study was conducted at the Sultan Qaboos University Hospital (SQUH) blood bank. The SQUH blood bank is an independent blood bank and the second-largest in the country. It has blood donor, blood component manufacturing, clinical transfusion and apheresis services, and serves the university hospital with a total of 452 beds. SQUH is located within the Sultan Qaboos University Campus, which is the largest university in Oman. The hospital is a tertiary care hospital that treats adult and pediatric patients and is a major referral center from all over the country with inpatient, outpatient, emergency, and operative services with high demand for blood and blood components. The hospital has the only allogeneic bone marrow transplantation unit in the country presently and has the largest adult and pediatric thalassemia daycare unit in Oman. The SQUH blood bank issues about 30 000 units annually, and 75–80% of these units are collected and processed in-house.

We conducted a prospective cross-sectional study at SQUH blood bank over two years (from January 2015 to December 2016). Omani blood donors attending the SQUH blood bank on Wednesdays each week were invited to be enrolled in the study. Blood donors needed to fulfill the local blood donation eligibility criteria, and informed consent was obtained from the eligible donors before enrollment. Inclusion criteria included male and female Omani blood donors consenting for their agreement to be enrolled in the study. Non-Omani blood donors and repeated blood donors attended the blood bank more than once during the study period were excluded.

Ethical approval was obtained from the local Research Ethics Committee at the College of Medicine and Health Sciences at the Sultan Qaboos University (MREC #928).

The ABO and Rh blood group and the extended RBC phenotype for the Rh (C, c, E, e antigens), Kell (K, k, $Kp^a$, $Kp^b$ antigens), Kidd ($Jk^a$, $Jk^b$ antigens), Duffy ($Fy^a$, $Fy^b$ antigens), Lewis ($Le^a$, $Le^b$ antigens), Lutheran ($Lu^a$, $Lu^b$ antigens), MNS (M, N, S, s antigens), and P1 were tested serologically on each enrolled blood donor.

ABO and Rh blood group was determined using forward and reverse serological grouping by gel cards using manufacture instructions. DiaClon ABD-confirmation for blood donors ID-cards (Bio-Rad©, Cressier Switzerland) were used to determine the ABO and Rh type using the standardized procedure as per the manufacture instructions. These gel cards detect DVI phenotype and assign the Rh-positive status to those that react positively. A 5% RBC suspension was prepared by mixing 25.0 µL of blood donor's packed cells with 0.5 mL of ID-Diluent 2 solution (Bio-Rad©, Cressier Switzerland) and 12.5 µL of the blood donor's red cell suspension was added to the microtubes of the ID-card using a calibrated pipette. The ID-card was centrifuged at 900 rpm for 10 minutes in the ID-centrifuge and results were recorded visually.

In the ID-card system, a (+++) to (++++) reaction indicates the presence of the D antigen. A (+) to (++) reactions indicate a possible weak or partial D. These weak positive reactions are further verified using the ID-DiaClon anti-D with the utilization of a monoclonal anti-D formulated to characterize weak D's and DVI. A 0.8% red cell suspension is made by mixing 1.0 mL of ID-Diluent 2 solution with 10.0 µL of packed red cells. A 50.0 µL of red cell suspension is added to the appropriate micro-tube and mixed with 50.0 µL of 'ID-DiaClon Anti-D' for confirmation of weak D by indirect anti-globulin test. The ID-card was incubated for 15 minutes at 37 °C in the ID-incubator. The ID-card was then centrifuged for 10 minutes in the ID-centrifuge and the results were then read and recorded. A positive reaction is defined by agglutinated cells forming a red line on the surface of the gel or agglutinates dispersed in the gel. RhD positive is defined by ++++ reaction, while Rh weak D/DVI is defined by a reaction that ranges between + to +++. A negative reaction is defined by a compact button of cells on the bottom of the micro-tube and indicates RhD negative. Known weak D positive and negative samples are included to validate the results. False positive results may occur in cases where the direct anti-globulin test (DAT) is positive. Therefore, DAT is performed in all positive results before assigning the RhD status.

Phenotyping was performed serologically using gel cards and Bio-Rad© monoclonal antisera on freshly drawn samples within 24 hours of collection. Phenotyping was performed serologically using

standardized procedures as per the manufacture instructions (Bio-Rad©, Cressier Switzerland). For the determination of the Rh and Kell phenotypes, the DiaClon Rh-subgroups +K ID-cards (Bio-Rad©, Cressier Switzerland) were used. A 5% RBC suspension was prepared from each sample by mixing 25.0 µL of the donor's packed cells with 0.5 mL of ID-Diluent 2 solution (Bio-Rad©, Cressier Switzerland). Then 12.5 µL of the donor's red cell suspension was added to all microtubes of the ID-card. The ID-card was then centrifuged at 900 rpm for 10 minutes in the ID-centrifuge, and results were recorded visually. For determination of the P, Lewis, Lutheran, Kell, and Kidd phenotypes, the ID-Antigen Profile I and ID-Antigen Profile II ID-cards were used, respectively, (Bio-Rad©, Cressier Switzerland). A 5% red cell suspension was prepared from each sample by mixing 50 µL of red cells in 0.5 mL of Diluent 1 solution. The cell suspension was incubated for 10 minutes at room temperature at 18–25 °C. About 12.5 µL of the cell suspension was added to each micro-tube of the phenotype ID-cards. The ID-cards were then centrifuged at 900 rpm for 10 minutes in the ID-centrifuge and read.

For the determination of the MNS and Duffy phenotypes, the ID-Antigen Profile III ID-cards (Bio-Rad©, Cressier Switzerland) were used. A 0.8% cell suspension was prepared from each sample by mixing 10.0 µL of red cells in 1.0 mL of ID-Diluent 2 solution. The cell suspension was used immediately by pipetting 50.0 µL of the cell suspension to each micro-tube of the phenotyping ID-cards. Afterwards, a 50.0 µL of ID-test sera was added to the appropriate microtubes. The ID-cards were then incubated for 10 minutes at room temperature (18–25 °C) and centrifuged at 900 rpm for 10 minutes in the ID-centrifuge. Phenotyping was performed using monoclonal antibodies for all phenotypes tested.

Reactions obtained were graded for each antigen phenotyped according to the standard practice.[14]

The laboratory internal quality control procedure involves daily and weekly quality control tests using ID-internal control reagents and manufacturer's instructions. Daily quality control tests include tests for ABO blood group and antibody screen. Weekly quality control tests include tests for ABO blood group, antibody screen, antibody identification, direct Coomb's test, and Rh phenotype. The blood bank also participates in the Royal College of Pathologists of Australia quality assurance program.

The sample size for this descriptive cross-sectional study was calculated based on the expected proportion and the desired maximum 95% confidence width. Assuming a phenotype proportion of 25% with a 10% precision and an alpha of 0.05 using binomial exact distribution, we estimated the need to test 300 Omani blood donors for the RBC phenotypes. A data sheet was generated to record the obtained phenotypes for each specific antigen in the studied blood group systems: Rh (C, c, E, e antigens), Kell (K, k, $Kp^a$, $Kp^b$ antigens), Kidd ($Jk^a$, $Jk^b$ antigens), Duffy ($Fy^a$, $Fy^b$ antigens), Lewis ($Le^a$, $Le^b$ antigens), Lutheran ($Lu^a$, $Lu^b$ antigens), and MNS (M, N, S, s antigens). Frequencies and percentages for each specific RBC antigens and the frequencies for each of the derived phenotype combinations was assessed. The frequencies of the antigens were estimated using direct counting of the positive observations. For antigen groups, different combinations were constructed and their frequencies were estimated using the direct counting method.

Frequencies of the derived phenotypes in the Omani blood donors was compared with what has been reported in European, African, and Asian populations.[14,15] We also aimed to compare the obtained phenotypes to what has been published in other populations from the region, including Iran and India.[7,8] Proportions were compared between different populations using chi-squared test or Fisher's exact test as appropriate. Results were analyzed on all blood donors regardless of gender. An alpha threshold of 0.05 was used for statistical significance. Statistical analysis was performed using the R statistics software, version 3.1.2 (Lucent Technologies, Ohio, USA).

## RESULTS

A total of 337 Omani blood donors were tested. The most common blood group was O+ (44.9%) followed by B+ (20.2%), A+ (17.4%), AB+ (6.8%), B- (2.7%), O- (7.4%), and A- (0.6%). Table 1 describes the frequencies of the Rh antigen phenotypes and predictable genotypes in the Omani blood donors compared to Europeans, Africans, and Asians.[15] Table 2 displays the frequencies of the obtained phenotypes in the different RBC blood group systems in comparison to the European and African populations.[14]

The obtained frequencies of the different Rh antigens among all blood donors were as follows:

**Table 1:** Rh phenotypes and predictable genotypes in Omani blood donors compared to the reported data in other populations.[15]

| Phenotype | | | | | Frequency (%) | | | |
|---|---|---|---|---|---|---|---|---|
| D | C | c | E | e | Omanis | Europeans | Africans | Asians |
| + | + | - | - | + | 22.8 | 18.5 | 0.7 | 56 |
| + | - | + | + | - | 1.5 | 2.3 | 1.3 | 3.5 |
| + | - | + | - | + | 5.7 | 2.1 | 58.9 | 0.2 |
| + | + | - | + | - | 0.0 | Rare | Rare | Rare |
| + | + | + | - | + | 32.6 | 34.9 | 13.2 | 8.4 |
| + | - | + | + | + | 8.1 | 11.8 | 18.3 | 2.1 |
| + | + | - | + | + | 0.0 | 0.2 | Rare | 1.1 |
| + | + | + | + | - | 0.3 | 0.1 | Rare | 0.3 |
| + | + | + | + | + | 18.3 | 13.4 | 2.1 | 28.1 |
| - | + | - | - | + | 0.0 | Rare | 0.1 | Rare |
| - | - | + | + | - | 0.0 | Rare | Rare | Rare |
| - | - | + | - | + | 10.5 | 15.1 | 4.1 | 0.1 |
| - | + | - | + | - | 0.0 | Rare | Rare | Rare |
| - | + | + | - | + | 0.3 | 0.1 | 1.3 | 0.1 |
| - | - | + | + | + | 0.0 | 0.1 | Rare | Rare |
| - | + | - | + | + | 0.0 | Rare | Rare | Rare |
| - | + | + | + | - | 0.0 | Rare | Rare | Rare |
| - | + | + | + | + | 0.0 | Rare | Rare | Rare |

c (77.3%), C (74.3%), e (98.3%), and E (28.2%). The majority (89.3%) of the tested blood donors were RhD positive with R1r being the most common Rh phenotype among RhD positive blood donors followed by $R_1R_1$ and $R_1R_2$. Among the RhD negative blood donors, rr was the most common Rh phenotype obtained, as described in the European and African populations [Table 1]. No RhD weak or RhD variants were found. In the Kell blood group system, 4.5% of the blood donors were K antigen-positive and 99.4% were k antigen-positive, while $Kp^a$ and $Kp^b$ antigens were positive in 2.7% and 100% of the tested donors, respectively. The most common phenotype in the Kell blood group system was K-k+ as described in the European and African populations.[14] As for the Duffy blood group system, $Fy^a$ and $Fy^b$ antigens were found at frequencies of 16.7% and 22.3%, respectively. The most common phenotype in the Duffy blood group system was Fy(a-b-), similar to that reported in the African population [Table 2].[14]

As for the Kidd blood group system, the frequencies of the $Jk^a$ and $Jk^b$ antigens were 82.4% and 64.3%, while the frequencies of the M, N, S, and s antigens were 89.0%, 51.8%, 64.0%, and 82.1%, respectively. The most common phenotype in the Kidd blood group system was Jk(a+b+) [Table 2]. The most common phenotype in the MNS blood group system was M+N-S+s+ reported at 22.6%, while the M+N-S-s- and M-N+S-s- phenotypes were not found. The $Le^a$ and $Le^b$ antigens were found in 21.7% and 67.3% of blood donors, respectively, while Le(a-b+) was found to be the most common phenotype as described in European and African populations [Table 2]. For the Lutheran blood group system, the frequencies of $Lu^a$ and $Lu^b$ antigens were 3.0% and 96.7%, respectively; while the frequency of the P1 antigen was 78.9% similar to what has been reported in the European population.[14] The frequency of the rare $Lu^b$- and K- phenotypes were 3.3% and 0.6%, respectively.

When compared to other populations in the region, there was a statistically significant difference in the proportion of all blood group phenotypes among the Omani blood donors to what has been reported in the Iranian and North Indian populations, with exception of the Kidd blood group system [Table 3].[7,8] There were insufficient reports among other populations in the region for the comparison.

**Table 2:** Red blood cell phenotype frequencies among Omanis in compared to reported data in other populations.[14]

| System | Phenotype | Frequency (%) | | |
|---|---|---|---|---|
| | | Omanis | Europeans | Africans |
| Kell | K+k- | 0.0 | 0.2 | Rare |
| | K+k+ | 4.5 | 8.8 | 2 |
| | K-k+ | 94.9 | 91 | 98 |
| | K-k- | 0.6 | Exceedingly rare | Exceedingly rare |
| | Kp(a+b-) | 0.0 | Rare | 0 |
| | Kp(a+b+) | 2.7 | 2.3 | Rare |
| | Kp(a-b+) | 97.3 | 97.7 | 100 |
| Duffy | Fy(a+b-) | 9.2 | 17 | 9 |
| | Fy(a+b+) | 7.4 | 49 | 1 |
| | Fy(a-b+) | 14.9 | 34 | 22 |
| | Fy(a-b-) | 68.5 | Very rare | 68 |
| Kidd | Jk(a+b-) | 35.4 | 28 | 57 |
| | Jk(a+b+) | 47.0 | 49 | 34 |
| | Jk(a-b+) | 17.3 | 23 | 9 |
| | Jk(a-b-) | 0.3 | Exceedingly rare | Exceedingly rare |
| MNSs | M+N- | 48.2 | 28 | 26 |
| | M+N+ | 40.8 | 50 | 44 |
| | M-N+ | 11.0 | 22 | 30 |
| | M-N- | 0.0 | NA | NA |
| | S+s- | 17.9 | 11 | 3 |
| | S-s+ | 36.0 | 45 | 69 |
| | S+s+ | 46.1 | 44 | 28 |
| | S-s- | 0.0 | 0 | < 1 |
| Lewis | Le(a+b-) | 21.4 | 22 | 23 |
| | Le(a-b+) | 67.0 | 72 | 55 |
| | Le(a-b-) | 11.3 | 6 | 22 |
| | Le(a+b+) | 0.3 | Rare | Rare |
| Lutheran | Lu(a+b-) | 0.6 | 0.15 | NA |
| | Lu(a+b+) | 2.4 | 7.5 | NA |
| | Lu(a-b+) | 94.3 | 92.35 | NA |
| | Lu(a-b-) | 2.7 | Very rare | NA |
| P | P1 | 78.9 | 79 | 94 |

*NA: not available.*

## DISCUSSION

To the best of our knowledge, this is the first study to describe the prevalence of blood group phenotypes in Omani blood donors. Group O is the most common group among Omanis. There is limited complete data published on the frequency of blood groups in other populations in the region. Group O has been found to be the most common blood group in some populations including Lebanon,[16] Bedouin Arabs of Jordan,[17] Kuwait,[6] Egypt,[18] Kurds,[19] Kurdish population of Iraq,[20] and North East Iranians.[8] In contrast, group A is the commonest blood group reported in a publication from Turkey.[21] Our data showed that group B is the second most common blood group with overall frequency of 22.9%, comparable to what has been described in Africans,[14] which could be explained by the past links of Oman with Africa.[15,22] Group B is the second commonest

**Table 3:** Red blood cell phenotype frequencies among Omanis compared to reported data in North East Iranian[8] and North Indian[7] populations.

| System | Phenotype | Omanis (n = 337) | Iranians (n = 522) | *p*-value (Omanis vs. Iranians) | Indians (n = 1240) | *p*-value (Omanis vs. Indians) |
|---|---|---|---|---|---|---|
| Kell | K+k- | 0.0 | 2.3 | 0.003 | 0.0 | 0.041 |
| | K+k+ | 4.5 | 5.7 | | 5.68 | |
| | K-k+ | 94.9 | 92.0 | | 94.32 | |
| | K-k- | 0.6 | 0.0 | | 0.0 | |
| | Kp(a+b-) | 0.0 | 3.3 | < 0.001 | 0.0 | 0.027 |
| | Kp(a+b+) | 2.7 | 6.0 | | 0.95 | |
| | Kp(a-b+) | 97.3 | 90.7 | | 99.05 | |
| Duffy | Fy(a+b-) | 9.2 | 47.7 | < 0.001 | 43.85 | < 0.001 |
| | Fy(a+b+) | 7.4 | 26.4 | | 42.9 | |
| | Fy(a-b+) | 14.9 | 22.8 | | 13.25 | |
| | Fy(a-b-) | 68.5 | 3.4 | | 0.0 | |
| Kidd | Jk(a+b-) | 35.4 | 34.7 | 0.361 | 33.4 | 0.711 |
| | Jk(a+b+) | 47.0 | 44.4 | | 49.21 | |
| | Jk(a-b+) | 17.3 | 20.7 | | 17.35 | |
| | Jk(a-b-) | 0.3 | 0.0 | | 0.2 | |
| Lewis | Le(a+b-) | 21.4 | 35.3 | < 0.001 | 20.82 | 0.002 |
| | Le(a-b+) | 67.0 | 55.2 | | 60.57 | |
| | Le(a-b-) | 11.3 | 1.6 | | 18.61 | |
| | Le(a+b+) | 0.3 | 7.9 | | 0.0 | |
| Lutheran | Lu(a+b-) | 0.6 | 2.5 | < 0.001 | 0.0 | 0.016 |
| | Lu(a+b+) | 2.4 | 6.3 | | 0.95 | |
| | Lu(a-b+) | 94.3 | 88.5 | | 95.9 | |
| | Lu(a-b-) | 2.7 | 2.7 | | 3.15 | |
| P | P1 | 78.9 | 66.2 | < 0.001 | 71.92 | 0.010 |

blood group reported among the general population in Kuwait following group O.[6]

Our data shows some similarities in the RBC frequencies in the Rh blood group system when compared to the European population.[15] The R1r phenotype was found to be the most common phenotype, as described in other populations in the Middle East.[6,8,18] This is in contrast to the Asian and African populations in whom the most common Rh phenotypes reported are $R_1R_1$ and $R^0r$, respectively.[15] The most common phenotypes in the Kell, Kidd, Lewis, Lutheran, and P1 appear to be similar to what has been reported in the European population. Our data showed a statistically significant difference in the frequencies of the phenotypes in the other blood group systems, with the exception of the Kidd blood group, when compared to the Iranian and Indian populations. This suggests the influence of the ethnic background in the RBC phenotypes. The most common phenotype observed among Omani blood donors in the Kell blood group system was K-k+ and Kp(a-b+), resembling what has been described in other populations, including Europeans, Africans, Kuwaitis, Egyptians, North East Iranians, and North Indians.[3,6,8,14,18,23,24]

The obtained phenotypes in the Duffy blood group system shows an interesting profile resembling the African population with higher frequency of the null Fy(a-b-) phenotype. The *FY* gene encodes for a membrane glycoprotein on which $Fy^a$ and $Fy^b$ antigens are located. This glycoprotein is a receptor for *Plasmodium vivax* merozoites for RBC penetration, and individuals whose RBCs lack the $Fy^a$ and $Fy^b$ antigens confer malaria resistance. This is hypothesized to explain the higher prevalence of the Fy(a-b-) phenotype among individuals from malaria-endemic areas, approaching 70% in Africans and close to 100% in West Africans.[15] In comparison,

the incidence of $Fy^a$ and $Fy^b$ in Europeans is 68% and 80%, respectively.[15] The higher prevalence of this RBC phenotype among Omanis can be explained by the past endemic status of malaria in Oman before the 1990s before major eradication steps were undertaken.[25] Moreover, the past and current connections of Oman with East Africa may explain the resemblance of the Duffy phenotype frequencies with what has been reported in the African population.[22] Both of these factors may explain the high prevalence of the Duffy null phenotype in Omani blood donors, which has also been observed in other Middle Eastern populations.[6,23] The high prevalence of the Fy(a-b-) phenotype further explains the high past endemicity of *P. falciparum* infections over *P. vivax* in Oman.[25]

The frequencies of the Kidd antigens in the Omani blood donors is similar to Europeans and North Indians and appear to be different compared to Africans.[26,27] The most common Kidd phenotype in Omanis is Jk(a+b+), similar to what has been reported in Europeans, North Indians, North East Iranians, and Egyptians.[3,8,14,18,27] The Jk(a-b-) phenotype is found in only 0.3% of the Omani blood donors, in line with observations of the rare prevalence in different populations except in Polynesians and Finns.[3] The most common phenotypes of the Lewis and Lutheran blood group systems in Omani blood donors were Le(a-b+) and Lu(a-b+), similar to what has been described in the Europeans.[14] These phenotypes are also the most commonly reported in both blood group systems in North East Iran and North India.[8,27] We observed a higher prevalence of the Le(a-b-) phenotype compared to what has been reported in Europeans, but lower than what was reported in Africans.[5,14] Additionally, the Lu(a-b-) phenotype was at 2.7%, while this was found to be very rare in Europeans.[14] Similar rates were reported among North East Iranians and North Indians.[8,27] The frequency of the P1 antigen in this study was also the same as in the European population.[14] The above findings support the racial variability in the expression of the blood group antigens.

There are many influences that explain the close resemblance of the RBCs phenotypes with many of the populations in the region. Oman had its close connections with India in the 19th century and had controlled Mombasa and Zanzibar in East Africa for many years, beside its prior trade connections. In addition, there was a great influence of the Sassanian monarchs from Persia in the region in 200–600 CE.[22] Part of Oman's population originates in these former colonies,[28] and such influences can explain the epidemiologic presence of the different hemoglobin S haplotypes in Oman.[22] However, it is possible that the malaria endemicity in Oman had a strong influence, supported by the prevalence of the most common Duffy phenotypes as discussed above.

This study has some limitations. The sample included represent blood donors presenting a single hospital-based blood bank. Secondly, the study is not powered enough to study the difference in the geographical differences in the phenotype of blood donors from different regions of Oman, due to the limited grant obtained. The reported frequencies of the RBC phenotypes herein need to be confirmed on a larger national multi-center study since our sample in this study was affected by bias of ascertainment from one center. Additionally, the study was not powered to accurately assess the estimates of the frequency for the small subgroups. Thirdly, the method of blood donor enrollment is based on convenient sampling due to the limited availability of the student research assistant to obtain consent from the blood donors for enrollment. That said, this study is the first to describe the RBC blood group phenotypes among the Omani blood donors, albeit from a single institution in the Muscat region. Furthermore, there is scant of data of RBC phenotypes in the region and variation in the methods used for reporting the obtained frequencies,[5,6,16] limiting the ability to compare the results we obtained with what has been published elsewhere in the Middle East.

## CONCLUSION

This study describes the antigen and phenotype frequencies among Omani blood donors and compares these to what has been described in the literature in other ethnic populations. This is the first study to provide information on the frequencies of the common RBC antigen phenotypes in the Omani population. The difference of the reported phenotypes compared to other ethnic groups highlights the importance of having well-structured blood donor recruitment programs, a local registry of phenotyped blood donors and a frozen inventory of rare blood donors to enable the provision of compatible components to alloimmunized patients. This is particularly important with the

high incidence of hemoglobinopathies in the region such as sickle cell disease and thalassemia, for which blood transfusion is considered one of the cornerstones of management. Moreover, this study raises the importance of developing a locally-driven red cell reagents for antibody screening tests for local blood banks, since what is available in the market is derived from donors of other ethnic backgrounds. We advocate for a larger nation-based study involving blood donors from different regions in Oman to confirm our findings and to assess for any region-specific variations in the RBC phenotypes considering the heterogenous geography of the country.

*Disclosure*

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