Oral Abstracts will take place on April 25th at 2:45 pm				
2:50 PM	Oral	Sol Haberman Award Brett Dunlap LifeShare Blood Centers	Determining the Clinical Signifcance of anti-M Using Monocyte Monolayer Assay	T/S
3:05 PM	Oral	E Weiner / V Chrebtow	Increasing Compatibility of Apheresis Platelet Collections with Pathogen Reduction While Maintaining Current Split Rates	T/S
3:20 PM	Oral	KM Prioli / JK Karp / JH Herman / LT Pizzi / C Robbins	Cost Implications of Pathogen Reduced Platelets: A Hospital Budget Impact Model	T/S
	Poster	K Bowman / P Anderson / K Billingsley / M Kalvelage / J Fry	Autoanti-G Found in the Absence of Anti-D and Anti-C	T/S
	Poster	A Delk / R Gammon	Using Technology For Advanced Search Functions - End User Survey	T/S
	Poster	M Gannett / R Gammon	Direct Agglutination by an IgM anti-Ku	T/S
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ORAL ABSTRACTS

Sol Haberman Award Winner

Determining the Clinical Significance of anti-M Using Monocyte Monolayer Assay

Author: Brett C. Dunlap, LifeShare Blood Center

The MNS system is one of the 36 blood group systems currently recognized by the International Society of Blood Transfusion (ISBT). This blood group system (ISBT 002) was the second blood group described by Landsteiner and Levine in 1927. The MNS system consists of 48 antigens of which the M, N, S, s antigens are commonly encountered. These antigens are only expressed on red cells and are fully developed on fetal red cells. The MN determinants are carried on the transmembrane protein glycophorin A (Gagandeep, Sabita, Paramjit, & Ravneet, 2012).

Anti-M antibodies are usually not reactive at 37°C and hence are generally ignored in transfusion practice. However, when anti-M is active at 37°C, M antigen negative, crossmatch compatible red cell units should be selected for transfusion. The anti-M antibody is known to show dosage effect, reacting more strongly with homozygous cells (M+ N-) than heterozygous cells (M+ N+). This phenomenon is well demonstrated throughout this study. The prevalence of anti-M in donor sera is found to be 1 in 2500 units when tested with homozygous (M+N-) cells. This is reduced by half (i.e. 1 in 5000) when heterozygous (M+ N+) cells were used; indicating that some weaker examples of anti-M may be missed with heterozygous cells. Anti-M has also been described as a naturally occurring antibody in individuals whose red cells lack the M antigen and have no previous history of sensitization (Gagandeep, Sabita, Paramjit, & Ravneet, 2012).

Rarely has anti-M been implicated in immediate and delayed hemolytic transfusion reactions, which are supported by the results of 51Cr survival tests and monocyte phagocytosis assays. These examples demonstrate that anti-M can at times be of clinical importance and hence interpretation of test results should be done with caution (Gagandeep, Sabita, Paramjit, & Ravneet, 2012).

Anti-M antibodies have often been detected in antenatal mothers. Anti-M has been reported to cause hemolytic disease of the fetus and newborn (HDFN) of varying degrees of severity which may range from neonatal jaundice requiring exchange transfusion to intrauterine death. Reports in the literature describe this antibody as the second most common non-Rh antibody after anti-K (Kornstad, 1983). An unusual case in *Archives of disease in childhood: Fetal and neonatal* describes an anti-M of IgM and IgG nature responsible for neonatal red cell aplasia with substantial reduction in proliferation of erythroid cells in culture. It postulates that like an anti-K, anti-M may cause HDFN primarily by destroying erythroid progenitors rather than mature erythrocytes (Hinchliffe, Nolan, Vora, & Stamps, 2006).

This research outlines an experiment using monocyte monolayer assay (MMA) to discern whether the anti-M is clinically significant in 34 samples taken from thawed donor plasma known to contain anti-M.

Materials and Methods : RBC antibody testing technique

The presence of the anti-M antibodies in the sample plasma was tested and confirmed by using routine no enhancement media indirect antiglobulin (IAT) tube test. Two Medion (Grifols, Barcelona, Spain) screening cells, which were both homozygous for the M antigen, were selected to test the donor sera by IAT.

Sol Haberman Award Winner (*continued*)

Treatment of plasma with 0.01M DTT (dithiothreitol)

The preparation of fresh 0.01M DTT was conducted by dissolving 0.154g of DTT powder in 100mL of a working solution of PBS at a pH of 7.3. To treat the plasma samples, a 1:1 ratio of 0.01M DTT and plasma were incubated at 37°C for 60 minutes. Using the previously stated antibody test method, the mixture was then retested. For each sample, a control tube was prepared using a 1:1 ratio of plasma sample to 0.9% isotonic saline and tested in parallel with the test tubes. To ensure the removal of IgM antibodies, the control tube should remain positive, while the "test" tube should be negative.

Monocyte Monolayer Assay technique

This section describes the method for preparation of a monocyte monolayer, preparation of antibody-sensitized red cells, a combination of monolayer monocytes with sensitized red cells, and a calculation for the Monocyte Index (MI). The first step is to collect and separate the monocytes: two freshly drawn 7mL EDTA tubes are drawn from a healthy volunteer. The contents of the EDTA tubes are transferred to a 50mL sterile conical tube with equal parts phosphate buffer solution (PBS). In a separate 50mL sterile conical tube, 15mL of Sigma-Aldrich Histopaque® - 1077 is added and then tilted to a 30° angle. The diluted blood is slowly layered onto the Histopaque® - 1077 and then brought to a total volume of 45mL using PBS. After centrifugation for 60 minutes, the supernatant is removed and discarded. The white cell layer is carefully removed, transferred to another sterile 50mL tube and then diluted to a total volume of 45mL with PBS. After an additional 60-minute centrifugation step, the supernatant PBS is removed and discarded. 5mL of 5% Sigma-Aldrich® Fetal Bovine Serum (FBS) is then added to the remaining white blood cell pellet. This suspension is divided into equal amounts in each chamber of a tissue culture slide and incubated for one hour at 37°C in an atmosphere of 4-5% CO2.

Aliquots from four units of donor blood, type O Rh negative, homozygous for M antigen were chosen for sensitization. The aliquots were manually washed three times with PBS and suspended to a 3% concentration. The test plasma was added to the 3% red cell solution to a ratio of 1:2 red cells/plasma and incubated at 37°C for 60 minutes. To allow for activation of complement, fresh ABO compatible, non-immune serum was added to the red cells being sensitized with the test plasma and this mixture incubated for an additional 15 minutes at 37°C. After three more wash cycles with PBS, the mixture was suspended with 0.9% isotonic saline to a concentration of 3%. A drop from each tube was tested using Immucor Gamma anti-IgG and Immucor Gamma anti-C3b/C3d. The tubes were centrifuged once more, and the supernatant removed and discarded. The cells were then suspended in RPMI-1640 medium with 5% Sigma-Aldrich® Fetal Bovine Serum (FBS). The white cell monolayer was overlaid with the sensitized red cells and incubated at 37°C in an atmosphere of 4-5% CO2 for 60 minutes. After incubation, the excess fluid was aspirated from each chamber on the tissue slide. The slide was removed from the media chambers and washed with 0.9% saline, then dried at 24°C. After drying, the slide was stained with a Wright stain and allowed to dry at 24°C. The monocytes were counted using a microscope with oil immersion lens. The slide was examined for at least 400 monocytes with a separate count of which cells were adhered (Adherence Index) and which were phagocytized (Phagocytized Index). Then, the Monocyte Index (MI) is calculated using the following formula:

MI = (total # of monocytes with adhered RBCs + total # of monocytes with phagocytized RBCs) X 100 total number of monocytes counted.

To calculate the Adherence Index (AI) use the following formula: AI = (total number of monocytes with adhered RBCs) X 100 total number of monocytes counted.

To calculate the Phagocytized Index (PI) use the following formula: PI = (total number of monocytes with phagocytized RBCs) X 100 total number of monocytes counted.

Table1. Significance of MI Values

0	No adhered or phagocytized red cells
<5%	Incompatible blood can be given without significant risk of hemolytic transfusion reaction (HTR) or hemolytic disease of the fetus and newborn (HDFN)
>20%	Significant risk of HTR and HDFN

RBC antibody titration technique

Anti-M titration studies were conducted by the selection of a cell, which was homozygous for the M antigen, and then performing a serial dilution of the plasma samples. These samples reacted at the anti-human globulin (AHG) phase in the routine antibody screen. No enhancement media was added while performing the titrations. The titration endpoint was defined as the tube with the highest dilution demonstrating 1+ reactivity macroscopically. The titer was reported as the reciprocal value of this dilution.

Results: Antibody screens were performed on 34 donor plasma samples using no enhancement technique. These samples were previously known to contain anti-M. Twenty-four of the 34 samples had room temperature reactivity and no reactivity at AHG phase; therefore, no additional testing was performed on these samples. The remaining 10 samples, which did yield reactivity at the AHG phase, were treated with a freshly made working solution of 0.01M DTT, to remove any IgM component that might have been present in each sample. After the DTT treatment, an additional antibody screen was performed using no enhancement to determine which samples had IgG components. Five of the 10 samples treated yielded reactivity at AHG (see Table 2). These five samples are the group used for MMA testing (see Table 3 for MMA results).

Table 2. Serological Results of the Test Plasma				
Sample ID	Neat Plasma		DTT-treated Plasma	
	Screen Cell I	Screen Cell II	Screen Cell I	Screen Cell II
W2801	3+	2+	0/0✔	0/0✔
W4716	2+	2+	0/0✔	0/0✔
W2397	2+	1+	2+	1+
W1643	Micro+	Micro+	0/0✔	0/0✔
W6840	2+	2+	W+	W+
W9437	2+	2+	1+	1+
W8793	3+	3+	0/0✔	0/0✔
W2370	1+	1+	1+	1+
W1552	2+	2+	3+	3+
W1202	1+	1+	0/0✔	0/0✔

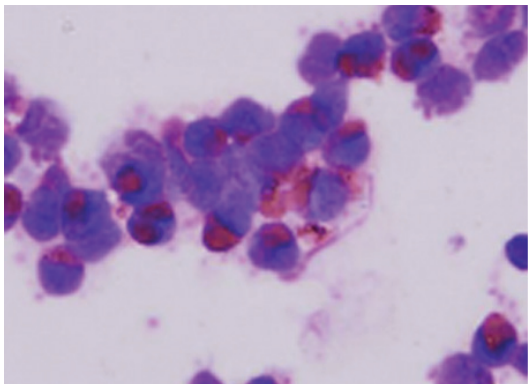

Image of MMA demonstrating RBC adherence and ingestion. Photograph credit: In house

Additionally, antibody titrations were performed on these five samples in order to correlate the clinical significance of the titer with the MMA results (see Table 4 for titer results).

Table 3. MMA Results				
RBC Source	Antigen Profile	AHG IgG	AHG C3b/ C3d	Monocyte Index
Positive control	D+ M+N=	3+	Not done	60.25
Donor W9437	M+N=	1+	Micro+	4.5
Donor W2370	M+N=	2+	1+	5.5
Donor W2397	M+N=	2+	Micro+	2.5
Donor W6840	M+N=	1+	1+	0.75
Donor W1552	M+N=	2+	1+	15.5

Table 4. Antibody Titrations of anti-M present in MMA test samples	
Donor	Titer
Donor W9437	Titer = 8
Donor W2370	Titer = 16
Donor W2397	Titer = 16
Donor W6840	Titer = 4
Donor W1552	Titer = 32

Sol Haberman Award Winner *(continued)*

Prior to this study, using the MMA to determine the clinical significance of anti-M had yet to be performed. This study has the potential to uncover antibodies to the M antigen which are clinically significant and may not be detected by serology alone.

The low titers on samples from Donor W9437 and Donor W6840 are associated with the low MI percentages, which provides further proof that the antibodies present are not clinically significant. The higher titration result in the sample from Donor W1552 is in direct relation to the higher MI percentage, which indicates that this anti-M is more abundant and has more potential for clinical significance.

References

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Rampersad, G., Suck, G., Sakac, D., Fahim, S., Foo, A., Denomme, G. L., & Branch, D. (2005). Chemical compounds that target thiol-disulfide groups on mononuclear phagocytes inhibit immune mediated phagocytosis of red blood cells. Transfusion, pp. 384-393.

T/S Poster

Autoanti-G Found in the Absence of Anti-D and Anti-C

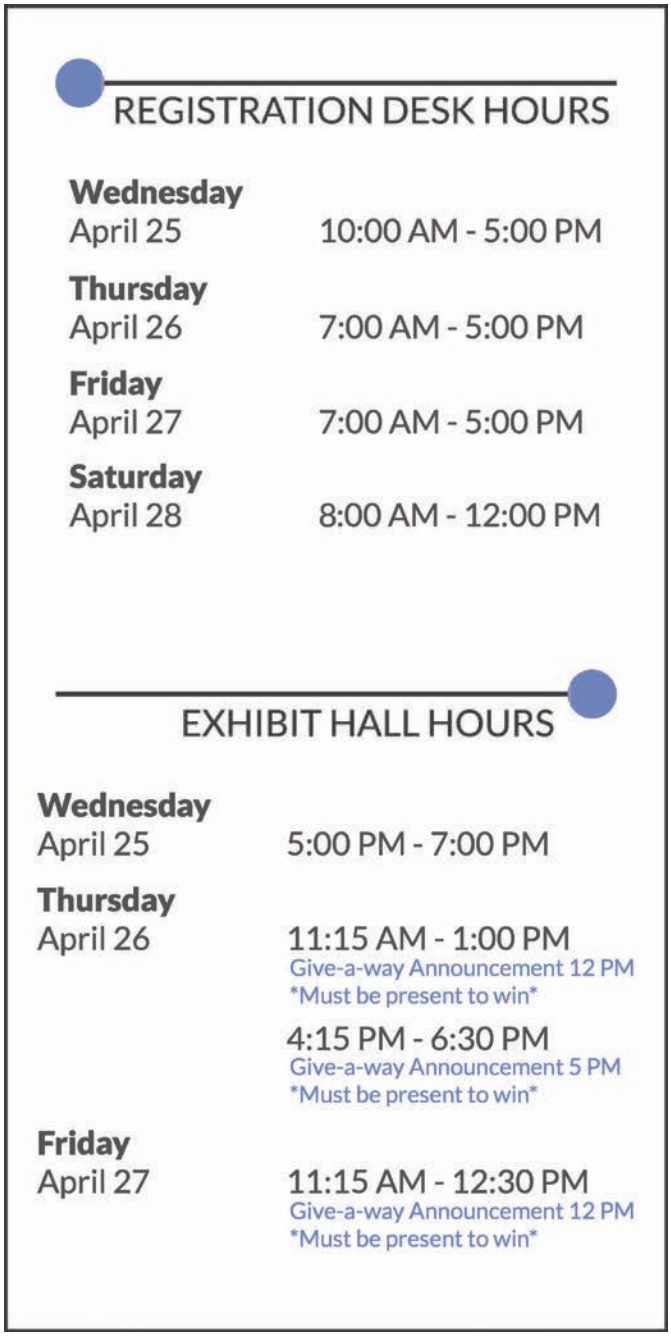
Author: K. Bowman, P. Anderson, KL. Billingsley, M. Kalvelage, J. Fry

Background/Case Studies: A 15 year old Caucasian male diagnosed with hemolytic anemia and no previous transfusions was referred to the Immunohematology Reference Laboratory (IRL) for antibody identification and RBC genotyping. Initial serologic testing by the referring facility and the IRL demonstrated anti-D, anti-C and/or anti-G specificity with a positive auto control and IgG DAT. Anti-G has an anti-D, -C specificity and is most frequently found in rr individuals exposed to r'r cells. The G antigen is present on RBCs expressing either RhD and/or C and very rarely on D-C-G+ (rGr) cells. Both RHCE*C and RHD genes encode Ser103 which determines G expression. Rare RhD variant antigens lacking Ser103 are G-.

Study Design/Methods: Serologic evaluation included tube testing using Gamma LO-IONTM (Immucor, Inc., Norcross, GA) enhancement, elution studies (Gamma ELU-KIT® II (Immucor, Inc.)), EDTA glycine acid treatment (Gamma EGATMKit (Immucor, Inc.)), allogeneic adsorptions with papain treated intact RBCs, reagent and patient-derived RBCs and antisera. Molecular testing was performed with BioArray Precise Type IVD HEA Assay (Immucor, Inc.).

Results/Findings: Molecular testing revealed an RHCE*cE genotype (with a C-E+c+e- predicted phenotype) and an otherwise unremarkable RBC typing report. Serologically, the antibody(ies) demonstrated an anti-D, -C, -G specificity in the serum and eluate using Ror, R2R2, r'r, rGr and rr cells. This patient is predicted to be R2R2 (DcE/DcE) therefore, anti-C is possible but an allogeneic anti-D or -G is exceptionally unlikely. Allogenic adsorptions using papain treated Ror and r'r cells excluded anti-C and anti-D, leaving anti-G as the only explanation of the initial findings. Reactivity with the patient's EGA treated (DAT negative) cells against the "neat" serum, eluate and anti-G antisera confirmed auto anti-G.

Conclusion: Warm autoantibodies are common findings and often have an Rh specificity; however, these antibodies usually demonstrate a broad but weaker specificity in the eluate or in the serum when enhancements are used. This anti-G had no reactivity with G- cells. The differentiation of anti-G from anti-D and anti-C is generally academic as transfusion recommendations are the same: provide RhD-, C- units. It is relevant and clinically important to determine the presence or absence of anti-D in RhD negative women of childbearing age who present with an anti-G specificity. If anti-D is excluded these women should receive RhIG as part of their prenatal care. In this case differentiating anti-D, -C from an auto anti-G was necessary to provide transfusion recommendations. Providing RhD- and C- units to give serologically compatible RBCs could result in formation of an allogeneic anti-e.



T/S Oral

Increasing Compatibility of Apheresis Platelet Collections with Pathogen Reduction While Maintaining Current Split Rates

Author: E. Weiner, V. Chrebtow

Background: Platelet Components (PC) can only be Pathogen Reduced (PR, INTERCEPT, Cerus, Concord, CA) if within approved criteria for volume, concentration and platelet (plt) dose. PR results in ~10% dose loss which must be accommodated when collecting plt donations to ensure the US plt dose of $\geq 3.0 \times 10^{11}$ is met. Currently, Triple Set kits for PR are only approved in Europe. Plt loss, and adjusted apheresis targeting parameters may impact split rate (SR) or products per apheresis procedure. Inventory suitable for PR without impacting US blood center SRs warrants evaluation and optimization.

Methods: 1,000 apheresis collections from 4 centers with different SRs were analyzed. A baseline SR for conventional PC was calculated assuming i) a minimum plt dose (allowing for production loss) of 3.1×10^{11} for single (S), 6.3×10^{11} for double (D), and 9.5×10^{11} for triple (T) conventional PCs, ii) concentration and volume requirements from apheresis device manufacturer were used.

For each collection, dose, volume, and concentration were assessed for PR kit compatibility, based on storage medium (PAS or 100% plasma) assuming i) a minimum dose (allowing for production loss) of 3.5×10^{11} for S and 6.7×10^{11} for D for PR units, ii) removing small quantities from units with excess volume or dose to meet PR specifications, iii) if all or part of an out of parameter D or T collection could be divided into one or more kits for PR, eligible parts undergo PR, and the remainder is treated conventionally, iv) collections not meeting PR specs or decreasing SR if PR treated are counted as conventional PCs.

Results: See chart below.

Conclusion: Depending on current blood center SRs, PR can be adopted to treat as high as 99% of the plt inventory without affecting SRs. PR compatibly increases further when T and large D donations are divided. Percent achievable depends on blood center practices and proportion of S, D, T collection. Changes to D and T collection parameters, optimized donation, plt count accuracy, and volume reduction can improve PR compatibility further. Individual analysis is warranted for each blood center.

Site	A	B	C	D
Collection	100% Plasma	65% PAS	65% PAS	100% Plasma
SR	2.25	2.01	1.72	1.48
S Products (%)	128 (12.8)	201 (20.1)	404 (40.4)	525 (52.5)
D Products (%)	982 (49.1)	1178 (58.9)	956 (47.8)	950 (47.5)
T Products (%)	1143 (38.1)	630 (21.0)	354 (11.8)	0 (0)
Total Products per 1000 collections	2253	2009	1714	1475
S PR compatible* (%)	113 (88.3)	176 (87.6)	364 (90.1)	522 (99.4)
D PR compatible* (%)	236 (24.0)	318 (27.0)	384 (41.2)	940 (99.0)
D PR adjusted** (%)	91 (9.3)	77 (6.5)	49 (5.1)	2 (.2)
T PR adjusted** (%)	627 (54.9)	359 (57.0)	188 (53)	0 (0)
PR Compatible Products* (%)	**349 (15.5)**	**494 (24.6)**	**748 (43.6)**	**1462 (99.1)**
PR Adjusted Products (%)**	**718 (31.9)**	**436 (21.7)**	**237 (13.8)**	**2 (.1)**
Total PR Products (%)	**1067 (47.4)**	**930 (46.3)**	**985 (57.5)**	**1464 (99.3)**

*Natively compatible; ** Divided into compatible doses.

T/S Poster

Using Technology For Advanced Search Functions- End User Survey

Author(s): A. Delk, R. Gammon

Background/Case Studies: Recently our two Immunohematology reference lab (IRL) antisera inventories were merged. An Excel based synergistic method to organize, store, and search the inventory was developed and launched. A dynamic/static freezer box storage system that was inter-box static and intra-box dynamic was being used and had been effective. Antisera assigned to a box remained in that box, but may be moved within the box. The box itself may be moved among freezers. Box movement and vial movement within the box were monitored by the custom rare frozen antisera spreadsheet. ABO group, desired and unwanted antibodies filters allowed quick search for appropriate antisera. The spreadsheet also had hyperlinks to scanned instructions for use (IFU). After implementation, the end users were surveyed.

Study Design/Methods: A six-question survey was administered to our IRL technologists to determine: awareness of (question 1), ease of use (question 2), and adoption rate of (questions 3, 4, and 5) the electronic system. A final question (6), measured manual vs. electronic spreadsheet end user preference.

1. Do you know that the IRL had a rare frozen antisera spreadsheet?
2. Do you know how to use the rare frozen antisera spreadsheet?
3. Since 7-01-2017 (implementation date) have you needed to use rare antisera? (not including the commonly used antisera)
4. If yes to #3, did you use the rare frozen antisera spreadsheet to perform a search?
5. If yes to #3, did you use the rare frozen antisera spreadsheet to view and/or print the IFU?
6. Do you prefer an electronic spreadsheet to a manual card file?

Results/Findings: The results of the survey are listed below (table). All 23 of the IRL technologists were aware of the program. While the majority either 5/23 (21.7%) fully or 11/23 (47.8%) somewhat knew how to use the electronic spreadsheet, 7/23 (30.5%) did not. During the time period of the survey, 12/23 (52.2%) needed to find rare antisera. Of those 1/12(8.3%) used the program and 11/12(91.7%) chose to search manually. Of the 11 that choose to search manually only 1/11 (9.1%) fully knew how to use the program, but preferred the manual method. The remaining 10/11 (90.9%) either did not or only somewhat knew how to use the electronic spreadsheet. 18/23 (78.3%) of the IRL technologists preferred an electronic spreadsheet to a manual method. In contrast, of those that needed to search for rare antisera only 7/12 (58.3%) preferred the electronic spreadsheet.

Conclusions: Although all of the IRL technologists were aware of the electronic spreadsheet only 21.7% fully knew how to use it. Many of the subset of technologists who needed to search for rare antisera preferred and chose to use a manual method. Since a significant majority of the IRL technologists (78.3%) surveyed preferred an electronic method, this implied that additional training and end user input into spreadsheet design may increase adoption rate.

Survey Responses N=23, Number and (Percent)

	Question 1	Question 2	Question 3	Question 4	Question 5	Question 6
Yes	23(100)	5(21.7)	12(52.2)	1(4.3)	1(4.3)	
Somewhat		11(47.8)				
No		7(30.5)	11(47.8)	12(52.2)	12(52.2)	
N/A				10(43.5)	10(43.5)	
Electronic						18(78.3)
Manual						5(21.7)

N/A – Not applicable

T/S Poster

Direct Agglutinating by an IgM anti-Ku

Author: M. Gannett, R. Gammon

Background: Several Kell system antibodies are known to cause direct agglutination (DA). However, some specificities such as anti-Ku have not been reported to cause DA. This case study evaluates a DA anti-Ku. In late 2015, an unidentified cold antibody and a "possible anti-Ku" 3+ reactive at PeG and Ficin-IAT were identified by another facility. Molecular testing by an outside facility confirmed the patient to be homozygous for IVS3+1g>a, probable KEL*02N.06 associated with a Kell null phenotype. Due to the patient's condition the hospital had to provide blood incompatible with anti-Ku. After transfusion of four units the patient developed a delayed serologic transfusion reaction with a weak positive DAT IgG by Gel with anti-Ku eluted. In October 2016 when samples were received in our immunohematology reference laboratory (IRL), initial testing showed 3+ to 4+ reactions at Saline-IS, LISS-37°C, LISS-IAT, and PeG-IAT with all Kell phenotypes tested including k-, Kp(a+b-), and Js(b-). The autocontrol and K^0K^0 cells were negative at all phases consistent with an anti-Ku. Kx- cells (McLeod Phenotype) were w+ at most phases, as expected since Kell is weakened without the XK protein. 0.2M Dithiothreitol (DTT) treated RBCs were used to identify an anti-E and for the ABO reverse type. Since we were unaware of any published reports of a DA anti-Ku, we performed additional testing to evaluate if the reactivity was caused by IgM or high titer IgG antibodies as a result of the incompatible RBC transfusions.

Methods: The DA nature of this anti-Ku was evaluated by titration studies. These were performed after the plasma was treated with 0.01M DTT at 37°C for 30 minutes, untreated plasma was tested in parallel for comparison. The titrations were tested with several Kell phenotypes for comparison at Saline-IS, 15′ Room Temp (22°C), 60′ 37°C, and then at the AHG phase. Titration studies started at a titer of 4 to conserve sera and required only 150µl of plasma to test k-, Kp(a+b-), and Js(b-) cells. In addition, because of the potency and value of this rare antibody, the plasma was recovered after the 37°C reading prior to washing for the AHG phase to preserve antisera.

Results: The DA was diminished with 0.01M DTT treated plasma when compared with the saline controls, but the AHG titer was not weakened. The Table displays the testing results with k- cells, which were typical for all phenotypes tested. The DA nature of this anti-Ku appeared to be of both an IgM and IgG nature.

Conclusion: To our knowledge this is the first reported case of Direct Agglutination by an IgM anti-Ku.

Table:

Phase	Cell	Test	4	8	16	32	64	128	256	512	1024	2048	4096	8192	Titer	Titration Score
Saline-IS	k-	Saline Control	3+	3+	1+	0	0	0							16	25
Saline-IS	k-	0.01M DTT	3+	3+	1+	0	0	0							16	25
15′ RT (22°C)	k-	Saline Control	2+	2+	2+	1+	0	0	0						32	29
15′ RT (22°C)	k-	0.01M DTT	1+	1+	0	0	0	0							8	10
60′ RT (37°C)	k-	Saline Control	4+	3+	3+	3+	1+	0	0						64	48
60′ RT (37°C)	k-	0.01M DTT	0	0	0	0	0	0							<4	0
AHG 60′ RT (37°C)	k-	Saline Control	4+	4+	4+	4+	4+	4+	4+	4+	2+	2+	0	0	2048	113
AHG 60′ RT (37°C)	k-	0.01M DTT	4+	4+	4+	4+	4+	4+	4+	3+	3+	2+	1+	0	4096	117

S-Strong

T/S Oral

Cost Implications of Pathogen Reduced Platelets: A Hospital Budget Impact Model

Authors: C. Robbins, K.M. Prioli, J.K. Karp, J.H. Herman, L.T. Pizzi

Background: An FDA draft guidance has highlighted the need to reduce bacterial contamination risk of platelet components (PC) via pathogen reduction (PR) or secondary rapid testing (RT). An interactive model was created for hospitals to analyze cost and budget impact when considering different interventions.

Study Design/Methods:
An Excel model was built and populated with base case costs and probabilities identified through literature search as well as through a survey administered to 27 US hospital transfusion service directors. The model was reviewed and refined by a panel of 7 transfusion medicine physicians. Three scenarios were generated to compare annual costs of PC acquisition, testing, wastage, dispensing / transfusion, sepsis management, shelf-life, and reimbursement for a hospital that purchases all of its PCs: 100% RT-PC, 100% PR-PC, and 50% RT-PC / 50% PR-PC.

Model Assumptions:
- 5,500 apheresis PC purchased/year
- Shelflife: • RT-PC: 7-day • PR-PC: 5-day
- 60% RT-PC are gamma irradiated
- PR replaces irradiation, CMV testing, bacterial detection (BD)
- Unit cost: • Conventional PC: $525 purchase • Conventional PC irradiated/CMV tested: $630 purchase • PR-PC: $640 purchase
- RT per test cost: $25
- 26% of PC transfusions are for outpatients (reimbursable)

Results/Findings: See Table 1 below.

Conclusion: The model predicts a modest (~3%) cost increase for PR-PC compared to RT-PC, which includes cost offsets such as elimination of BD and irradiation, and reimbursement. In an annual overall hospital blood budget, PR-PC only represents a ~0.06% increase in budget versus RT-PC. This small increase may be justified by additional safety provided by PR, which helps mitigate transfusion-transmission of viruses, parasites and emerging pathogens, and potentially transfusion associated graft-vs-host disease. PR also provides the benefit of not having to perform additional testing within the hospital. The effective PC shelf-life is potentially increased with RT, but platelets can be available sooner with PR due to elimination of BD, depending on NAT turnaround time.

Table 1: Annual Costs, Reimbursement, Shelf Life

	100% RT PC	100% PR PC	50% RT PC / 50% PR PC
Acquisition	$3,243,240	$3,527,680	$3,385,460
RT costs	$113,041	$0	$56,520
Wastage	$87,929	$167,644	$127,787
Dispensing / transfusion	$260,721	$260,721	$260,721
Sepsis (excludes septic shock cases that result in MODs/mortality/morbidity)	$8,095	$0	$4,047
Outpatient reimbursement	$878,572	$1,037,508	$958,040
Total hospital cost (less reimbursement)	$2,834,453	$2,918,536	$2,876,495
% of Blood budget*	2.18	2.24	2.21
PC age when placed in inventory (hours)	48	33	NA
Max. usable shelf-life (hours)	120	87	NA

*Assumes an annual blood budget of ~$130 million, including platelet, plasma, red blood cell components.

A Poster

Improving Patient Outcomes in the Golden-Hour

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Background/Case Studies: In emergency medicine, "the golden hour" refers to the critical one-hour time period following traumatic injury in which the patient has a higher likelihood of survival. Nearly half of all trauma related deaths occur in the first hour after injury -- half of those deaths are the result of major hemorrhaging. Rapid administration of blood products is vital to the survival of these patients.

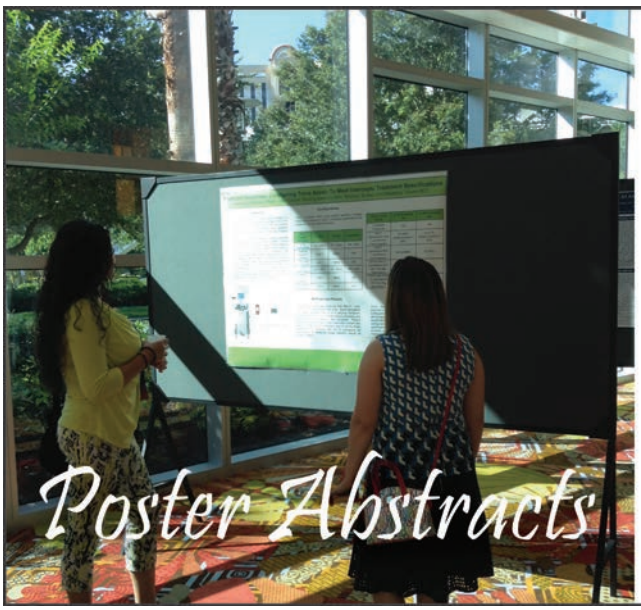
We implemented BloodTrack Emerge (Haemonetics, Braintree, MA) in our Trauma emergency department (ED) as part of a quality improvement initiative to more efficiently provide group O red blood cells (RBCs) and group A thawed/liquid plasma (plasma) for incoming trauma patients to support ratio-based transfusions and ensure the proper handling and traceability of this regulated resource.

Study Design/Methods: We treat approximately 30-40 trauma patients monthly. An assessment of our current blood supply chain revealed a multi-step, manual process that took about 8 minutes to prepare and physically transport a cooler from the blood bank to the ED. Coolers of blood were provided for incoming trauma patients, whether they ended up needing transfusions or not. This practice worked to ensure available blood supplies during critical moments, but resulted in inefficiencies and unnecessary inventory tie-ups, with only 10 percent of coolers fully used. It also consumed valuable staff time as technologists typically made 20-45 trips per month from the blood bank to the ED. Plus, there was no effective way to maintain traceability, control access to coolers or monitor usage.

Results/Findings: From January to August 2017, 189 RBCs and 27 plasma units (216 total) were removed by the ED staff. Between 151 and 173 units were transfused (70-80% utilization). The remainder were either returned to the Emerge system or rotated by the blood bank. During the same time period, 0.5% RBC units were discarded due to improper handling by ED nursing when returning to the Emerge system. No liquid plasma was wasted.

BloodTrack Emerge has freed up technologists to perform important tasks, tightened traceability and inventory control procedures and contributed to the Medical City Plano's verification as a Level 1 trauma center. Rather than preparing coolers of blood in case they may be needed in emergency situations, BloodTrack Emerge provides ED staff ready access to emergency units whenever they're actually needed — and frees up an estimated 6-10 hours of tech time per month during which they can perform other tasks. Audio and visual alerts notify the blood bank when emergency units are removed, allowing a quick response. Plus, by stocking emergency blood supplies in the ED, the blood bank isn't unnecessarily tying up group O RBC units. Today, the blood bank stocks and maintains 2-4 units of group O RhD negative, 4 units of O RhD positive RBCs, and 4 units of group A plasma in BloodTrack Emerge.

Conclusion: Implementing BloodTrack Emerge has enabled us to more effectively provide blood products for incoming trauma patients to support ratio-based transfusions, improve staff efficiencies and proactively respond to emergency situations.



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