

Winter 2007



Page 2
Featured Article
Page 3
Case Study
Page 4
Advertisements
Page 5
Case Study Cont.
Page 6
Word Find

President's Message-DONNA RATLIFF^{MT(ASCP)}

It has been an honor to be the President of KABB. It has been a pleasure to work with such enthusiastic and supportive Board Members. I would like to say thanks to all who help make KABB successful.

KABB is an important organization for those associated with transfusion medicine. KABB offers continuing education credits not only in blood banking but also in quality assurance and administrative areas. I would like to encourage you to take the opportunity to become involved in the meetings and the educational experiences KABB has to offer.

I hope you are aware of the KABB web site. Go to www.kabb.org to read about the history of KABB, names of the Board Members, information on future meetings, job opportunities and case studies.

If you have any suggestions for future topics or would like to become more involved with KABB, please contact any of the board members or me at donna.ratliff@kctcs.edu.

President-Elect Message:Sharon Noble, MT (ASCP)

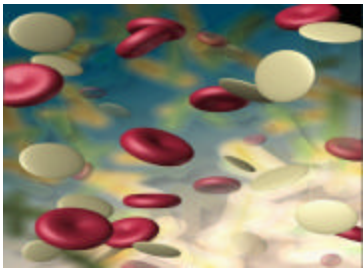
Welcome readers, I am Sharon Noble and will become president of the KABB this year. I am currently working at Kentucky River Medical Center in Jackson, KY as their Blood Bank Supervisor. I hope that this New Year will be good for each of you.

We are looking forward to all the upcoming events planned by the KABB for 2007. We are always looking for new ideas for our meetings and would be happy to have suggestions from each of you on topics. You can reach me by email sajnob@aol.com.

This year we have changed our meeting schedule and our small meeting will be held on March 10, 2007 at the Natural Bridge State Park located at Slade, KY just off of the Mountain Parkway. The room rate is \$59.95 a night. This is a one-day session that I am sure all will enjoy and benefit from attending. For more information on this and upcoming events please check our web site at www.kabb.org. As always all our meetings offer opportunities to get any CEU's that you may need.

Our fall event will be the **KABB/KSCLS Joint Meeting** held on September 11th and 12th. The meeting will be at Louisville Marriott East in Louisville, KY. This is the Hurstborne exit. Room rates here are \$79.95 per night for this meeting.

I look forward to this year and hope that each of us can work together to have a couple very good meetings.



The Duffy Blood Group System

Submitted by Dr. Elpidio Pena: Medical Director for Central Kentucky Blood Center.
For questions or comments pertaining to this article, please email Dr. Pena at:
epena2@email.uky.edu

The discovery of the ABO blood group system at the dawn of the 20th century led researchers to look for other blood groups. Now, a hundred years later, we know of at least 26 blood groups, some of them with more than fifty specificities.

Blood groups antigens are discovered by the presence of antibodies directed against antigens, and the Duffy blood group is no different. The Duffy blood group system was discovered in 1950 with the detection of an antibody in a hemophiliac patient by the name of Duffy. The antigen corresponded to what we know now as Duffy-A (Fy^a), and soon after the specificity for Duffy-B (Fy^b) was discovered on a multiparous female.

The Duffy blood group consists of six antigens Fy^a , Fy^b , $Fy3$, $Fy4$, $Fy5$, and $Fy6$. The gene that codes for the basic protein is in chromosome 1 and it contains two alleles coding for Fy^a and Fy^b . These two antigens and their corresponding antibodies are the ones we frequently encounter in our transfusion practices. The two alleles are expressed codominantly, so there are four possible phenotypes for Duffy: $Fy(a+b-)$, $Fy(a+b+)$, $Fy(a-b+)$, and $Fy(a-b-)$.

The Fy^a and Fy^b antigens are common in the Caucasian population (66% and 83% respectively) and much less common in Blacks (10% and 23%). The $Fy(a-b-)$ phenotype is common in blacks, probably as a selective advantage against malarial infection. The Duffy glycoprotein is a receptor for *Plasmodium vivax* merozoites, and red blood cells with the $Fy(a-b-)$ phenotype are more resistant to infection.

Clinically significant antibodies against the Duffy blood group system are mainly IgG1 and may cause hemolytic transfusion reactions (delayed and acute, ranging from mild to severe), and hemolytic disease of the fetus and newborn. Most of the antibodies will be anti- Fy^a and less commonly anti- Fy^b . Personally I have seen two cases of acute hemolytic transfusion reactions due to anti- Fy^a .

Antibodies directed against other Duffy antigens are very rare. Within this group the better documented ones are directed against $Fy3$ and $Fy5$. Both of these antibodies are present in people with a $Fy(a-b-)$ phenotype. Antibodies against either of these two antigens can cause delayed hemolytic transfusion reactions. Anti- $Fy3$ can cause acute hemolytic transfusion reactions. However, $Fy3$ in opposition to other Duffy antigens is resistant to enzyme degradation including the commonly used ficin.

References:

1. Daniels, Geoff: *Human Blood Groups. Second edition, Blackwell Science, Oxford, UK, 2002*
2. Dean, Laura: *Blood Groups and Red Cells Antigens* in <http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=rbcantigen>



CASE STUDY???

Karla Smith, MT(ASCP)SBB, Reference Laboratory Technologist,
Central Kentucky Blood Center, Lexington, KY.

Case Study:

Patient: 30 year old female admitted for reconstructive surgery following a motorcycle accident the prior spring.

Patient's medical history includes: 1 pregnancy in which she received 2 packed cells after delivery. Received multiple blood products in April 2006 following her accident.

Medications: None.

A specimen was received in the Blood Bank for a 2 unit crossmatch

Preliminary Blood Bank Laboratory Results:

Patient typed B Negative with a positive antibody screen. A panel was performed and the results follow.

| | Rh-hr | | | | | | | Kell | | | | | | Duffy | | Kidd | | P1 | Lewis | | MNSs | | | | N-HANCE | | |
|----|-------|---|---|---|---|---|----|------|---|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|----------------|-----------------|-----------------|------|---|---|---|---------|------|-----|
| | D | C | c | E | e | f | Cw | K | k | Kp ^a | Kp ^b | Js ^a | Js ^b | Fy ^a | Fy ^b | Jk ^a | Jk ^b | P ₁ | Le ^a | Le ^b | M | N | S | s | IS | 37°C | AHG |
| 1 | + | + | 0 | 0 | + | 0 | 0 | + | + | 0 | + | 0 | + | + | + | 0 | + | + | 0 | + | + | 0 | + | 0 | 0 | 0 | 2+ |
| 2 | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | 0 | + | + | 0 | + | + | + | 0 | + | 0 | + | 0 | + | 0 | 0 | 2+ |
| 3 | + | 0 | + | + | 0 | 0 | 0 | 0 | + | 0 | + | 0 | + | + | 0 | + | + | + | + | 0 | + | 0 | 0 | + | 0 | 0 | 2+ |
| 4 | + | 0 | + | 0 | + | + | 0 | + | + | 0 | + | 0 | + | 0 | 0 | + | + | + | + | 0 | 0 | + | 0 | 0 | 0 | 0 | 2+ |
| 5 | 0 | + | + | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | 0 | 0 | + | 0 | 0 | + | + | + | 0 | 0 | 0 | 0✓ |
| 6 | 0 | 0 | + | + | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | + | 0 | + | 0 | + | + | 0 | 0 | + | 0 | 0 | 0✓ |
| 7 | 0 | 0 | + | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | 0 | + | + | 0 | 0 | + | + | 0 | + | 0 | 0 | 0✓ |
| 8 | 0 | 0 | + | 0 | + | + | 0 | 0 | + | + | + | 0 | + | 0 | + | 0 | + | 0 | + | 0 | + | 0 | 0 | + | 0 | 0 | 0✓ |
| 9 | 0 | 0 | + | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | 0 | + | + | + | + | 0 | + | + | + | + | 0 | 0 | 0✓ |
| 10 | + | W | + | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | + | 0 | + | + | 0 | 0 | + | + | 0 | 0 | + | 0 | 0 | 2+ |
| 11 | + | + | 0 | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | + | 0 | 0 | + | + | + | 0 | 0 | + | + | + | 0 | 0 | 2+ |
| AC | | | | | | | | | | | | | | | | | | | | | | | | | 0 | 0 | 0✓ |

1. After looking at the panel, what antibody (ies) do you suspect?

2. What is the next step?

The following discussion is based on ruling out 17 commonly encountered antibodies in the blood bank. The 17 corresponding antigens we will be looking at are: D, C, c, E, e, K, Fy^a, Fy^b, Jk^a, Jk^b, P₁, Le^a, Le^b, M, N, S, and s. We will also rule in and rule out using 2 homozygous cells.

From the first panel we can rule out the following: c, e, Fy^b, Jk^a, Jk^b, P₁, Le^b, M, and s using 2 homozygous cells. Initially, the antibody specificity appears to be an Anti-D. At this point the D, C, E, K, Fy^a, Le^a, N, and S cannot be excluded.

Next, a selected cell panel could be set up to rule out the antibodies we could not exclude in the above panel. Another option would be to run a panel using a different type of enhancement media.

The panel cells from the first panel were treated with ficin and then tested with the patient's serum. See the panel on page 5.



www.unitedpharma.org

MacoPharma supported project entitled: "BLOOD BAGS WITH INTEGRATED RFID-LABELS TO PROVIDE A COMPLETE TRACEABILITY FROM PRODUCTION TO PATIENT: FIRST ROUTINE EXPERIENCES AND FUTURE PROSPECTS" is underway. The aim of this project is to develop a range of RFID (Radio Frequency Identification Devices) tags for both tracing of blood products from donor to recipient and close temperature monitoring. This system will be designed to meet the strict guidelines of cGMP and future EU and FDA directives on blood products. To learn more about this project contact:

YarivSivan@UnitedPharma.org

The Exclusive United States Importer and Distributor of
MacoPharma Branded Products & Services
770-270-6867 www.unitedpharma.org



Attention: 2007 Institutional Members

As part of your membership, you may post your job openings on our website and in our quarterly newsletter. For additional information, please e-mail jobs@kabb.org

SPECIAL REMINDER!!

2007 Spring Meeting
Saturday, March 10, 2007
Natural Bridge State Resort Park
Slade KY



Book a room by Tuesday, February 6, 2007, to get the KABB room rate of \$59.95.
Call 800-325-1710

For additional information including the Meeting Brochure, go to www.kabb.org

CASE STUDY CONTINUED from pg 3

| | Rh-hr | | | | | | | Kell | | | | | | Duffy | | Kidd | | P1 | Lewis | | MNSs | | | | FICIN | |
|----|-------|---|---|---|---|---|----|------|---|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|----------------|-----------------|-----------------|------|---|---|---|-------|-----|
| | D | C | c | E | e | f | Cw | K | k | Kp ^a | Kp ^b | Js ^a | Js ^b | Fy ^a | Fy ^b | Jk ^a | Jk ^b | P ₁ | Le ^a | Le ^b | M | N | S | s | 37°C | AHG |
| 1 | + | + | 0 | 0 | + | 0 | 0 | + | + | 0 | + | 0 | + | + | + | 0 | + | + | 0 | + | + | + | 0 | + | 0 | 2+ |
| 2 | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | 0 | + | + | 0 | + | + | + | 0 | + | 0 | + | 0 | + | 0 | 0✓ |
| 3 | + | 0 | + | + | 0 | 0 | 0 | 0 | + | 0 | + | 0 | + | + | 0 | + | + | + | + | 0 | + | 0 | 0 | + | 0 | 0✓ |
| 4 | + | 0 | + | 0 | + | + | 0 | + | + | 0 | + | 0 | + | 0 | 0 | + | + | + | + | 0 | 0 | + | 0 | 0 | 0 | 2+ |
| 5 | 0 | + | + | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | + | 0 | + | 0 | 0 | + | + | + | 0 | 0 | 0✓ |
| 6 | 0 | 0 | + | + | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | + | 0 | + | 0 | + | + | 0 | 0 | + | 0 | 0✓ |
| 7 | 0 | 0 | + | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | + | 0 | + | 0 | 0 | + | + | 0 | + | 0 | 0✓ |
| 8 | 0 | 0 | + | 0 | + | + | 0 | 0 | + | + | + | 0 | + | 0 | + | + | 0 | 0 | + | + | 0 | 0 | + | 0 | 0 | 0✓ |
| 9 | 0 | 0 | + | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | 0 | 0 | + | + | + | + | 0 | + | + | + | + | 0 | 0✓ |
| 10 | + | W | + | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | + | 0 | + | + | 0 | 0 | + | + | 0 | 0 | + | 0 | 0✓ |
| 11 | + | + | 0 | 0 | + | + | 0 | 0 | + | 0 | + | 0 | + | + | 0 | + | + | + | + | 0 | 0 | + | + | + | 0 | 0✓ |

1. After looking at the Ficcin panel, what antibody (ies) do you suspect?
2. What would you do next?
3. Based on the results in ficcin, do you now expect a different antibody to be reacting in the first panel? If so, what could it be?

From the Ficcin Panel, the patient appears to have an Anti-K. The Rh, Kell, Kidd, P1, and Lewis system antigens are either not affected or are enhanced with ficcin treatment. The highlighted columns on the panel above indicate antigens (Duffy and MNS system antigens) destroyed by Ficcin. Therefore, we cannot rule in or out antibodies in the Duffy or MNS system with a ficcin treated panel. From the Ficcin panel, the following can be excluded: D, C, c, e, JK^a, JK^b, P₁, Le^a, and Le^b.

Next, looking at both panels, we can exclude with 2 homozygous cells: D, C, c, e, Fy^b, Jk^a, Jk^b, P₁, Le^a, Le^b, M, and s. The reactivity in Ficcin appears to be an Anti-K. Looking back at the first panel, the Anti-K can account for 2 of the 6 reactive cells. The other activity appears to be due to an Anti-Fy^a and not due to an Anti-D. If it were due to an Anti-D, the antibody should have shown up in the ficcin panel. Since we know the Fy^a antigen is destroyed by ficcin, the antibody would not react when testing serum with a ficcin panel.

4. Now, we think we have an Anti-K and Anti-Fy^a. What is the next step?
5. What type of red cells would be provided for this patient?

First, we need to run a selected cell panel to exclude the E, N and S. Below is a selected cell panel. As you can see, we were able to exclude these three antibodies.

| | Rh-hr | | | | | | | Kell | | | | | | Duffy | | Kidd | | P1 | Lewis | | MNSs | | | | N-HANCE | | | |
|---|-------|---|---|---|---|---|----|------|---|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|----------------|-----------------|-----------------|------|---|---|---|---------|------|-----|----|
| | D | C | c | E | e | f | Cw | K | k | Kp ^a | Kp ^b | Js ^a | Js ^b | Fy ^a | Fy ^b | Jk ^a | Jk ^b | P ₁ | Le ^a | Le ^b | M | N | S | s | IS | 37°C | AHG | |
| 1 | + | + | 0 | 0 | + | 0 | 0 | 0 | + | 0 | + | 0 | + | 0 | + | 0 | + | + | 0 | + | 0 | + | 0 | + | 0 | 0 | 0 | 0✓ |
| 2 | + | + | 0 | + | 0 | 0 | + | 0 | + | 0 | + | 0 | + | 0 | 0 | + | + | + | 0 | + | 0 | + | + | + | 0 | 0 | 0 | 0✓ |
| 3 | + | 0 | + | + | 0 | 0 | 0 | 0 | + | 0 | + | 0 | + | 0 | 0 | + | + | + | + | 0 | + | 0 | 0 | + | 0 | 0 | 0 | 0✓ |

We have ruled out the possibility of additional antibodies being present. Now, we must see if we have ruled in each antibody using 2 cells. In the initial panel, we have 2 reactive cells that are Fy^a positive and K negative. In the ficcin panel, we have 2 reactive cells which are K positive and Fy^a negative.

Next, the patient is typed for the K and Fy^a antigens and found to be negative. We have now confirmed the patient has an Anti-K and Anti-Fy^a. We should provide this patient crossmatched red blood cells antigen negative for K and Fy^a.

TRANSFUSION MEDICINE WORD SEARCH

BY SHARON NOBLE

S E N S I T I Z A T I O N N
Y D D L T N P Z R G N Y K I
M N E F B K H E T A G Q N L
T J Y L Q J A E I M W I R U
R B X J A C Y L R L M O D B
A L K Y T Y E B B I T J N O
N N V I H D E L D P T B L L
S F O B N G G D E M F E T G
F N K E R T B C T P Y B D I
U W M R Y M E F X J A H N T
S B H O F R A I R A L A M N
I H M N F G E N O T Y P E A
O M N O U D D O M I N A N T
N Z M D D T N K L R P D K L

FIND THE WORDS BELOW:

ANTIGLOBULIN
DELAYED
DOMINANT
DONOR
DUFFY
FYA
FYB
GENOTYPE
IgG
INHERITED
MALARIA
MENDELIAN
REACTION
RECEPTOR
SENSITIZATION
TRANFUSION

Channels-KABB
c/o Danny Thacker
7437 Pin Oak Circle
Bristol, VA 24202

www.KABB.org