Hello!
My name is:
Joe
What to Expect

• Today: Immunohematology I
  - Materials at BBGuy.org/LLU
  - Password: LLUPathology

• 1/22: Immunohematology II (PT testing, Ab ID)
  - Interactive session!

• 2/5: Blood Products and Their Uses

• TBD: Transfusion Reactions
Immunohematology I

- Basic antigen-antibody testing
  - Basic tests
  - Principles to know

- Blood Groups
<table>
<thead>
<tr>
<th>Best at body temp (“warm”)</th>
<th>Best below body temp (“cold”)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coats RBCs</td>
<td>Agglutinates/lyses RBCs</td>
</tr>
<tr>
<td>Pregnancy, transfusion</td>
<td>“Naturally occurring”</td>
</tr>
<tr>
<td>Crosses placenta (HDFN)</td>
<td>Does not cross placenta (no HDFN)</td>
</tr>
<tr>
<td>Protein-rich antigens</td>
<td>Carbohydrate-rich antigens</td>
</tr>
</tbody>
</table>
Basic Reactions

4+ → 0

Tube

Solid phase

Gel (column agglutination)
Three “Phases”:

Immediate Spin
37C Phase
AHG Phase

Tube Testing

Immediate Spin Phase

Test RBCs

Do not incubate, incubate, spin again

Add potentiator, incubate, spin again

37C Phase

AHG Phase

Patient Serum

Test RBCs

Do not incubate, incubate, spin again

Add potentiator, incubate, spin again
Gel Testing

Images Courtesy of Bio-Rad
Solid Phase Testing

- Microwell
- Lysed RBC antigens
- Patient antibody
- Indicator RBC with anti-IgG

Image: Courtesy of Immucor
Solid Phase Testing

4+ Positive Result
3+ Positive Result
2+ Positive Result
1+ Positive Result
0 Negative Result
Indirect Antiglobulin Test

IAT: Antibody coating happens in test system

Direct Antiglobulin Test

IAT: Antibody coating happens in test system
DAT: Antibody coating happens in the body

Types of Antiglobulin

- **Anti-IgG, -C3d**
  - “Polyspecific”
- **Anti-IgG**
- **Anti-C3d**
  - IgM hemolysis, CAD
<table>
<thead>
<tr>
<th>Antibody</th>
<th>RBCs</th>
<th>Reaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-Fya</td>
<td>Fy(a+b−)</td>
<td>3+</td>
</tr>
<tr>
<td>Anti-Fya</td>
<td>Fy(a+b+)</td>
<td>0-1+</td>
</tr>
</tbody>
</table>

Kidd Duffy Rh
Proteolytic Enzymes

Papain/ficin/bromelain cleave proteins

<table>
<thead>
<tr>
<th>Anti-M vs M+ (pre)</th>
<th>Anti-M vs M+ (post)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3+</td>
<td>0-1+</td>
</tr>
</tbody>
</table>
Proteolytic Enzymes

Papain/ficin/bromelain cleave proteins

<table>
<thead>
<tr>
<th>Anti-D vs D+ (pre)</th>
<th>Anti-D vs D+ (post)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2+</td>
<td>4+</td>
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</table>
Proteolytic Enzymes

Papain/ficin/bromelain cleave proteins

<table>
<thead>
<tr>
<th>Anti-K vs K+ (pre)</th>
<th>Anti-K vs K+ (post)</th>
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<tbody>
<tr>
<td>3+</td>
<td>3+</td>
</tr>
</tbody>
</table>
“Over 300 Antigens?!”

Source: International Society for Blood Transfusion (ISBT); Accessed Jan 2019
### Enzyme Classification

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<tr>
<th>Enhanced</th>
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<tbody>
<tr>
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<td>MNS System</td>
<td>Kell System</td>
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<td></td>
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<td>-P1PK/GLOB</td>
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<td></td>
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<td>Rh System</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kidd System</td>
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</tbody>
</table>
ABO-related Systems

- Type 1 chains

Secretions, primarily glycoprotein
Plasma, primarily glycolipid
ABO-related Systems

- Type 2 chains

RBC Membranes, glycoprotein/glycolipid
$H$ (FUT1)

- $H$ and $h$ alleles
- Fucosyltransferase enzyme
- Near 100% of us have at least one $H$
Se (FUT2) - “Secretor”

- Se and se alleles
- Fucosyltransferase enzyme
- 80% of us can make H in secretions

H antigen

Gal

GlcNAc

R

Type 1 Chain

Fuc

Plasma (65%)
White blood cells
and platelets (+15%)
Red blood cells (45%)
H Antigen

- Precursor to A or B - Just add sugar!

Type 1 H

- Gal
- GlcNAc
- R
- Fuc

Type 2 H

- Gal
- GlcNAc
- R
- Fuc
Blood Group A

A antigen

A allele product: GalNAc transferase

Blood Group B

B antigen

B allele product: Gal transferase
ABO Antigens

- Antigens start early (6 weeks EGA)
- Still poorly formed at birth
- Adult levels by age 4
- Antigens are not limited to RBCs
  - Platelets
  - Endothelial and epithelial cells
    - Lung, GI tract, heart, kidney, etc.
  - Plasma and secretions
ABO Antibodies

- Babies have only Mom’s IgG
- Best from Group O
- Production by 6 months of age
- Adult levels by age 10
"Landsteiner’s Law"
# ABO Testing

<table>
<thead>
<tr>
<th>Cell Group</th>
<th>Serum Group</th>
<th>ABO Interp.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-A</td>
<td>Anti-B</td>
<td></td>
</tr>
<tr>
<td>4+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>0</td>
<td>4+</td>
<td>4+</td>
</tr>
<tr>
<td>4+</td>
<td>4+</td>
<td>0</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>4+</td>
</tr>
</tbody>
</table>

*Images: Harmening D “Modern Blood Banking & Transfusion Practices”*
# ABO Types by Race

<table>
<thead>
<tr>
<th>Type</th>
<th>Whites</th>
<th>Blacks</th>
<th>Asians</th>
<th>Native Americans</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>45%</td>
<td>49%</td>
<td>40%</td>
<td>79%</td>
</tr>
<tr>
<td>A</td>
<td>40%</td>
<td>27%</td>
<td>28%</td>
<td>16%</td>
</tr>
<tr>
<td>B</td>
<td>11%</td>
<td>20%</td>
<td>27%</td>
<td>4%</td>
</tr>
<tr>
<td>AB</td>
<td>4%</td>
<td>4%</td>
<td>5%</td>
<td>&lt;1%</td>
</tr>
</tbody>
</table>
Group O

- Most common across racial lines
- Antigen = H
  - *Ulex europaeus* lectin
- 3 mostly IgG antibodies = anti-A, anti-B, anti-A,B
  - Most common form of HDFN
  - Antibodies tend to be stronger than grp A or B
Group A

- Antigens = A, H
- Varying amounts depending on subgroup
- Antibody = anti-B
  - Naturally occurring IgM
Group A Subgroups

- A₁ (80%) and A₂ (~20%)
  - A₁ has 5x more A antigen than A₂
  - 1-8% A₂, 25% A₂B form anti-A₁
  - Insignificant unless at 37 C
  - Dolichos biflorus lectin + with A₁ not A₂

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<td>Anti-A</td>
<td>Anti-B</td>
</tr>
<tr>
<td>A₁ RBC</td>
<td>B RBC</td>
</tr>
<tr>
<td>4+</td>
<td>0</td>
</tr>
<tr>
<td>1+</td>
<td>4+</td>
</tr>
</tbody>
</table>
Group AB

- Least frequent across all racial lines
  - 4%
- Antigens = A, B (little to no H)
- Subgroups of A (A₁B, A₂B, etc.)
- Antibodies: NONE
  - “Universal recipient”
ABO Discrepancies

- Red cell grouping ≠ serum grouping
- Something’s wrong with antigen testing
- Something’s wrong with antibody testing
- Somebody messed up the testing

- Bottom line: **Until you KNOW, give Group O!**
Lewis System

- *Le* allele

![Diagram of the Lewis System with the following sugars: Fuc, GlcNAc, Gal, Lea, and R. The Type 1 Chain is indicated on the right side.]
Lewis System

- Wait, Se works here, too!

Type I H antigen

H and Le\textsuperscript{b}
Lewis Antigens

Le(a-b+)

RBCs

Plasma Glycolipids

Lipoproteins
Mismatch

- Antibodies are insignificant (cold IgM)
- HTR’s are rare (anti-Le\textsuperscript{a})
- HDFN not seen
  - IgM antibodies
  - Fetal RBCs lack Le antigens
    - Cord blood is Le[a-b-]
I System

Type 2 chain
I product is enzyme

ABO antigens get stronger in parallel with I
I System

“Big I in big people, little i in little people”
Classic Associations

• Auto-anti-I
  - Mycoplasma pneumonia
  - Cold Agglutinin Disease
    ✓ CLL, NHL, Waldenstroms
• Auto-anti-i
  - Infectious mononucleosis
  - Cold Agglutinin Disease
P1PK/GLOB Systems

• The weirdest blood group around
• Three historic antigens:
  - P1 and P^k in P1PK system
  - P now a globoside ("GLOB") antigen
  - Missing all 3: “p phenotype”
• P1 famous: Hydatid cysts and pigeon eggs!
• P: Parvovirus point of entry
P1PK Antibodies

- Antibodies usually cold, insignificant IgMs
- Auto-anti-P: “Biphasic IgG hemolysin” (next)
- p phenotype antibodies:
  - Rare anti-PP1P\textsuperscript{k}
    - ✓ Acute HTRs
    - ✓ Spontaneous abortions (placenta)
P1PK Antibodies

- Paroxysmal Cold Hemoglobinuria
  - Biphasic IgG autoanti-P (Donath-Landsteiner)
    ✓ *Cling* when it’s cold, *hurts* when it’s hot
  - Seen after viral infection in children
Donath-Landsteiner Test

A: 4C to 37C
B: 4C only
C: 37C only
New Plan

• 1/22 (today!): Finish Immunohematology I
• 2/5: Immunohematology II (Antibody ID, cases)
• 3/5: Blood Components
• 3/26: Transfusion Reactions

• Don’t forget: BBGuy.org/LLU is your page!
  • (PW: LLUPathology)
  • Notes, slides, new stuff!
### Enzyme Classification

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</table>
Interpret ABO Testing

Forward

L: A1 RBCs, R: B RBCs

Interp: Group B

Reverse

Interp: Group B
Which association is correct?

Auto-anti-I: Mycoplasma pneumonia
Anti-i: Paroxysmal Cold Hemoglobinuria
Auto-anti-P: Infectious mononucleosis
Most common HDFN: Blood group A mother

Total Results: 0
A 23 year old new mother has the genotype \textit{hh, sese}. What ABO type will she be, and what ABO type will her newborn be?

Mom: Any type; Baby: O only

Mom: Either A or B; Baby: Either A or B

Mom: O; Baby: Any type

Mom: Any type; Baby: Any type

Total Results: 0
Bombay O\textsubscript{h} Phenotype

- Genotype: \textit{hh, sese}
- No H, A, or B due to no \textit{H} or \textit{Se}

<table>
<thead>
<tr>
<th>Red Cell</th>
<th>Serum</th>
<th>ABO Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-A</td>
<td>Anti-B</td>
<td>A\textsubscript{1} RBCs</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>4+</td>
</tr>
</tbody>
</table>

H lectin (\textit{Ulex europaeus})
Rh System

• Five main antigens (50+ overall)
  - D, C, E, c, e
  - “Rh+” means “D+”
  - Rh– described as “d”
Rh System Genetics

Chromosome 1

- **RHD**: D+, D-
- **RHCE**: Ce, cE, ce, CE
Rh System

Mom

D

RHD

C e

RHCE

DCE = R₁

Dad

d

RHD

(Mutated/deleted)

c e

RHCE

dce = r

“Haplotype”
<table>
<thead>
<tr>
<th>$R_1$</th>
<th>$D$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$R_2$</td>
<td>$D$</td>
</tr>
<tr>
<td>$R_0$</td>
<td>$D$</td>
</tr>
<tr>
<td>$R_z$</td>
<td>$D$</td>
</tr>
</tbody>
</table>

There's no such thing as “d”

**RHD Options**

- **D**
- **d**
$R_1^\circ = \begin{array}{c|c|c}
D & Ce & \text{DCE} \\
\hline
\end{array}$

$R_2^\circ = \begin{array}{c|c|c}
D & cE & \text{Dce} \\
\hline
\end{array}$

$R_0^\circ = \begin{array}{c|c|c}
D & ce & \text{Dce} \\
\hline
\end{array}$

$R_z^\circ = \begin{array}{c|c|c}
D & CE & \text{DCE} \\
\hline
\end{array}$

$r'' = \begin{array}{c|c|c}
\text{d} & \text{CE} & \text{dCE} \\
\hline
\end{array}$

$r'' = \begin{array}{c|c|c}
\text{d} & \text{cE} & \text{dce} \\
\hline
\end{array}$

$r_y = \begin{array}{c|c|c}
\text{d} & \text{CE} & \text{dCE} \\
\hline
\end{array}$
<table>
<thead>
<tr>
<th>$R_1$</th>
<th>$D$ $Ce$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$R_2$</td>
<td>$D$ $cE$</td>
</tr>
<tr>
<td>$R_0$</td>
<td>$D$ $ce$</td>
</tr>
<tr>
<td>$R_z$</td>
<td>$D$ $CE$</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>$r'$</th>
<th>$d$ $Ce$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$r''$</td>
<td>$d$ $cE$</td>
</tr>
<tr>
<td>$r$</td>
<td>$d$ $ce$</td>
</tr>
<tr>
<td>$r^y$</td>
<td>$d$ $CE$</td>
</tr>
</tbody>
</table>

BloodBankGuy.org
The “Fantastic Four”

- \( R_1, R_2, R_0, \) and \( r \) (97%)

<table>
<thead>
<tr>
<th></th>
<th>Whites</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( R_1 )</td>
<td>( r )</td>
<td>( R_2 )</td>
<td>( R_0 )</td>
</tr>
<tr>
<td>Blacks</td>
<td>( R_0 )</td>
<td>( r )</td>
<td>( R_1 )</td>
<td>( R_2 )</td>
</tr>
</tbody>
</table>

1. \( R_0 \) 1st in blacks, last in whites
2. \( r \) always second
3. \( R_1 > R_2 \)
The “Fantastic Four”

- R₁, R₂, R₀, and r (97%)

<table>
<thead>
<tr>
<th></th>
<th>Whites</th>
<th>Blacks</th>
<th>Asians</th>
</tr>
</thead>
<tbody>
<tr>
<td>R₁</td>
<td>R₂ &gt; R₀</td>
<td>R₂ &gt; R₁</td>
<td>R₂ &gt; R₀</td>
</tr>
<tr>
<td>R₂</td>
<td>R₀ &gt; r</td>
<td>R₁ &gt; R₂</td>
<td>R₀ = r</td>
</tr>
<tr>
<td>R₀</td>
<td>r &gt; R₁</td>
<td>R₂ &gt; R₀</td>
<td></td>
</tr>
<tr>
<td>r</td>
<td>R₁ &gt; r</td>
<td></td>
<td>R₁ &gt; R₂</td>
</tr>
</tbody>
</table>
RhD-negative

- Mutations and deletions, not $d$
- Caucasians most likely D-negative
- Asians RARELY D-negative

<table>
<thead>
<tr>
<th>Caucasians (15%)</th>
<th>African-Americans (3-5%)</th>
<th>Asians (&lt;0.1%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deleted $RHD$</td>
<td>Deleted $RHD$ “Psi” pseudogene</td>
<td>Mutated $RHD$ DEL</td>
</tr>
</tbody>
</table>
Rh Antibodies

- Exposure-requiring warm IgG
- Rh antigens (esp. D) stimulate antibodies well
  - Old: 80% of D– make anti-D
  - New: 22% of D– hospital pts make anti-D
- Consequences:
  - HTRs, primarily extravascular
  - Prototypical HDFN with anti-D
  ✓ Anti-c: Severe HDFN, others mild
What’s “Weak D?”

**Routine D Testing (Steps 1-4)**
1. Anti-D
2. Pt. RBCs
3. Spin
4. Read

Mix of IgM/IgG

5. Incubate 15-30 min, spin
6. Wash, add AHG, spin

**Weak D Test (Steps 5-6)**
Weak D

Typical D+

True Weak D

“Serologic Weak D”

Current AABB Recommendation:
Consider Rh genotyping with serologic weak D in Tx service
- Saves Rh neg supply
- Saves RhIg injections for moms

Recipients: OK to call D neg
Donors: MUST NOT call D neg
Partial D

• **Qualitative** D defect
  - Parts of D on outside of RBC via *RHD* mutation
• Abs vs. missing parts ~ anti-D
  - Classic: Anti-D in a D+ person

Weird Stuff

- Rh$_{\text{null}}$
  - Mutation in $\text{RHAG}$ gene (3rd Rh gene)
    ✓ Structural problem
  - No Rh antigens
  - Stomatocytic hemolytic anemia
Kidd System

• Antigens:
  - $Jk^a$ and $Jk^b$
    ✓ $Jk^3$: Absent in $Jk(a^b^-)$
  - Urea transport antigen
    ✓ $Jk(a^b^-)$ resistant to 2M urea
Urea Transport

= Urea
Screen for $\text{Jk}(a-b-)$

- $\text{Jk}(a+)$ or $\text{Jk}(b+)$
- $\text{Jk}(a-b-)/\text{Jk}3-$
Screen for Jk(a-b-)

Hi, there!
Kidd Antibodies

- Warm IgG (+/- with IgM component)
  - May fix complement
- Rarely found alone
- Marked dosage
- Disappear over time
  - “Hide and seek”
- Delayed (or immediate) hemolytic reactions
  - Undetectable antibody that comes roaring back
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</tr>
</tbody>
</table>
MNS System

• 49 recognized antigens; 5 are most important
• **Glycophorin A and B**
  - Glycophorin A carries M and N
  - Glycophorin B carries S, s, and U
  - Both are receptors for malaria parasite
    ✓ *P. falciparum* attaches here
The diagram illustrates the membrane proteins GPA\(_M\) and GPA\(_N\), with amino acid changes indicated:

- GPA\(_M\): Ser \(\leftrightarrow\) 1 \(\leftrightarrow\) Leu
- GPA\(_N\): Gly \(\leftrightarrow\) 5 \(\leftrightarrow\) Glu

For the other proteins, the changes are:

- GPB\(_S\): Met \(\leftrightarrow\) 29 \(\leftrightarrow\) Thr
- GPB\(_S\): U \(\leftrightarrow\) U

The notation 'N' indicates a specific region or feature of the protein.
# MNS Antibodies

<table>
<thead>
<tr>
<th>Anti-M and -N</th>
<th>Anti-S, -s, &amp; -U</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Natural”</td>
<td>Exposure</td>
</tr>
<tr>
<td>IgM/IgG</td>
<td>IgG</td>
</tr>
<tr>
<td>Cold-reactive</td>
<td>37 and AHG</td>
</tr>
<tr>
<td>Us. Insignificant</td>
<td>Significant</td>
</tr>
</tbody>
</table>

Occasional anti-M at 37°C
“Naturally Occurring”

M

N

S

26 AA’s

Outside

Inside

GPA_M

GPA_N

GPB_S

GPB_S

Naturally Occurring
Exposure Required
Exposure Required

Outside

Inside

$\text{GPA}_M$

$\text{GPA}_N$

$\text{GPB}_S$

$\text{GPB}_S$
MNS Antigens

• S-s-U-
  - 1% of African-Americans, never in Caucasians
  - Anti-U is significant risk
    ✓ Can cause HTR and HDFN
Duffy System

- **Fy\(^a\)**, **Fy\(^b\)** main antigens (*FY*A, *FY*B alleles)
  - Carried on RBC membrane protein (ACKR1)
  - Also on endothelial cells of:
    - Heart, lung, kidney, spleen, GI tract
- *FY*B is commonly mutated in African-Americans
- This mutation impacts Fy\(^b\) expression ON RBCs
- **BUT NOT everywhere else!**

Fy(a-b-)

<table>
<thead>
<tr>
<th></th>
<th>C</th>
<th>AA</th>
</tr>
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<tbody>
<tr>
<td>Fy(a+b-)</td>
<td>17</td>
<td>9</td>
</tr>
<tr>
<td>Fy(a+b+)</td>
<td>49</td>
<td>1</td>
</tr>
<tr>
<td>Fy(a-b+)</td>
<td>34</td>
<td>22</td>
</tr>
<tr>
<td><strong>Fy(a-b-)</strong></td>
<td>rare</td>
<td><strong>68</strong></td>
</tr>
</tbody>
</table>

Fy(a–b–) individuals of African descent rarely make anti-Fy\textsuperscript{b}

Fy(a–b–) individuals of African descent are resistant to *P. vivax*

Duffy Antibodies

• Exposure-requiring warm IgG
  - Marked dosage (like Kidd)
  - Can disappear (like Kidd)
  - Delayed HTRs (like Kidd)
  - Anti-Fya >> anti-Fyb
## Enzyme Classification

<table>
<thead>
<tr>
<th>Enhanced</th>
<th>Decreased</th>
<th>Unaffected</th>
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<tbody>
<tr>
<td>ABO-related</td>
<td>MNS System</td>
<td>Kell System</td>
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<td>-ABO/H</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Lewis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-P1PK/GLOB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rh System</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kidd System</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Dithiothreitol (DTT)

Kp\textsuperscript{a}/Kp\textsuperscript{b}/Kp\textsuperscript{c}

K/k

RBC

Membrane

Xk  Kell

Js\textsuperscript{a}/Js\textsuperscript{b}

BloodBankGuy.org
Kell Antigens

- 36 antigens!
- Most important: \textbf{K} (aka KEL1, NOT “Kell”)
  - 9% Caucasians, 2% African-Americans
- High frequency:
  - k (aka KEL2, “Cellano”); 99.8%
  - $\text{Js}^b$, $\text{Kp}^b$
Kell Antibodies

- **Anti-K**
  - After D, most immunogenic non-ABO antigen
    - ✓ 1/3 of non-Rh antibodies!
  - Exposure-requiring warm IgG
  - Severe HTRs
  - Severe HDFN*

- **Anti-k**
  - Like anti-K, just very uncommon
**Anti-K “HDFN”**
- Suppression
- Reticulocytopenia
- ANEMIA

Titers hardly matter

**RhD HDFN**
- Hemolysis
- Reticulocytosis
- Hyperbilirubinemia
- Anemia

**vs.**
Ku = Kell “universal”
McLeod Neuroacanthosis

<table>
<thead>
<tr>
<th>McLeod Phenotype</th>
<th>McLeod Syndrome</th>
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<tr>
<td>RBCs lack Kx</td>
<td>RBCs lack Kx</td>
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<td>RBCs have decreased Kell Ags</td>
<td>RBCs have decreased Kell Ags</td>
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<td>Acanthocytic hemolytic anemia</td>
<td>Acanthocytic hemolytic anemia</td>
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<tr>
<td>X-linked CGD (minority)</td>
<td>X-linked CGD (minority)</td>
</tr>
<tr>
<td>Chorea, seizures</td>
<td>Chorea, seizures</td>
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<tr>
<td>Psychiatric disorders</td>
<td>Psychiatric disorders</td>
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<tr>
<td>Muscle wasting</td>
<td>Muscle wasting</td>
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<tr>
<td>Cardiac arrhythmia</td>
<td>Cardiac arrhythmia</td>
</tr>
</tbody>
</table>

Image: Blood 108:5;1433 (Maslak P)
Pretransfusion Testing

Blood Supplier Actions (yellow)
- ABO Type RhD (+ weak D) Antibody Screen
- Infectious Disease Screening
- Reconfirm donor: ABO RhD (no weak D)

Transfusion Service Actions (blue)
- Proper Specimen Collection
- ABO Type RhD (no weak D) Antibody Screen
- Antibody ID (if necessary)
- Previous Records Check
- Select Components
- Compatibility Check
- Label with Patient Info

ISSUE PRODUCT!
Recipient Testing

- Request forms
  - Proper ID critical!
  - No ID labeling errors should be corrected
    ✓ 1997 study: samples 40X more likely to have issues if mislabeled
  - What’s needed and when needed
  - Provider
  - Modifications
Specimen

- Labeled at bedside
- Red top (serum) or lavender top (plasma) OK
  - Current technologies: Plasma
    ✓ Serum: Debris interference
    ✓ Plasma may inhibit C-dep. Abs
- Two separate samples required by AABB/CA if first time seeing patient in your facility
Type and Screen

- Test ordered when RBCs *might* be needed
- Records check for comparison only
- ABO testing
- Rh typing (no weak D)
- Antibody detection / “screen”
Antibody Screen

- Pt plasma/serum vs. 2/3/4 group O donors
- MUST read at AHG
- 18 antigens required by FDA
  - D, C, c, E, e, Jk^a, Jk^b, M, N, S, s, Fy^a, Fy^b, K, k
  - Le^a, Le^b, P1
### Antibody Screen

<table>
<thead>
<tr>
<th>Cell</th>
<th>Rh-hr</th>
<th>D</th>
<th>C</th>
<th>E</th>
<th>c</th>
<th>e</th>
<th>f</th>
<th>C*</th>
<th>K</th>
<th>Kp</th>
<th>Kp</th>
<th>Js</th>
<th>Js</th>
<th>Jk</th>
<th>Jk</th>
<th>Fy</th>
<th>Fy</th>
<th>Le</th>
<th>Le</th>
<th>Le</th>
<th>M</th>
<th>N</th>
<th>S</th>
<th>s</th>
<th>P1</th>
<th>Lu</th>
<th>Lu</th>
<th>Results</th>
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<tbody>
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</tbody>
</table>
Type and Crossmatch

• When RBCs are *most likely* or *definitely* needed
• Everything in T&S PLUS crossmatch
• **Major** crossmatch
  - Recipient serum vs. donor RBCs
  - ABO compatibility
  - Required if ≥ 2 ml **RBCs** in product
• **Minor** crossmatch
  - Donor serum vs. recipient RBCs
Crossmatch Types

- **Immediate Spin (IS)**
  - Rapid ABO check only
  - Only with no antibodies

- **Electronic (computer)**
  - Same as IS; no antibodies allowed
  - Validated system

- **AHG (“Full crossmatch”)**
  - Required with significant antibodies now or ever
“Convert” T&S to T&C

• If antibody screen negative:
  - ABO check only is needed
  - IS or computer crossmatch
  - Should be very quick!
The q 3 Day Rule

If transfused or pregnant in preceding 3 months:

<table>
<thead>
<tr>
<th>Sun</th>
<th>Mon</th>
<th>Tues</th>
<th>Wed</th>
<th>Thurs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Draw Sample 2 pm</td>
<td></td>
<td></td>
<td></td>
<td>New Sample</td>
</tr>
<tr>
<td>Sample used for crossmatch for any transfusion thru Wednesday Midnight</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

0 1 2 3 0
Variations for Neonates

• Babies < 4 months
  - Baby’s antibodies = mom’s antibodies
  - Check baby ABO/Rh, mom’s plasma for Abs
  - No XM or serum grouping (if giving O RBCs)