~ A Blood Bank Road Trip

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Immunohematology Reference Laboratory

American Red Cross

Points of Interest (Objectives)

Discuss the analogy between a Road Trip and a Blood Bank Work-Up.

Look at the signs that indicate we may be dealing with a high prevalence antigen/antibody.

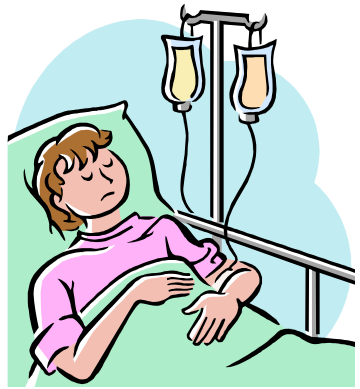
Review the tests that will help aid in the identification of a high prevalence antigen/antibody.

Examine a case study involving an antibody to a high prevalence antigen.

Blood Bank Work-Ups are a lot like Road Trips….

The first thing you need to decide when planning a Road Trip is your final destination. Where do you want to go?

For a Blood Bank Work-Up, the answer is easy… our final destination is to obtain the safest possible transfusion for our patients because in the end, it is really all about them.



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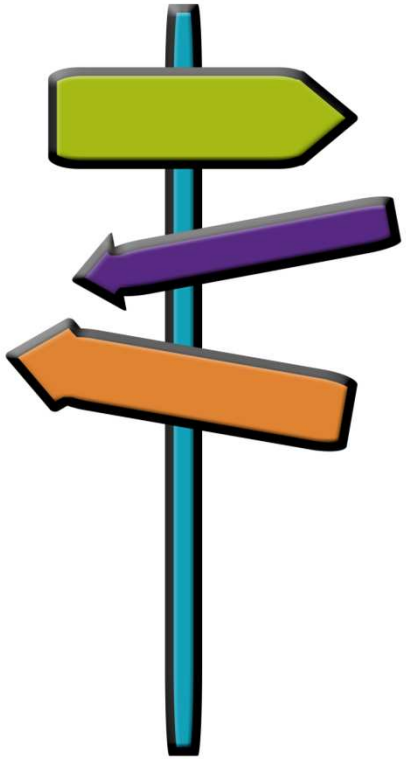
Blood Bank Work-Ups are a lot like Road Trips….



Signs along the way on our Road Trip help point us in the right direction to reach our final destination.

For Blood Banks Work-Ups the signs we use are the

- Patient's previous antibody history
- Patient's previous transfusion and/or pregnancy history
- Patient's race
- Direct antiglobulin test (DAT)/auto control results
- Antibody screen/panel phase of reactivity and/or pattern of reactivity

Blood Bank Work-Ups are a lot like Road Trips….

For our Road Trip we may use a navigation system to lead us on the correct path.

For our Blood Bank Work-Up, the navigation system is our work instructions or procedures that tell us how to perform the correct tests to find the answer.

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Blood Bank Work-Ups are a lot like Road Trips….

Even with a good navigation system, it is always good idea to have a map just in case the navigation system doesn't actually match up with the road you happen to be traveling.

For a Blood Bank Work-Up, this map is an overall picture of what the "signs" are telling us and the what the possible outcome may be.

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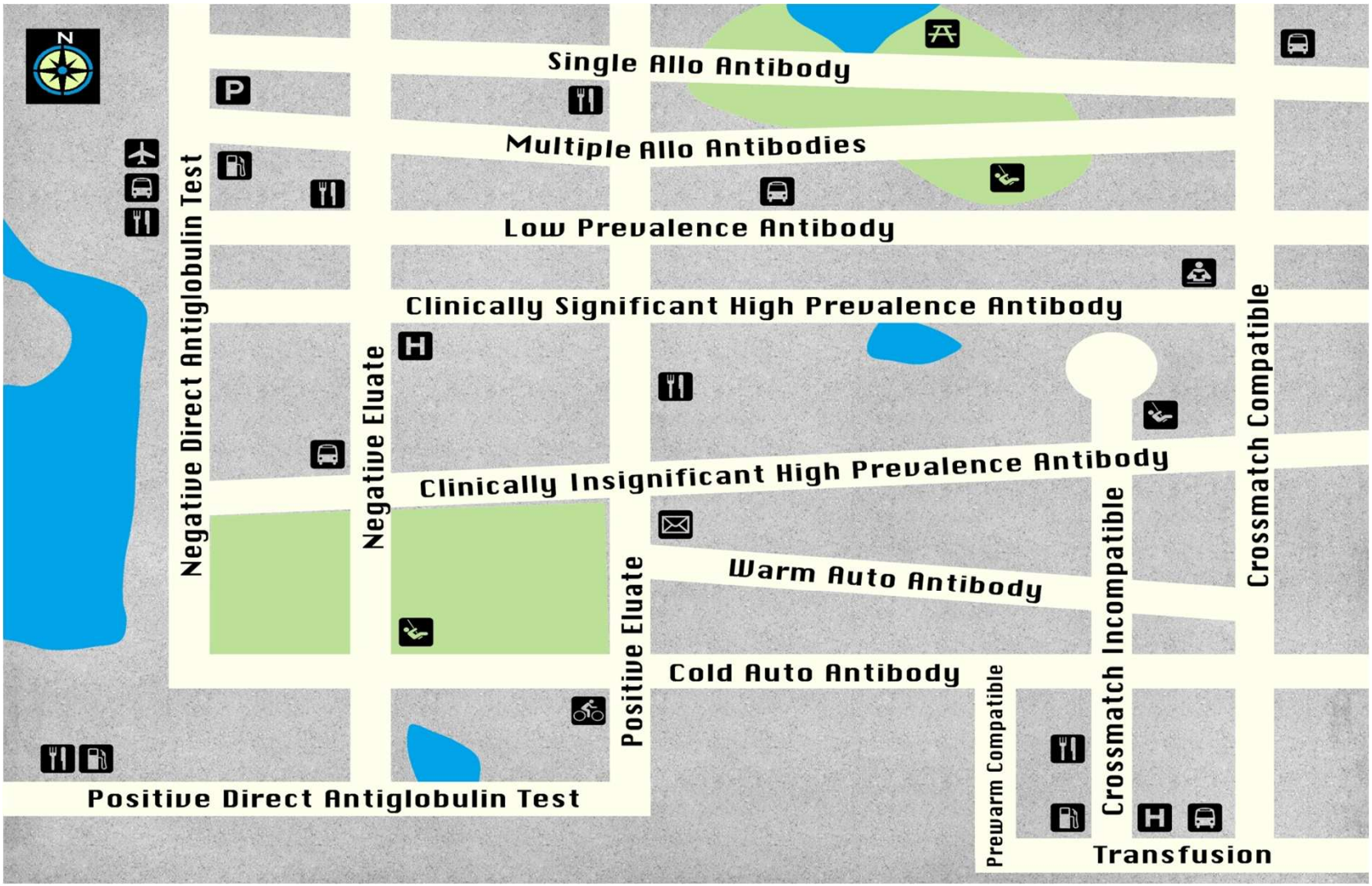
Our Blood Bank Road Map…



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Blood Bank Work-Ups are a lot like Road Trips….

On Road Trips, even with navigation and a map, sometimes we just need to stop and ask for directions when we are lost and the signs are not matching our map. Maybe, someone more familiar with the area can help us interpret our map better.

For Blood Banks Work-Ups, we do this when we ask our co-workers or supervisors for guidance on which way they think we should go next when test results are not giving us a straight forward picture.

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Blood Bank Work-Ups are a lot like Road Trips….

Even with all that guidance, we are bound to still come upon

DETOURS & ROAD BLOCKS

on our Road Trip and in our Blood Bank Work-Up.

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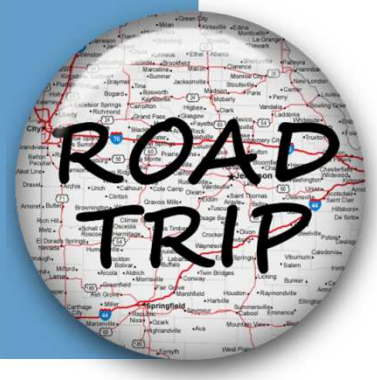
We Will Be Traveling the "High" Road on Our Blood Bank Road Trip.



The antibodies to high prevalence antigens can be split into two groups:

- Clinically Significant
- Clinically Insignificant

  (those that do not cause shortened red cell survival of antigen positive units)

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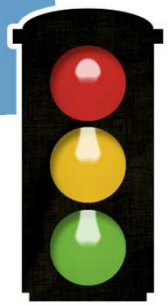
Definition/Classification of High Prevalence Antigen/Antibody



Antigens are considered to be high prevalence if more than 98% of the population is positive for the antigen.

Currently, there are 30 blood group systems and several blood group collections. High prevalence antigens can be found in most of these systems and collections. However, there are eight high prevalence antigens that are not eligible for classification into a system or collection. These 8 antigens are collectively called the 901 series.

Antibodies that are directed against these antigens are called “high prevalence antibodies” in our lab. However, the antibodies are quite uncommon since most people are positive for the antigen and therefore can not make the antibody.

Examples of High Prevalence Antigens

System/ Collection	MNS		Rh			Lutheran		Kell			Duffy	Kidd	Yt	Colton
Antigen or Phenotype	U	En^a	hr^S	hr^B	Rh29	Lu8	Lu20	k	Kp^b	Js^b	Fy3	Jk3	Yt^a	Co^a
Ethnic origin	Black	Finn> Canadian	Black	Black			Israeli	Caucasian> Black>any	Caucasian> Japanese	Black	Black (32%)	Polynesian	Arabs> Jews	
Percentage	99	99.9	98	98	100	99.8	100	99.8	100	99	100	100	99.8	99.9
DTT	Pos	Var	Pos	Pos	Pos	Neg	Neg	Neg	Neg	Neg	Pos	Pos	Neg	Pos
Enzyme	Pos	Pos	**Pos**	**Pos**	**Pos**	Pos	Pos	Pos	Pos	Pos	Pos	**Pos**	Pos	Pos
Trypsin	Pos	Var	Pos	Pos	Pos	Neg	Neg	Pos	Pos	Pos	Pos	**Pos**	Var	Pos

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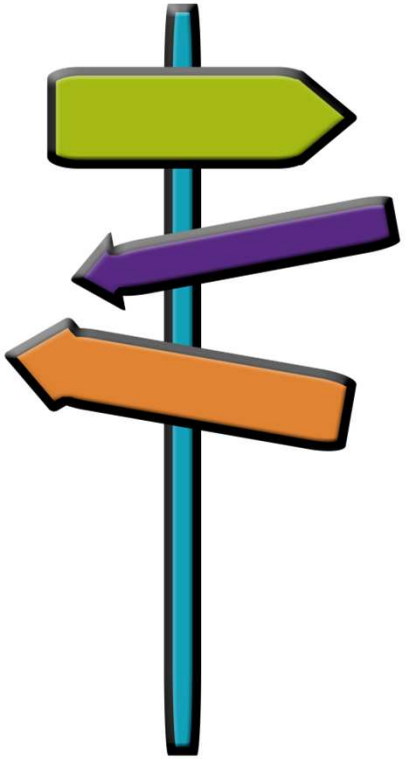
Signs That We May Be Dealing With a High Prevalence Antibody



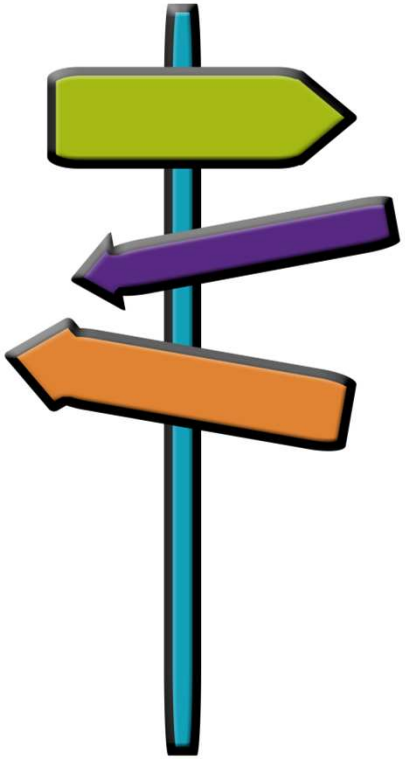
- **Direct antiglobulin test (DAT)/auto control results**

  The DAT and auto control are usually negative.

  DETOUR: This may not be true if the patient has been recently transfused and the antibody is newly made and coating the transfused cells.

  ROAD BLOCK: Patient's may have multiple things going on in their serum. It is possible for a patient to have both a cold or warm autoantibody along with the high prevalence antibody.

Signs That We May Be Dealing With a High Prevalence Antibody



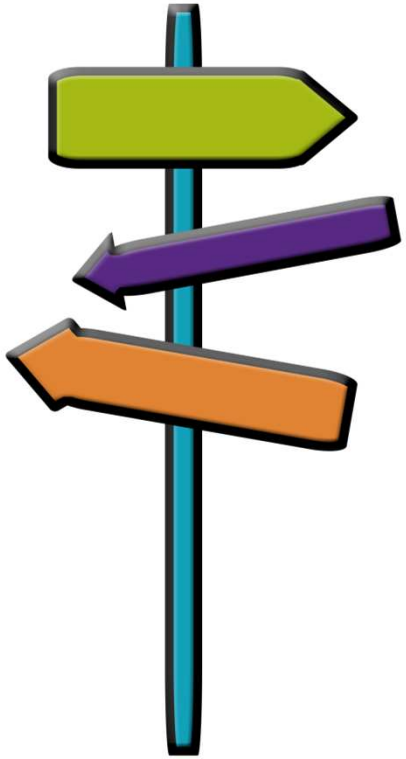
- **Antibody screen/panel phase of reactivity and/or pattern of reactivity**

  Generally, when a high prevalence antibody is present, the pattern of reactivity is uniform with all panel cells and donor red cells.

  DETOUR: You may see variable reaction strength if the patient has other alloantibodies besides the high prevalence antibody.

  ROAD BLOCK: You may see variable reaction strength if the high prevalence antibody is showing dosage and you test panel cells with a heterozygous expression of the antigen.

Signs to Help Us Determine the Identity of the High Prevalence Antibody



- **Patient's previous antibody history**

  Obviously the easiest way to ID a high prevalence antibody is by obtaining the antibody specificity from another facility or the patient themselves.

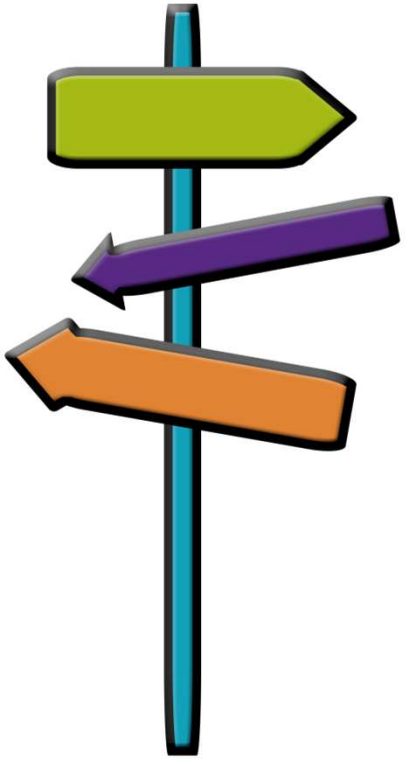
  ROAD BLOCK: The patient knows that it was hard for the facility to find blood for him the last time he was transfused, but he doesn't know exactly why. He also doesn't remember where he was when he had that problem.

- **Patient's previous transfusion and/or pregnancy history**

  The transfusion history can aid in ID of the high prevalence antibodies by letting us know whether or not we need to perform cell separation techniques prior to phenotyping the patient for suspected high prevalence antigens.

  DETOUR: The patient is incoherent or can not be reached to obtain this information.

Signs to Help Us Determine the Identity of the High Prevalence Antibody

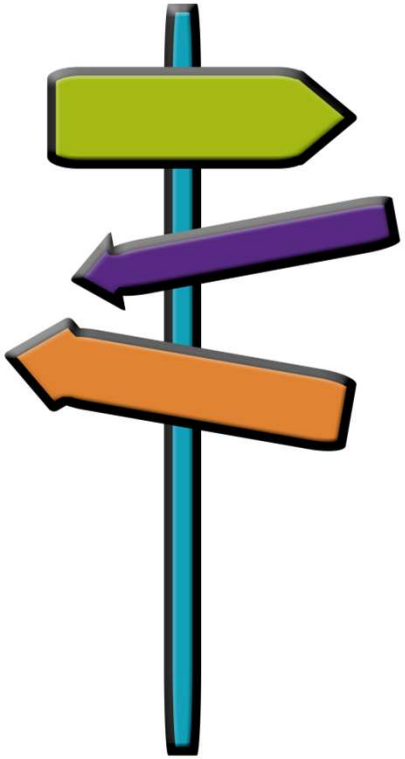


- **Patient's race**

  The ethnic origin of the patient is actually a very helpful clue to determine which high prevalence antibodies should be explored first. Lacking certain high prevalence antigens is commonly linked to specific populations. Knowing the patient's race gives a starting place for which specificities to attempt.

  ROAD BLOCK: Information is not supplied by submitting facility or not able to obtained in a timely fashion.

Signs to Help Us Determine the Identity of the High Prevalence Antibody



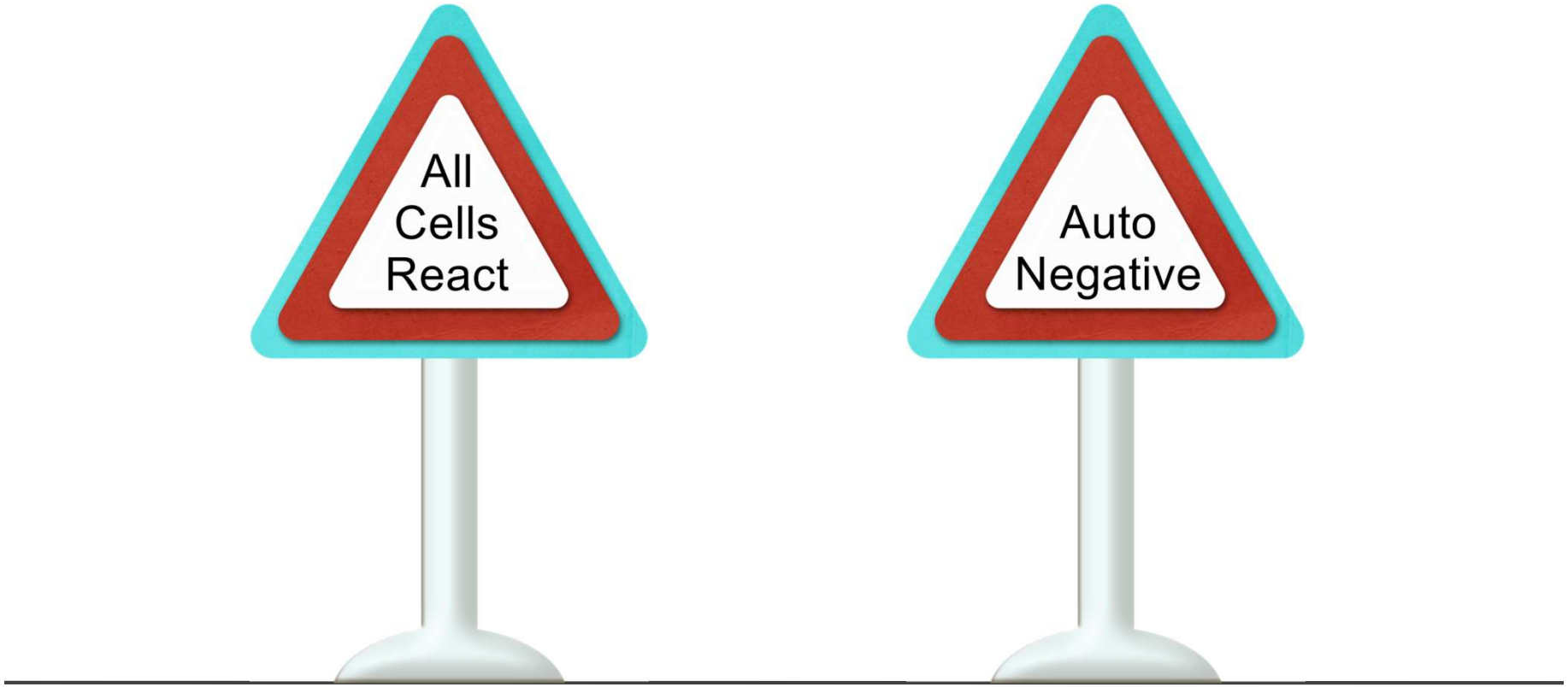
- **Antibody screen/panel phase of reactivity and/or pattern of reactivity**

  Serologic characteristics play a huge part in providing clues to high prevalence antibody IDs. Reactions at IS or RT suggest that the antibody is IgM in nature and an antibody specificity that prefers colder temperatures should be investigated. If in vitro hemolysis is present, Anti-P,P1,P^k, -Vel, or -Jk3 should be investigated. Further testing with enzyme or chemically treated cells also aids in determining which antibody specificities to test.

  DETOUR: Not all antibodies react by the book or the book is not clear.

  ROAD BLOCK: Having more than one antibody present may cause misleading results to the antibody ID of the high prevalence antibody.

One more lap…
What are the two “Signs” that we are dealing with an antibody to a high prevalence antigen?



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Our Road Trip Destination…

Transfusion of Patient UL

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Patient Information Received From Referring Hospital

- 79 year old Caucasian Female
- Diagnosis: Port Revision
- Current Hgb/Hct: 11.1/34.5
- Date of last transfusion: December 14, 2006
- Previous antibody history: Nonspecific antibody in Gel.
- Sent to IRL as Life Threatening need and surgery postponed until results are received from our lab. Two units on hold are requested for surgery.
- Referring hospital results: O Pos DAT: Negative
  All panels cells 3+ positive at Gel-IgG
  Auto control is reactive

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IRL Initial Testing

Blood Type: O Positive

Rh Phenotype: D+ C- E+ c+ e+ (R2r)

DAT: Negative

Initial Panel: all 11 cells tested at Gel-IgG react 3+; auto control is 1+

Essentially, we are seeing the same results as the referring facility

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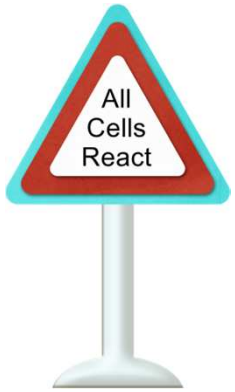
Do we see any signs that we are dealing with an antibody to a high prevalence antigen?



Yes, all cells are reacting and their reaction strength is uniform

No, the auto control is positive, but the DAT is negative

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We have come to our first Road Block

Our signs are not direct. We need to run more tests to get a better picture of what we are dealing with.

What other roads could we possibly be traveling based on the results of our initial tests?

Warm Auto Antibody?

It is unlikely that a patient with a warm auto antibody has a negative DAT.

Cold Auto Antibody?

Definite possibility

Multiple Alloantibodies?

Probably not since we do not see any variability

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What Next

Run more tests

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Further Testing to Exclude Other Possible Roads to Take

Additional tube tests were performed to further investigate the negative DAT. This included testing with multiple sources of Poly AHG and Anti-IgG and Anti-C3. All of these tests were negative. A warm autoantibody does not seem likely and was not pursued further.

A selected cell panel which rules out all common clinically significant alloantibodies (a rule out panel) along with the auto control was tested at 15' RT and 15' 4°C. No reactivity was seen at these phases. This eliminates the possibility that we are dealing with a cold autoantibody.

A complete phenotype was performed on the patient. Several panel cells which are phenotypically similar to the patient were tested. These cells were all 3+ at Gel-IgG. If we are only dealing the common alloantibodies, then these cells would be nonreactive.

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More testing is needed…

Complete Phenotype: M+ N+ S+s-; K-; Fy(a+b-); Jk(a+b-); P1+; Le(a-b+)

~Unfortunately, there is nothing in the phenotype that jumps out and screams, “It’s Me!” However, we do know which alloantibodies our patient can make.

So far, we have only tested an original panel in Gel and a cold panel. In our lab, we are most experienced with testing in tubes. So, when we reach the point of puzzlement, we always switch to tube testing.

NAME UL
NO. 4050-63108
INSTITUTION
BLOOD GROUP
ANTIBODY IDENTITY
TECH AQ DATE 08162011

PANOCELL -10, FICIN-TREATED
Master List

MO-IL Regional Red Cross
4050 Lindell Blvd.
St. Louis, MO 63108

IMMUCOR, INC. Norcross, GA 30071 USA
US LICENSE NO: 886
LOT NO: 27063
EXPIRES: 2011/09/09

			Rh - Hr							Kell						Duffy		Kidd		Lewis		P	MN				Luth-eran		Xg	PATIENT'S SERUM TEST RESULTS TEST METHODS						
VIAL	Special Type	Donor	D	C	c	E	e	V	C^w	K	k	Kp^a	Kp^b	Js^{a*}	Js^b	Fy^a	Fy^b	Jk^a	Jk^b	Le^a	Le^b	P_1	M	N	S	s	Lu^a	Lu^b	Xg^{*a}		455					
1		R1R1 B2821	+	+	0	0	+	0	0	+	+	0	+	0	+	0	+	+	+	0	0	0	0	+	0	+	0	+	+	1	15	37	46				
2	Co(b+)	R1wR1 B7641	+	+	0	0	+	0	+	0	+	0	+	0	+	+	+	0	+	+	0	+	+	+	0	+	0	+	0	2						
3		R2R2 C3566	+	0	+	+	0	0	0	0	+	+	+	0	+	+	+	0	+	0	+	+	+	+	+	+	+	+	0	3	0	0	W+			
4		Ror D619	+	0	+	0	+	0	0	0	+	0	+	+	+	0	+	+	0	0	+	+	+	0	+	0	0	+	+	4						
5		r'r E403	0	+	+	0	+	0	0	0	+	0	+	0	+	+	0	+	+	0	+	+	+	0	0	+	0	+	0	5						
6		r''r F796	0	0	+	+	+	0	0	0	+	0	+	0	+	+	0	0	+	+	0	+	+	0	+	+	0	+	+	6						
7		rr G1096	0	0	+	0	+	0	0	+	+	0	+	0	+	0	+	+	+	+	0	0	0	+	0	+	0	+	+	7	0	0	W+			
8		rr H643	0	0	+	0	+	0	0	0	+	0	+	0	+	+	0	+	0	+	0	+	+	+	0	+	0	+	+	8	0	0	W+			
9	Yt(b+)	rr N1579	0	0	+	0	+	0	0	0	+	0	+	0	+	+	+	0	+	0	+	+	+	0	+	0	0	+	+	9						
10	Co(b+)	R1R1 B7636	+	+	0	0	+	0	0	0	+	0	+	0	+	+	+	+	0	0	+	0	+	0	0	+	0	0	+	+	10	0	0	W+			
TC	Di(a+)	R1R1 B4869	+	+	0	0	+	0	0	0	+	0	+	0	+	+	+	0	+	0	+	+	+	0	+	+	0	+	0	TC						
		Patient's Cells																											PC	0	0	0			

Direct Antiglobulin Test				Eluate Result
	Poly	IgG	C3	
LOT				
RESULT				

NOTES:
An antigen designated with a 'w' represents a weakened expression of the antigen that may or may not react with all examples of the corresponding antibody.

TC: The Diego system antigen Di^b is a high incidence antigen in all populations studied. Its antithetical partner, Di^a, is extremely rare in Caucasians, although it occurs more frequently in populations of Mongolian extract. Anti-Di^a has been reported to cause hemolytic disease of the newborn and transfusion reactions. Ref: Race RR, Sanger R. Blood groups in man. 6th ed. Oxford: Blackwell Scientific, 1975:372-8.

PATIENT'S SERUM:	PANOSCREEN LOT										
REVERSE GROUPING CELLS	I										
	II										
	III										
	A1										
	A2										
	B										

* Indicates those antigens whose presence or absence may have been determined using only a single example of a specific antibody.

What have we learned from this panel?

Remember our Gel results were 3+ indicating that this antibody reacts pretty strongly. However, when tested in the tube with LISS enhancement, we only see weak reactions at LISS-IgG. That is quite a different picture. Other key things to notice…

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Phenotypically Similar Cells

Patient's Name UL
Patient's Number 4050-63108
Date: Collected 08162011 Date: Tested 08162011

SELECTED PANEL
American Red Cross, Missouri-Illinois Region
4050 Lindell, St. Louis, MO 63108
Technologist: AJ.

	Supplier Lot #	Donor/ Vial#	RhHr								MN				P	Lew		Lut		Kell						Duf		Kid		X	Additional Antigens	Patient's Plasma Test Results
			D	C	E	c	e	f	V	Cw	M	N	S	s	P1	Lea	Leb	Lua	Lub	K	k	Kpa	Kpb	Jsa	Jsb	Fya	Fyb	Jka	Jkb	Xga		LISS IgG
1 OOD	Ortho A RA799	116787 4	+	0	0	+	+	+	0	0	0	+	+	0	+	0	0	0	+	0	+	0	+	0	+	0	0	+	0	+		WH
2 OOD	Imm-20 13792	N2412 19	0	0	0	+	+	+	0	0	0	+	+	0	+	0	0	0	+	0	+	0	+	0	+	0	0	+	0	+		WH
patient phenotype			+	0	+	+	+				+	+	+	0	+	0	+			0						+	0	+	0			

The phenotypically similar cells that reacted at Gel-IgG are also reacting at LISS-IgG, but much weaker.

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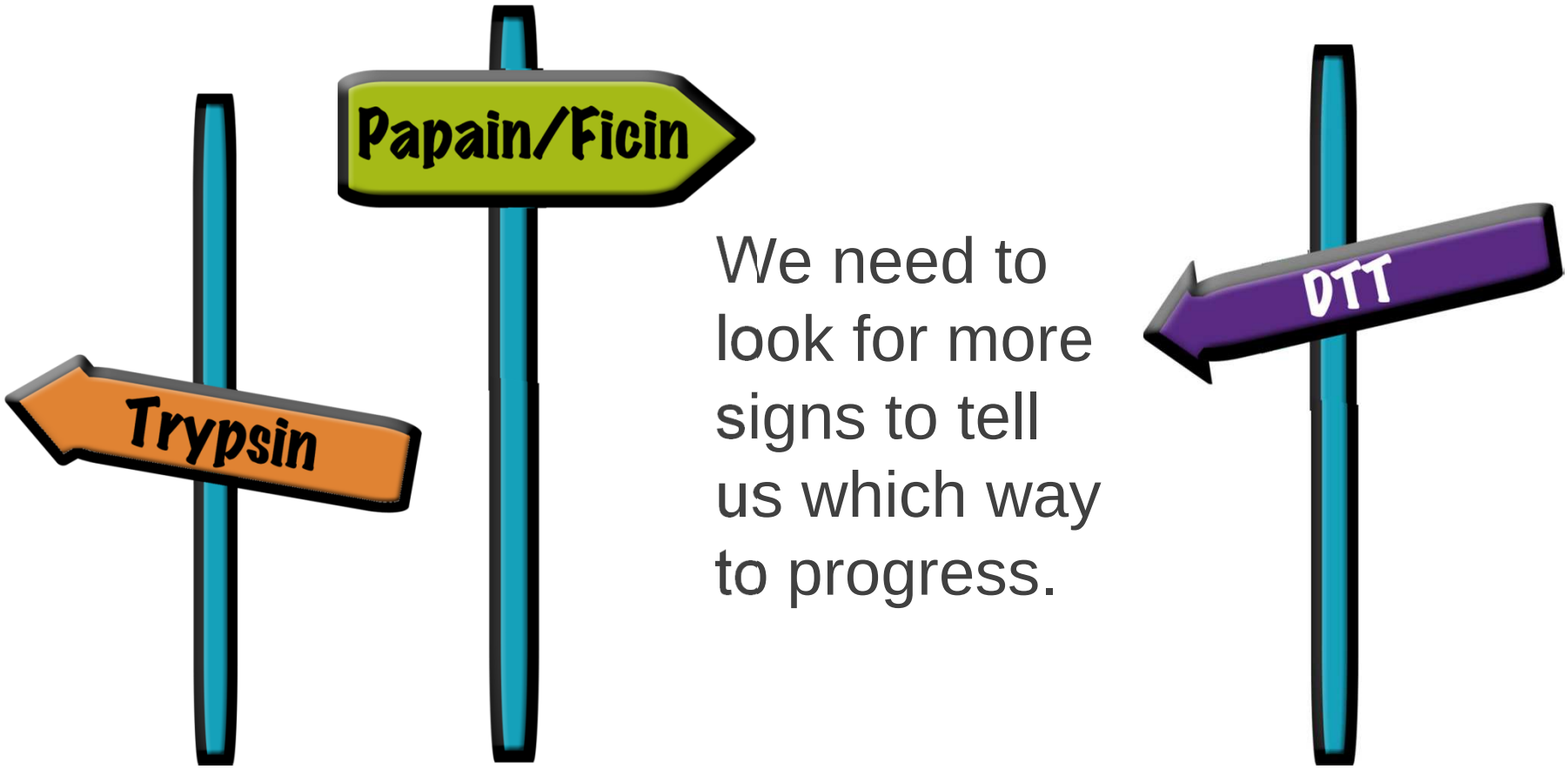
We are back on course,
But we still have lots of road left to travel.



We need to look for more signs to tell us which way to progress.

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VIAL	Special Type	Donor	D	C	c	E	e	V	C^w	K	k	Kp^a	Kp^b	Js^{a*}	Js^b	Fy^a	Fy^b	Jk^a	Jk^b	Le^a	Le^b	P_1	M	N	S	s	Lu^a	Lu^b	Xg^{*a}		FICIN 37 IgG	TRYPSIN 37 IgG	IAT LISS-IgG		Polybrene RT	Polybrene IgG
1		R1R1 B2821	+	+	0	0	+	0	0	+	+	0	+	0	+	0	+	+	+	0	0	0	0	+	0	+	0	+	+	1						
2	Co(b+)	R1wR1 B7641	+	+	0	0	+	0	+	0	+	0	+	0	+	+	+	0	+	+	0	+	+	+	0	+	0	+	0	2						
3		R2R2 C3566	+	0	+	+	0	0	0	0	+	+	+	0	+	+	+	0	+	0	+	+	+	+	+	+	+	+	0	3	W+	W+	W+		0	MS ⊖
4		Ror D619	+	0	+	0	+	0	0	0	+	0	+	+	+	0	+	+	0	0	+	+	+	0	+	0	0	+	+	4						
5		r'r E403	0	+	+	0	+	0	0	0	+	0	+	0	+	+	0	+	+	0	+	+	+	0	0	+	0	+	0	5						
6		r"r F796	0	0	+	+	+	0	0	0	+	0	+	0	+	+	0	0	+	+	0	+	+	0	+	+	0	+	+	6						
7		rr G1096	0	0	+	0	+	0	0	+	+	0	+	0	+	0	+	+	+	+	0	0	0	+	0	+	0	+	+	7	W+	W+	W+		0	M+ ⊖
8		rr H643	0	0	+	0	+	0	0	0	+	0	+	0	+	+	0	+	0	+	0	+	+	+	0	+	0	+	+	8	W+	W+	W+		0	M+ ⊖
9	Yt(b+)	rr N1579	0	0	+	0	+	0	0	0	+	0	+	0	+	+	+	0	+	0	+	+	+	0	+	0	0	+	+	9						
10	Co(b+)	R1R1 B7636	+	+	0	0	+	0	0	0	+	0	+	0	+	+	+	+	0	0	+	0	+	0	+	0	0	+	+	10	W+	W+	W+		0	M+ ⊖
TC	Di(a+)	R1R1 B4869	+	+	0	0	+	0	0	0	+	0	+	0	+	+	+	0	+	0	+	+	+	0	+	+	0	+	0	TC						
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PATIENT'S SERUM:								
PANOSCREEN LOT	I							
	II							
	III							
REVERSE GROUPING CELLS	A1							
	A2							
	B							

* Indicates those antigens whose presence or absence may have been determined using only a single example of a specific antibody.

Possible specificities based on reactivity

- A long list…
  - A,B; H; P1; Lewis; Rh; Kidd; En^{a}FR; U; Fy3; Di^{a},Di^{b},Wr^{a},Wr^{b}; Colton; Ok^{a}; I,i; P,P^{k},LKE; AnWj; At^{a}; Cs^{a}; Er; Jr^{a}; Lan; Vel; Sd^{a}; PEL;

- We start by selecting high prevalence antigen negative cells from ***liquid*** reagent red cell panels that fit the reactivity pattern we obtained.

Selected cells

Patient's Name UL
Patient's Number 4050-63108
Date: Collected 08162011 Date: Tested 08162011

SELECTED PANEL
American Red Cross, Missouri-Illinois Region
4050 Lindell, St. Louis, MO 63108
Technologist: AJ

	Supplier Lot #	Donor/ Vial#	RhHr								MN				P	Lew		Lut		Kell						Duf		Kid		X	Additional Antigens	Patient's Plasma Test Results						
			D	C	E	c	e	f	V	Cw	M	N	S	s	P1	Lea	Leb	Lua	Lub	K	k	Kpa	Kpb	Jsa	Jsb	Fya	Fyb	Jka	Jkb	Xga		USS J96						
1 OOD	Imm-16 34360	G242 15	0	0	0	+	+	+	0	0	+	+	+	0	+	0	+	0	+	+	0	0	+	0	+	+	+	0	+	+	Cs(a+w)	WH						
2 OOD	Biotest 8117011	600026 4	+	0	0	+	+			0	+	+	+	0	0	0	0	0	+	0	+	0	+	0	+	+	0	0	+	0	Cs(a-) Yk(a+w) Co(b+) Co(a-)	O+						
3 OOD	Imm-20 32348	B6654 2	+	+	0	0	+	0	0	0	+	0	0	+	0	0	+	0	+	0	+	0	+	0	+	+	0	0	+	+	Vel var	WH						
4 OOD	Imm-16 24019	F567 6	0	0	+	+	+	+	0	0	+	+	0	+	+	0	+	0	+	0	+	0	+	0	+	+	+	+	0	+	Sd(a-)	WH						

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Almost There? Could It Be? Anti-Co^{a}?

- Before thawing rare reagent cells, we decide to phenotype our patient.

Co^{a}		
UL	Pos Cont	Neg Cont
2+	2+	0✓

WHAT?

American Red Cross

Additional Sources

Co[a]			
	UL	Pos Cont	Neg Cont
Source #1	2+	2+	0✓
Source #2	1+	1+	0✓
Source #3	2+	1+	0✓

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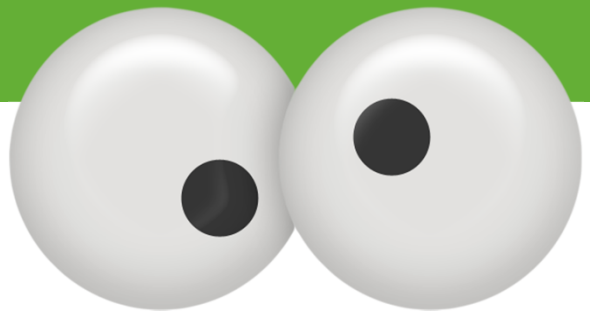

Time to switch drivers - a new set of eyes…

Patient's Name UL
Patient's Number 4050-63108
Date: Collected 08162011 Date: Tested 08162011

SELECTED PANEL
American Red Cross, Missouri-Illinois Region
4050 Lindell, St. Louis, MO 63108
Technologist: AJ

	Supplier Lot #	Donor/ Vial#	RhHr								MN				P	Lew		Lut		Kell						Duf		Kid		X	Additional Antigens	Patient's Plasma Test Results						
			D	C	E	c	e	f	V	Cw	M	N	S	s	P1	Lea	Leb	Lua	Lub	K	k	Kpa	Kpb	Jsa	Jsb	Fya	Fyb	Jka	Jkb	Xga								
1 OOD	Imm-16 34360	G242 15	0	0	0	+	+	+	0	0	+	+	+	0	+	0	+	0	+	+	0	0	+	0	+	+	+	0	+	+	Cs(a+w)	LISS IgG						
2 OOD	Biotest 8117011	600026 4	+	0	0	+	+			0	+	+	+	0	0	0	0	0	+	0	+	0	+	0	+	+	0	0	+	0	Cs(a-) Yk(a+w) Co(b+) Co(a-)	WH O+						
3 OOD	Imm-20 32348	B6654 2	+	+	0	0	+	0	0	0	+	0	0	+	0	0	+	0	+	0	+	0	+	0	+	+	0	0	+	+	Vel var	WH						
4 OOD	Imm-16 24019	F567 6	0	0	+	+	+	+	0	0	+	+	0	+	+	0	+	0	+	0	+	0	+	0	+	+	+	+	0	+	Sd(a-)	WH						

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A good source of antisera to type patient for Cs^a is not available, so we thaw rare frozen cells and test

Cell ID	D	C	E	c	e	K	k	M	N	S	s	Fy^a	Fy^b	Jk^a	Jk^b	Le^a	Le^b	P1	Additional Antigens	LISS IgG		PEG IgG
440C	+	0	+	+	+	0		+	0	0	+	+	+	+	+	0	+	+	Cs(a-)	0✓		0✓
212C	+	+	0	0	+	0	+	+	+	0	+	0	+	0	+	0	+	+	Cs(a-)	0✓		0✓
136C	+	+	0	+	+	0		+	+	0	+	+	+	0	+	0	+	+	Cs(a-)	0✓		0✓
168C	+	+	0	+	+	+	+	+	0	0	+	+	0	+	+	0	+		Cs(a-)	0✓		0✓

Patient negative for C, K, s, Fy^b, Jk^b

American Red Cross

One Additional Test

60' 37°C IgG	1:1	1:2	1:4	1:8	1:16	1:32	1:64	1:128	1:256	1:512	Diluent Control
Immucor 27063 #8	w+	w+	w+	w+	w+	w+	~~0~~ m+	~~0~~ m+	0✓	0✓	0✓

Anti-Cs^{a} antibodies are known to be HTLA-like. That is, they react weakly and do so to a high dilution. This particular example has a titer of 128 and shows weak reactions across four or more dilutions.

Important Facts About Cs^a

- Antibody was discovered and named in 1965 by Giles et al
  - Two most fully studied examples were from patients names Copeland and Stirling
- The antibody is usually
  - IgG and optimally reactive by IAT
  - Reactions are usually weak and some examples react at high dilutions (HTLA-like)
  - Clinically insignificant (does not cause decreased red cell survival)
- Belongs to the Cost blood group collection
  - Two antithetical antigens in the Cost collection: Cs^a and Cs^b
  - The antigens are serologically related to those of the Knops, but don't appear to be located on CR1
  - The antigens are resistant to treatment with Ficin/Papain, Trypsin, and DTT

Key Points to Identifying Anti-Cs^{a}

- Do not usually cause positive DAT or auto controls
- Weakly reactive at IAT phase with all cells tested
- React with all cell treatments (i.e. Ficin, DTT, Trypsin)
- Often react at high dilutions
- Difficult to rule out common clinically significant alloantibodies
  - Test with rare Cs(a-) cells
  - Rule out using valid autologous red cell phenotype
  - Give antigen negative for those alloantibodies unable to be excluded

Final Report on Patient UL

- **CELL TYPING**
  - **Blood Type**: O Positive
  - **Phenotyping**: D+ C- E+ c+ e+; M+ N+ S+ s-; K-; Fy(a+b-); Jk(a+b-)
  - **DAT**: Negative

- **SERUM TESTING**
  - Anti-Cs^a

- **TRANSFUSION RECOMMENDATIONS**
  - Transfuse incompatible units

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What Have We Learned?

- Reaching our final destination may not be as direct as the maps and/or navigation systems lead us to believe.
- We may have to deal with road blocks and detours along the way, but there are valuable resources out there for us to utilize.

- Our Blood Bank Road Trip Directions…

  Take Negative DAT south and then left on Clinically Insignificant High Prevalence.
  Take a right onto Crossmatch Incompatible and left on Transfusion.
  Your final destination will be on your right.

- Questions?