DISCLAIMER: The opinions expressed on this episode are those of my guest and I alone, and do not reflect those of the organizations with which either of us is affiliated. Neither Dr. Fontaine nor I have any relevant financial disclosures. D. Joe Chaffin, MD (4/18/16)

Joe Chaffin: Welcome to the Blood Bank Guy Essentials Podcast! I'm really excited to have my guest for today, Dr. Magali Fontaine from the University of Maryland! Welcome Magali!

Magali Fontaine: Thank you for having me.

Joe: Dr. Fontaine is the Director of Transfusion Services and the Cellular Therapy Processing Laboratory at the University of Maryland Medical Center in Baltimore. She's also an Associate Professor of Pathology and Medicine at the University of Maryland. Her background is spectacular! Very few people that I've talked to on this podcast or in blood banking, in general, have the background that Magali has in general surgery, as well as extensive time in research. She's done lots of work on mesenchymal stem cells and pancreatic islet cells, and honestly, there's just too much to tell! She's served in numerous leadership capacities with AABB, including a stint as the chair at the AABB Standards Committee for cellular therapy. When you look at her CV, she's published more than 50 peer reviewed articles, more than 50 abstracts, but there's one thing, Magali, I have to tell you that really jumped out at me because I am a teacher at my core, and I love the fact that you were honored for excellence in teaching in 2011 while you were at Stanford, which was your most recent stop before Maryland. So, obviously you have an interest in teaching as I do and with all that background—-my goodness—-I have to ask, how the heck did you get involved in blood banking? What interested you about blood banking?

Magali: So my original interest was in transplantation and immunology. I wanted to become a transplant surgeon which is why I spent a couple of years doing an internship in general surgery and eventually was pulled in the research lab during my internship and wanted to spend more time in the lab and I was offered to complete a PhD in Pathology, while also finishing AP/CP Pathology residency. Which basically, I decided to take on and drop surgery. And at the end of that training, I realized that transfusion medicine was really at the crossroads of transplantation immunology and blood banking and I thought that was the best subspecialty I could take on and here I am!

Joe: (laughs) Well, you've certainly made a huge difference! I understand, you still though, obviously I've seen some of the papers that you've published, you

still do work with pancreatic islet cells and reporting on transplant issues in that field as well, right?

Magali: Right. I do have a small laboratory in University of Maryland and pursuing some preservation studies on islet encapsulation for delivery in patients with Type I diabetes.

Joe: Well you're a very busy person obviously, and I can't thank you enough for spending a little bit of time with us today and helping us understand some things. Today our topic is going to be about: "Choices in Management of Inventories in the Transfusion Service." Magali, you and your fellow faculty members at Stanford…boy, a few years ago, you guys were very busy publishing multiple papers about inventory management, and you said some things that were a little different, perhaps than people were used to, and maybe surprised some people, but before we get to those details, I just want to step back for a second and keeping in mind, who we're talking to here—our target audience. I think we need to establish a little bit of the ground work. So let's start at the beginning. Why is Inventory Management in the transfusion service complicated? I think that trainees and people that are new in the blood bank think it's simple: You've got blood on the shelf, people order blood—-you give the blood. Why is this hard? Why is it complicated?

Magali: Well, so you first of all are dealing with different products with different shelf life. The shorter the shelf life the more challenging the inventory management strategy because you have a limited amount of time to turn around the products. So you have to be very efficient in understanding the exact amount that is needed on a daily basis so you don't waste and expire any products on the shelf. Which is basically correlated with mathematical model that supply chain experts use on a daily basis in industrial company starting with grocery stores trying to not outdate milk bottles on the shelf. So we are trying to do the same thing for platelets, for example, which has a shorter shelf life in the transfusion service compared to red cells.

Joe: Ok, so I guess that's why, when we look at the products that we have to deal with the most in terms of how intensely we have to manage their inventory, as you said the shorter the shelf life the bigger the issue. So platelets and red cells more of an issue than plasma, I'm assuming?

Magali: Correct, although plasma, once you have thawed it, it's also very short-lived, so you have to manage that very carefully as well, And especially when you have a busy trauma center like ours, liver transplants, kidney transplants—-we have to make sure we have blood available at all times, for any potential severe bleed and not exaggerate the inflation of blood that's available, so you don't expire it at the end of those 5 days.

Joe: I see, okay. That's something I think that perhaps people miss with plasma because we think of it as a one year shelf life, no big deal. But you're right, once you thaw it, suddenly you've got a product that's essentially has the same shelf life as platelets and that is a bigger issue.

Magali: Right, right exactly.

Joe: So, we aren't going to talk a whole lot—in fact, I think we may have just already talked about plasma, as much as we're going to talk about it today. We're going to focus mostly on the products that we deal with, perhaps, obviously in a major trauma center as you're describing, plasma is a large issue, of course it is. But for most smaller places, the platelets and red cells seem to be the biggest issue. So I'd like to talk to you starting off, just a little bit about platelet inventories and how to manage those. Obviously, not only at tertiary medical centers, where these issues can be acute, but also, kind of throughout the supply chain in transfusion services all over. What would you say are some of the biggest challenges that people have to deal with when managing platelet inventories?

Magali: So the platelet inventory is basically dependent on the supplier, which is usually a regional blood center or a collection center at the site of the transfusion center, which is not always the case. But at our center we have our blood center, being the Red Cross, which is basically a few miles away. When I was at Stanford we had the Stanford Blood Center, which was actually also a few miles away. So you have to establish a very close collaboration with your blood center leadership and have them be a part of your transfusion management— platelet transfusion management program. So they understand which day of the week, for example, your transfusion, your number of platelets are the highest, so they can accordingly collect more platelets for those particular days. And if you have specific needs, that we, I think we're going to talk about—-like CMV or HLA requirements, or ABO requirements we can also get help from our supplier in meeting those patients needs at the bedside. So I think that's probably the key challenge is to really establish a core relationship with the blood center.

Joe: I completely agree with you, in my current role as a blood center medical director, I certainly appreciate that, as I did when I was on the other side in transfusion services. That's something that people miss. Establishing that close relationship and making sure that there is a clear understanding **both** directions of what the capabilities are, what the needs are, again from both sides, it makes a huge difference in keeping things running smoothly, don't you think?

Magali: Oh yeah, absolutely! We really had that capacity both at Stanford and at Maryland. So we meet on a regular basis, and again, we try to exchange

data and understand how each other can improve efficiency, as well as increased quality of patient care.

Joe: Agreed. So obviously, we start off with just the general need to have adequate supply on hand, to meet everyday needs and to meet special needs, and we'll talk about the special needs here, in just a second. I think that, again considering our audience, it's often unappreciated among new people in blood banking—well, I don't like to say it this way because it sounds a little scary, but it's quite common in blood center world and in transfusion service world, to hear the words, "We're short on platelets." And those are words that nobody likes to say, nobody likes to hear but obviously with the short shelf life of the product, it does happen from time to time. So, just from the practical side, on the transfusion service side, before we get into the special needs, when you hear that, when you get a comment about, "Wow, we're short on platelets or the blood center is short on platelets", is there anything practically that you guys do proactively to try and manage your inventory in those scenarios?

Magali: Yes. So whether we are short or not, I would say that we actually are constantly *thinking* that we are short. We never know what tomorrow brings. So we actually, we are on a constant—-especially in a trauma center like ours…so we pretty much screen all the platelet orders to make sure that they meet transfusion guidelines that we have at our institution, which is usually 10,000 for a cancer patient who's stable, 20 if the patient is maybe febrile or has other pathologies like coagulopathy, and then 50 or 100 in surgical patients or in our surgical patients, respectively. So when those guidelines are not met, we talk it over with the clinician. So we don't transfuse a platelet that we could have transfused to a trauma patient coming in the next hour. So we have constantly that mindset that we are short! And we often say all the time to our clinicians, if they argue with us, because that's our state of mind.

Joe: Right! I think that especially in your scenario, where you never know who's going to be rolling into the ER in the next 5 minutes. That's essential that you manage it proactively like that!

Magali: Yeah, yeah. So I'm not sure that all centers do it because it takes a lot of staff energy to do that. We actually have our residents involved, and it's a very good experience for them to communicate with the clinical team and it's part of our blood management strategy as well. So we all learn from it and I think our clinicians eventually improve on patient care.

Joe: And you have the added benefit, potentially, Magali, of helping to educate your clinicians about appropriate—not necessarily guidelines, well, that maybe too strong, because you and I both know that patients are patients and these thresholds are guidelines and they're not absolute hard and fast rules

in most cases. But it helps to educate the clinicians about what you're thinking about appropriate platelet transfusion, right?

Magali: Right. We try to remember and actually keep in mind that a transfusion that's not needed is one less transfusion reaction, basically, that can possibly happen. And transfusion reactions, associated cost is huge. At most institutions, it increases length of stay, it can potentially actually can have implications admitting a patient who's an outpatient setting to begin with for the transfusion, etc. etc.

Joe: Yep. Well, that in it of itself is a topic for another day. We could explore that another time, but I would love to talk a little about several issues with special platelet products. In particular, let's see if we can break this down 3 ways: let's talk about issues with cytomegalovirus (CMV), issues with patients that are platelet refractory, and start off with some discussion about ABO issues. Let's start with CMV, and if we can just start from the beginning. With CMV, why are clinicians concerned about cytomegalovirus potentially being transmitted through platelet transfusion? What's the big deal with CMV?

Magali: Yes, CMV really is almost present in 60% of our blood donors, and so having a CMV negative inventory at all times means "CMV-seronegative" because we don't test for DNA in the donors. We had, at Stanford, been acknowledging the potential risk of CMV in immunosuppressed patients who are, I think, the most likely at risk of developing CMV disease, which is extremely rare in immunocompetent patients and has pretty much no significance. But in immunocompromised patients like a premature baby or a cancer patient, highly immunosuppressed or transplant patients, we have a risk anywhere between 10 and 15% of CMV disease developing. So, clinicians are not all in agreement with the rate of transmission of CMV by blood products. Some institutions actually totally ignore CMV transmission as long as the product is leukoreduced, and I think that's become now pretty much the standard across the nation. But there remain some institutions that really think that CMV transmission in a product that is CMV negative is decreased. So we had, at the time of the first transfusion of an immunosuppressed patient, we had mandated that patients be tested for CMV serology, which was very hard to coordinate at the time at Stanford where we wanted to respect those special needs. So we implemented reflex testing on the same platform as the platform we did type and screen to get a serology immediately at the time of the type and screen. We could potentially triage immediately those patients that really needed CMV negative products if they were CMV negative and immunosuppressed. That was one way for us to improve on the efficiency and not unnecessarily transfuse a CMV negative product to a patient who would potentially develop CMV disease but was not tested for CMV.

Joe: The idea basically being that if one of those immunocompromised patients, when you tested them, you found that they were already CMV positive, then the issue became less concerning with giving them CMV negative products, right?

Magali: Exactly.

Joe: Again, just for clarity: You mentioned the discussion about leukocyte-reduced and can you just give me the quick thumbnail, we're not talking about that greatly today, but why has it become, as you said, generally the standard of care (though people disagree), that leukocyte-reduced products are equivalent to CMV-seronegative?

Magali: Well, because CMV virus is really transmitted through white blood cells, so there is usually very little CMV circulating freely in the blood. There may be some DNA circulating freely, but we don't capture those with CMV testing, because we test for antibodies. So technically, a CMV antibody positive now could be considered, in the light of some virologists and immunologists, *protective*, and it's now actually become almost obsolete at some centers to ask for CMV antibody negative, because if you are in that early window period of infection, the donor would not have CMV antibodies yet, and could potentially be more infectious. Leukocyte-reduced products have actually been shown in some bone marrow transplant patients, in studies from Europe, that leukoreduced was actually equivalent to CMV negative products in the context of developing CMV disease post-bone marrow transplant. So, really, I think there's going to be more to this story, but it's become less of a concern. We've now been, at Maryland, able to convince most of our clinicians that a leukoreduced product is just as good.

Joe: Excellent! OK, let's move on. We've covered CMV issues, let's talk about platelet refractoriness patients, patients who aren't getting an adequate response to platelet transfusion. I should tell you, I recently did a full podcast on this with Dr. Pat Kopko from UCSD, so we don't have to cover everything about platelet refractoriness. But, there was something that you were involved in that is unique, I think, and shed some very new light on this whole process. When you have a patient who is not getting a response to platelet transfusions, one of the ways to work them up has historically been to screen them for HLA antibodies, and there are several strategies that can come from that, in patients who have HLA antibodies. So let me stop there and throw it to you: What was the strategy that you were using when you were at Stanford and potentially still now, in terms of how best to treat those patients with identified HLA antibodies, and what did you discover that somewhat changes the traditional thought a little bit?

Magali: The cancer center at Stanford and actually at U Maryland are dealing with patients chronically transfused, becoming refractory like you said, most of the time due to HLA antibodies. So, the key question in managing the inventory for those patients is "which platelet is really a problem for those patients?" If you have a test result on HLA antibody screen coming from your HLA lab showing a list of 100 antibodies, with having to avoid all of these antigens corresponding to antibodies, you never have platelets available for those patients. Then you have to potentially get an HLA match platelets which is very difficult, so we discussed this with the director of the HLA lab Dolly Tyan at Stanford, and she had recently implemented a refined HLA antibody screen that instead of looking at only the platelets that were binding IgG antibodies, we wanted to actually identify those platelet IgG antibodies that also bound complement. And that actually reduced the list of antibodies on the HLA antibody screen and allowed us to more easily find compatible platelets. So our "antigen avoids list" was decreased by 50%. So what we found some patients who actually had a list of 50 antigens to avoid, because they had antibodies corresponding to these antigens, that all were IgG-bound but not knowing if they were complement also bound, when we actually only looked at those 50 that also bound complement we went sometimes down to zero, none of them would bound complement, or only maybe five. Interestingly enough, you had still a pretty good platelet response if you were transfusing platelets that were not compatible, that still had antigens corresponding to those IgG-bound antibodies. So we figure that **maybe if the antibody is specific to complement binding then avoiding those would only be good enough for them to have a platelet response** and so we found out that was just good enough so we were able to increase the availability of products based on reducing the antigens to avoid for those patients because we only avoided those antibodies that bound complement.

Joe: So in terms of—well it's 2 things— first, in terms of the specific part of you were testing for, all complement components or one specific complement component? Let me start there, was it one specific complement component you were looking at, Magali?

Magali: So the one specific complement component is the C1q, so Dolly Tyan had an assay which is the Luminex platform, where the fluorescent beads expresses a specific HLA antigen. We incubate the patient's serum with that bead, the antibody binds to the bead and expresses a fluorescence only if we have a secondary antibody that's complement specific. And so, the fluorescence intensity was obviously less if you were looking for antibody complement specific vs. fluorescence intensity for any kind of IgG bound antibody. So the fluorescence intensity decreased and we could then decrease our list of antigens to avoid.

Joe: That is exciting because I think we in "blood center land" certainly deal with this a lot where we have patients from our hospitals that have a list of antibodies, as you said, could be 100 antibodies and when we look at the number of people that could be potentially compatible, generally doing a calculated PRA, which again, is a topic for another day, we basically have zero shot of finding anything compatible for them. But this allows you to potentially to narrow the list dramatically of antibodies you really have to worry about, right?

Magali: Yes and I'm glad you mentioned PRA or CPRA which is more relevant, a calculated PRA, which is basically understanding what the percentage in a normal donor population of HLA antigen is reactive to your patient serum. So if that reactivity is close to 100%, you have very little chance of finding a compatible donor. And using the classical method of HLA antibody screen that was not specifically looking at C1q or complement C1q binding, we had a lot of refractory patients with a PRA of 100% and when we repeated their blood sample screen using the C1q secondary antibody, then sometimes these percentages drop significantly, sometimes even down to 0 or 5%. We were then able to transfuse platelets and those platelets just did fine and patients walked out the door with platelet counts that were acceptable.

Joe: So correct me if I'm wrong, Magali, this testing is not—-true or false—-it is not yet widely available? Is that a true statement or am I wrong about that?

Magali: So the test I think is available. It's basically—it's a specific bead assay and HLA labs can implement it. We have included it at Maryland, we use it as well.

Joe: Awesome, awesome. That is a great tip! That is something to have the potential to open things up. So I'd love to spend more time on that but we're starting to get a little short on time. I want to move on, and talk just briefly about ABO issues for platelet inventory and specifically with ABO, let's talk first about the so-called major incompatible products, meaning that the platelets carry an ABO antigen against which the recipient has an antibody. For example, if you gave group A platelet to group B or a group O recipient, let's talk about that first before we go into the reverse scenario. I know that some people don't care and don't worry about this, but what is your experience and the data show about that? Does it make a difference when you give platelets like that?

Magali: Yes. So I think it all depends again on the size of your institution and what we observed at Stanford and here at Maryland, as well, is that when you don't care about ABO your patient will still present with a good corrected increment, providing they're not highly HLA antibody immediate refractory. So the response with ABO incompatible platelets, like the examples you just mentioned, an A platelet into an O patient for example, if a patient has not HLA antibody that

response will be fine. It will potentially be a little less than an A platelet into an A patient, but it will just be fine. The patient will respond. Now if you have a patient who is refractory, has a significant amount of HLA antibodies and you don't have an HLA-compatible platelet and you don't have an ABO-compatible platelet, the platelet response is probably going to be minimal, probably close to zero. If you do have an ABO-compatible platelet still not HLA compatible the response maybe slightly better. So that's what we observed. But if you don't have again, if you don't have an ABO-compatible platelet and your patient doesn't have any HLA antibody, and you have a platelet that's expiring on the shelf, we don't take a chance in wasting that platelet, we just transfuse it if possible.

Joe: Sure. And that's obviously a function of, as you were saying, we've got a product with a finite shelf life and evidence that if you give patients out of group platelets in situations like that where the antigens are incompatible, you'll still get a response. It may not be as dramatic, but generally speaking, good inventory management says, "You don't want to throw away that unit, if you have a chance to use it". Right?

Magali: Exactly. So basically the ABO role is not significant enough to make it a priority for us to take a chance in wasting platelets on the shelf because of that. We struggle because our staff sometimes wants to respect that and at a very high cost, so if it's not needed we don't.

Joe: So that brings us then to a topic that's been discussed a lot over the last, well at least 10 years or so, with a flurry of publications, including some that you have been a part of. What about the reverse situation? Before we were talking about the so-called "major incompatible", the platelet antigens your transfusing are incompatible. What about the opposite situation where your transfusing ABO incompatible *plasma* to a recipient? So it's reversed. For example, a group O platelet with all of it's anti-A and anti-B in the plasma, going into a recipient who's group A or group B. Is that a bigger deal and should we be more worried about that?

Magali: Yes. I think the ABO-incompatible plasma is an issue in smaller blood volume, especially pediatric patients. We have to acknowledge that it's really important to have a policy limiting the amount of ABO incompatible plasma and volume-reduce for sure, platelets for those patients if we cannot find an ABO match, an ABO plasma compatible. But in the case, for example, a platelet that has to be HLA-compatible and for which we don't have the ABO plasma compatible component, it can be a risk if we don't know what the anti-A and anti-B titers are in these platelet products. We have had a case of severe hemolytic reaction in a bone marrow transplant patient that received a platelet that had a very high titer anti-A. As a result of that, we started actually first,

volume-reduce our platelets if the plasma was incompatible, ABO incompatible. And then we actually implemented a titering method, to prevent the risk of hemolysis.

Joe: Before we talk about titers, Magali, just for clarity, can you thumbnail for us what it means to volume-reduce a platelet?

Magali: In order to volume-reduce a platelet, you just spin it down and you usually have to keep a certain amount of plasma in that platelet to prevent large volume of plasma to be infused. Usually 50 cc is the minimum amount of plasma you have to keep in the platelet product. You just basically do a simple spin down, a very light spin, and then you have to still keep that platelet rested for at least 45 minutes to an hour before transfusion. So it does increase the turnaround time of issue of that product and in an emergency situation we actually don't do that. We don't take a chance. But in elective cases, cancer patients, who are coming in for prophylactic platelet transfusion in infusion center, we do those procedures.

Joe: Does that process mess with the platelets at all? Do you get any less of a response as a result of doing that?

Magali: Yes. There's 2 issues: 1) There's the issue of labor in the transfusion service, I think that is one. 2) And the other issue is the corrected count increment that has actually been looked at, published by the Hopkins group, shown to be less than if you were not manipulating that platelet. If you wash them it's even worse, the corrective count increment. So we decided in our institution, again, with a close collaborative effort with the blood supplier, asking them, can you maybe give us a titer on those platelets. And they listened to us. They said, "well I think we can do it." And they implemented an automated platelet titering method on the instrument used for the ABO typing on the donors. An additional run was performed after the ABO typing on each platelet donation, and that second run would give us a threshold indicator of a high or low titer on that platelet product. It would not give us a titer, but we had validated a method to flag for us those platelets that potentially had a high ABO titer. So, once these platelets were on the shelf, and if they were transfused to a patient who would potentially receive incompatible plasma, we would not do that unless it was an emergency or we had time to volume-reduce it.

Joe: Got it. So, the titer theoretically being predictive…the higher the titer, in other words, the stronger the ABO antibodies, the more the potential risk for that reverse type of hemolysis where the plasma goes in and destroys the recipient's red cells?

Magali: Right, exactly. So the risk really is probably minimal, but if it happens, it can be a potential serious hemolytic reaction that you want to avoid at all costs.

Joe: One last question about that, Magali: Traditionally, we have talked about the risk of high titer antibodies being mostly with blood group O people, and not as much with group A or B donors. Can you talk a little bit about what you guys found at Stanford? Is it limited to just group O people with high titer, or is it across the blood groups?

Magali: It's mostly O donors, and we have actually just recently published a study where we looked at two years worth of platelet donation, indicating that most younger and female donors are presenting with those higher titers, and potentially with other manipulation you can do at the donor center using plasma additive solution instead of plasma, that would reduce the volume of plasma, and dilute out the antibodies. You can potentially try to target those specific donors to be processed with that particular additive solution instead of plasma. That's what we suggested in my paper. It's no surprise that women would be more at risk of developing anti-A and anti-B because of pregnancy history. Similarly, we see female donors having a higher risk of HLA antibodies.

Joe: Makes sense. We've talked for a long time about platelets, and really I think that's with good reason! I just want to close just spending a couple of minutes on red cell inventory, Magali. Since we don't have a ton of time to do this, you and the group at Stanford have published some really cool and interesting work on managing red cell inventory in terms of managing it based on the age of the red cell unit. So, why don't I just be quiet for a second, and if you can just quickly summarize for us, first, what the issue is, why do we care about the age of red cells, and second, what did you guys decide to do and how that might apply for other centers?

Magali: We had embarked on a collaborative effort with supply chain engineers at Stanford who helped us with our platelet inventory strategies. Concomitantly, the data from the Cleveland Clinic came out, showing that there could potentially be harm to transfuse older red cells in some categories of patients such as cardiovascular post-op surgical patients. We elaborated on what if we were to have a similar limited shelf life for red cells as we currently have for platelets, and how would we manage such an inventory? So, we basically looked at a series of red cell donations and allocated this inventory differently for different patients based on the age of the red cells, whether it was less than seven days or less than fourteen days or greater than 21 days. Obviously, the results were expected: The availability of product was going to be limited for each category, and the waste, the outdate rate was also going to be increasing as you decreased the age of the red cells. So, we published that

mathematical model, which remains to be considered if that theory of having to transfuse fresher red cells in some categories of patients becomes a reality. Currently, we don't know yet if there is really any harm to transfuse old blood in some patients. It seems like, based on the "ABLE" and "RECESS" studies, we cannot conclude at this time that cardiovascular surgical patients absolutely require this restriction. But there is now interest in trauma patients, not so much in the same perspective that was looked at in the studies such as ABLE and RECESS, but more interestingly, looking at the amount of red cells, or the number of units that would be older in trauma patients during the resuscitation. Would more than ten "old" red cells be a problem in a patient, which is often the case in trauma patients? So, there is some interest in looking at that, as well. I think we haven't finished hearing about the age of red cells as a possible challenge in the future.

Joe: Sure. I think that's a great point. When the 2 studies you mentioned, the RECESS study and ABLE study came out, in early 2015 as I recall, both in the New England Journal, I think a lot of people in our blood banking world said,"Ok, that's it. Great, it's done. It's put to bed, we don't have to worry about age of blood anymore!" I'm certain, and I think you would agree based on what you just said, we're not done with this issue. There's more to look at and there's definitely people that still feel like this is a bigger issue than RECESS and ABLE were "able," no pun intended, to show.

Magali: And there's even more refinement that clinicians are willing to look at that we haven't yet mentioned, one of which is age of donor. That actually is an interest in massive transfusion resuscitation.

Joe: It's getting complicated, Magali!

Magali: Yeah! (laughs) It is definitely getting worse, definitely need to work closely with our mathematician, understanding how to manage these inventories.

Joe: You bet. Well, we are out of time and I really honestly just can't thank you enough for spending time with us today, Magali, and helping us to understand some of these inventory issues. I want to let everyone know we'll have some references on the show page on the Blood Bank Guy website with some of the references to the articles that Magali mentioned and has published with her team at Stanford during our talk together. So Magali, again, just thank you so much for taking your time to be on The Blood Bank Guy Essentials podcast.

Magali: Thank you and best wishes!