



Association Bulletin #24-02

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To: AABB Members

From: Aaron A. R. Tobian, MD, PhD – President
Debra BenAvram, FASAE, CAE – Chief Executive Officer

Re: Use of Rh Immune Globulin and Considerations in the Setting of Supply Shortages and Limited Availability

Association Bulletins provide a mechanism for publication of documents approved by the Board of Directors for distribution to individual and institutional members. Association Bulletins can communicate:

- New or revised standards that were adopted after publication of the most recent edition of *Standards* and have become requirements for accreditation.
- Statements of AABB policy.
- Recommendations to address emerging trends and new information supporting patient and donor safety.
- Guidance, best practice, reports that have been developed by AABB Committees, and other important information.

This bulletin does not contain specific recommendations, nor does it create a standard or accreditation requirement. This bulletin:

- Was developed by an ad hoc working group of AABB member physicians with relevant expertise in transfusion medicine, including members of the Clinical Transfusion Medicine Committee, and the Transfusion Medicine Subsection Coordinating Committee.
- Provides important information and resources to AABB members to inform clinical practice decisions and inventory management strategies during unexpected and/or prolonged shortages of Rho(D) Immune Globulin.

Background:

In December 2023, the Food and Drug Administration (FDA) posted information regarding a shortage of Rho(D) Immune Globulin (RhIG) on the Center for Biologics Evaluation and Research (CBER) webpage for Regulated Products: Current Shortages.¹ The current shortage, resulting from factors affecting the anti-D plasma market and manufacturing deviations, initially involved one manufacturer in the United States (US), but has since expanded to include other manufacturers of RhIG.²



RhIG was created to reduce the risk of RhD alloimmunization and the devastating fetal and neonatal outcomes of hemolytic disease of the fetus and newborn (HDFN) caused by anti-D.³ In the mid-20th century, HDFN secondary to anti-D was a major cause of perinatal death. Proper use of RhIG immunoprophylaxis reduces the risk of D immunization from approximately 16% to <0.1% and significantly reduces perinatal morbidity and mortality. Therefore, even during shortages, it is important that pregnant D-negative patients continue to receive RhIG per established policies.

AABB members have expressed a need for guidance on considerations to support effective management of shortages of RhIG in the US. We recognize that FDA-approved suppliers of RhIG also provide products to customers outside of the US. Shortages may, therefore, also have effects beyond US borders.⁴ This bulletin summarizes typical use of RhIG and provides approaches facilities may consider in the event of supply shortages.⁵

Key Considerations:

- 1) Transfusion medicine professionals in collaboration with other local stakeholders should consider developing institutional policies for RhIG use. For example, RhIG should be prioritized for higher risk situations, such as pregnant D-negative patients, particularly those who are postpartum or at later gestational ages during times of shortages.⁶
- 2) Institutional stakeholders should regularly monitor RhIG inventory and establish policies and procedures for RhIG allocation during various levels of inventory shortages to ensure and prioritize RhIG availability to pregnant D-negative patients whenever possible.
- 3) Transfusion services should have policies for appropriate use of RhIG prophylaxis for D-negative patients of child-bearing potential who have been exposed to D-positive Red Blood Cells (RBCs) per AABB's *Blood Banks and Transfusion Services Standards*.⁷

Background on Use of RhIG:

With the discovery of the Rh system and its association with HDFN in 1940 and 1941, respectively,⁸ scientists looked for ways to prevent alloimmunization in pregnant D-negative individuals. An effort by multiple researchers in the United Kingdom (UK) and US⁹⁻¹¹ contributed to the discovery¹² that iatrogenic administration of anti-D could prevent the maternal immune system from the formation of alloanti-D. This finding led to the production of RhIG, first licensed in 1968, to prevent RhD immunization in the setting of pregnancy in D-negative patients. Initially, a single dose of 300 µg RhIG was administered postnatally in D-negative patients following the birth of D-positive or D-unknown newborns in the US. This measure prevented anti-D formation in approximately 90% of cases. Given that a percentage of pregnant patients (12-16%) still formed a D alloantibody even with the use of post-delivery RhIG, an additional 300-µg dose for all D-negative patients at 28-32 weeks of gestation was introduced based on results from the McMaster Conference on the Prevention of Rh Immunization in 1977.¹³ This additional measure reduced the frequency of D sensitization to 0.1%.¹⁴ Although



anti-D HDFN has not been eradicated, severe morbidity and mortality resulting from anti-D HDFN has been reduced by 50% worldwide since RhIG entered the market. As a result, the creation of RhIG as a preventive medicine has been described as a landmark development in the history of obstetric medicine.⁵

RhIG is a human-derived product collected by apheresis from volunteer plasma donors with high titers of anti-D. The collected plasma is pooled and fractionated by commercial manufacturers [at the time of writing: Grifols (HyperRHO[®] S/D), CSL Behring (Rhopylac[®]), Kamada (WinRho[®] SDF), and (RhoGAM[®]) in the US] and prepared in varying doses.¹⁵⁻¹⁸ Originally, RhIG was primarily collected from D-negative patients who formed anti-D following D-positive pregnancies, however as RhIG use increased worldwide, the number of these individuals decreased significantly. Manufacturers thus resorted to deliberately alloimmunizing D-negative male donors with D-positive RBCs to ensure an adequate supply for customers.⁸ Throughout the course of their donation career, it is often necessary to boost these donors with D-positive RBCs to elevate antibody titers and thereby support manufacturing of an effective product.¹⁹ To date, efforts by researchers to make effective recombinant anti-D have not been successful.

The most commonly available dose of RhIG in the US is 300 µg (1500 IU) of anti-D packaged as a single-use syringe administered via an intramuscular injection; this dose is available from all four US manufacturers. Each 300-µg dose effectively prevents RhD alloimmunization after exposure to up to 30 mL of D-positive whole blood or 15 mL of D-positive RBCs.²⁰ In the US, the 300-µg dose is commonly administered to pregnant patients at risk for D-alloimmunization at 28 weeks of gestation and postpartum when a D-positive infant is delivered. Intravenous formulations with different doses are also available. (**Table 1**).

Table 1. Rh Immune Globulin Products Available in the United States The CBER-Regulated Products: Current Shortages webpage provides current information on availability. ¹			
Dose	Product	Manufacturer	Route
300 µg	RhoGAM [®] Ultra Filtered PLUS	Kedrion Biopharma ¹⁸	IM
	WinRHO [®] SDF	Kamada ¹⁷	IV
	Rhopylac [®]	CSL Behring ¹⁶	IM or IV



	HyperRHO® S/D Full Dose	Grifols ¹⁵	IM
50 µg	MICRhoGAM®	Kedrion Biopharma ¹⁸	IM
	HyperRHO® S/D Mini-Dose	Grifols ¹⁵	IM
1000 µg, 3000 µg	WinRHO® SDF	Kamada ¹⁷	IV
IM – intramuscular; IV-intravenous			

As early as the 1990’s, concerns were raised about potential shortages⁶ of RhIG as the number of alloimmunized donors declined worldwide.²¹ In Australia, an extended shortage of the Australian manufactured product in the mid-1990’s necessitated the importing of RhoGAM®. At that time, some experts proposed limiting use of RhIG in the first trimester, while others expressed concerns about withholding RhIG in any instance of possible fetomaternal hemorrhage (FMH).²² Not long after the shortage in Australia, UK experts authored a publication on supply shortages. The authors referenced the previous critical shortage in Australia and advocated for renewed efforts to increase the national supply of plasma collected from donors with high titer anti-D.²³ The current RhIG shortage, beginning in December 2023, is the worst seen in the US. The following considerations may support optimal use of this resource during times of shortages.

Considerations for Management of RhIG Use During Times of Shortage:

Healthcare systems that routinely prescribe and administer RhIG should develop and maintain policies for its use.

Transfusion medicine professionals along with institutional stakeholders (eg, Obstetrics, Pharmacy, Hematology-Oncology, Emergency Medicine/Trauma, etc) involved with RhIG administration decisions should develop, approve, and operationalize site-specific policies for appropriate use. Recommendations from other health and state organizations^{24,25} may be considered to support policy development.

The policy may also include an institutional review process for when high doses of RhIG are requested. This policy should be consistently followed, even beyond times of RhIG shortage. For instance, when a transfusion service or pharmacy receives requests for unusually large doses of



prophylactic RhIG for FMH, consideration should be given to repeating the quantitative test used for determining the dose (whether flow cytometry or Kleihauer-Betke).²⁶ Causes of unusually high percentages of fetal hemoglobin such as hereditary persistence of fetal hemoglobin or other hemoglobinopathies should also be entertained depending on the specific patient circumstances.

Prioritize RhIG for pregnant D-negative patients, with patients at later gestational ages receiving highest priority (eg, postpartum, then at 28 weeks of gestation, then first trimester).

The American College of Obstetricians and Gynecologists (ACOG) currently recommends administration of RhIG at 28 weeks of gestational age to unsensitized D-negative patients and again within 72 hours of birth if the fetus/newborn is determined to be D-positive; these practice recommendations are based on high-quality scientific evidence⁶ and are considered the standard of care in the US.

First prenatal visit: Before administration of RhIG to any pregnant patient, the patient should be typed for the D antigen. If a pregnant patient has an indeterminate D type on serologic testing, molecular testing should be considered to reduce unnecessary allocation of RhIG to patients with molecular variants (eg, weak D type 1, 2, 3, 4.1) that are not at risk for D alloimmunization.^{27,28} This practice should be followed during times of sufficient RhIG supply and during local or national shortages. Following molecular testing, patients should have access to their D-genotyping results to reduce unnecessary repeat testing in future pregnancies.

28-week dose: For patients who are D-negative or have a D-variant²⁹ at risk for anti-D alloimmunization, current practice recommends a 28-week RhIG dose given that the fetal D-antigen status is unknown. However, during times of RhIG shortages, it may be advisable to determine fetal D-antigen status and administer RhIG only to women carrying a D-positive fetus. On April 24, 2024, ACOG issued a practice advisory that aligns with this strategy.²⁵ The advisory proposed that if paternity is certain and the father or sperm donor is known to be D-negative, antenatal prophylaxis may be avoided. The ACOG advisory also commented on the antenatal use of cell-free fetal DNA (cffDNA) analysis³⁰ to determine fetal D-antigen status during RhIG shortages to ensure that the limited supply of RhIG is administered only to patients at risk for anti-D alloimmunization.²⁵

The use of cffDNA for determination of fetal D status has been increasing worldwide,³¹⁻³⁴ although availability and implementation in the US have been relatively limited. In Europe for the past 15 years, national programs have increased. Routine use in Europe first began in 2010 in Denmark with national routine cffDNA screening of D-negative patients at 25 weeks of gestation.³⁵ Other countries shortly followed suit although with some variation in the recommended timing of testing (ie, Netherlands at 27 weeks of gestation since 2011,³⁶ Norway at 24 weeks of gestational age since 2016,³⁷ and Switzerland as early as 18 weeks of gestation since



2020).³⁴ Additionally, noninvasive fetal D status assessment is currently being performed regionally in Sweden,³⁸ and nationally in Finland,³⁹ France,⁴ and the UK).⁴⁰

Currently, cffDNA testing is available in the US with both sensitivity and specificity approaching 100%^{4, 41-43} The cffDNA test can be performed as early as 9 to 10 weeks of gestation.⁴³⁻⁴⁴ These tests offer the opportunity to non-invasively assess the fetal D status before birth, aid in avoiding unnecessary medical intervention (ie, RhIG prophylaxis when the fetus is D-negative), and optimize the use of a scarce resource.

After delivery: The use of RhIG after delivery should be guided by the blood type of the newborn. Consistent with the ACOG practice advisory, post-natal RhIG administration should be administered only after the infant has been typed for the D antigen. If the infant is D-negative (with serologic weak D testing also negative), a postpartum RhIG dose is unnecessary.²⁵ Additionally, should delivery occur within 3 weeks of the antenatal RhIG dose, postpartum dosing may be omitted in the absence of excessive FMH.⁶

In the event of a severe shortage, postpartum RhIG prophylaxis should be prioritized over the 28-week antepartum dose due to greater risk of D antigen exposure at the time of delivery. However, as previously stated, the residual risk of alloimmunization with only postpartum prophylaxis remains at 12-16%. Therefore, prioritizing obstetric patients should be considered to ensure all eligible pregnant D-negative patients receive RhIG during shortages (discussed below).

Within the obstetrics community, there is a lack of consensus regarding use of RhIG to prevent sensitization to the D antigen during the first trimester when a possibly sensitizing event occurs.^{3,45} Potentially sensitizing events include, but are not limited to: chorionic villus sampling, amniocentesis, cordocentesis, threatened/ diagnosed miscarriage, ectopic pregnancy, molar pregnancy evacuation, therapeutic abortion, antepartum hemorrhage, abdominal trauma, intrauterine fetal demise, external cephalic version (ECV), and delivery.⁶ **Table 2** summarizes recommendations from different US obstetric societies. Of note, ACOG also recommends RhIG following antenatal hemorrhage after 20 weeks of gestation and following fetal death in the second or third trimester.⁶ Given these inconsistencies, the decision on whether to provide RhIG following potentially sensitizing events in the first trimester should involve nuanced discussions between the obstetric providers and their patients.



Table 2. RhIG Dosing Recommendations after Sensitizing Events [from American College of Obstetricians and Gynecologists (ACOG), Society for Maternal Fetal Medicine (SMFM), and Society of Family Planning (SFP)]

US Society	First-Trimester Sensitizing Events	Recommended Dose	Priority
ACOG ⁶	<ul style="list-style-type: none"> • External cephalic version (ECV) • Evacuation of a molar pregnancy • Spontaneous first-trimester miscarriage (especially those in whom instrumentation has been used) • Either medical or surgical termination • Ectopic pregnancy • Abdominal trauma at any point in gestation 	<p><12 weeks: 50 to 120 µg</p> <p>>12 weeks: 300 µg</p>	<p>Prioritize available RhIG for pregnancies of later gestation when supplies are limited.^{3,25}</p>
SMFM ³	<p>Society statement addresses only spontaneous or induced abortion <12 weeks</p>	<p><12 weeks: 50 µg when available; otherwise, administer 300-µg RhIG dose</p> <p>Society statement addresses only spontaneous or induced abortion <12 weeks</p>	<p>Administer RhIG during the first trimester when supplies are available and when it does not hinder access to abortion care.³</p> <p>Prioritize available RhIG for pregnancies of later gestation when supplies are limited.^{3,25}</p>
SFP ⁴⁶	<ul style="list-style-type: none"> • Ectopic pregnancy • Sharp curettage • Other invasive procedures 	<p><12 weeks: None for spontaneous or induced abortion; 50 µg for other first-trimester sensitizing events</p>	<p>SFP committee consensus statement recommends providing RhIG only after 12 weeks of gestation for spontaneous abortion or medication or aspiration</p>



		13 to ≤18 weeks: 100 µg >18 weeks: 300 µg	abortion. If recommended dose is unavailable, a larger dose can be given. At >12 weeks, RhIG should be given only to patients who are outside the window of efficacy of any previous administrations.
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As in the US, Canada, the UK, Australia, and New Zealand, routine second-trimester dosing is recommended along with dosing within 72 hours of delivery if the infant(s) is determined to be D-positive.^{6,47-49} Additionally, national organizations [Society of Obstetricians and Gynaecologists of Canada, the Royal College of Obstetrics and Gynaecology (RCOG), and the Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG)] recommend prophylaxis for sensitizing events where FMH may have occurred.⁴⁷⁻⁴⁹ The general framework of these national recommendations are the same, but some details vary as shown in **Table 3**.

Table 3. RhIG Dosing Recommendations in United States, Canada, United Kingdom, Australia, and New Zealand			
Country	First Trimester or Other Sensitizing Events	Routine Antepartum	After Birth (within 72 hours of birth)
United States ⁶	50 µg or 120 µg if ≤12 weeks AND 300 µg if >12 weeks	300 µg at 28 weeks	300 µg + additional dose depending on FMH Can omit if <3 weeks after 28-week dose and no excessive FMH
Canada ⁴⁷	120 µg if ≤12 weeks AND 300 µg if >12 weeks	300 µg at 28 weeks OR 100- to 120-µg at 28 AND 34 weeks	300 µg + additional dose if FMH >30 mL whole blood OR 120 µg + additional dose if FMH >12 mL whole blood



United Kingdom ⁴⁸	50 µg if ≤20 weeks AND 100 µg if >20 weeks	300 µg at 28 weeks OR 100- to 120-µg at 28 AND 34 weeks	100 µg
Australia and New Zealand ⁴⁹	50 µg if ≤12 weeks AND 300 µg if >12 weeks	125 µg at 28 AND 34 weeks	125 µg at birth with additional dose possible depending on FMH

Consider whether RhIG should be given to D-negative recipients of D-positive platelets given the low risk of alloimmunization, particularly for apheresis platelets. During times of RhIG shortage, centers may strongly consider no RhIG administration for apheresis platelet transfusion exposures.

Due to limited supply of D-negative platelets, D-negative patients may require transfusion with D-positive platelets. Given the low rate of alloimmunization following D-positive apheresis platelet transfusion, provision of RhIG in the setting of D-positive apheresis platelets to D-negative recipients is not considered to be indicated in individuals without childbearing potential; this can be critical in the setting of severe RhIG shortages. Platelets do not express the D antigen; however, there is a small amount of residual red cells in apheresis platelet units (<0.001 mL) resulting in an anti-D alloimmunization rate estimate of 0.75% (95% CI: 0.2-1.6%) based on a recent 2024 systematic review and meta-analysis of prevalence. Whole-blood-derived pooled platelet units have higher red cell content (range: 0.03-0.6 mL) and are five times more likely to cause anti-D alloimmunization at a rate of 4.1% (95% CI: 1.7-7.4%).⁵⁰ Hubert et al also found that patients identifying as female had a two times higher rate of anti-D immunization following transfusion of whole-blood-derived pooled platelet units compared to patients identifying as male (3.6% vs 1.8%, respectively) whereas immunocompetent and immunosuppressed patients had comparable rates of anti-D alloimmunization (3.3% and 2.9%, respectively).⁵¹ Many centers do not provide RhIG in the setting of D-positive apheresis platelet transfusion.⁵² When supplies permit, depending on the clinical circumstances, RhIG may be considered for D-negative patients of childbearing potential following transfusion of D-positive platelets, for instance if transfusing whole-blood-derived platelets.

Consider avoiding RhIG administration to D-negative patients without or beyond child-bearing potential following transfusion of RBCs or whole blood.

Although the risk of RhD alloimmunization after RhD-incompatible transfusion was historically cited as 80% in D-negative male volunteers,⁵³ recent studies report that the risk may be as low as



22% in non-oncology patients,⁵⁴ and even lower in immunocompromised patients.⁵⁵⁻⁵⁷ For D-negative individuals who will not bear children and will be transfused only once in their lifetime, sensitization to the D-antigen could lead to the formation of anti-D IgG antibodies, but will likely have negligible clinical impact. If an alloimmunized patient is re-exposed to D-positive RBCs through RBC or whole blood transfusion, the consequences of extravascular hemolysis and a delayed hemolytic transfusion reaction are typically manageable; for the purpose of transfusion, anti-D is managed like other alloantibodies to red cell antigens, by issuing antigen negative units and providing supportive care if antigen-positive RBCs are transfused. In emergency settings, where receipt of RhD-incompatible transfusion is most likely, the risk of a hemolytic transfusion reaction after receipt of incompatible RBCs is <1%.⁵⁸ Further, there is an important difference between immunoprophylaxis in the setting of RBC component (eg, RBCs, whole blood) transfusion and pregnancy; if an attempt is made to prevent RhD alloimmunization following the transfusion of RBCs, a much higher dose of RhIG (approximately 6000 µg or the equivalent of 20 300-µg vials per unit of RBCs) is required compared to that which is routinely needed for immunoprophylaxis during pregnancy. Administration of such high doses can increase the risk of hemolyzing transfused D-positive red cells. In addition, high-dose RhIG has been associated with significant adverse events including headache, vomiting, hematuria, skin discoloration, neutropenia, and disseminated intravascular coagulation.⁵⁹⁻⁶² Finally, using RhIG in this setting can reduce the inventory availability for recipients most likely to benefit pregnant D-negative patients.

It is always better to provide D-negative RBCs to D-negative patients of childbearing potential, rather than transfuse D-positive units followed by RhIG immunoprophylaxis. This is especially true in cases of massive hemorrhage. If sufficient D-negative RBCs are not available to support a D-negative patient, then RhIG should not be administered without careful consideration of the risks associated with high-dose RhIG and the impact on the overall RhIG supply, as discussed above. In addition, the practice of performing red cell exchange to reduce the risk of D-positive red cell hemolysis is not recommended by the American Society for Apheresis.⁶³ However, if a patient of childbearing potential does become alloimmunized during a trauma resuscitation, modeling exercises have estimated the risk of fetal/neonatal death from HDFN at 0 - 6.5%, depending on factors such as age at the time of transfusion, age during pregnancy, and others.⁶⁴

The most certain means of preventing RhD alloimmunization in D-negative patients of childbearing potential is to avoid the transfusion of D-positive RBC components when possible. If policies allow for the transfusion of D-positive RBC components to D-negative and D-unknown patients of childbearing potential, it is essential that each institution carefully weigh the risks and benefits of including such patients in institution-specific clinical policies. Similarly, the risks and benefits of including these patients in clinical trials must be carefully considered and discussed among investigators, clinicians, and potential subjects. Inclusion of high-dose RhIG in such protocols is not suggested for the reasons stated above.



RhIG should not be given to D-negative recipients of D-positive hematopoietic progenitor cell products.

Donor and recipient pairs with D antigen mismatch are common in allogeneic hematopoietic stem cell transplantation (HSCT); however, there are only rare case reports of anti-D alloimmunization and no reports claiming severe or fatal clinical consequences due to formation of anti-D after D-antigen incompatible HSCT.⁶⁵ Because the immunomodulatory effect of passively transferred anti-D from RhIG on engraftment and immune reconstitution is also unknown, provision of RhIG is not advised close to HSCT.

RhIG is not first-line treatment for immune thrombocytopenia (ITP) in D-positive patients with an intact spleen.⁶⁶ Other treatments are available including corticosteroids, rituximab, and human thrombopoietin receptor agonists.⁶⁷

Management approaches in adult and pediatric patients with ITP are based upon the severity of bleeding symptoms and include observation, corticosteroids, IV immunoglobulin (IVIG), RhIG, rituximab, splenectomy, and thrombopoietin receptor agonists. Despite the increase in the number of available therapies, there are minimal data from randomized trials to guide management. In 2019, the American Society of Hematology (ASH) published guidelines⁶⁸ for the treatment of ITP that favored strategies aimed at avoiding medication side effects.⁶⁹ Of note, intravenous RhIG for treatment of ITP has not been available in most European countries since June 2009 because of concerns about the benefit-to-harm balance in this clinical setting.^{68,70}

For adults with a new diagnosis of ITP, the ASH guidelines recommend a therapy plan based on the platelet count and presence of minor mucocutaneous bleeding; therapy plan options include either management with observation or a short course of corticosteroids with or without hospitalization.⁶⁷⁻⁶⁸ IVIG may be used for some patients who do not tolerate, or have a contraindication to, corticosteroids. IVIG may be added to corticosteroids treatment for patients who require a more rapid increase in platelet count (ie, for an invasive procedure), as IVIG raises the platelet count more rapidly than corticosteroids.

Historically, RhIG has been used as an alternative to IVIG in ITP patients with an Rh(D)-positive blood type and an intact spleen.⁶⁹ The RhIG mechanism of action is thought to raise the platelet count by saturating Fc receptors with anti-D (RhIG)-coated D-positive red cells. In the prior recommendations from 2011, ASH guidelines recommended either IVIG or anti-D (in appropriate patients) as a first-line treatment if corticosteroids are contraindicated. Although not addressed in the 2019 guideline, there is a risk of intravascular hemolysis associated with administration of RhIG (FDA black box warning) and it is not considered first- or second-line therapy by the 2019 ASH guidelines.⁶⁸

The ASH guidelines made similar recommendations for pediatric patients with newly diagnosed ITP and little or no associated bleeding (ie, observation, corticosteroids).⁶⁷ For patients with life-



threatening hemorrhage, combination therapy with platelet transfusion, corticosteroids, and IVIG are recommended.⁷¹ In this setting, RhIG can be added to the therapy plan in D-positive patients with an intact spleen and a negative direct antiglobulin test (DAT); however, the additional benefit of this agent in this setting is uncertain.

RhIG has also been used in Rh(D)-positive pregnant patients with ITP.⁷² However, concerns about maternal hemolysis and anemia, as well as limited availability in some countries, limit its use.

Conclusion:

Transfusion medicine professionals should collaborate with colleagues in the pharmacy and clinical stakeholders to formulate policies for appropriate RhIG use. These institutional policies may incorporate modifications in the event of RhIG supply shortages, and the approaches described in this Association Bulletin may be useful to consider. Implementing these efforts especially during times of shortage can help maintain the benefit of this advancement in medicine and prevent morbidity and mortality associated with HDFN due to alloanti-D.

Acknowledgment:

This Association Bulletin was developed by an ad hoc working group of AABB member physicians with relevant expertise in transfusion medicine, including members of the AABB's Clinical Transfusion Medicine Committee (chair Ryan Metcalf, MD, CQA) and the Transfusion Medicine Subsection Coordinating Committee (chair Wen Lu, MD). They include:

Wen Lu, MD

Kerry O'Brien, MD

Nabiha Huq Saifee, MD, PhD

Alyssa Ziman, MD

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