Improving HEMOVIGILANCE Tracking
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The Future of Hemovigilance

Increased reporting, tracking of transfusion-related adverse reactions could benefit the field and the public.

AABB Provides 2 Pull-Out Pocket Quick Reference Guides for Hemovigilance

When unknown antibodies are a good thing.

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Potential Benefits of Improving Hemovigilance Programs

The United States has required the reporting of blood collection and transfusion deaths since 1976, although the only state currently requiring adverse event reporting is Massachusetts. Otherwise, the Centers for Disease Control and Prevention’s National Healthcare Safety Network’s Hemovigilance Module is a voluntary system. Yet hemovigilance reporting could help us better understand adverse events associated with blood collection and transfusion — thus leading to improvements.

This month’s first feature article, starting on page 6, examines the future of hemovigilance in the U.S. and abroad, and the benefits of tracking and reporting adverse transfusion events. The second feature article, beginning on page 11, provides two pull-out pocket quick reference guides for hemovigilance, the pocket guide to “Standard for Surveillance of Complications Related to Blood Donation” and the “AABB Quick Reference Guide to the NHSN Hemovigilance Module: Adverse Reaction Definitions.” These guides are designed to fit in lab coat pockets, and AABB is providing access to these guides to be used in daily practice as well as educational resources.

2021 AABB Annual Meeting

As you likely already know, AABB has decided to host a virtual annual meeting in place of an in-person event this year. AABB is pleased to be able to provide the most exceptional Annual Meeting experience while protecting the health and safety of our attendees, exhibitors and staff.

This year’s Virtual Annual Meeting will be held Oct. 17-19, 2021. In response to feedback we received last year, this year’s meeting will be held on Sunday, Monday and Tuesday to make it as convenient as possible for attendees to participate in as many sessions as possible. There will be a few pre-meeting events on Friday, Oct. 15 and Saturday, Oct. 16. As they did last year, meeting attendees will have access to the platform before the meeting begins.

As we look to October, we are excited to build on all the successes of last year. Although we won’t be able to see each other in person this year, the virtual platform will allow us to expand access to the meeting to colleagues throughout the world, many of whom might not have been able to attend an in-person event.

Registration for the meeting will open for AABB members on June 23 and general registration opens June 30.
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Antonella Nai, PhD, is the group leader of the Regulation of Iron Metabolism Unit at the San Raffaele Scientific Institute in Milan, Italy. Nai received a National Blood Foundation early-career Scientific Research Grant in 2018 for her project, “Targeting the second transferrin receptor for amelioration of severe malarial anemia.” AABB News spoke with Nai about her research and how receiving the NBF grant helped her advance her research and her career.

AABB News: How did receiving an NBF early-career Scientific Research Grant help your research and career?

Nai: I was awarded the NBF early-career grant in a critical stage of my career: the shift from senior post-doc to independent researcher. NBF funding, together with the awarding of other national and international grants, and my scientific production, was instrumental to my promotion, after the evaluation by the Commission of Appointment and Promotion of the Institute, to group leader of the Regulation of Iron Metabolism Unit at San Raffaele Scientific Institute. In addition to my career advancement, this grant provided me with the unique opportunity of investigating the role of transferrin receptor 2 (TFR2) in malarial anemia, contributing to the main goal of my current research activity, which is the validation of TFR2 targeting as a novel general “erythropoiesis-stimulating approach.”

AABB News: Can you elaborate on your findings regarding the relationship between TFR2 and malaria risk?

Nai: TFR2 is known as one of the genes responsible for hereditary hemochromatosis, an iron-overload disorder, since it acts as a regulator of systemic iron homeostasis in the liver. Less is known about the function of the protein in erythroid cells. We previously demonstrated that erythroid TFR2 is a negative modulator of erythropoietin signaling, and its deletion stimulates erythropoiesis in wild-type mice and improves anemia in a murine model of non-transfusion dependent beta-thalassemia. Our preclinical studies in murine models supported by the NBF grant suggest that selective inactivation of Tfr2 in the erythroid compartment delays the onset of anemia following Plasmodium chabaudi chabaudi (Pcc) infection. Also, recovery from anemia was faster and erythropoiesis was more effective than in control animals. Even more interestingly, the degree of parasitemia was strongly reduced in mice that lack erythroid Tfr2, suggesting that Tfr2 inactivation might not only ameliorate anemia due to malaria infection, but also limit Plasmodium growth. Overall, our results suggest that TFR2 might be a candidate therapeutic target for malarial anemia. However, despite promising, these findings are preliminary and require further confirmation. In addition, the mechanism of parasitemia reduction by Tfr2 deletion, as well as the definition of an appropriate tool for therapeutic targeting of TFR2 remain to be identified.

AABB News: Your research findings suggest that TFR2 could be a potential therapeutic target for malaria treatment. How would this potentially work?

Nai: First of all, since the erythropoietic response to circulating erythropoietin is dramatically reduced in malaria infection, the inactivation of Tfr2 in erythroid cells, where it acts as a brake of erythropoietin signaling, is expected to rescue — at least partially — this defect, thus increasing the production of new red blood cells. Furthermore, our finding that Tfr2 deletion, besides ameliorating anemia, might reduce red blood cells’ infection by Plasmodium opens new possibilities. Indeed, Tfr2 deletion in the erythroid...
compartment leads to the production of smaller red cells each containing less heme/hemoglobin than normal cells. Being heme essential for *Plasmodium* growth within erythroid cells, this is in keeping with the reduced parasitemia observed in our mice. In addition, reduced levels of heme would limit the production of hemozoin, a derivative of heme degradation by the plasmodium, which is toxic to erythroid cells and has been proposed to contribute to erythropoiesis inhibition during malaria infection. Thus, the benefits would be multiple.

**AABB News: What more needs to be studied to better understand TFR2?**

**Nai:** The precise mechanism of the TFR2-mediated regulation of erythropoiesis remains to be elucidated, and our studies are currently directed to this ambitious aim. In addition, the elucidation of the role of erythroid TFR2 in *Plasmodium* growth/invasion is critical and deserves further investigation.

**AABB News: Did the NBF grant open any additional opportunities for your career and research?**

**Nai:** As mentioned above, the NBF grant strongly contributed to the progression of both my career and research activity. Indeed, some of the results obtained thanks to this funding were used as preliminary results for the application to other funding agencies for projects aimed at evaluating Tfr2 targeting as a potential novel general “erythropoiesis erythropoietic approach” for different forms of anemia. In addition, all the results so far obtained will be instrumental for applying to specific malaria-related grants, to the aim of expanding our project of the role of TFR2 in anemia.

**AABB News: What is next for your research?**

**Nai:** The next step will be to consolidate the results and to initiate in-depth mechanistic studies required to unravel the precise mechanism through which Tfr2 inactivation ameliorates malaria anemia and reduces parasitemia. In addition, we are trying to identify a tool for translating our strategy of Tfr2 inactivation, which at present is achieved through the technique of bone marrow transplantation, into a pharmacologic approach. This, in addition to malaria, might benefit other forms of common anemias as beta-thalassemia and anemia of chronic kidney disease.

**AABB News: From your perspective, why is it critical that the NBF supports early-career research?**

**Nai:** Early-career researchers need dedicated support for addressing their innovative ideas and producing preliminary results for applying as principal investigator to more substantial grants. This is essential for their growth and for reaching scientific independence. Funding from NBF perfectly fulfills this requirement and for this reason I am grateful to NBF and hope that it continues to support the scientific career development of excellent young scientists.

Scientific contributions like these are possible thanks to the generous donations of NBF supporters. AABB encourages members to donate today to support early-career investigators and have an impact on the health and safety of patients and donors both in their community and worldwide.