Evaluation of the Mirasol reduction technology system against Babesia microti in apheresis platelets and plasma

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Evaluation of the Mirasol platelet reduction technology system against *Babesia microti* in apheresis platelets and plasma

Laura Tonnetti, Melanie C. Proctor, Heather L. Reddy, Raymond P. Goodrich, and David A. Leiby

**BACKGROUND:** *Babesia microti* is an intraerythrocytic parasite, transmitted naturally to humans by infected ixodid ticks, that causes babesiosis. In recent years, *B. microti* has been identified as a growing public health concern that has also emerged as a critical blood safety issue in the absence of appropriate interventions to reduce transmission by blood transfusion. Thus, we evaluated the ability of the Mirasol pathogen reduction technology (PRT; CaridianBCT), which uses riboflavin (RB) and ultraviolet (UV) light, to diminish the presence of *B. microti* in apheresis plasma and platelets (PLTs).

**STUDY DESIGN AND METHODS:** Apheresis plasma and PLT units were spiked with *B. microti*-infected hamster blood and subsequently treated using the Mirasol PRT system. Control and experimental samples were collected at different stages during the treatment process and injected into hamsters to detect the presence of viable parasites. Four weeks postinoculation, hamster blood was tested for *B. microti* infection by blood smear and real-time polymerase chain reaction analysis.

**RESULTS:** None of the blood smears from animals injected with samples from PRT-treated plasma or PLT units were positive by microscopy, while all the non–PRT-treated plasma and PLT units were demonstrably parasitemic. Parasite load reduction in hamsters ranged between 4 and 5 log in all PRT-treated units compared to untreated controls.

**CONCLUSION:** The data indicate that the use of RB and UV light efficiently reduces the presence of viable *B. microti* in apheresis plasma and PLT products, thereby reducing the risk of transfusion-transmitted *Babesia* potentially associated with these products. Based on this observed “proof of principle,” future studies will determine the efficacy of the Mirasol PRT in whole blood.

**ABBREVIATIONS:** PI = postinoculation; PRT(s) = pathogen reduction technology(-ies); RB = riboflavin.

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PRT system has been shown to effectively inactivate several parasitic pathogens including *Leishmania donovani*, *T. cruzi*, and *Orientia tsutsugamushi*, as well as viruses and bacteria (e.g., human immunodeficiency virus, West Nile virus, *Staphylococcus epidermidis*, and *Escherichia coli*). Additionally, PRTs offer potential protection against the risk of emerging infectious agents.

*Babesia microti*, the primary cause of human babesiosis in the United States, is an intraerythrocytic parasite transmitted naturally to vertebrate hosts by infected ixodid ticks. *B. microti* is endemic to the Northeast and upper Midwest, but the geographic range of this parasite continues to expand. Human infections range from asymptomatic and relatively mild acute cases to those with severe complications including hemolytic anemia, thrombocytopenia, hematuria, renal failure, and death. The intraerythrocytic location of *B. microti*, coupled with its survival in stored blood products for 21 to 35 days (in whole blood and red blood cells [RBCs], respectively), makes this parasite an ideal candidate for transfusion transmission. In recent years, *B. microti* has emerged as a critical blood safety threat, with estimates suggesting that more than 70 cases of transfusion-transmitted *Babesia* have occurred in the absence of appropriate interventions, including nine deaths reported from 1997 to 2007, at least five of which were associated with RBC units. While most transfusion cases have implicated RBC units, at least three published cases have involved whole blood-derived PLTs and transmission by apheresis PLTs is possible given reports of extracellular *Babesia*. Herein, we report on an evaluation of the efficacy of the Mirasol PRT to reduce the presence of *B. microti* in apheresis plasma and PLTs. This study demonstrates a “proof of principle” for *B. microti* pathogen reduction by the Mirasol PRT system that will be extended to RBCs in the near future.

**MATERIALS AND METHODS**

**Collection of apheresis PLTs and plasma**

Apheresis PLTs and plasma were collected under an approved human use protocol by the American Red Cross Holland Laboratory Research Blood Program. PLT units and concurrent plasma were collected simultaneously from single donors using an automated blood collection system (Trima Accel, CaridianBCT) and processed immediately after collection. The following PLT unit characteristics were required for this study: target concentration of \(4 \times 10^{12}\) PLTs per \(\mu\)L, with a minimum total yield of \(3.7 \times 10^{11}\) PLTs and a volume range of 264 to 321 mL. The volume range for plasma units was 250 to 275 mL. In the conduct of research where humans are the subjects, the investigator(s) adhered to the policies regarding the protection of human subjects as prescribed by Code of Federal Regulations Title 45, Volume 1, Part 219; and Title 21, Chapter 1, Part 50 (Protection of Human Subjects).

**B. microti propagation**

*B. microti*-infected hamster RBCs were acquired from ATCC (Manassas, VA) and injected intraperitoneally (IP) into one naïve 4-week-old female Golden Syrian hamster (*Mesocricetus auratus*) obtained from Harlan Sprague Dawley, Inc. (Indianapolis, IN). Four weeks postinoculation (PI), a drop of hamster blood was collected by tail snip to verify *B. microti* infection by blood smear. The *Babesia*-infected hamster was then subjected to cardiac puncture and the blood collected was injected IP into a group of four naïve hamsters, 0.5 mL per hamster, to establish a colony of infected animals (propagation group). The parasite was serially passed every 5 to 6 weeks by injecting a new group of four naïve hamsters with the blood from one or two *Babesia*-infected hamsters of the previous group. Parasitemia was verified periodically by microscopic examination of blood obtained by saphenous vein puncture. All hamster procedures were performed using a protocol approved by the American Red Cross Institutional Animal Care and Use Committee. In conducting research using animals, the investigator(s) adhered to the Animal Welfare Act Regulations and other Federal statutes relating to animals and experiments involving animals and the principles set forth in the current version of the Guide for Care and Use of Laboratory Animals, National Research Council.

**Treatment with the Mirasol PRT system**

The Mirasol pathogen reduction process consists of three main steps: the transfer of the PLTs or plasma to a Mirasol illumination bag, the addition of 35 mL of 500 \(\mu\)mol/L RB solution, and the illumination of the bag with UV light. In the experiments reported herein, PLT and plasma bags were injected with 1 mL of *B. microti*-infected hamster blood through a spike port to a final concentration that ranged between \(10^6\) and \(10^7\) parasites/mL. The infected blood was collected from animals with parasitemias ranging from 40% to 50% as determined by blood smears. The exact parasite concentration of the plasma and PLT units was calculated by polymerase chain reaction (PCR) analysis. PLT or plasma units were attached to the Mirasol illumination bag using a sterile dock and the contents transferred through the inlet line. Single-dose bags of RB were sterile docked to the Mirasol illumination bag containing the PLTs or plasma, and the RB was transferred though the inlet line. It was noted that the addition of RB (35 mL) induced a dilution of parasite concentration that was minimal and not measurable by PCR (data not shown). The RB bag was removed and inlet line was sealed using a hand welder, while the port was sealed using a port
isolation welder. The illumination bag was weighed, and its bar code was scanned into the illuminator and then secured in the illuminator drawer. The illumination bag was exposed to UV light (285-365 nm, majority of energy between 280 and 320 nm) with an energy dose of 6.24 J/mL (±8.4%) delivered during illumination. UV illumination typically required less than 10 minutes, but its length was dependent upon product volume. Once illumination was complete and the illuminator confirmed a successful run, the illuminated product was ready for testing and/or storage.

Experimental design
The experimental design is illustrated in Fig. 1. The procedures were identical for plasma and PLT samples with the exception of the storage conditions of the collected samples and the posttreatment blood products: 5 days at 22 to 24°C for PLTs and 7 days at −20°C for plasma. A 16-mL sample was removed from each PLT or plasma unit immediately after being spiked with parasite-infected RBCs (positive control) and later after adding RB (RB control). For these 16-mL samples, 1 mL was used for DNA extraction while 6 mL was injected IP into groups of six anesthetized hamsters (1 mL each). The remaining PLT volume was stored for 5 days in a PLT incubator at 22 to 24°C, while the remaining plasma was stored for 7 days at −20°C. After illumination, an 8-mL sample was removed from each PLT or plasma unit for subsequent injection IP into six anesthetized hamsters (1 mL each) and DNA extraction, while the rest of the treated units were stored for 5 days at 22 to 24°C for PLTs and for 7 days at −20°C for plasma. Mirasol PRT–treated PLTs were stored in the Mirasol illumination/storage bag, and Mirasol PRT–treated plasma was transferred to a plasma storage bag. After the designated storage time (i.e., 5 or 7 days), samples were inoculated IP into hamsters (six hamsters, 1 mL each) and used for DNA extraction as for the Day 0 samples.

Standard curve of *B. microti* infection in hamsters
To generate a standard curve of *B. microti* infection in hamsters, we injected 1 unit of PLTs (approx. 250 mL) with *B. microti*-infected hamster blood at a final concentration of 10⁷ parasites/mL. After RB was added to the infected PLT unit, a 20 mL sample was removed (untreated control) and the rest of the unit was illuminated after the standard Mirasol PRT protocol as described above. After illumination, a sample was removed from the treated unit and samples from both the treated and the untreated units were serially diluted in phosphate-buffered saline to a value calculated to be less than one parasite per mL. One-milliliter volumes from each dilution were injected IP into groups of anesthetized hamsters (n = 5). Four weeks PI, blood was collected by cardiac puncture and the animal euthanized.
Parasite concentrations were evaluated by real-time PCR.

Animal inoculation and blood collection
One milliliter of each sample (controls and illuminated/treated) was injected IP into groups of six hamsters, 1 mL per hamster. Approximately 2 weeks PI, blood was collected from hamsters injected with the positive controls by saphenous vein puncture or tail snip to monitor the development of parasitemia by blood smear. Four weeks PI, blood was collected by cardiac puncture from all groups and the animals were subsequently euthanized. Collected blood was used for blood smear, DNA extraction, and subsequent real-time PCR analysis.

Thin blood smears
For thin blood smears, a drop of blood was taken from the hamsters and spread across a large area of the slide. Slides were air dried, stained with HEMA 3 stain, and examined under the microscope at 400× and 1000× magnification using the immersion oil technique.

DNA extraction and real-time PCR
Hamster RBCs were lysed using 1× RBC lysis buffer (eBio-science, San Diego, CA) and parasite DNA was extracted from 200 µL of whole blood using the DNA blood mini kit (Qiagen, Valencia, CA) as per the manufacturer’s instructions. Subsequently, DNA samples were purified through columns (QIAquick, Qiagen) to eliminate possible PCR inhibitors. Final sample volume was 50 µL. Real-time PCR was performed using a thermal cycler (ABI 7500, Applied Biosystems, Foster City, CA). Two primers and one internal probe were designed on the small ribosomal subunit gene sequence using computer software (Oligo Design Primer Express, Applied Biosystems). The probe was labeled with the fluorescent probe FAM at 5′ and the nonfluorescent quencher TAMRA at 3′. The reaction was prepared using universal master mix (TaqMan, Applied Biosystems) to a final volume of 50 µL containing 45 µmol/L each of primer, F109 (5′-TTTTACATGGATAACCGTGGTAA-3′) and R176 (5′-AAACGCCACGGCAA-3′), 10 µmol/L of the probe DLP133 (5′-FAM/TCTAGGGCTAATACATGCTCGAGGCC/FAMSp/3′), and 1 µL of DNA. Each sample was tested in triplicate. The amplification was performed as follows: 2 minutes at 50°C, one cycle; 10 minutes at 95°C, one cycle; and 15 seconds at 95°C and 1 minute at 60°C, 35 cycles.

Parasite quantification
The estimated number of parasites in collected samples was calculated for each real-time PCR procedure by the ABI 7500 thermal cycler software, based on a standard curve present on the plate. The positive control for the standard curve was developed as follows. DNA extracted from B. microti-infected human blood was amplified using a classical PCR with primers targeting a large sequence of the small ribosomal subunit gene. The product of the PCR was purified using columns (QIAquick, Qiagen) and cloned into PCR 2.1-TOPO vector following the manufacturer’s protocol. The plasmids containing the target sequence were transformed in DH5α competent cells, selected, amplified, and extracted with a plasmid mini kit (HiSpeed, Qiagen). Given that each parasite contains one copy of the small ribosomal subunit gene, the plasmid copy number corresponds to the number of parasites present in the sample. The plasmid copy number was calculated using the following formula:

\[
\frac{g/mol}{\text{Avogadro's number}} = \frac{\text{copy number where (g/mol)}}{\text{(base pair size of plasmid + insert)}} = \frac{330 \text{ Da} \times 2 \text{ nucleotide/base pair}}{\text{mol/L each of}}.
\]

The standard curve loaded in the real-time PCR plate consisted of serial dilutions of the plasmids from 10^6 to 1 plasmids. Data collected during the testing of the plasmids demonstrated that the real-time PCR detects a minimum copy number of approximately 10 parasites.

Calculation of B. microti load reduction
The levels of B. microti load reduction were calculated by comparing the parasite load, as determined by PCR, in hamsters injected with the preilluminated and postilluminated samples at 4 weeks PI. In calculating the log reduction, we took into consideration the results of the standard curve experiment, where injection of 10^2 parasites did not induce a PCR-detectable parasitemia in hamsters at 4 weeks PI. Therefore, we considered 10^2 our postillumination baseline even though no parasites were detectable by PCR in the hamsters infected with the treated samples.

RESULTS

Standard curve of B. microti infection in hamsters
To determine the optimal concentration of parasites for injection into hamsters, we inoculated nine groups of five hamsters with serial, 10-fold dilutions of untreated or illuminated apheresis PLTs containing B. microti-infected hamster RBCs. None of animals injected with the treated (i.e., illuminated) samples developed an infection detectable by real-time PCR (Table 1). In contrast, at 4 weeks PI, all the animals inoculated with untreated samples containing 10^2 and 10^3 parasites developed infections detected by real-time PCR. Four of the five hamsters
inoculated with $10^5$ and $10^4$ parasites and three of the five inoculated with $10^3$ parasites were parasitemic, while none of the hamsters receiving doses of $10^2$ or fewer parasites were demonstrably positive by real-time PCR. All positive hamsters, although injected with different concentrations of the parasite, developed similar levels of parasitemia as detected by real-time PCR (data not shown).

Inactivation of *B. microti* in plasma

Five units of *B. microti*-infected plasma were tested in separate experiments. Four weeks PI, hamsters injected with Day 0 plasma-positive controls and RB controls showed a range of parasitemia, from 24% to 51% infected RBCs based on a count of 500 cells in blood smear (data not shown). In contrast, the parasite was not detectable in the blood smears from hamsters receiving samples subjected to RB and then illuminated (Table 2 and Fig. 2). By real-time PCR, all the treated units (RB plus illumination) were negative with a parasite load reduction in hamsters between 4 and 5 log (Table 3). Samples from four of the five experiments were stored at $-20^\circ$C for 7 days and then injected in groups of six hamsters following the same procedure as for Day 0 samples. None of the hamsters injected with plasma samples that had been frozen, including the positive controls, demonstrated evidence of infection (data not shown).

**Inactivation of *B. microti* in PLTs**

The Mirasol treatment was also performed on five PLT units, cocomponents of the previously described plasma units, but due to technical problems unrelated to the pathogen reduction procedures, data were only available from four experiments. Procedures were the same as for plasma samples, with the exception of the posttreatment storage conditions; PLT samples were kept for 5 days at 22 to 24°C. Microscopic analysis at 4 weeks PI of blood smears from hamsters inoculated with samples of treated units on Day 0 revealed no parasites. In all experiments, blood smears of animals inoculated with samples from either the positive control or the RB samples showed detectable parasites, except in one experiment where only four of six hamsters infected with the RB control had a positive blood smear (Table 4 and Fig. 2). By real-time PCR, no parasite DNA was detectable in the treated samples injected on Day 0, with a measurable parasite load reduction in hamsters between 4 and 5 log (Table 5).

The positive control samples injected after 5 days of storage produced positive hamsters in only one of four experiments, with parasites detectable by microscopy and real-time PCR. In this experiment only, animals receiving the treated samples were negative by smear and by real-time PCR with an observed parasite load reduction in hamsters of 5 log (Table 5). In the other three experiments, positive controls stored for 5 days did not efficiently infect hamsters. Thus, none of the hamsters injected with the positive controls contained parasites detectable by blood smears or real-time PCR (Table 5).

**DISCUSSION**

In March 2007, the Canadian Blood Services and HemaQuebec organized a consensus development conference to discuss the current status of pathogen inactivation. The main focus of this consensus conference was to provide recommendations for the transfusion medicine community regarding pathogen inactivation. Among the conclusions made by the consensus panel was the acknowledgment that although the present risk of transfusion-transmitted disease is acceptable, emerging transfusion-transmitted pathogens represent an increasing concern. The current reactive strategy of surveillance, pathogen identification, and test development permits an agent to spread widely before significant clinical disease is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized. A proactive approach—such as the use of pathogen inactivation technologies—would provide an extra layer of protection, thereby allowing time to develop a test, and/or other interventions, before the pathogen is recognized.
routinely spread through blood transfusion. The panel also recognized that the implementation of pathogen inactivation technologies could allow for changes in the present donor deferral policy, the end of selected screening tests, and a reduction in costs and blood shortages.

Pathogen reduction represents a possible approach to mitigate transmission risk associated with *B. microti*, an emerging blood-borne protozoan parasite. Indeed, *B. microti* is the parasitic agent most frequently transmitted by blood transfusion in the United States. *B. microti* is endemic to portions of the United States, particularly the Northeast and upper Midwest. With estimates of more than 70 transfusion cases attributable to *B. microti*, this agent represents a considerable and growing blood safety threat to transfusion medicine. During a recent (September 2008) meeting entitled “Workshop to Consider Approaches to Reduce the Risk of Transfusion-Transmitted Babesiosis in the United States” sponsored by the U.S. Food and Drug Administration (FDA), members of the transfusion medicine community discussed potential strategies to reduce, and possibly eliminate, the risk of transfusing *B. microti*-infected blood. While testing blood donors, especially those from *Babesia*-endemic areas, is an attractive solution, the absence of an FDA-approved blood screening test for *B. microti* will considerably delay implementation of such an approach. Although the Mirasol system is not FDA approved, it is approved for use in countries that accept the CE Mark and is currently being evaluated for approval in several other geographic locations. Without an available test, PRTs could provide a concrete and feasible near-term solution, particularly if feasibility is demonstrated in whole blood.

In this study, we demonstrated that the Mirasol PRT system is a suitable method to inactivate *B. microti* in apheresis plasma and PLTs. One of the challenges of testing the efficiency of a PRT method for *B. microti* is the lack of an in vitro culture system to measure parasite survival. In our study, we used the golden Syrian hamsters as the primary detection method, since hamsters have been shown to be the most susceptible experimental host for *B. microti* infection. Based on a pilot experiment, we determined that inoculating hamsters with $10^7$ or $10^8$ parasites followed by termination at 4 weeks PI resulted in detectably high and reproducible parasitemia in control infections. Similar to previously published work using amotosalen, no parasites were detected 4 weeks PI in blood smears from animals receiving plasma or PLT units that had been treated with RB and illuminated, while animals receiving untreated controls demonstrated evidence of significant *Babesia* infections. In contrast to previously published studies, we counted a minimum of 500 cells on each blood smear in an attempt to increase the sensitivity of smear detection.
We also performed *B. microti* real-time PCR, and the results were complementary to the data obtained from blood smear, with parasite load reduction in hamsters ranging between 4 and 5 log in all treated units. In one instance, a PLT sample containing RB (but not illuminated) that was injected into hamsters on Day 0 induced parasitemia detectable by blood smears in four of the six animals, while five of the six hamsters were positive by PCR (Experiment 3, Tables 4 and 5). This discrepancy could be related to the limits of detection for microscopic analysis and/or the possibility of the PCR detecting dead parasites.

Two additional observations from the study were of note. First, in four of the five plasma experiments we stored sample aliquots for 7 days at -20°C before injection into animals (same protocol as for Day 0 samples). None of these frozen samples, including the positive controls, induced measurable parasitemia in the hamsters, suggesting that the freezing process killed the parasites regardless of the treatment protocol. Second, RB alone, in the absence of illumination, could have a deleterious effect on parasite survival. In three experiments (two plasma and one PLT), not all the animals injected with *B. microti*-infected plasma or PLTs exposed to RB (before illumination) produced parasitemia detectable by blood smears.

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Positive control</th>
<th>RB control</th>
<th>Mirasol treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6/6 5.8 × 10^4</td>
<td>5/6 2.6 × 10^4</td>
<td>0/6 0</td>
</tr>
<tr>
<td>2</td>
<td>6/6 3.1 × 10^6</td>
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<tr>
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<td>6/6 2.1 × 10^5</td>
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<td>0/6 0</td>
</tr>
<tr>
<td>4</td>
<td>6/6 1 × 10^5</td>
<td>6/6 2.8 × 10^5</td>
<td>0/6 0</td>
</tr>
<tr>
<td>5</td>
<td>6/6 1 × 10^5</td>
<td>6/6 1.9 × 10^5</td>
<td>0/6 0</td>
</tr>
</tbody>
</table>

**TABLE 4. Microscopic analysis of blood smears from hamster blood collected 4 weeks PI of Day 0 and Day 5 PLTs samples**

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Positive control</th>
<th>RB</th>
<th>Mirasol treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 0</td>
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<td>5/6 1.4 × 10^6</td>
<td>0/6 0</td>
</tr>
<tr>
<td>2</td>
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<tr>
<td>4</td>
<td>6/6 3.3 × 10^6</td>
<td>6/6 6.2 × 10^6</td>
<td>0/6 0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Positive control</th>
<th>RB</th>
<th>Mirasol treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 5</td>
<td>0/3 0</td>
<td>0/6 0</td>
<td>0/6 0</td>
</tr>
<tr>
<td>4</td>
<td>0/6 0</td>
<td>0/6 0</td>
<td>0/6 0</td>
</tr>
<tr>
<td>5</td>
<td>0/6 0</td>
<td>0/6 0</td>
<td>0/6 0</td>
</tr>
</tbody>
</table>

* No data from PLTs were collected on Experiment 1.
† These groups had less than six hamsters because some animals died from causes unrelated to *Babesia* infection.
positive results by microscopic analysis or PCR (Experiments 1 and 3, Tables 2 and 3; Experiment 3, Tables 4 and 5). Interestingly, a similar hypothesis concerning the negative impact of RB against *Plasmodium falciparum* was previously suggested, but further investigation is needed to establish the effects on *B. microti*.

In summary, the results of this study indicate that *B. microti*, like many other pathogens investigated to date,1,4-6 is successfully inactivated by the Mirasol PRT system. While most cases of transfusion-transmitted *Babesia* have involved *B. microti*, two cases of transfusion-transmitted *B. duncanii* have occurred.25,26 Given the broad range of organisms killed by the Mirasol PRT system, it is likely that this procedure will also be effective against other species of *Babesia*, including *B. duncanii*.

While this study demonstrates the efficacy of pathogen reduction for *B. microti* in apheresis plasma and PLTs, it does not address the primary source of transfusion-transmitted *Babesia*, which is infected RBC units. Thus, further studies to evaluate the efficiency of the Mirasol PRT system for RBCs are needed in the near term. However, several cases of transfusion-transmitted *Babesia* involving whole blood–derived PLTs have been reported,15 and the Mirasol PRT system could represent a promising technology to increase the safety of our blood supply.

**ACKNOWLEDGMENTS**

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**CONFLICT OF INTEREST**

HLR and RPG are employed by CaridianBCT Biotechnologies (Lakewood, CO), the company that developed and sells the Mirasol pathogen reduction technology.

**REFERENCES**


