Table 2. Summary of deaths attributed to transfusion-transmitted *Babesia* infection that were reported to the US Food and Drug Administration.

Patient	Age, years	Sex	State of residence	Medical history	Presenting complaint	Clinical course	Donor information
1	81	Female	Maryland	Hypercholesterolemia, hypertension, severe nosebleeds requiring transfusion but otherwise in good health	Severe fatigue and lethargy	CBC showing a hemocrit of 21%, a platelet level of 21,000 platelets/mL, BUN level of 80 mg/dL, and a creatinine level of 2.5 mg/dL (indicating renal failure); examination for anemia and fatigue identified *Babesia* species on PB smear (positive PCR result); treated with quinine and clindamycin; developed signs of adult respiratory distress syndrome; experienced thrombotic cerebrovascular accident on day 5 of treatment with high fever	Resident of Maryland; traveled to New York (Long Island); positive PB smear result; *B. microti* IFA titer of 1:512
2	88	Male	Rhode Island	Chronic myelomonocytic leukemia with chronic anemia (transfusion dependent) and GI bleed	4-Day history of progressive weakness, fatigue, and anorexia with low-grade fever	*Babesia* species identified by PB smear; treated with atovaquone and azithromycin; died on hospital day 12 with persistent parasitemia, progressive renal failure, anemia, and altered mental status	Resident of Rhode Island; *B. microti* IFA titer of 1:1024
3	57	Male	Texas	Recent history of melena and previous hepatitis B and C virus infection, cirrhosis, coronary artery disease, congestive heart failure, receipt of coronary artery bypass grafts, and GI bleed requiring transfusion	10–12-Day history of fever, night sweats, chills, and other complaints of melena, weakness, dizziness, anorexia, and increasing ascites	*Babesia* species identified by PB smear; treated; developed altered mental status, respiratory distress and GI bleed	Resident of Texas; history of travel to Massachusetts; *B. microti* IFA titer of 1:256
4	76	Male	Minnesota	Transfusion-dependent acute myelocytic leukemia, rheumatoid arthritis, steroid-induced immunosuppression, and history of splenectomy, coronary artery disease, idiopathic thrombocytopenia, and multiple other medical problems	Several-day history of fever, cough, weakness, and dyspnea	Sepsis examination and broad-spectrum antibiotics started at hospital admission, with *Babesia* infection diagnosed (by PB smear) and treated on hospital day 2; death due to multiple medical problems	Resident of Minnesota; *B. microti* IFA titer of 1:256; negative PCR result
5	71	Female	Connecticut	Chronic liver disease (portal hypertension with gastroesophageal varices and hepatorenal syndrome), chronic transfusion-dependent anemia and diabetes, splenectomy, and cholecystectomy	Low-grade fever, complaints of chills and weakness for several days, with hemocrit decreasing from 29% to 23% at routine outpatient CBC monitoring	*Babesia* species identified by PB smear; treated; developed acute tubular necrosis, altered mental status, and progressive hypotension	Resident of Connecticut; *B. microti* IFA titer of 1:256; positive Western Blot result; negative PCR result
6	82	Female	Ohio	Receipt of coronary artery bypass grafts and aortic valve replacement with transfusion, atrial fibrillation, cerebrovascular accident, and hyperlipidemia	Several-day history of fever and chills, with anemia and thrombocytopenia diagnosed at hospital admission	*Babesia* species identified by PB smear; treated with clindamycin and quinine plus automated RBC exchange by apheresis, which reduced parasitemia from 26% to 5%; developed altered mental status; the patient died of multiple-organ failure, *Staphylococcus aureus* pneumonia, and acute myocardial infarction	Resident of Ohio; traveled to Connecticut 2 months before donating blood; *B. microti* IFA titer of 1:256
7	74	Female	New Jersey	Insulin-dependent diabetes, end-stage renal disease (receiving dialysis), coronary artery disease (receipt of coronary artery bypass graft), GI bleeding, and polyp removal with transfusion	Nausea, cough, vomiting, weakness, and fever	Low platelet count on CBC with 8% *Babesia* species found by manual PB smear, confirmed by PCR as *B. microti*; atovaquone, clindamycin, and quinine failed to prevent respiratory failure, hypotension, cardiac complications, and progressive shock	Resident of New Jersey; *B. microti* IFA titer of 1:128
8	61	Female	Indiana	End-stage renal disease (receipt of hemodialysis), congestive heart failure, GI bleed requiring transfusion at previous hospital admission	Nausea with fever while receiving hemodialysis	Initially treated for bacterial sepsis (vancomycin and ceftazidime), then parasitemia was treated with exchange transfusion; originally received a misdiagnosis of malaria; treated with clindamycin and quinidine; developed altered mental status and disseminated intravascular coagulation and died; positive PCR results and an IgG titer of 1:2048 for *B. microti*	Resident of Indiana; traveled to wooded areas of Wisconsin 2 months before donating blood; no known tick bite; IgG titer of >1:256 and IgM titer of 1:20 for *B. microti*; negative PCR result after donation
9	43	Female	Delaware	Congenital hypoplastic anemia (Diamond-Blackfan syndrome), splenectomy, hepatitis C virus infection, pulmonary hypertension, iron overload, and multiple transfusions	Fatigue, "shakes" for 4 days without fever, decreased appetite and loose bowel movements for 1 week, chronic dry cough with infiltrates on chest radiograph	Treated for pneumonia with levaquin, vancomycin, and oseltamivir; previous PB smear reexamined as positive for *Babesia* species treated with clindamycin and quinine and transferred to ICU because of respiratory distress	Resident of New Jersey; traveled to Rhode Island but no known tick bite; *B. microti* IFA titer of 1:1024; negative PCR result; donated RBC unit was frozen and deglycerolized before transfusion

NOTE. The information in this table was reported through a passive surveillance system; we report here the information provided. BUN, blood urea nitrogen; CBC, complete blood count; GI, gastrointestinal; ICU, intensive care unit; IFA, indirect immunofluorescence antibody assay; PB, peripheral blood.

Table 3. Timing of clinical events in fatal cases involving transfusion-transmitted *Babesia* infection reported to the US Food and Drug Administration.

Timing	Patient								
	1	2	3	4	5	6	7	8	9
Date of implicated blood unit transfusion	9 April 1998	16 November 2005	6 December 2005	24 August 2006	20 September 2006	6 September 2006	17 September 2007	20 September 2007	26 November 2007
Latency period,[a] days	35	38	50	30	18	30	29	43	41
Time to diagnosis,[b] days	43	42	50	34	19	36	31	57[c]	45
Time to death, days	49	55	57	42	26	41	34	50	47

[a] Periods from the date of implicated transfusion to the onset of symptoms are approximate (based on available estimated dates of symptom onset).
[b] Posttransfusion diagnosis of *Babesia* infection.
[c] The patient died prior to diagnosis of *Babesia* infection.

Babesia infection should be considered among potential etiologies for otherwise unexplained fever in patients who have recently received blood products. Because of the mobility of donors and transportation of blood products, babesiosis should be considered even beyond geographical regions with naturally occurring disease. As noted in table 2, several donors did not live in areas of endemicity but had traveled to these regions before donating blood.

Patients presented with symptoms (table 2) that were typical of natural infections. Most developed altered mental status, renal failure, or respiratory distress. The interval from blood transfusion to symptom onset was 2.5–7 weeks (table 3). An earlier article reported a 1–9-week time frame for transfusion-transmitted babesiosis [17]. These ranges of latency periods contrast with the natural infection incubation time of 2–4 weeks.

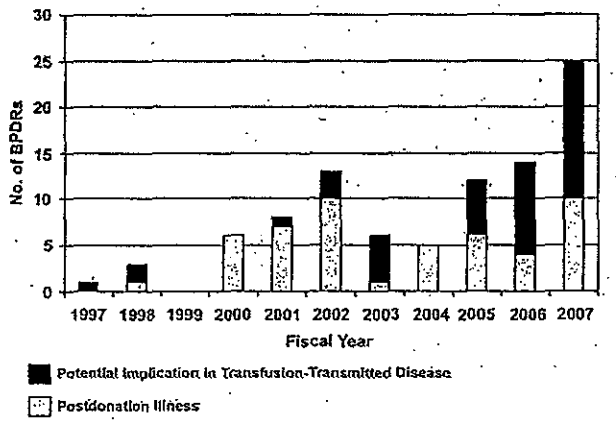

Figure 1. Summary of babesiosis-related Biological Product Deviation Reports (BPDRs) received by the US Food and Drug Administration (FDA) during fiscal years 1997–2007 (the FDA fiscal year is from 1 October through 31 September). These data do not include reports of infected donors identified prospectively through antibody assay research trials. BPDRs may include >1 recipient, unit, or donation. Potential implication in transfusion-transmitted disease refers to reports that indicate the safety of a blood component unit that may have been affected (e.g., instances when a blood transfusion recipient received a diagnosis of babesiosis, but the donor could not be contacted for confirmation). Postdonation illness refers to illness in donors who notified the blood collection establishment after donation that they had received a diagnosis of babesiosis. Whether these donors were infected at the time of donation was unknown; all units (not yet transfused) from these donors were withdrawn; and the donors were indefinitely deferred.

With 1 exception, all patients received transfusions from August through December, consistent with the seasonality of *Babesia* infection. Chronic parasitemia in the donor may have accounted for the 1 case involving a blood transfusion in April.

Implicated donations were identified in all cases; the donors tested positive by peripheral blood smear or immunofluorescence antibody assay. Four donors' samples also tested by PCR had negative results. They may have been convalescent and no longer parasitemic or were PCR negative because of the small sampling volume. All donors were asymptomatic at donation and remembered no tick bite.

Because many babesiosis cases may escape recognition, questioning donors has limited preventive value [17]. *Babesia* species can survive blood banking procedures, including refrigeration, leukoreduction, and filtration; pathogen transmission through transfusion of RBCs, deglycerolized RBCs, or platelets can occur [1, 18–21]. *Babesia* parasites can survive in frozen RBCs, because the glycerol treatment prevents lysis.

In view of the short periods between symptom onset and death (5–17 days) (table 3), examination of a peripheral blood smear (or other testing, depending on availability and the level of clinical suspicion) for possible *Babesia* species should be considered early in the evaluation of unexplained fever during the first few weeks after transfusions, particularly in asplenic or otherwise immunocompromised patients. Infectious disease consultation may be required to microscopically distinguish *Babesia* species from *Plasmodium* organisms.

Although babesiosis is not nationally notifiable, reporting transfusion-transmitted *Babesia* infections to public health authorities can allow investigators to identify infected donors and interdict remaining units. Investigation of prior donations also allows testing of associated recipients.

Similarly, blood collectors should immediately report postdonation babesiosis to the transfusion facilities to expedite

prompt withdrawal of potentially infected unexpired blood components. We remind blood establishments of the requirement to submit fatality and BPDRs to the FDA.

Our data cannot distinguish whether the increase in the numbers of deaths and reports to the BPDR system reflect an increasing incidence of babesiosis and/or improved diagnosis and reporting. State Health Departments (e.g., in New York and Connecticut) have also seen an increase in the number of babesiosis case reports over the past 10 years [22–25].

Each year, >5 million recipients receive >14 million transfusions of whole blood or RBCs [26]. Transfusion-transmitted babesiosis appears to be rare, but increased clinician awareness of the possibility of babesiosis in febrile transfusion recipients may facilitate earlier diagnosis and more successful treatment. It will also trigger timely public health investigations to interdict extant infected units and alert other associated recipients, protecting others from this potentially fatal blood-borne pathogen.

Addendum. During final revisions of this article in late September 2008, the FDA received a report of another death associated with transfusion-transmitted babesiosis. An elderly woman in Minnesota died ~3 weeks after receipt of 2 units of RBCs. One of the donated units' retention segments was positive for *Babesia* species by serologic testing and PCR.

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医薬品 研究報告 調査報告書

識別番号・報告回数			報告日	第一報入手日	新医薬品等の区分	総合機構処理欄
				2008. 11. 20	該当なし	

一般的名称	新鮮凍結人血漿	研究報告の公表状況		公表国
販売名(企業名)	新鮮凍結血漿「日赤」(日本赤十字社) 新鮮凍結血漿-LR「日赤」(日本赤十字社)		Seed C, Kee G, Ismay S, Wong T, Keller A.. AABB Annual Meeting and TXPO 2008; 2008 Oct 4-7; Montreal.	オーストラリア

研究報告の概要

○マラリア抗体検査-輸血伝播マラリア(TTM)のリスクを最小に抑える安全かつ有効な戦略

背景:マラリアのスクリーニングに関して、オーストラリア赤十字(ARCBS)は2005年7月から、従来の医療歴、渡航歴の収集から、リスクのある供血者に対し、リスクへの暴露を特定したときから最低4ヶ月間のマラリア抗体の検査を実施する代替戦略を導入した。

方法:マラリアに罹患後回復した、あるいは過去3年間にマラリア流行国へ渡航・居住した供血者の血液を、市販のマラリア抗体EIAを用いて検査した。陰性血液は輸血用として供給され、供血者は再度供血可能とされた。EIA反復陽性(RR)の血液は追加検査(リアルタイムプラスモジウムPCR及び 免疫抗原クロマトグラフィー)に供された。追加検査陰性の供血者は現在の感染を示す証拠がない「抗体陽性」と見なされた。追加検査で陽性となった供血者は感染の可能性があると見なされ、直ちに臨床診断に紹介された。

結果:2005年7月～2008年2月に合計122,713の供血血液のEIA検査が実施され、そのうち117,900(96.1%)は陰性であり、ARCBSは159,287本のRBCおよび17,815本の血小板を供給した。EIA RR 4,813(3.9%)のうち1例で、PCRによる低レベルのプラスモジウムDNA が検出された(初回検体31、追加検体50copies/mL)が、抗原は陰性であった。この供血者はリベリア移民で幼少時にマラリアの既往歴があったが、追跡調査時には症状はなかった。

結論:この検査戦略の開始以降、既存の供血者に由来する輸血可能製剤の製造効率は著しく向上し、TTM症例の報告もなかった。

報告企業の意見	今後の対応
オーストラリア赤十字(ARCBS)は2005年7月から、マラリア感染のリスクがある供血者に対し、リスクへの暴露を特定したときから最低4ヶ月間のマラリア抗体のスクリーニングを実施する代替戦略を導入した結果、既存の供血者に由来する輸血可能な製剤の製造効率が著しく向上し、輸血伝播マラリア症例の報告もないとの報告である。	日本赤十字社では、輸血感染症対策として問診時に海外渡航歴の有無を確認し、帰国(入国)後4週間は献血不適としている。また、マラリア流行地への旅行者または居住経験者の献血を一定期間延期している(1～3年の延期を行うとともに、帰国(入国)後マラリアを思わせる症状があった場合は、感染が否定されるまでの間についても献血を見合わせる)。今後も引き続き、マラリア感染に関する新たな知見及び情報の収集、対応に努める。

使用上の注意記載状況・その他参考事項等

新鮮凍結血漿「日赤」
新鮮凍結血漿-LR「日赤」

血液を介するウイルス、
細菌、原虫等の感染
vCJD等の伝播のリスク

these findings, and showed both haplotypes have altered hr^S and hr^B antigen expression, and the associated c and e antigens are altered to the extent that alloanti-c and alloanti-e were produced. We also describe a homozygous proband with an alloantibody to the high-prevalence antigen that is antithetical to JAL. **Methods:** Samples from 15 JAL+ persons (11 probands and 4 family members including two Caucasian, six African American Blacks, one Puerto Rican Black, and six Brazilian Blacks) were tested. Hemagglutination studies were performed by standard methods using reagents from multiple sources. **Results:** The JAL+ status of the RBCs was determined with three anti-JAL (J Pas., Allen, MacD). RBCs from both Caucasian JAL+ probands had the (C)(e) haplotype and altered C, e, hr^S, and hr^B antigens. RBCs from Black JAL+ persons had the (c)(e) haplotype and expressed altered c, e, f, V, hr^S, VS, and hr^B antigens. Of six anti-C reagents, all agglutinated the two Caucasian samples moderately. Of nine anti-c reagents tested with RBCs from the non-Caucasian probands, reactivity ranged from strong to negative; notably, clone MS42 reacted strongly, MS33 and BS240 reacted moderately, and Gamma clone 951 was non-reactive. Of 12 anti-e reagents, reactivity ranged from strong to negative; notably, clone MS62 and MS63 reacted moderately, MS69 and HIRO38, 41, and 43 reacted weakly to moderately, and MS16 (depending on the formulation) was weakly reactive or non-reactive. All RBC samples were tested with at least two examples of anti-V, -hr^S, -VS, and -hr^B; some gave weak reactions while others did not react. Plasma from one proband contained alloanti-c, from another contained alloanti-e, and from a third contained an alloantibody that failed to react with D- – and Rh_{null} RBCs. This apparent anti-Rh17 recognizes the high-prevalence antigen antithetical to JAL that we have named CEST. **Conclusions:** The presence of the JAL antigen has a quantitative and qualitative effect on the expression of, in Caucasians C, e, hr^S, and hr^B antigens, and in people of Black African ancestry c, e, f, V, hr^S, VS, and hr^B antigens. The qualitative effect on two antigens is revealed by two patients who received blood transfusions and made alloanti-c or alloanti-e. This is the first description of an antibody to the high-prevalence antigen antithetical to JAL.

Disclosure of Conflict of Interest

Christine Lomas-Francis, Connie Westhoff, Denden Alcantara, Pamela Nickle, Joan Uehlinger: Nothing to Disclose
Marion E. Reid: Not Specified

S36-020D

The JAL Antigen (RH48) Is Encoded by an ARG114TRP Mutation

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Background: Two JAL-positive (Rh48+) haplotypes have been described. One, found in Caucasians, has altered C and e antigens [(C)(e)], and the other, found in people of African Black ancestry, has altered c and e antigens [(c)(e)] (Lomas, et al., Vox Sang 1990;59:39). The purpose of this study was to determine the molecular basis associated with expression of the JAL antigen. **Methods:** Samples from fifteen JAL+ probands; two Caucasian, six African American Blacks, six Brazilian Blacks, and one Puerto Rican were included in the study. Hemagglutination studies were performed by standard methods. DNA, extracted from peripheral blood leukocytes, was amplified by PCR and analyzed by sequencing. Reticulocyte RNA isolation and Rh-cDNA analysis was performed by standard molecular methods. **Results:** Samples from Caucasian JAL+ probands had RHCE*Ce, and those from Black JAL+ probands had RHCE*ce, but all had a 340C > T (Arg114Trp) missense mutation in exon 3 of RHCE regardless of the Ce or ce background allele. The JAL-encoding RHCE*ce allele also had 733C > G (Leu245Val) and red cells from informative heterozygotes and homozygotes did not express f, hr^S, or hr^B antigens, and had dramatically weakened V, and VS antigens. Expression of c antigen was also altered, as the RBCs were non-reactive with one of three monoclonal anti-c tested. In kind, expression of C on the Ce allele was altered, as was the e antigen expressed from both the ce or Ce allele that encoded JAL. **Conclusions:** Expression of the JAL (Rh48) antigen results from 340C > T (Arg114Trp) on either a Ce or ce background. Although the Ce allele encoding the JAL antigen has been reported as CeMA by Noizat-Pirenne et al., (Transfusion 2002;42:627) and the ce allele as ceS(340) by the same authors (Blood 2002; 100:4223), these alleles have not previously been recognized as encoding the JAL antigen. Those samples were studied because of altered expression of C or e antigen, respectively. We show here for the first time that, in addition to altered expression of C or c and e, the 114Trp mutation encodes the low-prevalence Rh antigen, JAL.

Disclosure of Conflict of Interest

Connie Westhoff, Sunitha Vege, Christine Lomas-Francis, Kim Hue-Roye, Lilian Castilho, Marion E. Reid: Nothing to Disclose

Transfusion Transmitted Diseases: Malaria and Chagas Disease

S37-020E

Malaria Antibody Testing—A Safe and Efficient Strategy to Minimise the Risk of Transfusion Transmitted Malaria (TTM)

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Background: Until recently, owing to the lack of a suitable test, screening for malaria in Australian blood donors involved collecting a medical and travel history and excluding the 'cellular' blood components from donors with a potential malaria exposure. This strategy minimised the residual risk of TTM to less than 1 in 10 million but resulted in the unavailability for transfusion of approximately 35,000 red blood cells (RBC) per annum. In July 2005 the Australian Red Cross Blood Service (ARCBS) implemented an alternative strategy based on screening donors with potential malaria exposure for malaria antibodies a minimum of 4 months after their last identified risk exposure. **Methods:** Since July 2005, donations from donors identified as having had malaria and recovered, or having travelled to or resided in a malarial country within the last 3 years are eligible for testing. RBC from these donors were collected, quarantined and samples tested using a commercial malaria antibody EIA. Non reactive (NR) donations were then issued for transfusion and the associated donors re-instated for cellular component manufacture. EIA repeatedly reactive (RR) donations were not issued and subject to supplementary testing (real-time plasmodial PCR and antigen immuno-chromatographic test). Concordantly supplemental test negative donors were considered 'antibody positive' without evidence of current infection. Donors reactive on either or both supplemental tests were considered potentially infected and referred immediately for clinical assessment. **Results:** A total of 122,713 donations were tested by EIA between July 2005 and February 2008, of which 117,900 (96.1%) were NR and the remaining 4,813 (3.9%) repeat reactive (RR). From donors re-instated by a NR EIA, ARCBS issued for transfusion an additional 159,287 RBC and 17,815 platelets representing the combined return from their tested donation, and subsequent donations made during the period of their original restriction. Of the 4,813 EIA RR samples, one had low level but reproducibly detectable plasmodial DNA by PCR (index sample 31 and follow-up sample 50 copies/uL) but was antigen negative. The donor, a migrant from Liberia had a history of malaria during childhood but was asymptomatic at follow up. **Conclusions:** The testing strategy has delivered in excess of 50,000 additional RBC and 5,000 additional platelets annually since inception. This has markedly improved manufacturing efficiency by increasing the proportion of transfusible components from existing donors. The lack of a reported TTM case since implementing the new strategy suggests that this dividend has been achieved without measurably impacting recipient safety.

Disclosure of Conflict of Interest

Clive Seed, Glenda Kee, Susan Ismay, Timothy Wong, Anthony Keller: Nothing to Disclose

S38-020E

Malaria Deferrals: Time to Lessen the Impact of Travel Deferrals

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Background: Human malaria, caused by five species of the intraerythrocytic protozoan parasite Plasmodium, remains a blood safety concern. However, blood banks are adversely impacted by ongoing donor loss due to an increasing number of malaria deferrals. Among the 1% of donors lost because of malaria deferrals, the majority (91%) were associated with travel to malaria endemic areas. In contrast, the only 2 transfusion-transmitted malaria cases reported in the US since 1998 were attributed to donors with past residence in an endemic region. This study investigated the relationship