医薬品 研究報告 調査報告書

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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)		Young C, Losikoff P, Chawla A, Glasser L, Forman E. Transfusion. 2007 Mar;47(3):540-4.	米国

研究報告の概要

○輸血によるTrypanosoma cruzi感染
背景:神経芽細胞腫(ステージ4)を発症した3歳半の女児が複数の血液成分製剤投与を受けた後、Trypanosoma cruziによるシャーガス病と診断された。
試験デザインおよび方法:輸血された製剤の全供血者に対し、再度採血機関に足を運びT. cruzi抗体検査のための血液検体を提供するよう依頼した。
結果:初回供血者1名がT. cruzi抗体陽性であることが判明した。当該供血者は、ボリビア出身であり、17年前に米国に移住した。移住後は母国に帰国していない。
結論:本症例は、米国・カナダでの輸血によるシャーガス病感染の7例目の報告である。ニューイングランド地方では発生が予想されていなかったが、実際には発生した。本症例は、疾患が輸血感染に関係することから患者の免疫状態が重要であること、疾患伝播における地理的移動の影響、米国の血液供給において認可されたシャーガス病スクリーニング検査が必要であることを示している。

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

報告企業の意見	今後の対応
神経芽細胞腫の3歳半の女児が、17年前にボリビアから米国に移住した供血者由来の血液の輸血を受けた後、Trypanosoma cruziによるシャーガス病と診断されたとの報告である。	日本赤十字社は、輸血感染症対策として献血時に海外渡航歴・居住歴の有無を確認し、シャーガス病の既往がある場合には献血不適としている。今後も引き続き情報の収集に努める。

Transfusion-acquired *Trypanosoma cruzi* infection

Carolyn Young, Phyllis Losikoff, Anjulika Chawla, Lewis Glasser, and Edwin Forman

BACKGROUND: A 3 1/2-year-old girl with Stage 4 neuroblastoma received multiple blood components and was subsequently diagnosed with Chagas disease, which is caused by *Trypanosoma cruzi*.

STUDY DESIGN AND METHODS: All blood donors of the units that were transfused were requested to return to the collection facility for a blood sample to be tested for antibodies to *T. cruzi*.

RESULTS: One first-time donor was found to be positive for the presence of *T. cruzi* antibodies. This donor was originally from Bolivia and immigrated to the United States 17 years previously. She had not returned to her native country since her emigration.

CONCLUSIONS: This is the seventh reported case of Chagas disease transmission by blood transfusion in the United States and Canada. Although this would not be expected to occur in New England, it did, and this case demonstrates the significance of the immune status of patients as it relates to transfusion-acquired infections, the impact of geographic mobility in disease transmission, and the need for a licensed screening test for Chagas disease for the US blood supply.

CASE REPORT

A 3 1/2-year-old girl with Stage 4 neuroblastoma presented to a teaching hospital with a temperature of 40°C and neutropenia with a white blood cell (WBC) count of 200 per µL and an absolute neutrophil count of 120 per µL. Her pulse was 124 bpm and her blood pressure was 75/48 mmHg. She had an unusual 2.5- to 3-cm erythematous macule on her left hip, vulvar swelling, and evidence of a marrow biopsy performed on her left iliac crest 1 month before admission. She had no respiratory distress, headache, mental status changes, vomiting, or diarrhea. She complained of mild abdominal pain consistent with her operative history. Before her admission, she had completed five courses of nonmyeloablative chemotherapy that had required platelet (PLT) support, and she had gross disease seen on exploratory laparotomy performed 2 weeks previously. One month before her admission she had undergone a peripheral blood progenitor cell (PBPC) harvest. She never received these PBPCs.

A blood smear was performed due to the hospital policy of performing a complete blood count with a manual differential when a patient is neutropenic. The technologist examining the slide noted some unusual parasites. The clinical pathologist, LG, reviewed the slide and identified the parasites as trypomastigotes of *Trypanosoma cruzi*, the organism that causes Chagas disease (Fig. 1). The hospital blood bank physician reported transfusion-associated Chagas disease to the blood collection physician and gave the unit numbers transfused.

The patient was given 15 mg per kg per day nifurtimox divided in three daily doses after *T. cruzi* was identified on the blood smear. The child's parasite load dropped from 500,000 to 800 per µL over the first 48 hours of therapy, determined by a manual count with a hemocytometer counting chamber, and became undetectable after 14 days of treatment. *T. cruzi* antibody titer was positive 3 weeks after discharge at 1:64 and 3 months later increased to 1:1024. Although the diagnosis was made by identification of the parasite in the peripheral blood, further testing by polymerase chain reaction (PCR), immunofluorescent antibody assay antibody testing, and culture were performed for the purpose of following the patient's response to treatment (Table 1). After discharge

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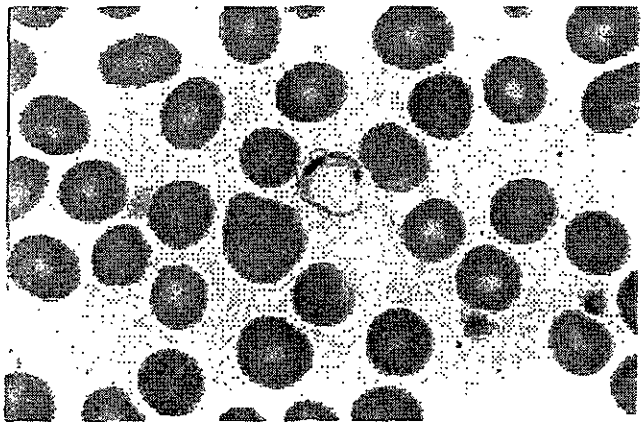

Fig. 1. Peripheral blood smear showing a typical trypanosome. The organisms usually appear C- or U-shaped with a centrally placed nucleus and a dotlike kinetoplast at the posterior end (Wright's stain; original magnification, ×500).

from the hospital she continued to receive chemotherapy with topetecan and cyclophosphamide in 4- to 6-week cycles. Nifurtimox was continued beyond the standard 3 months' course due to her compromised immune status. An autologous PBPC transplant was not performed based on her gross disease burden and concern about further immunocompromise. Three years after the diagnosis of Chagas disease, the patient expired in December 2005 due to progression of her primary disease.

MATERIALS AND METHODS

Seventy-eight units of leukoreduced and irradiated PLT concentrates and red cells (RBCs) had been transfused to this patient. All 78 donors were asked to return to the blood center for samples to be collected for testing for antibodies to *T. cruzi* performed by the Centers for Disease Control (CDC). Individual donor registration records were reviewed. We found that one donor completed a Spanish registration form and one completed a Portuguese registration form. The donor who had completed the Spanish registration form did not respond to written requests in both English and Spanish to return for testing. Phone calls to the donor's home revealed that the phone had been disconnected with no forwarding information. Spanish-speaking staff were sent to her home to obtain samples for testing because the medical director thought that she was the most likely source of transmission after reviewing all the donor registration records and she had not returned to the blood center. The CDC performed an immunofluorescent antibody assay for *T. cruzi*, which was positive at 1:256 on a sample that was obtained about 12 weeks after donation. Consignees of the RBCs and fresh-frozen plasma (FFP) were contacted. The FFP had been discarded at the hospital approximately 1 week after its receipt due to issues unrelated to this case. The recipient of the RBC unit was immunocompetent and was contacted by the hospital. A blood smear was performed that did not reveal any parasites. Antibody testing was planned; however, the patient did not follow through. He had a myocardial infarction in 2003. This recipient was last seen at the hospital in June 2006.

Additional samples were acquired from the donor for PCR testing on one subsequent occasion, approximately 14 weeks after donation, which was negative. PCR was performed with primer pairs that amplify the minicircle sequence, the flagellar protein locus, and the nuclear repeat sequence.[1,2] These additional samples were acquired from the donor at her home with a blood center staff member who had emigrated to the United States from Bolivia and the blood center medical director, CY. The donor's history was taken at the time these samples were obtained, with his translation assistance.

Of the 78 donors, 48 returned for testing. Forty-seven were negative, one was seropositive for *T. cruzi* as stated, and 30 did not return for testing. Telephone screening for travel history and vertical transmission ruled out any risk for *T. cruzi* in 20 of these donors. The remaining 10 donors did not respond to letters and were not reachable by phone. All transfused units had been leukoreduced before storage and irradiated with 2500 cGy.

Implicated donor

The implicated donor was a 50-year-old, first-time donor in the United States who came from Bolivia 17 years earlier. She had donated once before over 20 years prior, in Bolivia, as a directed donor for the child of a friend. She had never returned to Bolivia or other endemic Chagas disease areas. She had never been diagnosed with Chagas disease. When shown the picture of the reduviid bug, she immediately recognized it and said she had seen many in Bolivia. She recalled that she had been screened for Chagas disease a few times in her childhood by visiting health-care teams in her village and that the results had been negative. Her travel history included trips to Paraguay in her youth. Her medical history was not significant for any symptoms of Chagas disease, except occasional constipation, which she had attributed to her age and diet. After the CDC results were obtained, the donor was followed by her physician in consultation with the CDC. Her PLT concentrate was transfused to the patient approximately 6 weeks (September 4, 2002) before the child presented with symptoms of fever and neutropenia (October 19, 2002) and was diagnosed with Chagas disease.

DISCUSSION

In endemic areas, that is, rural areas of Mexico and Central and South America, *T. cruzi* is transmitted when mucous

TABLE 1. Patient's results for *T. cruzi*

Date collected*	Peripheral blood	Titer†	Culture	PCR
Presentation	Positive	Negative	Positive	Positive
2 weeks	Negative	1:32	Not done	Weak positive
4 weeks	Negative	1:64	Negative	Positive
6 weeks	Negative	1:64	Negative	Weak positive
10 weeks	Negative	1:256	Positive	Positive
14 weeks	Negative	1:1024	Positive	Positive
16 weeks	Negative	1:1024	Negative	Negative
20 weeks	Negative	Not done	Not done	Negative
23 weeks	Not done	Not done	Negative	Negative
6 months	Not done	1:512	Negative	Negative
3 years, 2 months	Negative	1:32	Not done	Negative

* Date of specimen collection recorded with respect to initial presentation.
† Antibody titers measured by indirect immunofluorescence performed by the CDC; a positive result is considered equal to or greater than 1:64.

membranes are contaminated by feces of infected triatomine bugs. Congenital infection by vertical transmission, transmission by blood transfusion, and organ transplantation are well described modes of infection in endemic areas. The acute illness, usually a childhood illness, is mild with nonspecific symptoms; however, meningoencephalitis and myocarditis can occur in the acute stage of infection. Infected persons then enter an intermediate phase, which is generally asymptomatic and characterized by elevated *T. cruzi* titers and intermittent parasitemia. Decades later 30 percent of those chronically infected will develop cardiomyopathy or gastrointestinal manifestations such as megaesophagus or megacolon. Patients with chronic *T. cruzi* infection who become immunosuppressed may have a reactivation of acute disease which can be more severe than in immunocompetent individuals.[3]

Despite a handful of cases of Chagas disease acquired in the United States, the United States is not considered endemic for *T. cruzi*.[4] Millions of immigrants from *T. cruzi*-endemic areas, however, live in the United States, and it is estimated that 50,000 to 100,000 of these people are chronically infected with *T. cruzi*.[5] These chronically infected individuals are a potential source of *T. cruzi* transmission via blood transfusion or organ transplant.[6] Because estimates for seroprevalence of *T. cruzi* antibodies in selected US blood donor populations varies widely and ranges from 0 to 4.9 percent, the risk for receiving a *T. cruzi*-contaminated unit may be different geographically.[7-11] A conservative estimate of the nationwide risk for transfusion acquired Chagas disease suggests that more than 600 potentially infectious components are entering the US blood supply annually (D. Leiby, AABB 2004 meeting). This translates into many more possible *T. cruzi* infections that are going undiagnosed in transfusion recipients, even without 100 percent transmission.

The US Census Bureau reported that the US resident population was 281,421,906 in 2000. Of this population, Hispanics accounted for 12.5 percent, up from 9 percent in the 1990 census, making it the fastest growing minority group in America. In response to the growing Hispanic population in Rhode Island, the blood center initiated blood donor recruitment targeting this group a few months preceding this case. Recruitment efforts included translating the donor registration record and predonation brochure into Spanish, hiring Spanish-speaking personnel, and adding Spanish to our automated phone answering tree. As a result of these efforts, our Hispanic donor base increased. This individual never gave blood in the United States before this donation and gave blood at a blood drive targeted toward Hispanic donors. Although New England would not be considered a high-risk area, compared to Los Angeles, Texas, or possibly New York, our donor, who resided in Rhode Island, was seropositive for the presence of Chagas disease. Nationwide, the number of Hispanic donors will likely increase as recruitment efforts continue to focus on this growing population. In fact, America's Blood Centers, an organization that consists of more than 70 community blood centers, supports a national recruitment program aimed at educating the Hispanic community about donating blood.

Currently, based on donor surveys performed in six regional blood centers, 2.5 percent of the donor population were born in Chagas-endemic areas. This survey did not ask whether the donor's mother was born in Chagas-endemic areas so does not include the risk for congenital infection (D. Leiby, 2004 AABB Meeting).

It has been demonstrated that the majority of blood donors seropositive for the presence of *T. cruzi* antibody have low-level parasitemia (L. Kirchhoff, University of Iowa, Iowa City, IA, personal communication). In this case, the donor did not have detectable parasitemia when tested by PCR by the CDC on a subsequent blood sample obtained approximately 13 weeks after donation. Additional blood samples at various intervals may have detected parasitemia had they been collected. The patient had no risk factor for Chagas disease, however, other than blood transfusions, and further samples were not obtained. We concluded that the donor's parasitemia must have been intermittent or the parasite load below the level of detection by PCR at the time the subsequent sample was obtained, because no risk factor for Chagas disease other than this transfusion was ever determined in the patient.

There is currently no licensed screening test for Chagas disease for blood donations in the United States. Current screening methods for the disease rely on the donor registration interview which asks whether a prospective donor has ever had Chagas disease. Unlike travel

or residence in malarial-endemic areas, which may impose a set deferral interval of 1 to 3 years, a previous diagnosis of Chagas disease incurs an indefinite, that is, permanent, deferral period. Currently, the only registration questions regarding previous residence or travel that impose an indefinite deferral are related to new variant Creutzfeldt-Jakob disease and HIV Group O. Blood screening manufacturers are working on implementing a screening test for Chagas disease based on an antibody assay, and the confirmatory test will likely be another antibody test like radioimmune precipitation assay, rather than a PCR-based test, which may not have the sensitivity required (L. Kirchhoff, Unviersity of Iowa, Iowa City, IA, personal communication). This may delay its launch, because it should be licensed simultaneously to prevent the unnecessary deferral of healthy blood donors who may have a false-reactive screening test.

There have been six previously reported cases of transfusion-acquired Chagas disease in the United States and Canada, all of whom, including our patient, were immunocompromised.[12-16] The majority of these patients suffered a fulminant course of acute Chagas disease. Only one other reported case in addition to ours had an essentially asymptomatic course.[16]

At this time, the recipient of the RBCs from the same blood donation may be in the indeterminate phase of Chagas disease. In the vast majority of patients who have Chagas disease with acute symptoms, the illness resolves in 4 to 8 weeks without treatment, and then they enter the indeterminate phase of infection that is characterized by a lack of symptoms, undetectable parasitemias, but usually detectable antibodies.[17] In this patient, no acute symptoms were seen so detection of parasites by a blood smear would not be expected. Unfortunately, samples for further testing were recommended but not obtained due to the patient not appearing for appointments.

In a recent Chagas disease study in Guadalajara, nine recipients of blood from radioimmune precipitation assay–positive donors provided blood samples for follow-up testing, and four of the nine were radioimmune precipitation assay–positive. Of the four, two had received whole blood, and two had received PLT transfusions.[18] If this recipient of the RBCs has indeterminate Chagas disease, it will be the first incidence of RBCs-associated Chagas disease in North America. Renewed efforts to obtain a sample from this recipient for further study are under way. Although there is a lack of consensus in whether to treat indeterminate and chronic phase, asymptomatic Chagas disease patients, in this patient, it would be helpful given his history of cardiovascular disease, to know whether he has Chagas disease at minimum for the purpose of following his cardiac status.

Had our pediatric patient not been neutropenic, a buffy-coat smear would not have been performed and *T. cruzi* may have been unrecognized. She never exhibited symptoms of Chagas disease. By the same token, other cases of transfusion-acquired *T. cruzi* likely occur in immunocompetent hosts and are not diagnosed due to the mildness and even asymptomatic nature of some acute Chagas disease.

In five of the six previously reported cases of transfusion-acquired *T. cruzi*, the donor was from a country where *T. cruzi* is endemic. This is also the pattern in our case. All of the donors were asymptomatic at the time of blood donation and none of the donors knew they had Chagas disease. In five of the six previous cases, a PLT transfusion was implicated which was also the source of infection in our patient.[12-16] PLT concentrates are prepared under centrifugation conditions that concentrate *T. cruzi* and are also stored under conditions that favor parasite survival (20-24°C; shelf life of 5 days, in enriched plasma). Also, although the component in our case was irradiated with 2500 cGy and leukoreduced (WBC count, $<8.3 \times 10^5$) before storage, transmission still occurred. Therefore, standard-dose irradiation of blood components and prestorage leukoreduction do not eliminate *T. cruzi*.

In one of the six previous cases, neither a specific donor nor a specific unit of blood or blood component was ever found to be the source of the Chagas disease infection.[13] In that case, an immunosuppressed 59-year-old woman with metastatic cancer developed acute and fatal Chagas disease. She had received numerous transfusions but an infected donor was never identified. In that case, her infection was assumed to be acquired from transfusion of a contaminated blood product. Our case would have followed a similar course of assuming that the patient acquired Chagas disease from a blood transfusion without identifying a donor, if staff had not been sent to obtain samples from the implicated donor's home. Immunocompromised patients may receive many transfusions, and the investigation of these cases is dependent on the willingness of donors to return for testing or the ability and resources of blood centers to go to the donors' homes or workplaces to obtain follow-up samples.

In conclusion, our patient is the youngest reported patient in a case of transfusion-acquired *T. cruzi* infection to date in the United States. Although this is only the seventh reported case of transfusion-acquired Chagas disease in the United States and Canada, the low numbers of reported cases may be due to the immunocompetent status of the majority of transfusion recipients. There may be many unreported cases of Chagas disease in immunocompetent individuals who are asymptomatic. As mentioned previously, the incidence of seropositive donors may vary region to region, but the number of potentially infectious components annually reaching the blood supply nationwide may be in the thousands. This case lends support to the view that Chagas disease must be recognized as a threat to the US blood supply. Neither leukoreduction nor irradiation at 2500 cGy prevent trans-

mission of Chagas disease by blood transfusion. A licensed assay is needed to aid in preventing the potential spread of *T. cruzi* to both immunocompromised as well as immunocompetent transfusion recipients.

*Subsequent to this manuscript submission, the FDA licensed an antibody test for *T. cruzi* on December 13, 2006. Confirmatory testing has not been licensed.

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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	機構処理欄
				2006. 11. 20	該当なし	

一般的名称	(製造承認書に記載なし)	研究報告の公表状況		公表国
販売名(企業名)	合成血「日赤」(日本赤十字社) 照射合成血「日赤」(日本赤十字社) 合成血-LR「日赤」(日本赤十字社) 照射合成血-LR「日赤」(日本赤十字社)		A. Armengaud, F. Legros, I. Quatresous, H. Barre, P. Valayer, Y. Fanton , E. D'Ortenzio, F. Schaffner. Eurosurveillance. 2006 Nov 16.	フランス

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○フランス国内感染三日熱マラリアの1症例、コルシカ、2006年8月
2006年8月にコルシカ島で診断された三日熱マラリアの1症例である。患者Yはフランス南東部出身の59歳の男性で、2006年の初夏～盛夏にかけて南コルシカのポルトに滞在していた。8月初めに発熱と消化器症状を発症、数日後に入院した。血小板減少症(血小板数40,000/㎣)からマラリアが疑われ、急性三日熱マラリアの治療を受けて回復した。患者はマラリア流行地域への渡航歴はなく、少なくとも10年間空港内に入ったことはなかった。本土からコルシカへは船で移動し、ポルト地区周辺を旅行した。ポルト地区で最近報告されたマラリア症例があるかを調査した。患者Xはマダガスカルの南東部と南西部に滞在後6月末にフランスに到着した。その後コルシカに渡り、ポルトに2週間、7月初めまで滞在した。コルシカに渡って1週間後に発熱、さらに1週間後に入院し、三日熱マラリアと診断された。治療を受けて完治し、ポルトに戻って8月1日まで滞在した。
どちらの症例も、フランス国立マラリア調査センターでP. vivax感染が確認された。後方視的疫学調査では、6月1日～9月4日の間に上記の2例の他は輸入症例のみ4例が検出され、原虫の循環を示唆するような三日熱マラリア症例は発見されなかった。マダガスカルは熱帯熱マラリアと三日熱マラリアの流行地域である。この2症例は、コルシカのハマダラカによってP. vivaxの国内伝播が起こったことを示唆している。ポルト地区で7月27日に蚊の繁殖場所が発見され、この仮説が裏付けられた。
本症例は、この地域で報告されたマラリアの地域内伝播の1972年以来初の症例であり、蚊の根絶と媒介動物対策を最大限の注意を払って行うべきであることを示している。コルシカの住民および旅行者にマラリア予防策を指示する根拠とはならない。系統だった旅行医学的アドバイスが非常に重要である。

報告企業の意見	今後の対応
フランス、コルシカ島でマラリア流行地域への渡航歴のない患者が三日熱マラリアを発症し、マダガスカル渡航歴のある同じ地区の患者からの地域内伝播が疑われたとの報告である。	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供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

合成血「日赤」
照射合成血「日赤」
合成血-LR「日赤」
照射合成血-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

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A case of *Plasmodium vivax* malaria was diagnosed in Corsica in August 2006. This is the first case of autochthonous transmission of malaria to be reported in the region since 1972 [1]. Malaria is a notifiable disease in mainland France (including the Mediterranean island of Corsica). Rare cases of malaria have been observed after bites from infected mosquitoes that had been imported from endemic areas into airports and sea ports or by transmission via contaminated blood transfusion or tissue grafts [2, 3, 4]. Corsica was endemic for malaria before 1953 and from 1965 to 1971. Because the *Anopheles* mosquitoes in Corsica may still have vectorial competencies for *P. vivax* [6], antivectorial measures (eradication of mosquitoes) are still taken. A small number of imported cases from endemic zones are reported in Corsica each year [1].

Epidemiological and entomological investigations were carried out into the case in August 2006. The patient, Patient Y, was a 59 year old man from southeast France who had stayed in Porto, a département of South Corsica, from early to mid summer 2006. At the beginning of August, he developed a fever and gastrointestinal symptoms, and after several days of illness, he was admitted to hospital. Malaria was suspected because of thrombocytopaenia (40 000 platelets/mm3). The diagnosis was made three days after admission and the patient was treated for acute *P. vivax* malaria and recovered. The patient had never travelled in endemic malaria areas and had not been inside any airport for at least 10 years. He had travelled to Corsica by ship from mainland France and had then travelled overland to the Porto area.

Possible link to earlier case of imported malaria?
Further epidemiological investigations found that another recent case of *P. vivax* infection had been reported in the Porto area. Patient X, who had previously visited southeast and southwest Madagascar, arrived in France at the end of June. He then travelled to Corsica, and stayed in Porto for two weeks at the beginning of July. After a week in Corsica, he developed a fever and was admitted to hospital a week later, where the diagnosis of *P. vivax* malaria was made. He was treated and recovered fully, then returned to Porto where he stayed until 1 August. Both cases of *P. vivax* infection were confirmed by the French national reference centre for malaria.

A retrospective epidemiological investigation undertaken from 1 June to 4 September 2006 by medical laboratories in Corsica, did not find any other *P. vivax* malaria cases that would suggest further circulation of the parasite in Corsica. So far, excluding the two *P. vivax* cases in Porto, only four imported malaria cases (one *P. ovale* and three *P. falciparum*) were detected in Corsica during the summer of 2006. This is within the expected range based on available data from previous years. From 4 to 20 September 2006, prospective active surveillance found no other autochthonous malaria cases.

Discussion
Madagascar is known to be an endemic country for *P. falciparum* and *P. vivax* [7]. The details of the imported case from Madagascar with onset in July, and of the case with onset in August, caused by the same species (*P. vivax*) and in the same place (Porto), in a patient with no travel to endemic areas suggests autochthonous transmission by local *Anopheles* mosquitoes [8,9].

This hypothesis was supported by the results of the initial entomological investigations in the Porto area, which found a mosquito breeding ground albeit with low numbers of *Anopheles* mosquitoes, in a fountain on 27 July 2006. The breeding ground was treated the same day using larvicide to eradicate the mosquito colony. An entomological investigation around the home of the Patient Y in southeast France at the beginning of September, did not find any *Anopheles* mosquitoes, but could not completely rule out the presence of this mosquito in May and June.

Conclusion
This autochthonous *P. vivax* malaria transmission on Corsica in August 2006, probably via the bite of a local *Anopheles* mosquito infected with *P. vivax* from a patient who had acquired infection in Madagascar, appears to have been unique because studies did not find other autochthonous malaria cases between 1 June and 20 September 2006.