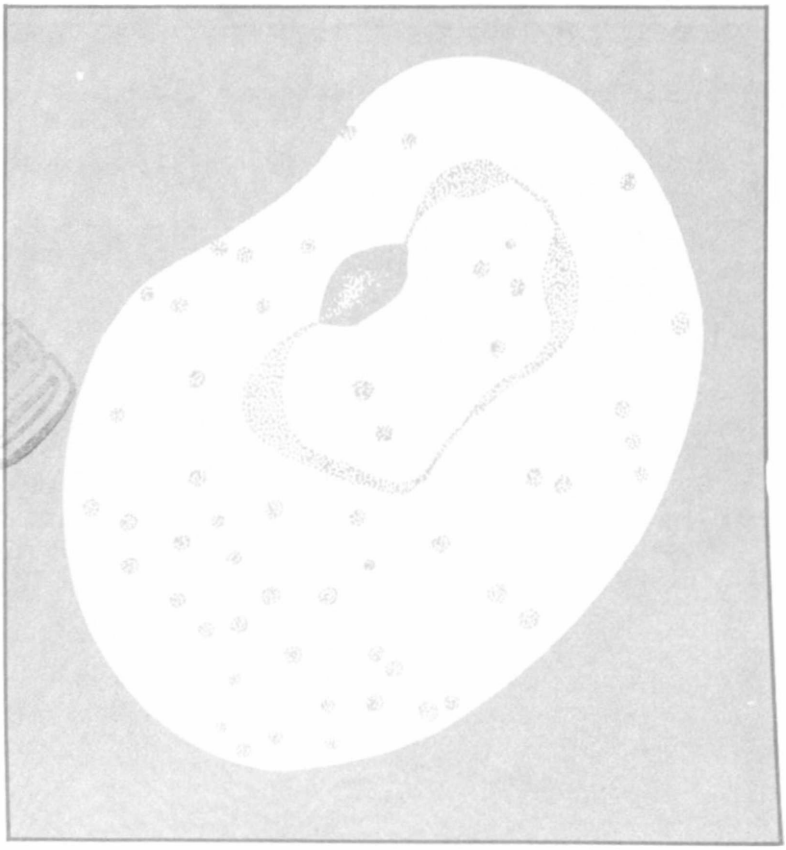
ANNUAL SUMMARY 1983

Issued October 1984

CENTERS FOR DISEASE CONTROL

MALARIA

SURVEILLANCE



U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES • Public Health Service

PREFACE

This report summarizes information received from state health departments, medical departments of the Armed Forces, and other sources. It is intended primarily for the use of those with responsibility for disease control activities. Anyone desiring to quote this report should contact the original investigator for confirmation and interpretation.

Contributions to the Surveillance Report are most welcome. Please address them to:

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SUGGGESTED CITATION

Centers for Disease Control: Malaria Surveillance Annual Summary 1983
Issued October 1984

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I. SUMMARY

During 1983, 803 cases of malaria diagnosed in the United States were reported to the Centers for Disease Control. This compares with 930 cases reported in 1982, a decline of 13.7%. A total of 468 cases with onset in 1983 were reported in foreigners compared with 574 in 1982, a 18.5% decrease.

Plasmodium vivax was the parasite identified in 56% of the 803 cases, and *P. falciparum* was identified in 30% of the cases. *P. malariae* and *P. ovale* were reported in 5% and 3% of cases, respectively. The species was not determined in the remaining cases.

Only 8 of the 803 cases involved persons who acquired their infection in the United States. Congenital transmission occurred in 2 cases, and infection through blood transfusion occurred in 5 cases. In 1 isolated case in Louisiana, the route of infection could not be determined.

Three deaths attributed to malaria were reported for 1983, compared with 2 deaths from malaria in 1982.

II. TERMINOLOGY

The terminology used in this report is derived from the recommendations of the World Health Organization (WHO)(1). The definitions of the following terms are included for reference purposes.

A. Autochthonous

1. Indigenous--malaria acquired by mosquito transmission in an area where malaria is a regular occurrence.

2. Introduced--malaria acquired by mosquito transmission from an imported case in an area where malaria is not a regular occurrence.

B. Imported

Malaria acquired outside a specific area (the United States, Puerto Rico, and Guam in this report).

C. Induced

Malaria acquired through artificial means, i.e., blood transfusion, common syringes, or malariotherapy.

D. Relapsing

Renewed manifestation (of clinical symptoms and/or parasitemia) of malarial infection, separated from previous manifestations of the same infection by an interval greater than any interval due to the normal periodicity of the paroxysms.

E. Cryptic

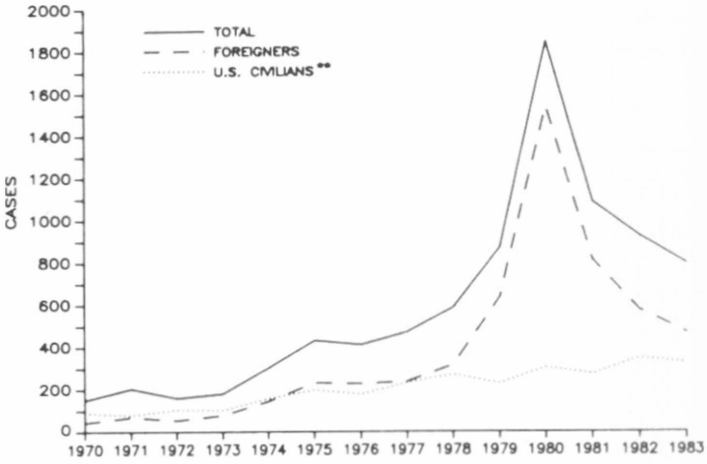
An isolated case of malaria not associated with secondary cases as determined by appropriate epidemiologic investigation.

III. GENERAL SURVEILLANCE

A total of 803 cases* with onset of illness in 1983 in the United States were reported to the Division of Parasitic Diseases, Center for Infectious Diseases, Centers for Disease Control (CDC); this represents a 14% decline compared with the 930 cases reported for 1982. Only 10 of the cases occurred in U.S. military personnel. Civilian cases have accounted for the majority of cases each year since 1973 (Table 1).

Malaria in foreign civilians accounted for 468 (58.3%) of all reported cases (Table 1), representing a 19% decline in the number of such cases in this group compared with 1982. The number of malaria cases in U.S. civilians decreased by 7% from 1982 (Figure 1).

FIG. 1 CASES OF MALARIA IN U.S. CIVILIANS AND FOREIGNERS, UNITED STATES, 1970–1983*



*INCLUDES PUERTO RICO, THE VIRGIN ISLANDS, AND GUAM
**INCLUDES 8 CASES ACQUIRED IN THE UNITED STATES

Table 1 All Primary Malaria Cases in Civilians and Military Personnel with Onset of Illness in the United States, 1966-1983*

Year	Military	U.S. Civilians	Foreign Civilians	Unknown	Total
1966	621	89	32	22	764
1967	2,699	92	51	15	2,857
1968	2,567	82	49	0	2,698
1969	3,914	90	47	11	4,062
1970	4,096	90	44	17	4,247
1971	2,975	79	69	57	3,180
1972	454	106	54	0	614
1973	41	103	78	0	222
1974	21	158	144	0	323
1975	17	199	232	0	448
1976	5	178	227	5	415
1977	11	233	237	0	481
1978	31	270	315	0	616
1979	11	229	634	3	877
1980	26	303	1,534	1	1,864
1981	21	273	809	0	1,103
1982	8	348	574	0	930
1983	10	325**	468	0	803

*includes Puerto Rico, the Virgin Islands, and Guam.
**includes 8 cases acquired in the United States.

*A "case" is defined as: 1) an individual's first attack of malaria in the United States, regardless of whether or not he/she had experienced previous attacks of malaria while outside the country, and 2) the presence of a positive peripheral blood smear examined in the local or state health department laboratory. Blood smears from doubtful cases were referred to the National Malaria Repository, CDC, for confirmation of the diagnosis. A subsequent attack in the same person caused by a different *Plasmodium* species is counted as an additional case. A repeated attack in the same person in this country caused by the same species is not considered to be an additional case.

The number of malaria cases in U.S. civilians declined from 348 cases in 1982 to 325 cases in 1983, a 6.6% decrease. Malaria in foreign civilians declined from 574 reported cases in 1982 to 468 cases in 1983, a 18.5% decrease.

Only 8 of the 803 cases involved persons who acquired their infection in the United States. Congenital transmission occurred in 2 cases, and infection through blood transfusion occurred in 5 cases. In 1 instance, the route of infection could not be determined.

The Plasmodium species could be determined in 748 of the 803 cases (93.2%). The proportion of cases caused by Plasmodium falciparum increased 9% compared with 1982. In 1983, P. vivax was identified in 56% and P. falciparum in 30% of the infected individuals (Table 2).

Table 2 All Malaria Cases by Plasmodium Species, United States, 1982 & 1983

Species	1982 Total	1982 Percent	1983 Total	1983 Percent
P. vivax	613	65.9	451	56.1
P. falciparum	189	20.3	237	29.5
P. malariae	43	4.6	36	4.5
P. ovale	25	2.7	20	2.5
Mixed	5	0.5	4	0.5
Undetermined	55	5.9	55	6.8
TOTAL	930	100.0	803	100.0

The countries of origin of the 803 cases are listed in Table 3.

The geographic distribution of the malaria cases within the United States is shown in Figure 2 by the State in which the patient first developed clinical symptoms of malaria.

FIG. 2 GEOGRAPHIC DISTRIBUTION OF MALARIA CASES WITH ONSET IN THE UNITED STATES, 1983

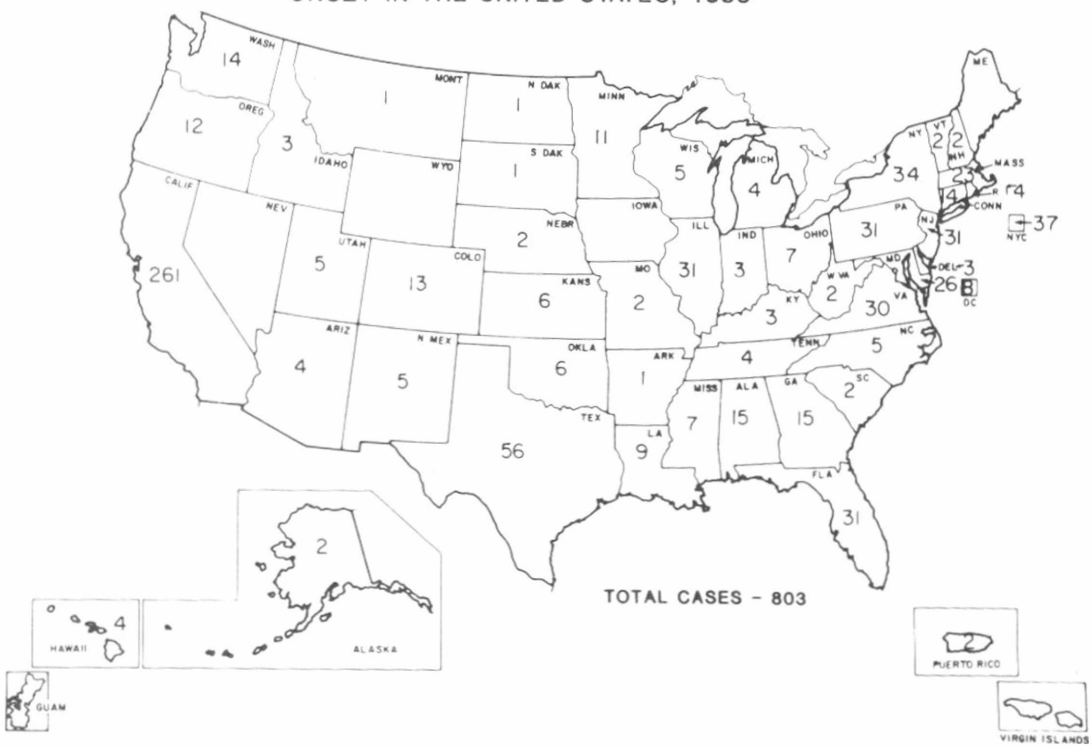


Table 3. Malaria Cases by Distribution of *Plasmodium* Species and Area of Acquisition, United States, 1983*

Area of Acquisition	*vivax*	*falciparum*	*malariae*	*ovale*	mixed	unknown	Total
AFRICA	54	167	14	17	0	20	272
Africa, East**	2	6	3	2	0	1	14
Africa, Central**	0	2	1	0	0	0	3
Africa, West**	1	5	1	1	0	0	8
Africa, South**	0	1	0	0	0	0	1
Africa, Unspecified**	9	11	1	1	0	3	25
Angola	0	3	0	0	0	0	3
Cameroon	1	1	2	2	0	0	6
Ethiopia	4	0	0	0	0	2	6
Gabon	0	1	0	0	0	0	1
Ghana	2	12	2	2	0	2	20
Guinea	0	1	0	1	0	1	3
Kenya	4	41	1	5	0	2	53
Liberia	4	5	0	1	0	1	11
Malagasy Republic	0	1	0	0	0	0	1
Malawi	1	1	0	0	0	0	2
Nigeria	6	31	2	1	0	5	45
Rwanda	0	1	0	0	0	0	1
Sierra Leone	0	7	0	0	0	2	9
Somalia	4	1	1	0	0	0	6
South Africa	0	1	0	0	0	0	1
Sudan	9	9	0	0	0	0	18
Tanzania	3	9	0	1	0	1	14
Uganda	1	0	0	0	0	0	1
Zaire	2	11	0	0	0	0	13
Zambia	1	6	0	0	0	0	7
ASIA	229	38	15	1	2	26	311
Asia, South East**	25	5	3	0	1	4	38
Pacific**	2	0	0	0	0	1	3
Middle East**	1	1	0	0	0	0	2
Afghanistan	3	1	0	0	0	1	5
India	159	21	9	1	0	16	206
Indonesia	8	2	0	0	0	0	10
Iran	2	0	0	0	0	0	2
Kampuchea	1	0	0	0	0	0	1
Pakistan	16	1	1	0	0	3	21
Philippines	4	3	1	0	0	0	8
Thailand	2	1	0	0	0	0	3
Vietnam	6	3	1	0	1	1	12
CENTRAL AMERICA AND CARIBBEAN	114	25	3	0	1	5	148
Caribbean, Unspec.**	1	0	0	0	0	0	1
Central Amer. Unspec.**	9	1	0	0	1	0	11
Belize	1	1	0	0	0	1	3
Dominican Republic	1	0	0	0	0	0	1
El Salvador	66	0	1	0	0	1	68
Guatemala	21	0	2	0	0	2	25
Haiti	0	14	0	0	0	0	14
Honduras	12	9	0	0	0	1	22
Nicaragua	1	0	0	0	0	0	1
Panama	2	0	0	0	0	0	2
NORTH AMERICA	33	2	2	1	0	2	40
Mexico	30	0	0	0	0	2	32
United States	3	2	2	1	0	0	8
SOUTH AMERICA	9	4	1	1	1	1	17
South America, Unspec.**	1	0	0	0	0	0	1
Brazil	3	1	0	0	1	0	5
Colombia	4	3	1	0	0	1	9
Peru	1	0	0	0	0	0	1
Venezuela	0	0	0	1	0	0	1
OCEANIA	11	1	1	0	0	1	14
Pacific, South**	3	0	0	0	0	0	3
New Guinea	6	1	1	0	0	1	9
Solomon Islands	2	0	0	0	0	0	2
UNKNOWN	1	0	0	0	0	0	1
TOTAL	451	237	36	20	4	55	803

*Includes Puerto Rico, Virgin Islands and Guam

**Country Unspecified

The interval between the date of arrival in the United States and the date of onset of illness was known for 602 of the imported cases for which the infecting Plasmodium species was also identified. Clinical malaria developed within 1 month after arrival in 75.9% of the patients with P. falciparum malaria and in 29.9% of the patients with P. vivax infections (Table 4). Only 25 (4.2%) of the 602 patients became ill 1 year or more after their arrival in the United States.

Table 4 Imported Malaria Cases by Interval between Date of Entry and Onset of Illness and by Plasmodium Species, United States, 1983

Interval (in months)	PLASMODIUM SPECIES									
	vivax	(%)	falciparum	(%)	malariae	(%)	ovale	(%)	Total	(%)
< 1	106	(29.9)	157	(75.9)	8	(29.7)	3	(21.4)	274	(45.5)
1-2	104	(29.4)	32	(15.5)	6	(22.2)	4	(28.7)	146	(24.2)
3-5	62	(17.5)	6	(2.9)	6	(22.2)	3	(21.4)	77	(12.8)
6-11	64	(18.1)	9	(4.3)	4	(14.8)	3	(21.4)	80	(13.3)
≥12	18	(5.1)	3	(1.4)	3	(11.1)	1	(7.1)	25	(4.2)
TOTAL	354	(100.0)	207	(100.0)	27	(100.0)	14	(100.0)	602	(100.0)

Three fatal cases due to malaria were reported in 1983 compared with 2 such cases in 1982. These cases are discussed in Section VII.

IV. MALARIA IN MILITARY PERSONNEL

Ten cases of malaria were reported in U.S. military personnel in 1983. The Army accounted for 6 cases, the Navy for 1, the Marine Corps for 2 cases, and for 1 case the Branch of Service is not known.

V. IMPORTED MALARIA IN CIVILIANS

Malaria in U.S. citizens accounted for 317 (40.4%) of the 785 imported cases in civilians, whereas 468 (39.5%) of the cases occurred in citizens of other countries (Table 5). Of the 317 imported cases in U.S. civilians, 164 (51.7%) were acquired in Africa and 78 (25%) were acquired in Asia.

Imported malaria in U.S. civilians who had been infected in Africa increased from 102 cases in 1981, to 129 cases in 1982, and 164 cases in 1983. Of the 164 infections imported from Africa, 57 (35%) were acquired in Kenya and Tanzania. Imported cases from these 2 countries increased from 26 cases in 1981 to 57 cases in 1983.

Table 5 Imported Malaria Cases in Civilians, by Area of Infection, United States, 1983

Area of Acquisition	United States		Foreigners		Total	
	Cases	Percent	Cases	Percent	Cases	Percent
Africa	164	51.7	107	22.9	271	34.5
Asia	78	24.6	230	49.1	308	39.2
Central America	29	9.1	100	21.4	129	16.4
Caribbean	12	3.8	4	0.9	16	2.0
Mexico	12	3.8	18	3.8	30	3.8
South America	10	3.2	7	1.5	17	2.2
Oceania	12	3.8	1	0.2	13	1.7
Unknown	0	0.0	0	0.0	0	0.0
TOTAL	317	100.0	468	100.0	785	100.0

Asia was the area of acquisition of infection in 230 (49%) of the 468 cases in foreign civilians. Infections acquired in India accounted for 150 of the malaria infections in foreign civilians during 1983 as compared with 172 cases in 1982. The 18% decline from 1982 of imported malaria cases in foreign civilians is due to further decline of malaria cases among refugees from South-East Asia (from 136 cases in 1982 to 60 cases in 1983) and India (from 172 cases in 1982 to 150 cases in 1983).

The principal changes in several categories of imported cases in U.S. civilians from the corresponding 1982 data are shown in Table 6. The major increases were observed among tourists (reported cases up 77%) and business representatives (up 83%).

Table 6 Imported Malaria Cases in U.S. Civilians, by Category, United States, 1982-1983

Category	1982		1983	
	Cases	(percent)	Cases	(percent)
Tourist	79	(23.9)	140	(44.3)
Business Representative	23	(6.9)	42	(13.2)
Government Employee	9	(2.7)	6	(1.9)
Missionary	32	(9.7)	34	(10.7)
Peace Corps	12	(3.6)	9	(2.8)
Seamen/Aircrew	4	(1.2)	3	(0.9)
Teacher/Student	32	(9.7)	15	(4.7)
Other	67	(20.2)	25	(7.9)
Unknown	73	(16.2)	43	(13.6)
TOTAL	331	(100.0)	317	(100.0)

VI. MALARIA ACQUIRED IN THE UNITED STATES

Two cases of congenital malaria, 5 transfusion (induced) malaria cases, and 1 cryptic case of malaria with onset of illness in 1983 were reported in the United States.

A. Congenital Malaria

Two cases of congenital malaria with onset of illness in the United States during 1983 were reported; both were caused by *P. vivax*. Both mothers were natives of Central America.

Case 1--A 22-day-old boy in California developed fever, diarrhea, and vomiting. *P. vivax* parasites were identified in a peripheral blood smear. The infant was treated with chloroquine and had an uneventful recovery. The mother had arrived in the United States from El Salvador in September 1982. She had experienced an episode of chills and fever 3 weeks before the delivery. Following the diagnosis of malaria in her son, *P. vivax* parasites were found in a peripheral blood smear. She was treated with primaquine.

(Reported by the Los Angeles County/USC Medical Center, Los Angeles, M. Tormey, M.P.H., Los Angeles County Department of Health Services, Los Angeles, and R. Murray, Dr.P.H., California Department of Health Services.)

Case 2--On April 21, 1983, a male newborn in Los Angeles, California, developed dyspnea and seizures. *P. vivax* parasites were identified in a peripheral blood smear. The newborn was treated with chloroquine and primaquine and had an uneventful recovery. The mother, a native from Guatemala, had arrived in the United States on May 25, 1982, and had experienced an episode of fever and chills on January 25, 1983, at 7 months of pregnancy; the illness was first diagnosed as influenza. The blood smears of the mother contained *P. vivax* parasites on April 27, 1983. She was treated with chloroquine and primaquine.

(Reported by the Los Angeles County Medical Center, Los Angeles, M. Tormey, M.P.H., Los Angeles County Department of Health Services, Los Angeles, and R. Murray, Dr.P.H., California Department of Health Services.)

B. Induced Malaria

Case 1--On March 8, 1983, a 42-year-old woman who resided in California developed chills and fever and was hospitalized on March 22, 1983. Peripheral blood smears showed *P. ovale* parasites. She was treated with chloroquine and primaquine. The patient had not been abroad but had received 7 units of packed red cells at the end of February for gastrointestinal bleedings. Serum specimens were obtained from 4 of the donors for serologic testing. One donor had indirect immunofluorescence (IIF) test titers of 1:4096 to *P. falciparum*, *P. malariae* and *P. ovale*, and of 1:64 to *P. vivax*. This donor was a 33-year-old engineer who had lived in Liberia. While in Liberia he had experienced an episode compatible with malaria, for which he had been treated. He came to the United States about 4 years prior to the blood donation.

(Reported by M. Ginsberg, M.D., S. Lojowski, P.H.N., San Diego County Health Department, D. Mc Pherson, R.N., B.S.N, University Hospital, San Diego, and R. Murray, Dr.P.H., California State Department of Health Services.)

Case 2--In October 1983, a 72-year-old man in Alabama underwent open-heart surgery. Fever and chills developed 14 days after the operation. P. falciparum parasites were identified on peripheral blood smears. The patient was treated with chloroquine and had an uneventful recovery.

The patient had travelled to Israel, Egypt, Turkey, and Greece in July, 1983, 3 months before his surgery. He had not taken malaria chemoprophylaxis. He first experienced fever and chills 14 days after his operation in October.

The patient had received 1 unit of whole blood and 1 unit of packed red cells on the day of his surgery, and 2 units of packed red cells 5 days after surgery. Because the interval between the transfusions and onset of symptoms is consistent with the incubation period of P. falciparum (8-17 days), the patient was considered to have been infected with malaria parasites as a result of the transfusions rather than as a result of his travel to Turkey or Egypt. The 4 donors were contacted. Three donors had never traveled outside the United States. The fourth donor was a 26-year-old student from Cameroon, who had come to the United States in September 1982. Thick and thin blood smears on all 4 donors were negative for parasites. Serum specimens from these donors were screened for the presence of antibodies to malaria. Only the serum from the African student was positive, with an IIF titer against P. falciparum of 1:65,536, P. vivax of $\leq$ 1:16, P. malariae of 1:1,024, and P. ovale of 1:4,096. The patient had received a unit of whole blood from this donor 14 days before the onset of symptoms. A repeat thick and thin blood smear from this donor in November was negative for parasites. The donor was contacted and chloroquine was prescribed.

(Reported by Joe Shaw, Jefferson County Department of Health, Karen M. Landers, M.D., Northwest Alabama Regional Health Department, Eoline I. McGowan, Red Cross Alabama Region Blood Service, Birmingham, Michael T. Reymann, M.D., Brookwood Medical Center, Birmingham, and the Alabama Department of Health.)

Case 3--A 37-year-old California woman received 10 units of blood folllowing an ileostomy on November 1, 1983. Six weeks later the patient developed chills and fever, and P. malariae parasites were identified in a peripheral blood smear. She was treated with chloroquine and had an uneventful recovery. Two of the 10 donors could be interviewed, and blood specimens were obtained for laboratory examination. One of these was a 34-year-old student from a central African country who had arrived in the United States in 1976. His IIF titer was $\geq$1:4096 to P. malariae, 1:1024 to P. falciparum, and 1:256 to P. vivax and P. ovale. He was treated with chloroquine and primaquine.

(Reported by A. Goodman, M.D., Eureka, California, Alex Taylor, M.P.H., San Bernardino County Health Department, and R. Murray, Dr.P.H., California Department of Health Services.)

Case 4--On April 4, 1983, a 63-year-old New York man developed spiking fevers and general weakness, for which he was admitted on April 15. P. malariae parasites were identified in a peripheral blood smear. The patient was treated with chloroquine and quinine and had an uneventful recovery. The patient had not traveled abroad and had no history of drug use. On March 1, 1983, he had received 2 units of packed red cells while undergoing total hip replacement. The 2 donors were identified and blood smears and sera were obtained. The blood films were negative. One donor, a 35-year-old man, had not traveled outside of the United States, and he had no history of malaria or unexplained febrile episodes. His serum did not contain demonstrable antibodies to malaria with the IIF test. The other donor was a 59-year-old woman who had come to the United States from Greece 10 years ago. She had no history of malaria or episodes of unexplained fever, but her serum was positive with an IIF titer against P. malariae of $\geq$1:4096, P. ovale of 1:1024, P. vivax of 1:64 and P. falciparum of 1:64. This donor had donated blood twice before but no malaria had developed in the recipients.

(Reported by Robert Reiff, M.D., Director, Hudson Valley Blood Bank, Valhalla, New York, and Rachel Stricoff, M.P.H., Epidemiologist, Bureau of Communicable Disease Control, New York State Department of Health, Albany, New York.)

Case 5--On August 28, 1983, a 70-year-old man who resided in Houston, Texas, began to experience chills and fever. P. vivax parasites were identified on a peripheral blood smear. He was treated with chloroquine and primaquine and had an uneventful recovery. The patient had no history of foreign travel but had received a transfusion with platelets on August 10, 1983.

The donor of the platelets was a 26-year-old male citizen of Mexico. He had an episode of fever on August 9, 1983. P. vivax parasites were found in a blood smear. He was treated with chloroquine and primaquine. The date of the blood donation and the date of his entry into the United States could not be ascertained.

(Reported by Jeffery Taylor, Bureau of Epidemiology, Texas State Health Department, Austin, Texas.)

C. Cryptic Malaria

A 48-year-old salesman in Denham Springs, Louisiana, developed chills and fever on April 13, 1983, for which he was admitted 6 days later to a local hospital. His temperature was 103° F and P. falciparum parasites were identified in his peripheral blood. The patient was treated with chloroquine and Bactrim** and recovered. The patient had not traveled outside the United States, had no history of unexplained fever or chills,

**Use of trade names is for identification only and does not imply endorsement by the Public Health Service or by the Department of Health and Human Services.

malaria, malaria therapy, and denied intravenous (i.v.) drug use. On April 1, 2 weeks prior to onset of his malaria attack, the patient had undergone cardiac bypass surgery in a Baton Rouge hospital. He had received 1 unit of whole blood on April 1, 1 unit of whole blood on April 2 and 2 units of packed red blood cells on April 3. He was discharged on April 8. The donors of the 4 units denied a history of malaria, malaria therapy, i.v. drug use or unexplained episodes of fever. One donor had lived in the Philippines from 1953 until 1960, and 1 donor had been stationed in the Mediterranean from 1965 to 1969. No malaria parasites could be found in blood smears from the 4 donors. Sera of 3 donors were negative for all Plasmodium species. Serum of 1 donor, who had never been out of the United States, was also negative against P. falciparum, but IIF titers of 1:64 were measured against P. vivax, P. ovale, and P. malariae. Thus, none of the donors could be incriminated.

The patient had not received any transfusions of blood or blood products other than those described. He lived in a rural area near Baton Rouge. No foreigners live near his home, and no residents are known to have traveled abroad. No cases of malaria had been seen in the local hospital. Hospital blood bank procedures, blood bank issue records for March 31 through April 3, and charts of all the patients who were in the operating room (OR) or the critical care unit (CCU) on those dates were reviewed; this was done to assess the possibility that the patient had inadvertently received a unit of blood intended for another patient. Each unit of blood or blood products released from the hospital blood bank was identified and traced by time of release from the blood bank, time of administration, blood type of the donor, blood type, and location of the patient. The donor of each unit was identified and his/her blood donor card was examined. The transfusion pack tags or sera of these donors were tested serologically. All test results were negative. The Baton Rouge Blood Center donor cards were examined of all donors who had donated blood on the 3 days that the 4 donors of the units received by the patient had given blood; donors who had given a history of foreign travel were identified, and their sera were tested with negative results. The blood collection, labeling, storage, and transportation procedures at the blood center were reviewed.

All units of blood issued for for patients in the OR or the CCU on March 31 through April 3 could be traced from donor to recipients, and only 1 donor was found to have a positive titer to P. falciparum. However, this unit, type B+, was given to a patient on the day before the 48-year-old salesman entered the hospital, and the salesman, who has blood type A+, did not have a transfusion reaction. Switching of a blood unit at the blood center also appeared unlikely. Furthermore, extensive search of the blood center's donor cards failed to identify a likely donor for blood with malaria parasites.

It is extremely unlikely that the patient was infected by a mosquito. The incubation period of P. falciparum ranges from 8 to 17 days. The patient had become ill on April 10, and if he had been infected by a mosquito bite, such inoculation probably would have occurred between March 21 and April 2. He was hospitalized during that time.

(Reported by Joseph L. Mass, M.D., Denham Springs, Louisiana, Louise McFarland, Dr.P.H., and Charles T. Caraway, D.V.M., Louisiana State Department of Health and Human Resources.)

Editorial comment: It is unlikely that the donor with the borderline IIF titers against the 3 heterologous Plasmodium species had a parasitemia at the time of blood donation in view of the negative P. falciparum IIF titer. It also appears very unlikely that the patient received a unit of blood intended for another patient. However, an unrecorded switch of a blood unit, either at the hospital or at the blood center would not be detectable, and this possibility therefore cannot be excluded. The route of infection could not be documented, and this case of malaria was classified as cryptic in accordance with WHO terminology.

VII. MALARIA DEATHS IN THE UNITED STATES

Three deaths in 1983 due to malaria were reported in the United States. All occurred in U.S. citizens who had acquired malaria abroad. Two had not taken chemoprophylaxis. One fatal case involved a patient who had used chloroquine and quinine while in East Africa.

Case 1--A 40-year-old businessman was admitted on June 9, 1983, to a hospital in Connecticut. On May 28, 1983, the patient had complained of fever, chills, and headaches, for which he was treated with erythromycin. The symptoms persisted until June 8, 1983, when P. falciparum parasites were identified in a peripheral blood smear. During the admission the patient became unconscious and was found to have a parasitemia of 40% infected red blood cells. Three hours after admission, a nasogastric tube was inserted to administer 650 mg of quinine, 600 mg of chloroquine, 50 mg of pyrimethamine, and 2 g of sulfisoxasole, all of which were given again 8 hours later. Subsequent antimalarial therapy consisted of i.v. quinine every 8 hours. An exchange transfusion totaling 5 units of whole blood was conducted during the first 12 hours of hospitalization. Despite these measures the coma increased in severity, with conservation only of brain-stem reflexes, necessitating assisted ventilation; the patient developed seizures, acute renal failure, and acidosis. The parasitemia decreased during this period to 10%, with further rapid decrease during the following 24 hours. No parasites were detected in the blood smear on the third day of hospitalization. Despite intense supportive therapy, including hemodialysis, the patient remained in coma, continued to have seizures, and the renal failure and metabolic acidosis persisted. The patient died on June 12, 1983.

The patient had traveled to Sidney, Australia, and Johannesburg, South Africa, from May 6 through 20, 1983, with airport stopovers in Mauritius on May 14 and in Monrovia, Liberia, on May 20. The stopover in Monrovia was from 12:25 a.m. to 1:45 a.m. It appears probable that the patient was infected during the stopover in Liberia (P. falciparum malaria does not occur on Mauritius). He had not taken malaria chemoprophylaxis. It is of interest that another U.S. traveler on the same plane also developed P. falciparum malaria after his return to the United States. Neither passenger had left the plane during the stopover.

(Reported by J. Slater, M.D., New Canaan, Connecticut, and the Connecticut State Department of Health.)

Case 2--A 70-year-old couple, residents of Florida, returned from a 1-month visit to Tanzania and Malawi on September 2, 1983. They had taken quinine and chloroquine as malaria prophylaxis. On September 7, 1983, both developed periodic episodes of fever, chills, diarrhea and myalgia. They were given nonspecific medications, and the chloroquine and quinine were discontinued. On September 16, 1983, both were admitted to the hospital after P. falciparum parasites were identified in peripheral blood smears. Their condition was considered stable, and chloroquine therapy was initiated. On the next day, September 17, 1983, the husband became comatose. He had a greater than 50% parasitemia and was promptly given quinine i.v., resulting in parasite clearance. However, he developed cardiomegaly, pulmonary edema, subcutaneous emphysema, adult respiratory distress syndrome, and renal failure and died on October 10, 1983.

The wife had a parasitemia of 5%-10%. Although she had initially also been treated with chloroquine, she was switched to a course of quinine and Fansidar. She developed an episode of congestive heart failure, cardiomegaly, and pulmonary vascular congestion which responded to treatment; she was eventually discharged from the hospital.

(Reported by Brian W. Elliott, M.D., and Michael Wood, M.D., St. Petersburg, Florida, and the Florida State Department of Health.)

Case 3--On November 11, 1983, a 29-year-old female missionary developed chills and fever while in a rural area of Colombia. The patient had been in Colombia 7 months and had not taken malaria chemoprophylaxis. The patient decided to return to her home and was admitted on November 16 to a hospital in Puerto Rico. P. falciparum parasites were identified in the peripheral blood films upon admission. She had a parasitemia of 6%-8.5%. The patient was treated on November 16 and 17 with quinidine, Septra, and i.v. fluids followed by quinine and Fansidar on November 17, 18, and 19. On November 18 no parasites were demonstrable in the peripheral blood, but the patient died on November 20, 1983.

(Reported by N. Hernandez, M.D., Rio Piedras, Puerto Rico, and the Health Department of the Commonwealth of Puerto Rico.)

VIII. PREVENTION OF MALARIA

Guidelines entitled "Prevention of Malaria in Travelers-1984" have been published in a supplement to the Morbidity and Mortality Weekly Report (MMWR). This issue provides information about countries and, where applicable, areas within each country, where malaria risk exists. In addition, areas in the world where chloroquine-resistant strains of P. falciparum are known to exist are listed.

IX. MICROSCOPIC DIAGNOSIS OF MALARIA

Early diagnosis of malaria requires a high level of clinical suspicion and, in particular, a comprehensive travel history taken from every patient with a fever of unknown origin. Once malaria is suspected, a Giemsa-stained smear of peripheral blood should be examined for the presence of parasites. Since the accuracy of diagnosis is dependent on the quality of the blood film, the following guide is offered for the proper preparation of thick and thin blood smears.

1. Manufacturers' "pre-cleaned" slides are not considered clean enough for use in malaria diagnosis. Before use, wash these slides in mild detergent, rinse them thoroughly in warm running water, then in distilled water, and dip them in ethyl alcohol (90%-95%). Then, wipe slides dry with a lintless cloth or tissue for immediate use or store them in 95% alcohol until needed.
2. Clean the patient's finger with alcohol and wipe the finger dry with a clean cloth or gauze.
3. After puncturing the finger with the blood lancet, allow a large globule of blood to form.
4. Place the cleaned surface of the slide against the drop of blood and, with a quick circular motion, make a film the size of a dime in the middle third of 1 end of the slide. Ordinary newsprint should be barely legible through such a wet drop (Figure 3). (Excessive mixing or stirring with a second slide leads to distortion of blood cells and parasites.)
5. Wipe the finger dry and gently squeeze a small drop of blood from the puncture, placing it at the edge of the middle third of the same slide (Figure 4).
6. Apply a clean "spreader" slide to the edge of the small drop at a 45° angle and allow the blood to extend about two-thirds of the slide width; then, keeping even contact, push the spreader forward along the slide. This will produce an even layer of red blood cells with a "feathering" at the lower edge (Figure 5).
7. While the thick blood film dries (minimum of 6 hours at room temperature)† , keep the film horizontal and protected from dust and insects.
8. Label the slide in the upper part of the thin film with the date and the name or initials of the patient as illustrated (Figure 5).

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†If a rapid diagnosis is desired, make the thick and thin films on separate slides. The thin film can be air dried, fixed with methyl alcohol, and stained immediately. If no parasites are found on the thin film, examine the thick film for organisms not detected on the thin preparation.

Fig. 3

in all their phases. The importance of the examination of blood films for the presence of malaria parasites will be fully understood

Fig. 4

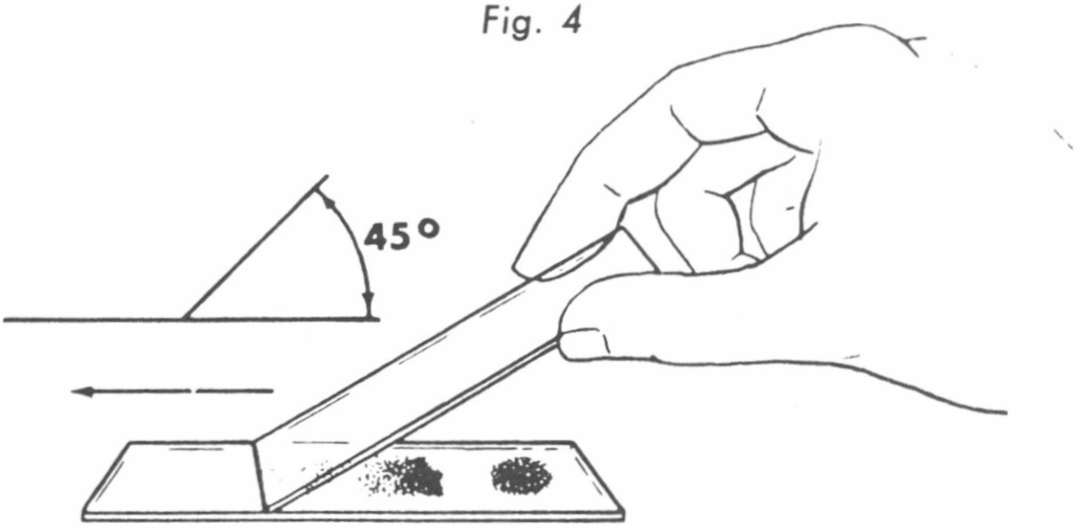
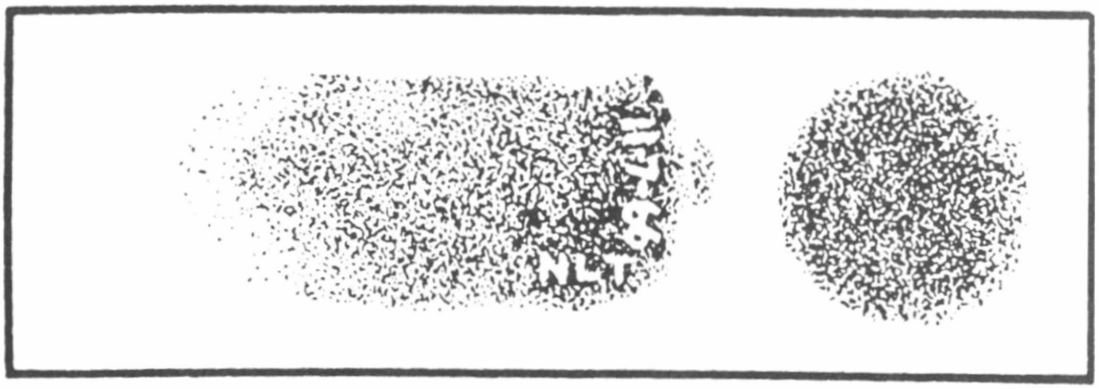


Fig. 5



ACKNOWLEDGMENT

The Malaria Surveillance Report, prepared annually at the Centers for Disease Control, is based on information provided in individual case reports. The excellent support given to malaria surveillance by state and local health departments and personnel of the preventive medicine services of the U.S. Army, Navy, and Air Force is greatly appreciated.

REFERENCE

1. World Health Organization. Terminology of malaria and of malaria eradication, 1963, World Health Organization, Geneva, p 32.

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* Formerly Trust Territory of the Pacific Islands