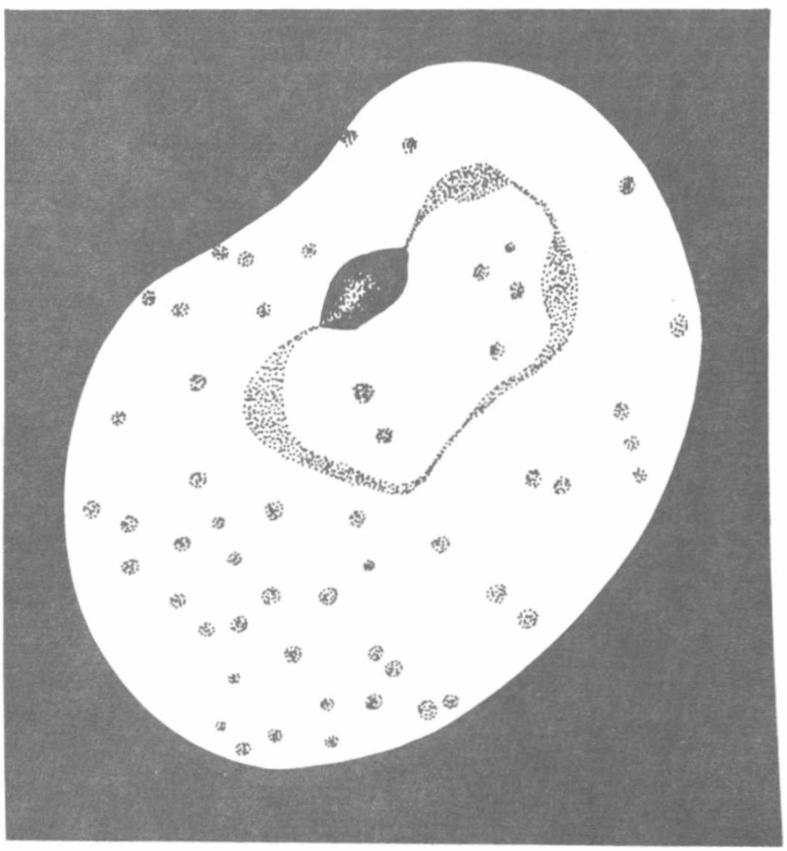
ANNUAL SUMMARY 1988

Issued November 1989

CENTERS FOR DISEASE CONTROL

MALARIA

SURVEILLANCE



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

P R E F A C E

This report summarizes information received from state health departments, medical departments of the Armed Forces, and other sources. It is intended primarily for those responsible for disease control activities. Before quoting this report, contact the original investigator for confirmation and interpretation.

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

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Guidelines for the prevention of malaria in travelers are published in HHS Publication No. (CDC) 89-8280, Health Information for International Travel 1989. This booklet also provides information about countries and, where applicable, areas within each country where malaria risk exists. Also listed are areas of the world where chloroquine-resistant strains of P. falciparum are known to exist. The booklet is available from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402.

SUGGESTED CITATION

Centers for Disease Control: Malaria Surveillance Annual Summary 1988
Issued November 1989

I. SUMMARY

A total of 1,023 cases of malaria with onset of illness in 1988 in the United States and its territories were reported to the Centers for Disease Control (CDC). This compares with 932 cases in 1987, an increase of 10%.

The number of reported cases with onset in the United States occurred in the following groups:

U.S. military personnel	33
U.S. civilians	540
Foreign civilians	440

Plasmodium vivax was the parasite identified in 43% of the 1,023 cases, and *P. falciparum* was identified in 46%. *P. malariae* and *P. ovale* were reported in 3% and 2% of the cases, respectively. The species was not determined in the other 7%.

Thirty-two of the 1,023 persons acquired the infection in the United States.

Six deaths attributed to malaria were reported for 1988, compared with 4 for 1987.

II. TERMINOLOGY

This report uses terminology derived from the recommendations of the World Health Organization (WHO)(1). Definitions of the following terms are included for reference.

A. Autochthonous

1. Indigenous--malaria acquired by mosquito transmission in an area where malaria occurs regularly.

2. Introduced--malaria acquired by mosquito transmission, from an imported case in an area where malaria does not occur regularly.

B. Imported

Malaria acquired outside a specific area (the United States and its territories 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 that is separated from previous manifestations of the same infection by an interval greater than those due to the normal periodicity of the paroxysms.

E. Cryptic

An isolated case of malaria ascertained by appropriate epidemiologic investigation not to be associated with secondary cases.

III. GENERAL SURVEILLANCE

This section covers 4 topics: the incidence of malaria, the *Plasmodium* species involved, the area in which infection was acquired and in which the onset of illness occurred, and how long it took for clinical malaria to develop after the patient's arrival in the United States.

A. Incidence

A total of 1,023 malaria cases with onset of illness in 1988 in the United States were reported to the Division of Parasitic Diseases, Center for Infectious Diseases, Centers for Disease Control (CDC), compared with 932 cases in 1987. In 1988, 32 of the 1,023 patients acquired the infection in the United States. Six fatal malaria infections were reported in 1988, as compared with 4 deaths in 1986. These cases are discussed in section VII.

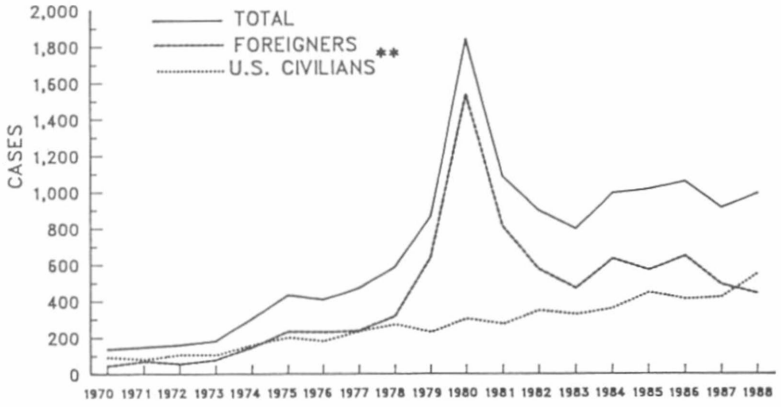
Table 1. All primary malaria cases* in civilians and U.S. military personnel with onset of illness in the United States, 1966-1988

Year	U.S. Military Personnel	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
1984	24	360	632	0	1,016
1985	31	446	568	0	1,045
1986	35	410	646	0	1,091
1987	23	421	488	0	932
1988	33	550	440	0	1,023

*A "case" is defined as: 1) a person'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) a positive peripheral blood smear examined in the local or state health department laboratory. Inconclusive blood smears were referred to the National Malaria Repository, CDC, for confirmation. 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 an additional case.

Only 33 cases occurred in U.S. military personnel. Civilians have accounted for most of the cases each year since 1973 (Table 1). The number of malaria cases in U.S. civilians increased from 421 in 1987 to 550 in 1988, exceeding, for the first time since 1974, the number of cases among foreign civilians. (Figure 1). Malaria in foreign civilians decreased from 488 reported cases in 1987 to 440 in 1988, a decrease of 10%.

FIG. 1 CASES OF MALARIA IN U.S. CIVILIANS AND FOREIGNERS, UNITED STATES,* 1970–1988



*Includes Puerto Rico, the Virgin Islands, and Guam

**Includes 9 cases acquired in the United States

B. Plasmodium Species

The Plasmodium species was identified in 957 (93.5%) of the 1,023 cases. In 1988, P. vivax was identified in blood from 43% of the infected persons and P. falciparum in blood from 46% (Table 2).

Table 2. Malaria cases by Plasmodium species, United States, 1987-1988

	1988		1987	
Species	Total	Percent	Total	Percent
P. vivax	437	42.7	409	43.9
P. falciparum	465	45.5	399	42.9
P. malariae	34	3.3	37	4.0
P. ovale	17	1.7	27	2.9
Mixed	4	0.4	2	0.2
Undetermined	66	6.5	58	6.4
TOTAL	1,023	100.0	932	100.0

C. Area of Acquisition and of Onset of Illness

The area in which each of the 1023 patients acquired the infection is listed in Table 3. From 1987 to 1988, cases imported from Mexico increased 90.5% and from Africa, 15.9%. Importation of malaria from Asia declined 35.0%. From 1978 through 1988, malaria infections acquired in Africa increased from 176 cases to 495 cases, a 181% increase.

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. Malaria cases with onset in the United States, by state, 1988

Table 3. Malaria cases by distribution of *Plasmodium* species and area of acquisition, United States, 1988

Area of Acquisition	vivax	falciparum	malariae	ovale	mixed	unknown	Total
AFRICA	42	393	12	16	1	31	495
Africa, East*	3	13	1	0	0	1	18
Africa, West*	1	20	1	2	0	1	25
Africa, Central*	2	0	0	0	0	1	3
Africa, Unspecified*	0	20	0	1	0	3	24
Benin	0	0	0	0	0	1	1
Botswana	0	3	0	0	0	0	3
Burkina Faso	1	2	1	0	0	0	4
Cameroon	1	12	1	0	0	0	14
Central African Rep.	0	2	0	0	0	0	2
Congo	0	1	0	2	0	0	3
Ethiopia	3	2	0	0	0	0	5
Gabon	0	3	0	0	0	1	4
Gambia	1	0	0	0	0	0	1
Ghana	2	31	2	0	1	2	38
Guinea	0	1	0	0	0	0	1
Guinea Bissau	0	2	0	0	0	0	2
Ivory Coast	0	12	0	1	0	1	14
Kenya	8	76	1	3	0	1	89
Liberia	2	16	0	3	0	1	22
Madagascar	0	2	0	0	0	0	2
Malawi	0	5	0	0	0	1	6
Mali	0	3	0	0	0	0	3
Niger	0	1	0	0	0	0	1
Nigeria	8	99	3	2	0	12	124
Senegal	0	2	0	0	0	0	2
Sierra Leone	2	10	0	1	0	2	15
South Africa	0	2	0	0	0	0	2
Sudan	4	10	1	0	0	1	16
Tanzania	1	3	0	0	0	0	4
Togo	0	12	0	0	0	0	12
Uganda	1	7	0	0	0	1	9
Zaire	0	9	0	1	0	1	11
Zambia	2	9	1	0	0	0	12
Zimbabwe	0	3	0	0	0	0	3
ASIA	145	36	9	0	2	18	210
Asia, Southeast*	6	1	2	0	0	1	10
Middle East, Unspec.*	0	0	0	0	0	1	1
Afghanistan	0	1	0	0	0	0	1
Burma	0	0	0	0	0	1	1
India	78	22	6	0	1	12	119
Indonesia	10	6	1	0	0	0	17
Lao People's Dem. Rep.	1	0	0	0	0	0	1
Nepal	0	0	0	0	0	1	1
Pakistan	10	0	0	0	1	1	12
Philippines	27	5	0	0	0	0	32
Sri Lanka	2	0	0	0	0	0	2
Thailand	6	1	0	0	0	0	7
Viet Nam	5	0	0	0	0	1	6
CENTRAL AMERICA AND CARIBBEAN	61	19	8	0	0	8	96
Central Amer. Unspec.*	4	2	0	0	0	0	6
Belize	6	1	0	0	0	0	7
El Salvador	12	1	2	0	0	1	16
Guatemala	19	5	0	0	0	0	24
Haiti	0	10	2	0	0	4	16
Honduras	15	0	1	0	0	1	17
Nicaragua	5	0	3	0	0	2	10
NORTH AMERICA	159	5	1	0	0	8	173
Mexico	129	3	1	0	0	8	141
United States	30	2	0	0	0	0	32
SOUTH AMERICA	5	3	0	0	0	1	9
Brazil	1	1	0	0	0	0	2
Colombia	2	0	0	0	0	0	2
Ecuador	2	1	0	0	0	0	3
Guyana	0	1	0	0	0	0	1
Venezuela	0	0	0	0	0	1	1
OCEANIA	21	7	4	1	1	0	34
Papua New Guinea	21	7	4	1	1	0	34
UNKNOWN	4	2	0	0	0	0	6
TOTAL	437	465	34	17	4	66	1023

**Country unspecified.

D. Interval Between Arrival and Illness

The interval between the date of arrival in the United States and the date of onset of illness was known for 650 of the patients for which the infecting Plasmodium species was also identified. Clinical malaria developed within 1 month after the patient's arrival in 89.0% of the P. falciparum cases and in 34.1% of the P. vivax cases (Table 4). Only 17 (2.6%) of the 650 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, 1988

Interval (in months)	PLASMODIUM SPECIES									
	vivax	(%)	falciparum	(%)	malariae	(%)	ovale	(%)	Total	(%)
< 1	99	(34.1)	290	(89.0)	7	(35.0)	2	(14.3)	398	(61.2)
1-2	61	(21.0)	23	(7.1)	4	(20.0)	4	(28.6)	92	(14.2)
3-5	66	(22.8)	9	(2.8)	3	(15.0)	7	(50.0)	85	(13.1)
6-11	51	(17.6)	1	(0.3)	5	(25.0)	1	(7.1)	58	(8.9)
≥12	13	(4.5)	3	(0.9)	1	(5.0)	0	(0.0)	17	(2.6)
TOTAL	290	(100.0)	326	(100.0)	20	(100.0)	14	(100.0)	650	(100.0)

IV. IMPORTED MALARIA IN MILITARY PERSONNEL

Thirty-three cases of imported malaria in U.S. military personnel were reported for 1988. The Army accounted for 10 cases, the Navy for 2, the Air Force for 1, and the Marine Corps for 20 cases.

V. IMPORTED MALARIA IN CIVILIANS

Of the 958 imported malaria cases in civilians, 546 (57%) were in U.S. citizens, whereas 412 (43%) were in citizens of other countries (Table 5).

Table 5. Imported malaria cases in civilians, by area of acquisition, United States, 1988

Area of Acquisition	United States		Foreign		Total	
	Cases	Percent	Cases	Percent	Cases	Percent
Africa	358	65.4	131	31.8	489	51.0
Asia	82	15.0	106	25.7	188	19.6
Central America	28	5.1	48	11.7	76	7.9
Caribbean	10	1.8	6	1.5	16	1.7
Mexico	30	5.7	110	26.7	140	14.6
South America	5	0.9	4	1.0	9	0.9
Oceania	31	5.7	3	0.7	34	3.5
Unknown	2	0.4	4	1.0	6	0.6
TOTAL	546	100.0	412	100.0	958	100.0

A. U.S. Civilians

Of the 546 cases in U.S. civilians, 358 (65%) were acquired in Africa and 82 (15%) were acquired in Asia (Table 5).

Imported malaria caused by P. falciparum in U.S. civilians infected in Africa has increased each year since 1981. For 1988, 291 such infections were reported, an increase of 34.7% over 1987, when 211 such cases were reported. Since 1981, these cases have increased 410%.

Most of the U.S. civilians were tourists (Table 6).

Table 6. Imported malaria cases in U.S. civilians, by category, United States, 1988

Category	Cases	Percent
Tourist	140	25.6
Business Representative	62	11.4
Government Employee	11	2.0
Missionary	67	12.3
Peace Corps	14	2.6
Seamen/Aircrew	1	0.2
Teacher/Student	28	5.1
Visiting friends/Relatives	66	12.1
Other	28	5.1
Unknown	129	23.6
TOTAL	546	100.0

B. Foreign Civilians

Of the 412 cases in foreign civilians, only 106 (26%) were acquired in Asia; infections acquired in India declined markedly from 149 in 1987 to 67 in 1988 (55%). A marked increase in infections acquired in Mexico was observed: from 60 to 110 cases (83%) between 1987 and 1988.

VI. MALARIA ACQUIRED IN THE UNITED STATES

A. Congenital Malaria

One case of congenital malaria with onset of illness in 1988 was reported. The infection was due to P. falciparum.

Case 1--On March 22, 1988, a blood smear of a 3-day-old infant showed rare ring forms of P. falciparum. The infant was treated with chloroquine and had an uneventful recovery. The mother had moved to Florida from her native Haiti 7 years earlier and had reportedly not returned to Haiti during that time. She denied having had malaria previously and blood smears were negative.

(Reported by E.R. Walters, M.P.H., Epidemiologist, and C. Levin, R.N., Broward County Public Health Unit, Fort Lauderdale, Florida.)

B. Induced Malaria

Case 1-- A 32-year-old male resident of New York underwent a renal transplant on August 21, 1988. On September 14, 1988, he developed intermittent fever episodes. P. falciparum parasites were identified, and he was treated with chloroquine, quinine, and pyrimethamine-sulfadoxine. He had moved to the United States from his native Ghana in September 1985 and had not returned to Ghana. He denied having had febrile episodes since his arrival in the United States.

The donor was the patient's sister who had traveled from Ghana to donate the kidney. Microscopic examination of several blood smears of the sister revealed the presence of a single P. falciparum parasite.

(Reported by R.L. Scheer, M.D., Syracuse, New York, and the New York State Department of Health, Albany, New York.)

C. Introduced Malaria

An outbreak of introduced P. vivax malaria was identified in San Diego County, California, in 1988. The episode represented the largest outbreak of introduced malaria since 1952.

On August 3, 1988, a migrant worker was diagnosed with P. vivax infection in a physician's office in Encinitas, 22 miles north of San Diego. That same day, a public health nurse accompanied the worker to the field where he was living; the nurse interviewed and referred for diagnosis 12 additional workers with symptoms suggestive of malaria. All had P. vivax parasitemia. As a result of this cluster of cases, health agencies intensified epidemiologic and entomologic surveillance efforts to define the extent of the outbreak and to guide control measures. Active case detection methods included contact with local hospitals, clinics, and physicians to identify unreported, laboratory-confirmed cases. Surveys for febrile illness in migrant workers in areas surrounding the outbreak focus were conducted. Local social networks and media were employed to disseminate information about malaria and to solicit reports of persons with malaria symptoms in the community.

The investigation identified a total of 30 outbreak-related symptomatic P. vivax infections with onset between July 24 and September 18, 1988 (Figure 3). Two cases were in local residents who had no apparent risk factors for malaria, and 28 were in migrant workers employed primarily in the agricultural businesses located near the Lake Hodges reservoir 25 miles north of San Diego. All patients denied previous malaria infection, intravenous drug use, or history of blood transfusions in the previous 3 years.

Nineteen of the migrant workers identified as cases lived in a canyon area several miles from Lake Hodges. Of the 11 individuals not living in the canyon, 7 were migrant workers employed at and living on a farm directly south of the canyon. Another case occurred in a pregnant migrant worker who worked at a tree nursery next to the farm and was required to be outside at dawn and dusk. An additional case was in a migrant worker who had lived in the canyon, but moved to a beach community before symptoms developed. The remaining 2 cases occurred in a married couple who were local county residents; their home was at the western edge of a development adjacent to the farm and tree nursery.

Of the 28 infected migrant workers, 27 could be located and interviewed; the remaining 1 had returned to Mexico immediately after diagnosis. All but 1 were males employed in a variety of agricultural activities. Seventeen (61%) came from the state of Morelos; 9 came from 4 other Mexican states, and 2 were from Guatemala. The infected workers' mean duration of stay in the United States before becoming ill was 17 weeks (range: 4 weeks to 3 years); 20 (71%) had been in the United States for at least 2 months.

Of the 38 individuals living in the canyon, 19 were diagnosed to have malaria, for an attack rate of 50%. The migrant workers in this area had diverted water flow from a canal to their north directly into and through their encampment; some of their dwellings (mostly plastic tarp and cardboard

shelters) were located within 5 feet of the water. Detailed questionnaires were administered to 31 (82%) of the migrant workers in this canyon (20 cases and 11 others); the remaining workers could not be located. No significant differences were noted between cases and non-cases in regard to shelter used for sleeping (open vs. enclosed), average number of daily hours spent by the water (lagoon, river, or canal), bathing sites, and time of day returning to the campsite.

On June 28, 1988, 8 carbon-dioxide baited light traps were placed along a river near the canyon where 4 days earlier a migrant worker with documented P. vivax malaria had slept for several days. The traps caught a total of 79 adult female Anopheles hermsi mosquitoes, a competent malaria vector. Traps placed in the canyon encampment area on August 4 contained between 1 and 11 adult female An. hermsi per trap. High An. hermsi counts (79 and 115 in 2 of the traps) were also documented next to an irrigation pond on the farm south of the canyon.

Control measures instituted on August 3 by the San Diego County Department of Health included active case detection and chloroquine and primaquine therapy of identified cases. In addition, groups considered to be at risk for continued exposure to infected mosquitoes were placed on weekly chloroquine prophylaxis. These included both the canyon migrant worker group and migrant workers living or working on the farm directly south of the canyon. Vector control efforts included larviciding in areas with standing water and insecticiding by both fogging and backpack spraying.

(Reported by S. Hunt, D. Maher, M. Ginsberg, M.D., M. Mizrahi, M.S., M. Thompson, Dr.P.H., D. Ramras, M.D., San Diego County Department of Health Services, R.R. Roberto, M.D., California Department of Health Services, Berkeley, California.)

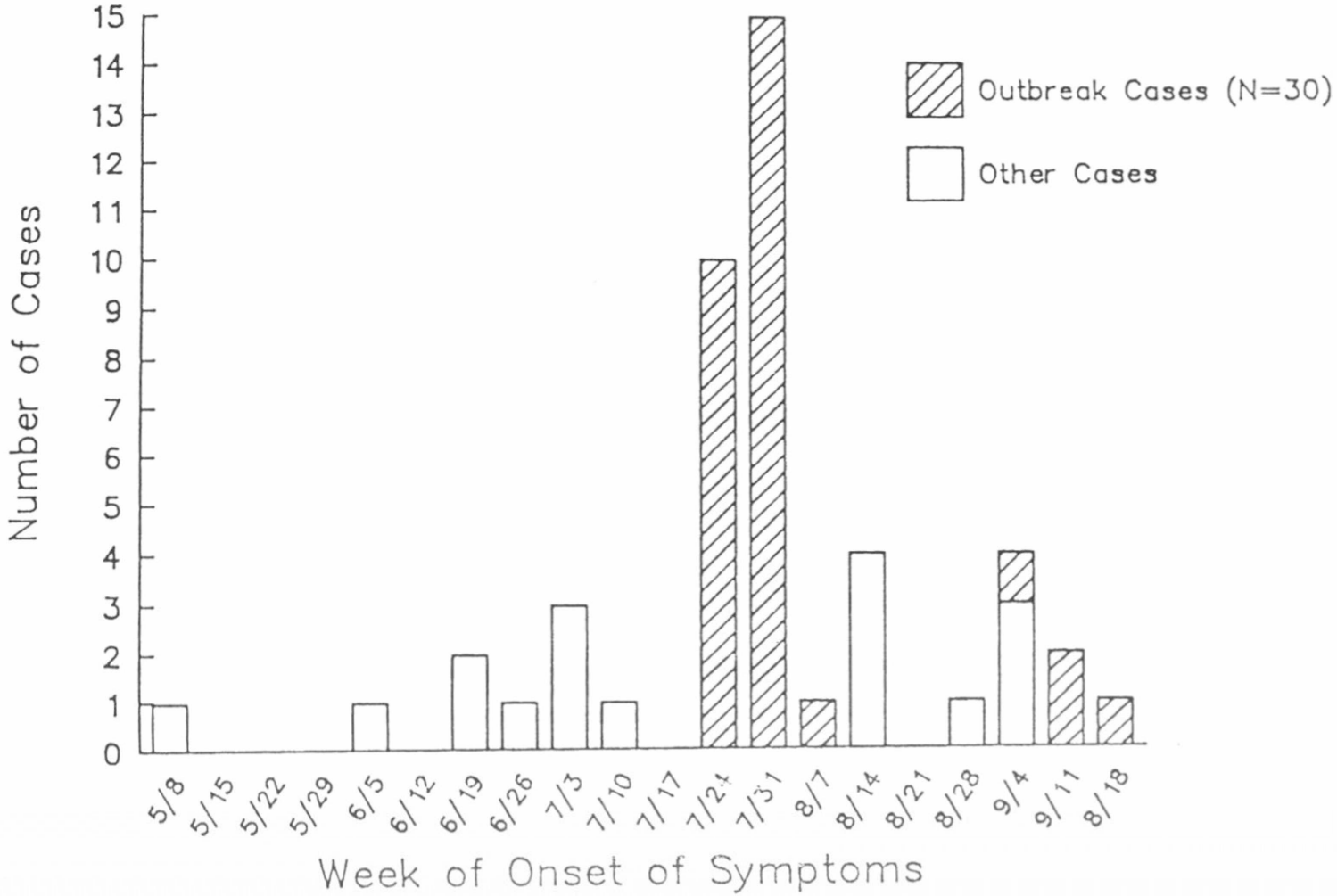
Comment: This outbreak of P. vivax malaria in San Diego county appears to have resulted from local transmission in the Lake Hodges area, an hypothesis strengthened by the occurrence of malaria in 2 local residents with no other risk factors for the disease. In addition, the geographical and temporal clustering of the cases from July 24 to September 18 suggests that infection was acquired locally in most, if not all, of the migrant worker cases. At least one individual with parasitemia was in the area approximately 30 days before this outbreak. This interval is consistent with the time for development of the parasite in host mosquitoes and transmission to humans. One other worker in the canyon encampment was ill for 4-5 weeks before diagnosis and may have been an additional source of P. vivax gametocytes infecting the local An. hermsi population.

This was the largest outbreak of introduced malaria in the United States since 1952, when 35 P. vivax infections were reported in a group of Camp Fire girls in California (2). The source of infection in that episode was presumed to be a serviceman who had returned from Korea. A second large outbreak in 1986 involved 27 migrant workers and 1 local resident in Carlsbad, 35 miles north of San Diego (3).

Since 1950, 13 of 20 episodes of introduced malaria in the United States, all due to P. vivax, have been reported in California; 6 of the 13 were identified in 1986 and 1988. Only 3 of the outbreaks involved more than 5 people, and 2 of these included migrant workers. The other 10 outbreaks were limited to 1-4 cases each, all involving California residents. The specific sources of the infections for the mosquitoes could not be identified in all outbreaks; however, in many such outbreaks agricultural workers from malaria-endemic countries were present in the affected areas. It is possible

Figure 3

Plasmodium vivax Cases
San Diego County, 1988
Week of Onset of Symptoms



that other limited outbreaks of introduced malaria have gone undetected, for 2 reasons: first, the diagnosis of malaria may not be considered in patients who have not traveled abroad, and second, malaria may go undetected in migrant workers and other groups who have limited access to medical care. Therefore, this outbreak in San Diego County underscores the fact that importation of malaria in the United States may result in outbreaks when infected persons, large numbers of competent mosquito vectors, and susceptible populations live in close proximity. Anopheline malaria vectors are found in many parts of California, making it nearly impossible for officials to predict where episodes of introduced malaria will occur.

Medical personnel should be aware that introduced malaria may affect migrant workers. When cases are identified, health care workers must obtain the date of arrival in the United States and a complete history of recent travel, past malaria infection, and intravenous drug use or blood transfusion. A cluster of malaria cases should prompt an immediate and aggressive investigation to determine whether local transmission has occurred.

VII. MALARIA DEATHS

Six deaths due to malaria were reported.

Case 1--A 63-year-old female resident of California returned from her native India on April 14, 1988, after a 1-month visit to relatives in the Bombay area. It is not known whether she used chemoprophylaxis in India. On April 16 she was seen in the emergency room with a history of fever, nausea, and diarrhea with onset during her return travel. She was diagnosed as having gastroenteritis. She returned to the emergency room on April 19 with the same symptoms. On April 22 she again came to the emergency room because of persistent nausea, vomiting, diarrhea, and fever. She had also become lethargic. Following admission she was found to have an infection with more than 15% of red blood cells containing *P. falciparum*. She was given oral chloroquine and primaquine. On April 23 she developed cardiac arrhythmia and died.

(Reported by A. Bathia, M.D., Santa Clara Valley Medical Center, San Jose, Connie Diggins, Nurse Epidemiologist, Santa Clara County Health Department, San Jose, and R.R. Roberto, M.D., California Department of Health Services, Berkeley, California.)

Case 2--A 52-year-old resident of California was admitted to a local hospital on April 17, 1988, with a 6-day history of fever and chills. Three days before admission he noted dark urine and yellowing of the skin. Physical examination indicated marked icterus, and enlarged spleen and liver. Blood smear examination revealed the presence of *P. falciparum* parasites. The patient was started on oral quinine but died shortly after admission.

He had traveled from March 27 to April 4 as a mechanic for an airline to Kenya, where he had gone on safari. Reportedly he had used chloroquine for prophylaxis. According to his wife he had been seen twice by a physician before admission but was advised that he had the flu.

(Reported by Marilyn Billimek, nurse epidemiologist, Ventura County Department of Health, Ventura, and R.R. Roberto, M.D., California Department of Health Services, Berkeley, California.)

Case 3--A 35-year-old male resident of Indiana visited Sierra Leone from December 16, 1987, until January 1, 1988. On January 20 he consulted a physician with a 10-day history of chills, fever, and diaphoresis. Blood smears made on that day revealed P. falciparum parasites. The patient did not return to his physician for follow-up and was found dead at home on January 23. It was estimated that he died on January 22. The autopsy report stated that he probably died of a massive myocardial infarct. He had used daily pyrimethamine for prophylaxis, from 2 weeks before departure until he visited the physician.

(Reported by G. Merkle, M.D., Blufton, and the Indiana State Board of Health, Indianapolis, Indiana.)

Case 4--Having been diagnosed to have P. falciparum malaria, a 35-year-old female resident of Washington, D.C., came to the emergency room of a District hospital on December 7, 1988. Treatment with intravenous quinine was started, but she died 2 hours after admission. She had traveled to Lagos, Nigeria, from November 15-19 and returned on November 20. She began to experience flu-like symptoms, fever and cough on December 1. She called a physician on December 3 and was prescribed erythromycin for "flu." She visited another physician on December 7, and a chest X-ray and a blood smear were ordered. The blood smear showed a 15% parasitemia with P. falciparum. She was then sent to the emergency room.

She reportedly had not used chemoprophylaxis, but a postmortem blood sample showed chloroquine levels consistent with therapeutic intake of chloroquine.

(Reported by M.S. Wolfe, M.D., and the District of Columbia Department of Human Services, Washington, D.C.)

Case 5--A 55-year-old male employee of a U.S. company returned from Nigeria on June 7, 1988. On June 20 he visited a physician and gave a 4-day history of daily rigors. Two days later he was found on the floor of his home in extreme respiratory distress. He was transported to the hospital but died in the emergency room. Blood smears made on June 20 contained P. falciparum parasites, but he had not received antimalarial therapy. Autopsy revealed P. falciparum parasites in the spleen, liver, kidneys, and brain. An antemortem blood smear indicated that 50% of red blood cells contained malaria parasites. Reportedly he had used chloroquine for prophylaxis during his travel to Nigeria.

(Reported by the West Virginia State Department of Health.)

Case 6--A 56-year-old female resident of California was admitted on May 30, 1988, to a local hospital and gave a 3-day history of fever, chills, and myalgia. She had also symptoms of generalized malaise, jaundice, and abdominal pain. She had anemia and renal failure. More than 20% of red blood cells contained P. falciparum parasites. She was treated initially with chloroquine and Fansidar^{R}, and later with intravenous quinidine. On June 4 she developed seizures and respiratory distress. The renal failure increased. On June 23 she developed adult respiratory disease syndrome. The patient died on July 4 of acute respiratory failure, acute renal failure and cerebral malaria. She had returned on May 26 from a 3-week trip to Kenya and had taken chloroquine for prophylaxis. No information was available on the dosage or compliance.

(Reported by R. Taylor, M.D., Upland, California, and R.R. Roberto, M.D., California Department of Health Services, Berkeley, California.)

A report was received in 1988 of an additional fatal case associated with malaria in 1987. This increases the total number of fatal cases in 1987 to 4:

Case 4--A 41-year-old female California resident returned from a 7-week trip to her native India and visited a clinic on July 30, 1987 with a 4-day history of chills and fever. P. falciparum parasites were identified in a blood smear. She was given 3 tablets of Fansidar but was unable to keep the medication down because of nausea and vomiting. She was admitted to a hospital on August 2 because of continuing chills and fever, and increasing weakness, dizziness and anorexia. Blood smears contained numerous Plasmodium parasites, considered to be P. falciparum. Subsequent examination of the blood slides by the Microbial Diseases Laboratory, California Department of Health, and by the Malaria Branch, CDC, identified only P. vivax parasites. The patient was treated with quinine, pyrimethamine and sulfadiazine. Three days after admission she developed acute pulmonary edema and hypotension. Subsequent chest x-ray showed diffuse bilateral fluffy infiltrates consistent with adult respiratory distress syndrome (ARDS). Her fevers persisted although no malaria parasites were found. Multiple cultures were negative. She was given antibiotics. The patient remained febrile and developed renal insufficiency, aplastic anemia, and disseminated intravascular coagulation. She died on August 16.

The autopsy diagnoses included interstitial nephritis, regenerating bone marrow, ARDS, and congestive heart failure. No malarial parasites were seen.

(Reported by J.A. Granzella, M.D., University of California Davis Medical Center, Sacramento, and R.R. Roberto, M.D., California Department of Health Services, Berkeley, California.)

VIII. MICROSCOPIC DIAGNOSIS OF MALARIA

Early diagnosis of malaria requires physicians to include malaria in the differential diagnosis and to take a comprehensive travel history from every patient with a fever of unknown origin. Once malaria is suspected, a Giemsa-stained smear of peripheral blood should be examined for parasites. Since the accuracy of diagnosis depends on the quality of the blood film, the following guide is offered for the proper preparation of thick and thin blood smears.

1. Manufacturers' "precleaned" slides are not considered clean enough for use in malaria diagnosis. Before using them, wash the slides in mild detergent, rinse them thoroughly in warm running water, rinse them in distilled water, and dip them in ethyl alcohol (90%-95%). Then, wipe the 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. Puncture the finger with the blood lancet, and 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 one-third area of the slide. Ordinary newsprint should be barely legible through such a wet drop (Figure 4). (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. Place the drop at the middle of the same slide (Figure 5).
6. Apply a clean "spreader" slide to the edge of the small drop at a 45^{o} angle, and allow the blood to extend about two-thirds, to the back of the slide. 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 6).
7. While the thick blood film dries (minimum of 6 hours at room temperature), keep the slide flat 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 6).

Note: For rapid diagnosis, make the thick and thin films on separate slides. Air dry the thin film, fix it with methyl alcohol, and stain it immediately. If no parasites are found on the thin film, wait until the thick film is dry and examine it for organisms not detected on the thin preparation.

Fig. 4

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

Fig. 5

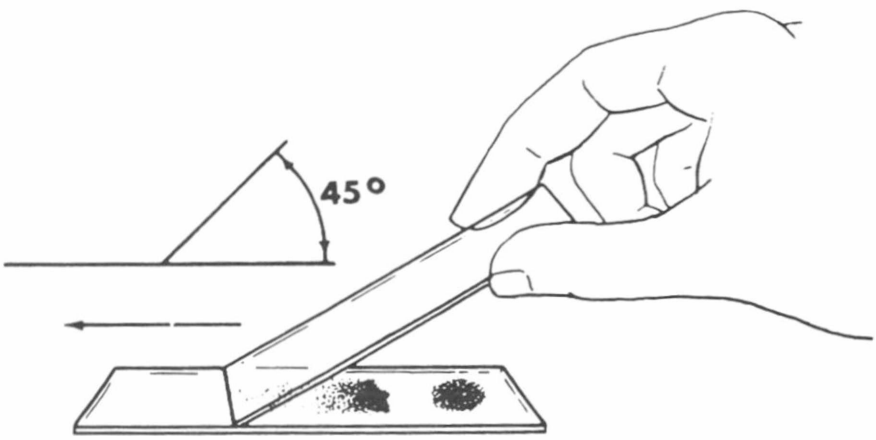
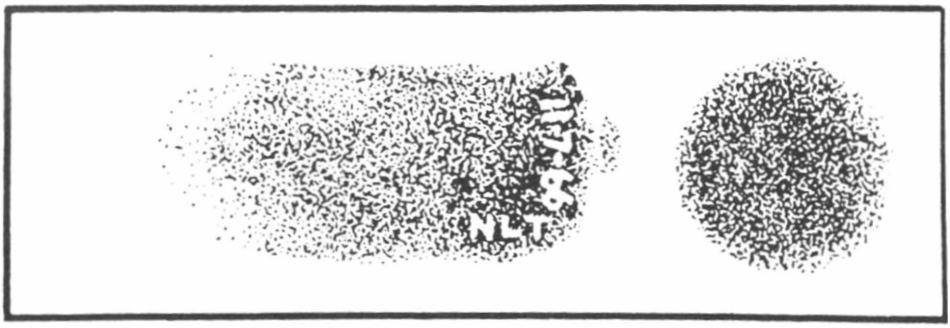


Fig. 6



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