malaria. It offered the opportunity to follow the natural history of *P. vivax* malaria in a non-immune population living in a non-endemic area.

All but two of the cases were confirmed by the laboratory to be *P. vivax*. The exceptions are included on clinical and epidemiologic grounds. None of the 26 latent cases had any symptoms of malaria prior to their onsets 226 to 307 days after exposure at Lake Vera, nor had they been given any suppressive therapy. None of them had any other known exposure—either previous or subsequent to their visit at the Lake.

A detailed account of the investigation, the findings and the control measures instigated are being published in the American Journal of Tropical Medicine and Hygiene. Because of the time limitation only the highlights of our findings will be presented today.

This outbreak fortifies Kortwieg's hypothesis that two-thirds of the *P. vivax* infections contracted in autumn remain clinically latent for eight to nine months. Being able to document the date of exposure and the date of onset was perhaps the most significant contribution of this investigation. As far as these infections are concerned suppressive therapy did not contribute to the long latent incubation period of this strain of *P. vivax*. Another interesting observation was the fact that seven of the original nine cases which had clinical malaria in 1952, later relapsed.

The illness in twenty-nine of the cases pursued the classical course of chills, fever, and sweating every forty-eight hours. Fifteen of them experienced premonitory symptoms, such as lassitude, headache, arthralgia, nausea, vomiting, chilliness, and fever from 1 to 7 days before their first rigor.

Two (SH and MT) of the six possible atypical cases developed daily paroxysms after three typical tertian reactions. Two others (ID and PC) had a prodromal stage of intermittent attacks of pharynigitis 41 to 48 days before their first paroxysm. In one girl (CP) the predominating symptoms and subjective findings pointed to pyelitis. These symptoms were also present at the time of her relapse in April. A two-day episode of fever, chills, arthralgia, nausea, vomiting, and headache occurred in one patient (SR) two weeks before the first classical attack.

Whether or not these cases represent the total remains to be seen. However, we do not believe that additional cases which come to our attention will change the incubation time. So far we have been able to verify that this latent period can be from seven to ten months. The two cases with onsets in July and August tend to substantiate this. One of the males (FP) moved to the vicinity of Lake Vera in September 1952. He became ill July 3, 1953, and assuming he became infected on arrival his incubation period would be nine and one-half months. The other person maintains a summer home at the Lake. She (RP) was subject to exposure from the time the mosquitoes were infected until October when spraying activities were conducted. Another case (PP) surreptitiously visited one of the camps for three days in mid-July. She became ill nine months later. Another (BC), the mother of one of the campers, came to the Lake the last two weekends in July 1952. She became ill in April 1953 or about eight months later. Twenty-one of the cases were definitely established as having onsets from eight to ten months after exposure (seven each eight, nine and ten months).

This observation has tremendous epidemiologic implications. Perhaps the cursory question of where were you three weeks ago will have to be revised. When dealing with temperate zone *P. vivax* infections in California, where were you three weeks ago *and* eight to ten months ago apparently will be more elucidating.

Obviously this outbreak demonstrates that this country is vulnerable to the introduction of malaria from abroad. As long as the vector is prevalent among our susceptible population transmission can occur. Our control endeavors to reduce this hazard should not overlook the possibility of secondary cases arising from those cases which have had their onsets months after original exposure.

NOTE: Acknowledgement is made to Roy Fritz, Senior Scientist, U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia, who is co-author of the report to be published.

Dr. Hackett: I'm glad that the mother was punished because you ought not to visit your daughters at these camps, but I'm sorry about that surreptitious girl.

I had a paper presented to the American Journal of Tropical Medicine from Fort Bennington by two doctors there, who said that they had a couple of soldiers who had come down with primary attacks of malaria eight or nine months after leaving Korea, but I rejected the paper because they told me that the soldiers had assured them that they had not taken any suppressive drugs while in Korea; I rejected it because you can't believe a soldier about suppressive drugs. But it is true that you can get the northern European and Asiatic malaria and suffer no symptoms whatever for eight to ten months before the attack. This outbreak has aroused a great deal of discussion among malariologists as to why it hasn't occurred more often.

What historical evidence have we that would bear on a determination of the vector of this outbreak. I hope Mr. Gray will throw light on some of these points.

INTERESTING ASPECTS OF THE MALARIA OUTBREAK AT LAKE VERA, CALIFORNIA

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At the outset, the epidemic of malaria among the Camp Fire Girls at Lake Vera, California, in 1952, must be characterized as an abnormality. The conditions were almost completely different from any usual setting for a malaria outbreak. In the first place, there was no housing in the usual sense, and the girls were freely accessible to the mosquitoes. In the second place, the girls were all non-immunes, coming from areas where malaria had never been endemic, or from areas where endemic malaria had been absent for many years. Third, the area in which the infections occurred had not had reported cases of malaria for many years. Fourth, the area in which the infections occurred had never, so far as I am informed, had any organized mosquito control work, nor an effective local health department. Fifth, efficient *Anopheles* vectors had always been numerous in the area—to my personal knowledge for over forty years.

These vectors are *Anopheles freeborni,* generally considered to be *the* malaria vector in the western part of the United States, and *Anopheles punctipennis,* generally considered to be an ineffective malaria vector except in special conditions, as it seldom enters houses.

Anopheles punctipennis tends to be relatively more numerous than *A. freeborni,* at least in the foothill areas,

in May, June and July, with *A. freeborni* generally becoming predominant in August, September and October. Since the infections in this outbreak were received in July, and since even in August at Lake Vera *A. punctipennis* adults were by observation more numerous than *A. freeborni,* as Mr. Fontaine will tell you, it appears that numerically *A. punctipennis* could be involved. And as it bites humans freely outdoors in the evening and also in the shade during the day, there was ample opportunity for this species both to obtain malaria parasites and to transmit them. On the basis of my own observations in similar situations in past years, and of the conditions occurring in this outbreak, it is my personal opinion that in the Lake Vera situation *A. punctipennis* could have been as efficient a malaria vector as *A. freeborni.*

Under these unusual conditions, all that was needed was the introduction, for a few nights, of a gametocyte carrier accessible to the *Anopheles* mosquitoes. The inevitable chain of results followed promptly.

BUT—and here is a big *BUT*—if the gametocyte carrier had slept in a screened house, possibly only a few *Anopheles* could have been infected; or if the girls had slept in screened houses, possibly only a few girls could have been infected. Or if the girls had come from areas where malaria was endemic, new infections would probably not have been suspected or noted as such—relapses would probably have been diagnosed, and the local origin of infection not discovered.

But, even if all these buts did not occur, the epidemic *could* have slipped by unsuspected as to source if Dr. Brunetti had not sensed a possible common origin in the nine initial cases of widely scattered home residence. It takes more than a routine shuffling of case history cards to ferret out the source of this type of an epidemic, and Dr. Brunetti deserves an accolade for having her feminine intuition in good working order.

One factor that we had to consider in this epidemic was an imponderable. What were the chances that it could occur again in 1953 if no mosquito control measures were attempted? On the basis of past experience it would appear that such an outbreak was unlikely to occur more than once in forty or fifty years. It is possible that no new cases of malaria would have occurred at Lake Vera in 1953, even without mosquito control. It is also possible that a girl who was infected there in 1952 might return in 1953, and in spite of medication she might still have gametocytes of malaria in her peripheral circulation. In that event another series of cases probably would have occurred, with consequent damage to Lake Vera's reputation and future use as a summer camp. In view of the Camp Fire Girls' investment in the camp, aside from any considerations of health, the chance was too great to risk. The decision to do mosquito control work for the 1953 season was therefore wise.

An interesting corollary to this outbreak would be far from some epidemiologist to work out the malaria picture in Nevada County in reverse—why and how did malaria die out in this region, after having been epidemic one hundred years ago, and endemic up to about thirty or forty years ago. Actually, the essential conditions that permitted this small outbreak to occur were very similar to those under which extensive and intense malaria occurred in the Sierra Nevada foothills a century ago—unhoused people freely exposed to a probably large Anopheline population, with gametocyte carriers present who brought their infections from the southern states or acquired them en route at Panama.

In this case, the control methods were simple, obvious and easy to apply. Fortunately, we had reasonably frightened Camp Fire Girls' executives to deal with, and not a lot of amateur health experts with pig-headed notions about DDT. We were able to persuade them to fill in the marginal shallows of the lake, from which came most of the *Anopheles.* Here, as almost always, the bulldozer was mightier than the spray can. After the event, I feel confident that the bulldozer work would have been sufficient to have prevented a recurrence of the outbreak, if applied to all the possible *Anopheles* sources. But some DDT spraying was necessary to mop up small residual sources. This was done with nearly 100% effectiveness. An encomium or two should go to Russ Fontaine for supervising the control job successfully.

It is probable that if a little additional bulldozer work is done to fill the marginal shallows along the lower end of Brush Creek, no *Anopheles* control work will be necessary in this area in the 1954 season, as a malaria preventive measure. And if a reasonably good job of *Aedes varipalpus* control is done in the spring of 1954 (which involves climbing all the deciduous trees in the camp area) further mosquito control work for the comfort of the campers may be unnecessary.

One sidelight on this outbreak especially intrigues me —but after many years of experience does not astonish me. It is the apparent utter indifference of the county officials, the health officer, the chambers of commerce, and the general public, to the implications of this outbreak. It seems to me that if I were a local chamber of commerce executive I would have been screaming to the board of supervisors and the health officer that such an outbreak was disadvantageous publicity for the area, and would be bad for business, and something must be done about it. And if I were a PTA member, or a hotel or a motorcourt proprietor, or any one in local business, I think my reactions would be the same. Possibly the real significance of this outbreak has been concealed from the local community mind by the far more spectacular and concurrent epidemic of encephalitis that summer. But this type of malaria epidemic can happen again, perhaps not at Lake Vera, but at any one of numerous localities in the Mother Lode area. It seems to me that this not only is a phase of recreational sanitation which has not yet been adequately considered by the State Department of Public Health, but it is also an area of local sanitation in which the State has not adequately stimulated the local health officials involved. Here is a field of action more useful and basic and valuable than some of the "modern" excrescences on the present corpus of public health practice.

Dr. Hackett: I would like to ask Mr. Gray whether Mexican agricultural labor goes up into Nevada County to pick fruit?

Mr. Gray: Probably only a few in that county, and probably none in the general vicinity of Lake Vera. There are a number imported in adjacent Placer County around Colfax, Auburn, Newcastle and Penryn.

Dr. Hackett: Any other questions on either of these first two papers? The surprise of this was, of course, that this hasn't happened before, and not that it should have happened at this time. When we consider the number of carriers which get into the Central Valley with 300,000 acres of rice growing *Anopheles freeborni,* you wonder

what it is that protects our people from malaria in the valley.

Mr. Russell Fontaine was sent up to Lake Vera so expeditiously that he got there before the end of August, and he made a survey, and also had charge of the preventive work there.

CONTROL MEASURES FOR THE MALARIA OUTBREAK AT LAKE VERA, CALIFORNIA

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One of the intriguing aspects of the Lake Vera malaria outbreak to the Culicidologist was, which one of the two prevalent *Anopheles* present in the area was the vector, *A. freeborni* or *A. punctipennis*? Of course both species may have been involved, but *Anopheles freeborni* is presumed to be. Perhaps the facts of the mosquito situation at Lake Vera as we found them when we first visited the area on August 30 ,1952 shortly after it was suggested that the Camp Fire Girls' camps were the source of the malaria will shed light on this question. Our search for *Anopheles* species in permanent buildings in the camps, particularly in washrooms and toilets, revealed both *A. freeborni* and *A. punctipennis* in abundance. However, *A. punctipennis* according to our crude counting methods outnumbered *A. freeborni* nearly two to one. Outdoors under the shade of the forest canopy both species were collected attempting to bite in the afternoon between 3:30 and 7:00 p.m. Other species noted included *Aedes varipalpus, Culiseta incidens* and *Culex tarsalis*. Although the *Anopheles* were found in considerable numbers at the four camps, the Piedmont camp which is located furthest from the lake on a hill had the lightest infestation. This finding was interesting because this was the only camp where malaria cases did not occur. A total of 8 *A. punctipennis* and 8 *A. freeborni* were dissected and examined for oocysts but all proved negative.

The major source of mosquitoes for the area was traced to the lake, which incidentally might be better classified as a pond because its area is only 15 acres. *Anopheles* larvae averaging about 10 per dip were taken in all the shallow areas less than two feet deep where emergent aquatic vegetation and floating mats of algae were present. These favorable mosquito source conditions comprised about 20% of the total lake area. Other sources were found at the outlets of Rock and Brush Creeks which flow into the upper part of the lake. A small sample of larvae and pupa were reared and identified as *Anopheles punctipennis* and *A. freeborni.*

Our findings in late August do not necessarily represent conditions existing during the July 4th weekend when the Korean veteran visited the lake. The only information available offering a clue to the mosquito situation in early summer is contained in a Bureau of Vector Vontrol report by Harvey I. Magy who made a survey at Lake Vera in early June of 1950 in response to a request of the Camp Fire Council directors who were interested in a control program at that time. Mosquitoes observed in the adult stage according to the report included *Anopheles punctipennis, Culex tarsalis, Culiseta incidens, Aedes varipalpus,* and *Aedes increpitus*. Both *Anopheles punctipennis* and *A. freeborni* were taken in the larval stage in the lake. Of particular interest is the fact that this report to the Camp Fire officials included pertinent recommendations for conducting a control program. Unfortunately the directors failed to forestall the consequences of their 1952 experience by not adopting these recommendations. If they had I'm certain this panel would not be seated here today.

There is of course nothing unusual about finding a preponderance of *A. punctipennis* in this Mother Lode county at 2500 feet elevation. Prof. W. B. Herms in his report on a state-wide mosquito survey carried out in 1916 also observed a considerably larger proportion of *A. punctipennis* in the Anopheline population of the Mother Lode counties and suggested that malaria among the miners in the early days may have been transmitted by this species.

The demands of the *Culex tarsalis* emergency control program incident to the encephalitis epidemic of 1952 forced us to limit our survey at that time to a single day's observations. However, the time spent appeared adequate to size up the major problem conditions and to draw general conclusions for a mosquito control program in preparation for the 1953 camping season. For example, it was evident that a project involving resloping and realignment of the shoreline and deepening or filling in shallow areas of the lake to eliminate the extensive, dense growths of emergent vegtation was a basic need. Fortunately there was no problem to accomplishing the job by bulldozing because of the routine practice of draining the lake in the fall by removing the flashboards at the dam, and refilling in the spring before opening of the camp season, allowed at least a six-month period when earthmoving equipment could be used.

The need for some larviciding work to supplement permanent control was clearly evident not only in the lake but for controlling the scattered, minor sources in Brush and Rock Creeks and in the area surrounding the camps. A residual DDT spraying of the permanent buildings also appeared desirable in order to effect an immediate reduction of the large population of *Anopheles.*

The Camp Fire officials were advised on control methods and procedures by the Bureau of Vector Control staff and eventually succeeded with considerable aid from the Alameda County Mosquito Abatement District in carrying them out to the letter, not only at the lake but for a one-mile radius surrounding the four camps.

Some of the highlights of the control activities and the results achieved in terms of mosquito reduction may be of interest.

The first operation performed was residual spraying of the permanent buildings in October 1952 resulting in complete disappearance of adults from all of the buildings shortly thereafter. The following spring on April 20 only two *A. freeborni* females were found after a prolonged search in nearly all of the buildings in the area. Undoubtedly they were overwintering survivors of the 1952 population. The first evidence of aquatic stages was observed on the same day. A moderate infestation of *Anopheles* 1st instar larvae and *Culiseta* 4th instar larvae, averaging about 4 per dip, were recovered in shallow seepage pools in the drained lake bottom. We are certain that this represented the first generation in the area. These pools were sprayed with DDT by the Camp Fire officials and no further larvae were noted until after the lake was refilled on June 18. Refilling of the lake was