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A FILARIASIS-CONTROL PROGRAM IN AMERICAN SAMOA*

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ABSTRACT: Clinical and blood surveys were made in four villages of Tutuila, American Samoa, in 1962 before and after treatment with diethylcarbamazine (DEC) in order to determine the current prevalence of filariasis, investigate continued transmission, assess the need for control, and evaluate dosages and schedules for drug administration. The microfilaria (mf) rate was 20.4%, the elephantiasis rate 3.4%, and no spontaneous decline of clinical or hematologic filariasis had apparently occurred during the previous 20 years. Therefore, mass treatment with DEC was recommended to a total dose of 72 mg per kg of body weight. This dose reduced the mf rate to 3.9% by the end of the first year, but, regardless of the schedule adopted for its administration, appeared insufficient for adequate control. However, because reductions of mf rate in two villages receiving identical regimens of DEC appeared proportional to their pretreatment level of mf infection, it was suggested that further treatment with DEC be administered to determine its subsequent effect upon mf rates and transmission.

In 1962, at the request of the Government of American Samoa, the Center for the Health Sciences, University of California, Los Angeles, conducted a field study to evaluate the public-health importance of subperiodic filariasis in the Island of Tutuila, American Samoa.

Complete clinical, blood, and mosquito surveys were performed in four experimental villages during that summer, for the purpose of: 1) collecting baseline data on the current prevalence of clinical and hematologic filariasis in the population, 2) evaluating the problem of continued transmission, 3) conducting preliminary therapeutic trials with diethylcarbamazine (DEC), and 4) assessing the potential need for mass treatment of the population with that drug. It was also hoped that the pilot control program would provide the basic guidelines needed for future treatment plans, should these become necessary.

This report is concerned exclusively with the results of the clinical and blood surveys performed in the four villages before and after treatment with DEC. The mosquito surveys have already been reported in part elsewhere.[1]

METHODS

As shown in Figure 1, two of the villages, Aoa and Amouli, are situated on opposite shores at the eastern end of the island and two, Malaeloa and Amanave, toward the western side, near the coast. Nearly the entire population of the villages, 1,008 persons, was examined; the 487 females and 521 males ranged in age from 1 to 82 years (1960 U.S. Census estimate: Aoa 202, Amouli 293, Amanave 269, and Malaeloa 336, a total of 1,100 persons.) The distribution of age and sex in the four villages was comparable and showed a preponderance of younger and older persons, with a relative scarcity of mature adults of both sexes, reflecting the steady emigration of persons of working age from the island.

A complete physical examination was made on all persons over 1 year of age in each village; subjects were interviewed by Samoan nurses regarding past and present history of filarial symptoms; the pertinent information was entered in the patients' records together with the results of the physical examination. Particular search was made for signs of lymphangitis, adenopathy, hydrocele, and elephantiasis of the limbs, genitalia, and breasts. However, only those patients exhibiting unequivocal signs of elephantiasis at any site or those with obvious gross hydrocele were included in the final tabulation of the data, borderline or minor cases being excluded.

The frequency of lymph-node enlargement was too high (about 50% of the population surveyed) to produce any significant correlation between this finding and filarial infection. Even when epitrochlear adenopathy alone was considered (about 25% prevalence) no significant correla-

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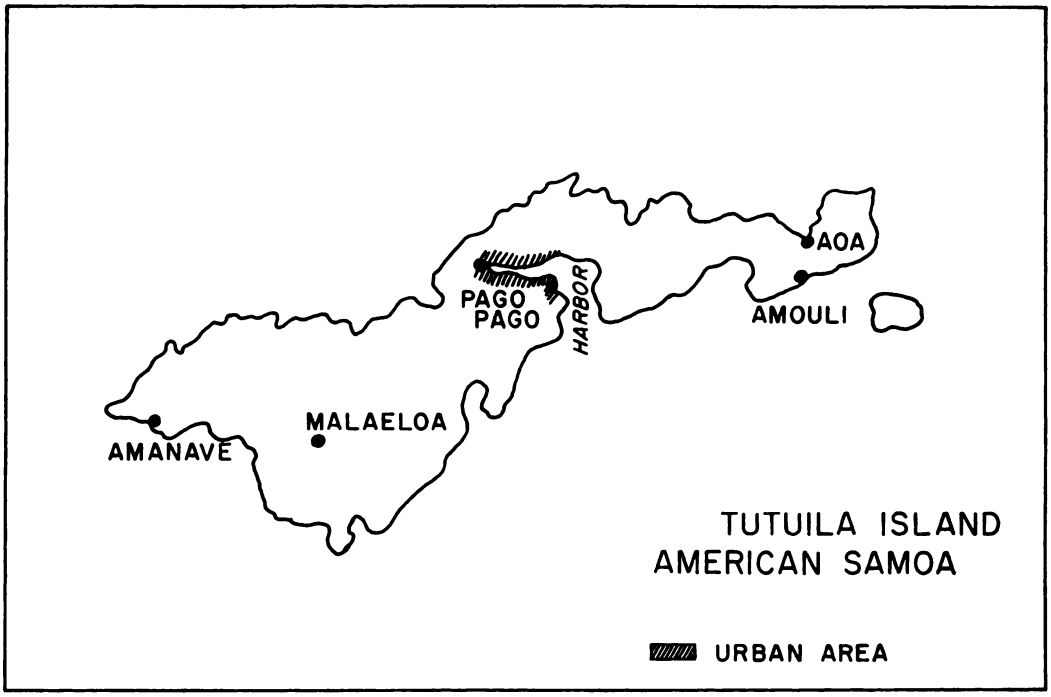

FIGURE 1. Map of Tutuila Island, American Samoa

tion resulted. Consequently, it was decided that these signs would be of little value in evaluating the epidemiology of filariasis in American Samoa. Likewise, the frequency of chyluria (one case) and deep filarial abscesses (three cases) in the sample population was too low to be of any statistical or prognostic value.

Blood tests were made on all persons immediately before treatment with DEC and repeated at regular intervals after treatment over a 3-year period. Each time two separate 20-cmm samples of blood were collected in Sahli pipettes from a finger puncture between the hours of 0800 and 2000. They were spread in thick films and stained according to the technique described by Beye *et al.*[2]

In this report, all counts are expressed as microfilariae (mf) per 20 cmm, the average of the number of mf counted in the two blood films.

After clinical and blood surveys were completed, each person examined, regardless of clinical state, was treated with oral DEC citrate in the amount of 6 mg per kg of body weight administered in a single daily dose. Subjects were accurately weighed on a scale, and the dose was calculated accordingly; adults received the drug in tablet form, children in a cherry-flavored syrup containing 50 mg of DEC per teaspoonful (5 ml).

Three different schedules of DEC administration were followed:

a) Once a day for 6 days; no treatment for 1 year; then repeat same dose once a day for another 6 days. This regimen was applied to Aoa.

b) Once a day for 6 days; no treatment for 6 months; then repeat the same dose once a day for 6 days. This regimen was applied to Amouli and Amanave.

c) Once a day for 6 days, followed by one monthly dose for 6 consecutive months. This regimen was applied to Malaeloa.

The choice of village for the various schedules of administration was random and not dependent upon previous epidemiologic information, but it was so that each person in the villages received a "loading dose" of DEC of 36 mg per kg over a 6-day period, followed by a second dose of 36 mg per kg distributed over a variable period of time. At the end of a year everyone had received an equal amount of the drug. Since no further treatment of any kind was given after the completion

TABLE 1

Prevalence of elephantiasis and hydrocele in Tutuila, American Samoa, 1943–1962

Village	Sex	Elephantiasis (Whole population) 1962 No.*	%	Dickson 1943 No.*	%	Murray 1945 No.*	%	Hydrocele (Male population) 1962 No.*	%	Dickson 1943 No.*	%
Aoa	M	6/83	7.2	↑				8/83	9.6	↑	
	F	2/92	2.1								
	All	8/175	4.5			8/149	5.4				
Amouli	M	4/145	2.5					11/145	7.5		
	F	1/123	0.8								
	All	5/268	1.8			1/121	0.8				
Amanave	M	6/145	4.1					5/145	3.4		
	F	4/124	3.2								
	All	10/269	3.7			3/118	2.5				
Malaeloa	M	7/148	4.5					13/148	8.8		
	F	5/148	3.4								
	All	12/296	4.0			4/166	2.4				
Totals	M	23/521	4.4	Not available by village				37/521	7.1	78/1,238	6.3
	F	12/487	2.5								
	All	35/1,008	3.4	57/2,171	2.6	16/554	2.8				
Population over 30 yrs.	All	29/243	11.9	54/503	11.0	16/171	9.3	34/115	29.5	54/356	15.1

* Numerator, number positive; denominator, number examined.

of the schedules above, it was hoped that some information would be gained regarding the most effective schedule for DEC administration in a mass control program.

RESULTS OF PRETREATMENT SURVEYS

Clinical Surveys

The results of the clinical examinations are summarized in Table 1, which shows the current prevalence of elephantiasis and hydrocele in each village compared with data obtained from observations made previously by Dickson[3] and by Murray.[4] The over-all prevalence of elephantiasis in the entire population was 3.4%, compared with 2.6% in 1943 and 2.8% in 1945. When only persons over the age of 30 were considered, the prevalence of elephantiasis in 1962 (11.9%) was similar to that reported by Dickson (11%) and by Murray (9.3%).

Elephantiasis rates were generally higher in males than females in the four villages. This fact had been recognized previously in Samoa[4] and other endemic areas and had been explained by the higher prevalence of filarial infection among males. The difference of 1.9% between the over-all rates in the two sexes was statistically significant ($z = 1.66$; $p = 0.097$) and therefore is probably real for this population sample, even though no difference in microfilaria (mf) rate by sex was found (*vide infra*).

The prevalence of hydrocele in the male population was 7.1%, not significantly different from that observed by Dickson in 1943 (6.3%).

Although the difference in hydrocele rates for males over the age of 30 between the two surveys, *i.e.*, 29.5% versus 15.1%, was statistically significant ($z = 3.092$; $p < 0.01$), the observation may only reflect the thoroughness of the present examination rather than an actual difference. The data nevertheless suggested that no significant decrease in clinical filariasis, as judged by elephantiasis and hydrocele rates, had occurred in the island during the intervening period of observation.

Acute lymphangitis or a history of lymphangitic attacks was recorded in about 10% of the population surveyed, a prevalence lower than expected considering the high prevalence of these condi-

TABLE 2

Frequency distribution of mf carriers by age and by sex in American Samoa, 1962

Age				Sex					
				No.*		% + mf rate		Median mf count/pos.	
Age groups (*years*)	No.*	% + mf rate	Median mf count/pos.	Male	Female	Male	Female	Male	Female
1–4	2/190	1.3	7	1/97	1/93	1	1	1	15
5–9	26/196	13.0	24	8/113	18/83	7	21	8	26
10–14	31/179	17.0	12	16/97	15/82	16	18	10	12
15–19	22/97	23.0	15	16/59	6/38	27	16	15	52
20–24	17/58	29.0	15	5/25	12/33	20	36	26	7
25–29	13/45	29.0	33	9/15	4/30	60	13	33	47
30–34	14/37	37.0	17	5/14	9/23	35	39	148	7
35–39	14/41	34.0	13	4/13	10/28	30	35	40	13
40–44	18/45	40.0	15	13/23	5/22	56	22	21	15
45–49	9/24	37.0	7	6/13	3/11	46	27	5	101
50–54	12/31	39.0	17	7/15	5/16	46	31	46	15
55–59	10/23	43.0	51	6/12	4/11	50	36	51	50
60+	16/42	38.0	30	7/25	9/17	30	53	48	26
Totals:	204/1,008	20.4	26	103/521	101/487	19.9	20.9	33	31

* Numerator, number positive; denominator, number examined.

tions in neighboring places, *i.e.*, as reported by Beye *et al.* from Tahiti.[5]

Blood Surveys

The distribution of mf rates and median mf counts by age and by sex is shown in Table 2. The age-group distribution of mf rates showed a progressive increase from childhood until about age 40, to remain relatively constant thereafter. The mf rate for the entire sample was 20.4%, with no significant difference in mf rates between males and females. Although the age-adjusted mf rates for females appear generally lower than for males, none of the observed differences was statistically significant, and the over-all rates in the two sexes were also similar: 19.9% and 20.9%, respectively.

The distribution of median mf counts by age groups (Table 2) disclosed no definite pattern in relation to any particular age group; likewise, the over-all median counts in the two sexes were similar, 33 and 31 mf, and close to the median count for the entire sample, which was 26 mf per 20 cmm of blood.

Therefore, it could not be shown that the distribution of median mf counts in this population followed any specific pattern with regard to age groups or sex.

The cumulative percentage distribution of mf carriers by actual counts and by sex is shown in Table 3; it can be seen that the 50th percentile mf count (MfD-50) falls between 20 and 30 mf per 20 cmm of blood, which is very close to the actual median mf count of the sample, *i.e.*, 26 mf per 20 cmm, but quite removed from the

TABLE 3

Cumulative percentage distribution of mf carriers in American Samoa, 1962

Mf count per 20 cmm blood	Cumulative distribution by mf counts (*No.*)	(*Cum. no.*)	(*Cum. %*)	Cumulative % by sex Males	Females
1	22	22	10.7	8.7	12.9
2	8	30	14.7	10.3	18.9
3	8	38	18.6	14.2	21.7
4	9	47	23.0	22.3	25.6
5	9	56	27.4	26.2	30.6
6	6	62	30.3	29.1	31.6
7	4	66	32.3	29.1	35.6
8	3	69	33.5	31.0	36.7
9	3	72	35.2	34.0	36.7
10	1	73	35.5	35.9	36.7
11–20	25	98	48.0	45.5	50.2
21–30	14	112	54.9	52.5	57.4
31–40	10	122	59.8	59.1	60.3
41–50	12	134	65.6	64.0	67.4
51–60	9	143	70.0	67.0	73.1
61–70	7	150	73.5	68.9	78.0
71–80	6	156	76.4	72.0	80.1
81–90	3	159	77.9	72.8	81.2
91–100	3	162	79.4	73.7	84.0
101–200	26	188	92.1	88.5	94.0
201–300	10	198	97.0	94.0	98.0
301–400	4	202	99.0	98.0	99.0
401–500	1	203	99.5	99.0	99.0
501–600	0	203	99.5	99.0	99.0
601–700	1	204	100.0	100.0	100.0

TABLE 4

Prevalence of microfilaremia above age five in Tutuila, American Samoa, 1948–1962

Village	1945				1962					
	No.*	% +	Total no. mf/ 20 cmm	Mean mf count per positive	No.*	% +	Total no. mf/ 20 cmm	Mf count per 20 cmm Mean	Median	Range
Aoa	35/149	23	2,702	77	31/140	22	2,356	76	33	1–670
Amouli	17/121	14	1,597	93	39/206	19	1,591	39	12	1–385
Amanave	24/118	20	857	35	58/223	26	4,399	75	33	1–384
Malaeloa	23/166	14	560	24	74/250	29	4,628	62	24	1–618
Totals:	99/554	17	5,716	57	202/819	24	12,974	64	26	1–670

* Numerator, number positive; denominator, number examined.

average (mean) mf density per positive, which was 64 mf per 20 cmm of blood. Thus it appears that the median mf count is a more realistic and comparable measurement than mf densities for the evaluation of mf infection in a population.[6]

The cumulative percentage distribution of mf counts by sex shows that the MfD-50 for males falls between 21 and 30 mf per 20 cmm and for females between 11 and 20 mf per 20 cmm, but in essence the two series parallel each other quite closely.

Table 4 compares the results of the present blood survey with those obtained by Murray in 1945[4] on persons over the age of 5 in each of the four villages. It can be seen that the over-all mf rate was 17% in 1945 compared with 24% in 1962 and that the mean mf density per carrier was 57 in 1945 compared with 64 in 1962. As the difference in mf rates observed was statistically significant ($z = 3.203$; $p < 0.01$), it seems justified to assume that some increase in mf rates had occurred in these villages between 1945 and 1962.

RESULTS OF POST-TREATMENT SURVEYS

Clinical Surveys

The percentage of persons who completed successfully the first 6-day course of treatment averaged 72% of the four villages, and 73% completed the second phase of the treatment as scheduled. Thus about three-quarters of the people completed the entire course, with the balance receiving less than the prescribed amount. Only 2% of the population escaped treatment entirely.

Reactions to the first administration of DEC were encountered in 53% of persons with positive blood tests and in 11% of those with negative films; a small number of the latter, however, had clinical signs of filariasis.

The frequency of drug reactions was highest during the first and second days of treatment with proportions as follows: chills and fever 29%, headache and myalgia 28%, nausea and vomiting 18%, anthelmintic effect 15%, and lymphangitis, edema, and pruritus, 10%. The percentage of adverse reactions reported with subsequent drug administration was less than 5% for the entire population, and these were of a minor nature.

In June 1964, a follow-up physical examination was made in order to detect any changes of the clinical parameters considered initially. The comparative results between the two surveys in a group of mf carriers who completed the required course of therapy are shown in Table 5. The data indicated little or no change in the percentage of persons with elephantiasis or hydrocele 2 years after treatment with DEC. A significant reduction in the frequency of reported lymphangitic attacks was noted in all villages, however, suggesting that perhaps some improvement of this condition can be achieved soon after

TABLE 5

Distribution of clinical filariasis before and after treatment with diethylcarbamazine in a group of mf carriers in American Samoa, 1962–1964

Clinical parameter	1962		1964	
	No.*	% +	No.*	% +
Acute lymphangitis	16/160	10.0	0/160	0.0
Elephantiasis	8/160	5.0	9/160	5.6
Hydrocele	10/160	6.2	8/160	5.0
Adenopathy	50/160	31.3	25/160	15.6

* Numerator, number positive; denominator, number examined.

TABLE 6

Distribution of mf carriers before and after treatment with diethylcarbamazine in four villages, American Samoa, 1962–1965

Village	Before treatment August 1962				1 week after treatment August 1962				January 1963			July 1963			June 1964			June 1965		
	No.*	% + mf rate	Mf/+ count mean median range	1st Rx No. completed Rx	No.*	% + mf rate	Mf/+ count mean median range	2nd Rx No. completed Rx	No.*	% + mf rate	Mf/+ count mean median range	No.*	% + mf rate	Mf/+ count mean median range	No.*	% + mf rate	Mf/+ count mean median range	No.*	% + mf rate	Mf/+ count mean median range
Aoa			76				1.4				6.0			8.2			1.0			4.4
	31/175	21.4	33	133/175	9/174	5.1	1	117/175	13/154	8.4	1	11/148	7.4	2	3/156	1.9	1	5/146	3.4	3
			1–670	76%			1–4	67%			1–49†			1–45†			0–1			1–10
Amouli			39				2.7				3.0			1.5			2.5			5.2
	40/268	15.0	12	177/268	11/266	4.1	1	204/268	19/216	8.8	2	10/211	4.7	1	6/210	2.8	1	7/214	3.2	1
			1–385	65%			1–10	76%			1–12			1–3			1–6			1–22†
Amanave			75				1.7				3.5			3.0			6.6			12.3
	59/269	21.6	33	196/269	22/265	8.2	1	208/269	26/240	10.8	3	7/200	3.5	2	19/234	8.1	3	25/233	10.6	2
			1–384	73%			1–5	77%			1–15†			1–9			1–24			1–107†
Malaeloa			62				1.4				9.0			1.5			2.1			5.4
	74/296	25.0	24	219/296	17/290	5.8	1	208/296	8/268	2.9	1	2/208	0.9	1	10/247	4.0	1	25/233	10.6	2
			1–618	74%			1–5	70%			1–62†			1–2			1–4			1–55
Totals:			64				1.8				4.5			4.3			4.3			8.1
	204/1,008	20.2	26	725/1,008	59/995	5.6	1	737/1,008	66/878	7.5	1	30/767	3.9	1	38/847	4.5	1	62/826	7.5	2
			1–670	72%			1–10	73%			1–62†			1–45†			1–24			1–107†

* Numerator, number positive; denominator, number examined.
† Includes untreated persons.

beginning treatment with DEC. The 50% reduction in palpable adenopathy observed 2 years after treatment could not be properly evaluated for reasons already stated.

Blood Surveys

After therapy, each village was surveyed *in toto* by means of blood films at regular intervals: 1 week after the initial course, 6 months (January 1963), 1 year (July 1963), 2 years (June 1964), and 3 years later (June 1965). The results obtained at each survey are shown in Table 6.

It should be noted that not all persons in the original cohort were available for examination at each subsequent survey; therefore the denominators shown in the Table are not constant; the percentages, however, were calculated in each instance from the available members of each original group and are therefore comparable.

The data in Table 6 show a prompt and pronounced decline in mf rates and densities 1 week after the first administration of DEC, of about the same degree in all four villages. Six months after the beginning of therapy there was a slight increase of mf rates in the three villages that had received no additional treatment and a further decrease in Malaeloa, which had received monthly doses of DEC; the median counts per positive, however, remained similar in all four villages. In July 1963, that is, 6 months after the administration of a second course of DEC to Amouli and Amanave, a substantial decline of mf rates was noted in these villages and also in Malaeloa, although the latter had received no additional therapy. In Aoa, which also had received no further treatment, the mf rate remained about the same.

In June 1964, the mf rate declined sharply in Aoa after the administration of the second six doses of DEC, while it rose slightly in Malaeloa and Amanave; a further decline also occurred in Amouli, even though no additional treatment had been given. In June 1965, the mf rates in Aoa and Amouli were comparably low, whereas in Amanave and Malaeloa they had risen to comparably higher levels. Median mf counts per positive, however, remained generally very low and did not vary significantly among the villages.

Of the two villages receiving the same treatment schedule, Amouli showed lower mf rates and counts than Amanave in June 1964 and 1965. The difference in mf rate between the two villages observed in 1965 was significant at the 1% level ($z = 3.185$; $p < 0.01$). As Amanave had a higher pretreatment mf rate and mf counts per positive than Amouli, it seemed reasonable to suspect that the observed difference was related to variances in the pretreatment level of mf infection between the two villages. The present data indicated that for an equal amount of DEC administered in the four villages, the one with lower initial rates of mf infection, Amouli, showed the best therapeutic results at the end of the observation period.

An analysis of the 62 mf carriers uncovered in June 1965 showed that 41, or 66%, were original mf carriers who received DEC therapy but remained in or reverted to a positive state subsequently, while 18, or 29%, were originally negative but had patent infections some time after treatment was given. Also three more persons, or 5%, who were originally negative and had escaped treatment, became positive at the end of the 3 years. These percentages are similar to those reported from Africa,[7] Japan,[8] and Tahiti[9] after DEC therapy and suggest that two-thirds of the recurrences are from relapses of pre-existing microfilaremia and one-third from newly acquired or latent infections that become patent during the period of observation. Thus it would seem more advantageous, whenever possible, to treat the entire population as suggested by Sasa *et al.*[10] rather than carriers alone.

It is also apparent from these data that the total amount of DEC of 72 mg per kg of body weight, regardless of how spaced, was not sufficient to eradicate filarial infections of the degree encountered in American Samoa and that additional doses will be needed if eradication is to be achieved.

DISCUSSION

Although no organized effort to control filariasis had been made in American Samoa during the past two decades, sporadic attempts at sanitation campaigns, garbage collection, mosquito abatement, and random administration of DEC to sick persons had occurred in Samoa during this interval.[11] The results in Tables 1 and 4, however, show that no significant reduction in the prevalence of clinical or hematologic filariasis has occurred in the villages tested since they were

surveyed by Dickson[3] and by Murray:[4] if anything, there may have been some increase of certain indices of filarial infection. Therefore it seemed warranted to recommend an island-wide control program based upon mass administration of DEC. Such a program was started in 1963.

The therapeutic trial given the four experimental villages amply confirmed the immediate and pronounced reduction of mf rates and densities that follows the first administration of DEC to an infected population. Improvement of certain clinical manifestations, such as episodes of acute lymphangitis, also was observed over a 2-year period. These results paralleled those reported from other endemic areas where mass treatment with DEC has already been applied.[8, 12, 18]

No demonstrable reductions in rates of hydrocele and elephantiasis were observed, but these chronic conditions usually are not affected directly by DEC treatment; these indices change much later, as noted by March *et al.*[14] and confirmed recently by Laigret *et al.*[15] when older infected persons disappear from a community in which fewer or no new cases occur.

Concurrently with these results, however, our long-term observations disclose a persistency or a slow return of very light mf infections in a number of persons treated with DEC, as reported also from other endemic areas[7, 8, 13, 15] where DEC has been used. The problem in Tahiti was reviewed by Laigret *et al.*,[9] who, reasoning from circumstantial evidence, labeled those cases as recurrences or relapses of microfilaremia rather than reinfections, or new infections, and related them to the interaction of a number of factors ranging from inadequate treatment of some carriers to the survival of adult parasites and their regained fertility. Our data show that most recurrences are likely to be relapses of pre-existing microfilaremia, *i.e.*, temporary failures of treatment resulting either from insufficient ratio between drug dosage and worm load or from inadequate blood levels of the drug. The latter could result from poor absorption, rapid excretion, or increased metabolic degradation of the drug, which, in turn, could conceivably lead to the progressive selection of DEC-resistant strains of mf, thus posing a serious threat to the future of filariasis-control programs. However, thus far no evidence to support this possibility has been produced, nor has the use of intramuscular DEC eliminated the problem of mf relapses,[16] as would happen if the defect were faulty intestinal absorption. Therefore, it seems likely that the mechanism involved is simply that of insufficient dosage of DEC in relation to the pre-existing worm load. In another study[17] it was shown that individual relapses of microfilaremia were closely correlated with the pretreatment mf density of each carrier, in that relapses after DEC treatment occurred most frequently among those carriers with initially high mf counts. The present observations suggest that there is a similar relation between the amount of drug administered and the pretreatment level of mf infection of the villages treated. For an equal amount of DEC administered in each village, the therapeutic results obtained at the end of a 3-year period of observation showed a statistically significant correlation with the degree of mf infection before therapy in at least two of the four villages. No correlation, however, could be found between the final results of treatment and the three schedules of DEC administration used in this study. Although it appeared that, in the initial phases of this study, a regimen of six monthly doses administered after a 6-day course produced a more pronounced and sustained decrease of mf rates than two 6-day courses administered 6 months apart, later on this advantage was lost.

The data in Table 6 show that in the long run, without any additional DEC therapy, similar results were achieved in two pairs of villages receiving entirely different schedules of DEC administration. These observations suggested that factors other than schedules of administration per se had influenced the results of the experiment, lending further support to the hypothesis that the pretreatment level of microfilaria infection in the population was the determining factor. Thus it seems justified to suggest that, in mass control programs, the total amount of DEC and the length of treatment be dictated by the pretreatment level of mf infection in the population at risk and that the schedule of administration be left to individual choice.

Further studies after additional administration of DEC in the four experimental villages should prove of great interest in assessing the correctness of these assumptions.

SUMMARY

Complete clinical and blood surveys were conducted in 1962 in four villages of Tutuila Island, American Samoa, with the purpose of assessing the public-health importance of filariasis in the island and evaluating the need for mass treatment of the population with diethylcarbamazine.

Pre-treatment surveys showed that no over-all decrease of clinical or hematologic filariasis had occurred since 1943–45, when the same villages were surveyed by Dickson and Murray, in the absence of a formal control program. The prevalence of elephantiasis in the population surveyed was 3.4% compared with 2.6% in 1943 and 2.8% in 1945. The hydrocele rate in the male population was 7.1% compared with 6.3% in 1943. The mf rate was 20.4%, and the mean and median mf count per positive were respectively 64 and 26 mf per 20 cmm of blood, compared with an mf rate of 17% and mf density of 57 in 1945.

Persons in the villages were treated with DEC to a total dose of 72 mg per kg distributed according to three schedules. In essence, all villages received one-half of the dose over a period of 6 consecutive days and the other half over the ensuing 6- to 12-month period. Post-treatment surveys indicated a prompt reduction of mf rates and densities in all villages, more pronounced initially in the village receiving monthly doses; later on, however, mf rates and counts reached comparable levels in the four villages regardless of the schedule adopted. It was concluded, therefore, that in long-term control programs and in the absence of additional therapy, the schedule of administration was not the most significant factor in the reduction of mf rates. The data, instead, suggested that there was some correlation between the degree of filarial infection in the villages before treatment and the results obtained 3 years after treatment, in that the best results appeared in the village with the lowest initial mf rate and counts, when given the same amount of DEC over the same period of time.

As the data also showed that most positives after DEC therapy occurred among original carriers, it was suggested that temporary therapeutic failures are probably the results of an inadequate ratio between dosage of DEC and pre-existing worm burden. The conclusion was therefore advanced that the total amount of DEC and the length of treatment should be guided by the degree of mf infection of the population at risk and that additional therapy be continued for as long as necessary to achieve eradication.

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REFERENCES

1. Kessel, J. F., 1967. Diethylcarbamazine in filariasis control. *Proc. & Papers 35th Ann. Cong. Calif. Mosq. Contr. Assoc., & Am. Mosq. Contr. Assoc.*, February, pp. 17–22.
2. Beye, H. K., Edgar, S. A., Mille, R., Kessel, J. F., and Bambridge, Ben, 1952. Preliminary observations on the prevalence, clinical manifestations and control of filariasis in the Society Islands. *Am. J. Trop. Med. & Hyg., 1:* 637–661.
3. Dickson, J. G., 1943. Annual sanitary report of the Public Health Department of American Samoa.
4. Murray, W. D., 1948. Filariasis studies in American Samoa. *U. S. Nav. Med. Bull., 48:* 327–341.
5. Beye, H. K., Kessel, J. F., Huels, J., Thooris, G., and Bambridge, Ben, 1953. Nouvelles recherches sur l'importance, les manifestations cliniques et la lutte contre la filariose à Tahiti, Océanie Française. *Bull. Soc. Path. Exot., 46:* 144–163.
6. WHO Expert Committee on Filariasis, 1967. *Wuchereria* and *Brugia* Infections. *World Health Organ. Tech. Rep. Ser.* 359, Geneva.
7. McGregor, I. A., and Gilles, H. M., 1956. Diethylcarbamazine control of Bancroftian filariasis. Follow-up of a field trial in West Africa. *Brit. Med. J., 1:* 331–332.
8. Sasa, Manabu, 1963. Pilot experiments in the control of Bancroftian filariasis in Japan and Ryukyu. *Bull. World Health Organ., 28:* 437–454.
9. Laigret, Jacques, Kessel, J. F., Malarde, Louis, Bambridge, Ben, and Adams, Herald, 1965. La lutte control la filariose lymphatique apériodique en Polynésie Française. *Bull. Soc. Path. Exot.*, 58: 895–916.
10. Sasa, M., Hayashi, S., and Tanaka, H., 1960. Experimental studies on filariasis control in Japan. *Ind. J. Malar., 14:* 441–456.
11. Annual Report of the Governor of American Samoa to the Secretary of the Interior, for

1961, pp. 43–45. U.S. Gov. Print. Office, Washington, D. C.

12. Kessel, J. F., and Massal, Emile, 1962. Control of Bancroftian filariasis in the Pacific. *Bull. World Health Organ., 27:* 543–554.
13. Marshall, C. L., and Yasukawa, Keitetsu, 1966. Control of Bancroftian filariasis in the Ryukyu Islands: Preliminary results of mass administration of diethylcarbamazine. *Am. J. Trop. Med. & Hyg., 15:* 934–942.
14. March, H. N., Laigret, Jacques, Kessel, J. F., and Bambridge, Ben, 1960. Reduction in the prevalence of clinical filariasis in Tahiti following adoption of a control program. *Am. J. Trop. Med. & Hyg., 9:* 180–184.
15. Laigret, J., Kessel, J. F., Bambridge, B., and Adams, H., 1966. Onze ans de chimioprophylaxie par la diéthylcarbamazine de la filariose lymphatique apériodique à Tahiti. *Bull. World Health Organ., 34:* 925–938.
16. Laigret, J., Davin, R., Bagnis, R., Outin, D., Cardines, R., and Bambridge, B., 1966. Rapport annuel de l'Institut de Recherches Médicales de la Polynesie Française. Personal communication.
17. Ciferri, F. E., and Kessel, J. F., 1967. Relation of age, sex, and microfilaria density to treatment of sub-periodic filariasis with diethylcarbamazine. *Am. J. Trop. Med. & Hyg., 16:* 321–328.