

Table 4. Rate of survival of first nymphal stage of *Pediculus humanus humanus* while fed on nonspecific hosts.

Time (days)	Host							
	Mouse	Hamster	Pig	Rat	Guinea pig	Chicken	Pigeon	
0	300	120	100	100	100	100	100	
1	280	113	88	—	—	59	35	
2	189	96	—	Nymph 1		20	11	
3	148	57	-----				4	1
4	106	38	-----				—	—
5	53	12	-----				Nymph 2	
6	22	5	-----				Nymph 3	
7	10	3	-----				Adult	
8	7	2	-----				Adult	
9	—	2	-----				Adult	
10	—	—	-----				Adult	

REFERENCES

- CABASSO, V. Reaction of the human body louse (*Pediculus humanus corporis*) to the ingestion of guinea pig blood. *Proc Soc Exp Biol Med* 64:437-39, 1947.
- CULPEPPER, G. H. Rearing and maintaining a laboratory colony of body lice on rabbits. *Am J Trop Med Hyg* 28:499-504, 1948.
- FAHRENHOLZ, H. Bemerkungen zu der arbeit G. Schwalbe's "Über die bedeutung de äusseren parasiten für die phylogenie der säugetiere und des menschen." *Z Morph Anthropol* 21:361-64, 1921.
- GEISSEL, H. Wie lange kann man kleiderläuse und schweineläuse bei dauerfütterung auf unspezifischen wirtten halten? Thesis, University of Heidelberg, 1970.
- HÄFNER, P., and H. W. LUDWIG. Eine methode zur membranfütterung der schweineläus *Haematopinus suis*. *Z Parasitenkd* 33:177-82, 1969.
- KRYŃSKI, STEFAN, et al. Badania nad istotą szkodliwego dziakania krwi świnki morskiej na wesz odzieżową. *Biul Panst Inst Med Morsk Trop Gdansku* 4:97-100, 1952.
- LUDWIG, H. W., and M. Thiemes. Zucht der schweineläus *Haematopinus suis* auf mäusen. *Z Parasitenkd* 30:176-78, 1968.

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Stefan Kryński.¹ *Influence of individual features and environmental temperature on the development of infection and intoxication in lice.* The present work attempts to provide an answer to several questions, namely: (1) do all lice belonging to one defined population react similarly to a harmful agent or are there any individual differences among them; (2) what role in the degree of the louse's sensitivity is played by sex, age, and the duration of starvation; (3) can a louse organism counteract an infection during its development, and (4) what is the influence of environmental temperature on

the response of lice to infection or intoxication?

Methods and materials

The lice that we use in our investigations originated from the laboratory colony founded by Weigl in 1918 (16, 17). In 1939 they were crossed with lice from the Laboratorio per la Profilassi e lo Studio delle Rickettsiosi (Laboratory for Rickettsiosis Prophylaxis and Research) in Addis Ababa, Ethiopia. They are kept at 32° C and fed once daily on human volunteers. Adult insects 12 days old are used for experiments 20 hours after the last feeding. Following intrarectal injection of the bacterial

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suspension or drug, lice are fed from one to three hours later (6).

Results

Individual differences in the responses of lice to infection and toxic action of antibiotics

Lice given 10^4 total lice infecting doses (LID_{100}) of *Rickettsia prowazekii* (10, 13) turn red and usually die between the fourth and sixth day of infection (4, 6, 8). Our strains, cultivated since 1945 exclusively in lice, kill them within four days. Lower doses prolong both the lag phase and the logarithmic one of *R. prowazekii* growth, causing the survival time of the infected insects to be correspondingly longer (13). The results presented in Table 1 demonstrate the existence of hundredfold differences in the degree of infection in red lice of the population inoculated with the same suspension. The appearance of a red color due to the permeation of undigested hemoglobin through the rickettsial toxin-damaged epithelium (2, 9, 18) is found in some lice by the number of

living rickettsial cells being equal to 10^5 LID_{100} , while in others the redness still does not occur at 10^7 LID_{100} (Table 2). The survival time of particular individuals differs, especially by lower inocula (Figure 1) and

Table 1. Degree of infection of red lice inoculated with 10^4 LID_{100} of *Rickettsia prowazekii* per louse.

LID_{100}	Day of infection	
	3d	4th
10^7	52	80
10^6	28	16
10^5	20	4

Table 2. Degree of infection of lice four days after inoculation with 10^2 LID_{100} of *Rickettsia prowazekii* per louse.

LID_{100}	Redness of lice	
	+	-
10^7	8	2
10^6	78	32
10^5	14	64
10^4	0	2

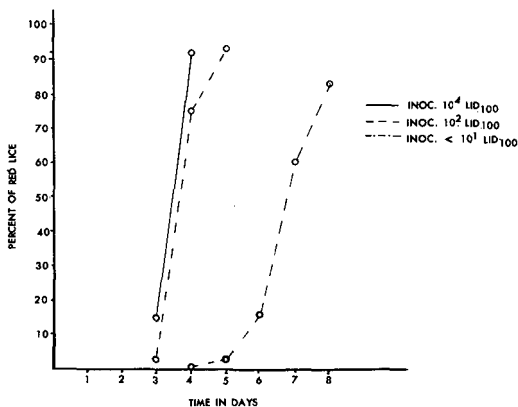


Figure 1. Mortality of lice in relation to inoculum size of *Rickettsia prowazekii*.

strains of *R. prowazeki* less virulent for lice. Doses higher than 10^5 LID₁₀₀ cause the occurrence of the toxic form of *R. prowazeki* infection (2, 4, 9). The louse becomes red and perishes within a few hours. It may be seen from Figure 2, however, that the toxic form does not emerge in all specimens. Some survive the first 24 hours, and the typical acute form (4) develops and leads to death only after 72 to 96 hours.

In infections due to the intrarectal injections of *Staphylococcus aureus* (14, 15), *Yersinia pseudotuberculosis* (11, 15), and *Y. enterocolitica* (12, 15) suspensions, the particular individuals of a given population display differences both in the survival time (Figures 3 and 4) and the number of the living bacterial cells recorded in them (Figures 5 and 6). The individual disparities relate not only to bacterial infections but also to the toxic action of antibiotics (Table 3).

Role of sex, age, and starvation in louse sensitivity to infection and toxic action of antibiotics

Males are more sensitive to some infections (Figure 5), and females to others (Figure 4). The development of an *R. prowazeki* infection does not show any differences in dependence on the sex of an insect. Survival time after a toxic dose of *R. prowazeki* is a little shorter among males. Becla (1), while investigating the doses of antibiotics lethal to lice, found them to be similar for both sexes.

Depopulation after a toxic dose of *R. prowazeki* is more rapid among older insects than among younger ones (Figure 7). Starvation has been proved to be an important factor affecting the sensitivity of lice to *R. prowazeki* toxin (2) and the toxic action of antibiotics (1, 7).

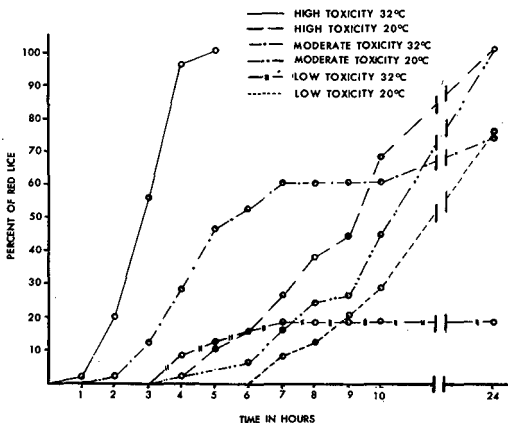


Figure 2. Effect of toxic action of *Rickettsia prowazeki* on lice in relation to environmental temperature.

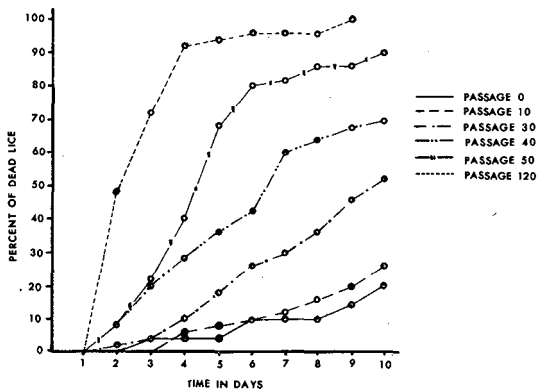


Figure 3. Depopulation among lice infected intrarectally with *Staphylococcus aureus*, strain IIP/Bor.

Counteraction of a louse organism to infection

Following the injection of *Y. pseudotuberculosis* (11, 15), some lice are observed to perish within a few days, whereas others not only survive but may become completely

bacteria-free. *S. aureus* (14, 15) infections are met with only by employment of strains weakly adapted to louse.

The fact that the louse becomes red and perishes in normal infection only when the number of viable *R. prowazeki* is equal to

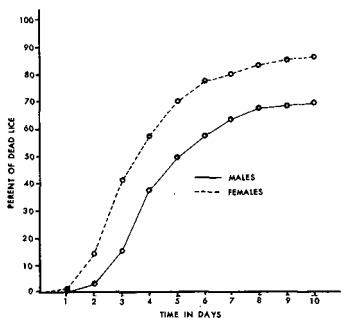


Figure 4. Depopulation among males and females infected intrarectally with *Yersinia enterocolitica*, strain Lucas 232.

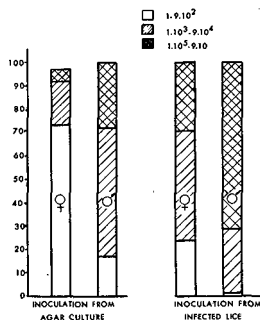


Figure 5. Number of viable bacteria cells in individual lice infected with *Staphylococcus aureus*, strain 1614.

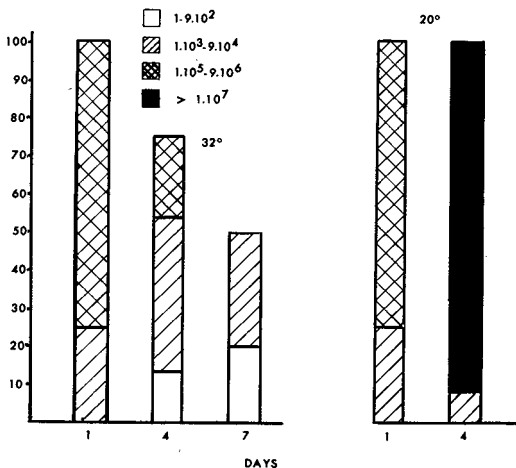


Figure 6. Number of viable *Yersinia pseudotuberculosis*, type I, cells in individual lice in relation to environmental temperature.

10^{6-7} LID₁₀₀ and in toxic form after injection of 10^5 LID₁₀₀ per louse, seems to indicate the appearance of antitoxic activity in intestinal epithelium cells. If lice previously infected with a suspension containing 10^{3-4} LID₁₀₀ per louse additionally receive injections of a suspension containing 10^5 LID₁₀₀ during the lag phase, they react similarly to the healthy individuals, whereas its introduction at the beginning of the logarithmic phase causes no appearance of red lice in the first 24 hours (2).

Influence of environmental temperature on louse response to infection and intoxication

The influence of temperature on the appearance of the toxic form of *R. prowazeki* infection (2, 9) in lice depends on the concentration of a suspension and the virulence of a strain (Figure 2). With higher numbers of viable rickettsiae and more virulent strains there is at 32° C a marked predominance of *R. prowazeki* over the ability of defense mechanisms of the louse. With weaker concentrations and less virulent strains at 32° C,

Table 3. Proportions of dead lice after various doses of antibiotics.

Antibiotic	5.2 ^a	2.6	2.1	1.6	1.0	0.5
Streptomycin	78.8	28.7			4.3	
Chloramphenicol		100	98.7	86.6	52.1	12.2
Oxytetracyclin			67.4	41.0	18.6	2.2
Neomycin		97.9	88.6	72.1	34.1	4.4

^a µg/mg of weight.

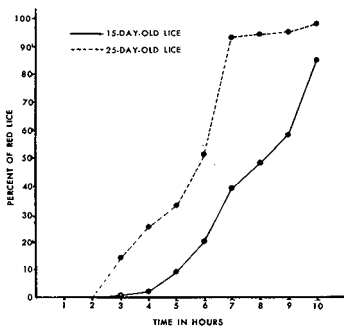


Figure 7. Influence of louse age on depopulation at 22°C after injection of toxic dose of *Rickettsia prowazeki*, strain Victoria.

which is the optimal temperature for lice, their defense mechanism is able to overcome the toxic action of *R. prowazeki*.

At 20° C, *Y. pseudotuberculosis* (11) multiplies in lice more rapidly and intensively, causing total depopulation within four days (Figure 6).

At 34° C, phenol of 0.5 per cent injected intrarectally kills about 6 per cent of lice and

at 20° C more than 90 per cent. Phenol of 0.3 per cent does not kill, but produces only paralysis, which at 34° C recedes considerably after one hour and totally after six hours, while at 20° C it recedes partially after six hours and totally after 24. Antibiotics also display higher toxicity for lice at 20° C (1).

Discussion

Even in our inbred population of lice there are apparent differences in the degree of their sensitivity to infection and intoxication. The differences are probably still greater among the population at large. The louse is not utterly defenseless against factors injurious to it, since it can oppose and counteract them. The most important factors are temperature and nourishment with regard to the frequency of feeding and the host's state of health (5). The level of hemoglobin and glucose in host blood is particularly relevant (3).

These properties of lice suggest that they should be treated not only as parasites and vectors of infectious diseases, but also as experimental animals in microbiologic and toxicologic investigations.

REFERENCES

1. BECLA, E. The determination of the toxic doses and level of some antibiotics in body louse. *Z Angew Zool*, in press.
2. KRYŃSKI, STEFAN. Investigations of the toxic action of *R. prowazeki* on lice and white mice. *Bull Inst Mar Trop Med Gdansk* 1:33-37, 1948.
3. ——— and S. WOYCIECHOWSKA. Investigations into problem of artificial feeding of lice by way of intrarectal injection as applied by Weigl. *Bull Inst Mar Trop Med Gdansk* 2:59-60, 1949.
4. ———. Forms of *R. prowazeki* infection in lice artificially infected by Weigl's method. *Bull Inst Mar Trop Med Gdansk* 2:231-35, 1949.
5. ———. Influence exerted by the feeder on the course of *Rickettsia prowazeki* infection in lice. *Bull State Inst Mar Trop Med Gdansk* 4:47-49, 1952.
6. ——— and J. D. RADKOWIAK. Principles of cultivation of *Rickettsia prowazeki* by Weigl's method. *Bull State Inst Mar Trop Med Gdansk* 4:213-44, 1952.
7. ———. Weigl's test: a new method of investigating the toxicity of chemotherapeutics. *Bull State Inst Mar Trop Med Gdansk* 7:140-44, 1956.
8. ——— and A. HERZIG-WEIGL. Influence de la température sur l'évolution de l'infection des poux. *Arch Inst Pasteur Tunis* 36:435-39, 1959.
9. ——— and E. BECLA. Influence de la température sur l'activité toxique de *R. prowazeki* chez le pou. *Arch Inst Pasteur Tunis* 37:3-12, 1960.
10. ——— and ———. The action of oxytetracycline on *R. prowazeki* in lice inoculated by the Weigl method. *Chemotherapy* 8:265-74, 1964.
11. ——— and ———. Infection of the body louse with *P. pseudotuberculosis* after intrarectal inoculation. *J Infect Dis* 114:379-85, 1964.

12. ——— et al. L'inoculation de *Yersinia enterocolitica* par voie intrarectale chez le pou. *Ann Inst Pasteur (Paris)* 110:779-84, 1966.
13. ——— and E. BECLA. Courbe de croissance de *R. prowazeki* dans l'intestin du pou infecté selon la méthode de Weigl. *Arch Inst Pasteur Tunis* 43:365-74, 1966.
14. ——— and ———. Staphylococcal infections in lice. *Postepy Mikrobiol* 5:327-29, 1966.
15. ———. Bacterial infections in lice injected by the Weigl method. *Wiad Parazytol* 13:615-18, 1967.
16. POKORNY, S. Biologia wszy: *Pediculus humanus corporis* w hodowli laboratoryjnej. *Przegl Epidemiol* 3:302-32, 1949.
17. WEIGL, R. Untersuchungen und experimente an fleckfieber läusen. *Beitr Klin Infect Kr* 8:353-76, 1919.
18. ———. Further studies on *Rickettsia rocha-limae*. *J Trop Med Hyg* 27:14-15, 1924.

OPEN DISCUSSION

Moderator: Dr. Busvine

Dr. Gaon: Dr. Busvine, why do you think head lice spread to other people more easily than body lice? Is that generally true or is it simply that the longer the hair, the more the head lice? Also, why do head lice seem to be on the increase, as you mentioned is happening in England?

Second, what is the likelihood of lice more readily attacking dirty, sweaty shirts or shirts covered with louse feces?

Dr. Busvine: First of all, Professor Buxton summarizes in his classic book a number of investigations in different parts of the world on the relationship between hair length and infestations. This was found to be correlated not only within populations but within different groups. For example, races of people who do not shave or cut their hair tend to be rather more infested than those who wear their hair short. Thus, one would readily expect hippies or young people who like long hair to be more prone to infestation and, in fact, a British newspaper made this suggestion quite recently.

Unfortunately for the theory, the difference in lousiness between girls, who in the past traditionally wore their hair longer, and boys still obtains. Girls are still more prone to lousiness than boys, even through many boys are wearing their hair as long as girls. There is also quite a big difference between very young girls and boys when it comes to lousiness.

As to the so-called preference of body lice for people with dirty shirts, I think that is probably an error of observation. People who don't wash their shirts do not affect lice, and therefore their shirts tend to be dirty. On the other hand, Prof. Vincent Wigglesworth did some experiments many years ago

in which he confined body lice in a choice chamber, on one side of which was a shirt that had been worn and was covered with perspiration and on the other side of which was a clean shirt. The lice preferred to sit on the dirty shirt.

Dr. Murray: You said you observed that lice were relatively scarce on lousy individuals. We feel that it is necessary for a large number of lice to be present on a Brill-Zinsser disease patient in order for such a person to infect enough lice to cause an epidemic. I would be interested in hearing about the experiences of Drs. Snyder, Wisseman, and Gaon in counting lice and getting estimates of the numbers of lice on individuals during an epidemic such as occurred in Egypt, Burundi, and Yugoslavia.

Dr. Busvine: There are one or two published accounts that give figures about counts of lice on infested people, though the counts may not necessarily have been made during epidemics. I think that counting lice under epidemic circumstances would be a very unpleasant, dangerous thing to do. The counts I have in mind were made among British troops in World War I and among indigent men in London in more recent times.

Dr. Wisseman: In Burundi we did not make counts, but in collecting lice to conduct resistance tests it was very easy to get the requisite number from the clothing of people who just crowded around. I think we got as many as 400 lice from one person, but this was an unusually large number. Nevertheless, they were easy to find, and it took very few pieces of clothing to get several hundred lice in satisfactory health with which to conduct the insecticide sensitivity tests. They were sufficiently numerous so that very often

they were seen crawling on the outer aspects of the clothing, which indicated a very heavy infestation. These lice came from people who visited a dispensary. We found it much easier to collect lice if we went to the jails, but prisoners are a special population, of course.

Dr. Fabrikant: The situation in Burundi made it virtually impossible for people to get rid of their own lice. They were too poor to have a change of clothing. Water supplies were distant, and water was carried in pots on heads. The insecticide louse eradication program was inadequate because of poor logistics. As Dr. Wisseman said, although we didn't count lice for that purpose, it was not at all difficult in the dispensary at Katara to acquire lice on oneself just by wandering among patients.

Dr. Busvine: I think the point about these numbers is that lousy people do something about their lice if they can, but at a certain point they become a little immune to the bites and don't trouble to remove the lice any more. But small numbers of lice are usually due to the efforts that people take to kill their own lice.

Dr. Woodward: One additional word about the degree of lousiness under epidemic conditions. I certainly support Dr. Wisseman's statement in that during the epidemic among the native population in North Africa in 1943 it was quite common to find two, three, or four hundred lice in the clothes of one patient. When Dr. Snyder was in Cairo, I would go into my typhus ward and ask for two lice for one cigarette. I would put 10 lice in a tube, send them off to him in Cairo, and I think he always isolated *Rickettsia prowazeki* from those dried, dead lice. Later, in Naples, one had but to turn the shirt back and pick off a hundred or more lice without any trouble at all.

Dr. Snyder: Just a comment to corroborate what Dr. Woodward has already said. It was usually assumed that louse infestation

would decrease in very hot places in the summertime. In Egypt during the summers of 1943 and 1944 we were able to get a tablespoonful of lice even on the hottest day by etherizing a villager's garment and shaking it lightly into a pan.

Dr. Kostrzewski: Dr. Busvine, were your data on louse infestation in Poland about school children?

Dr. Busvine: No, the data I presented were from Buxton's work and were about prisoners. My personal experience is only with infested vagrants in London.

Dr. Gear: Dr. Busvine, is there any evidence that the effectiveness of the host's immunity mechanism determines the number of lice or the degree of louse infestation? With the diminishing effectiveness associated with disease, old age, or starvation, is there any evidence that the lice increase in number? It has been noticed that physically and mentally defective people tend to be much more lousy than others. There are obvious physical reasons why this may be so. But are there any biologic reasons?

Dr. Busvine: Anyone who is a veterinarian will know that animals in poor health become heavily infested with parasites. There may be some parallel with man.

Dr. Wegner: With reference to Dr. Busvine's paper, I would like to note that in Poland Pokorny, in his research on *Pediculus humanus* biology, found that when lice were fed once a day and kept at 32° C under laboratory conditions, their life span was about 40 days. A female was able to lay between 50 and 60 eggs during its life. Regular egg-laying periods interrupted by two or three days of rest were observed. Daily egg production amounted to one to four eggs. Females of middle age laid eight, and very occasionally 13, eggs. Nymphs hatched on the fifth to seventh day after egg laying. Lower temperatures prolonged the hatching period.

When the influence of temperature on the hatching of nymphs was studied, it was found

that after the eggs were kept at room temperature for five days and then placed in a temperature of 32° C, between 90 and 100 per cent hatched. After 10 days at room temperature, only 60 per cent hatched; after 15 days, 20 per cent; and after 20 days, no nymphs at all. No nymphs hatched either after eggs had been kept at 30° to 40° C for only a few hours.

When investigating the influence of frequency of the population on the fertilization and fertility of *Pediculus humanus*, it was found that after a single population, about 70 per cent of the eggs remained unfertilized. Repeated population, although giving a higher percentage of fertilized eggs, did not influence the number of eggs laid.

Mr. Cole: Dr. Ludwig, did the lice that you fed on the nonspecific host or different hosts exhibit the redness that Dr. Busvine mentioned? Before you answer, I would like to comment that we have observed this redness in our lice when we were transferring them from human to rabbit feeders. At times we observed a high percentage of red lice, which always died quickly. We assumed this to be due to a rickettsia, but no one has ever worked it out. We corrected the situation merely by changing rabbit hosts, and as soon as we changed to a new rabbit host we got no further high percentage of red lice and high mortality.

Our conclusions were that this was either a higher percentage of favorable rabbits, or that the lice made a better adaptation to an unnatural host. We now find a higher percentage of rabbits that are favorable hosts when we buy them for our use in rearing.

Dr. Ludwig: All lice fed on guinea pigs become red and die. The redness is due to the rupture of the intestine, as Professor Kryński showed about 20 years ago. A certain percentage of lice fed on other hosts always die because of the redness. This percentage depends on the host's suitability as a substitute.

Dr. Brooks: Have you made any histologic examination of the lice that fed on blood containing antibiotics? Do you know if the symbiotes were affected?

Dr. Ludwig: No, we have not yet made a histologic examination, but we intend to. Our experience so far has only been with *Haematopinus suis*, but we want to investigate this problem in *Pediculus humanus* as well.

Dr. Ormsbee: We did one thing in investigating the possible involvement of animals in typhus spread in Egypt that may be of some interest. To simulate a situation that might occur, we reared lice on camels and men on alternate days. We succeeded in rearing head lice through such a cycle. This might be of some importance in other diseases.

Dr. Smith: Medical entomologists, I have noticed, have one thing in common with fishermen: when fishermen get together they always tell stories about who caught the biggest fish and where, and when medical entomologists get together they very frequently, if not invariably, get into a contest about who found the lousiest person and where.

I too have found some very lousy people, and I too have an observation I would like to make. I collected lice in various places in Africa, one of which was Freetown, Sierra Leone, where the climate is very humid. I had extreme difficulty finding lice: I found lousy people, but only one to 10 or 20 lice per person. In places where the humidity was not so high, such as Saint-Louis, Senegal, I found quite heavily infested people. The market beggars I examined in Freetown were just as filthy and had had their rags on just as long as those in Saint-Louis, but they were much less infested.

The only conclusion I could reach was that humidity was deleterious to lice. We know that in our rearing cabinets the need is not to maintain high humidity, as for many other insects, but to decrease it. No one else

has mentioned this point, but I would like you to keep it in mind as you observe louse infestations in different environments.

I would also like to confirm what Dr. Ormsbee said about alternating hosts. This can be done not only by alternation between a favorable and an unfavorable host, but between several unfavorable hosts. In the early days of the Gainesville body louse colony we found that there were certain rabbits on which the strain could be main-

tained day after day. There were other rabbits on which the colony would die over a period of days. But if one took four of these rabbits, none of which individually would support a louse colony, and fed the lice first on No. 1, the next day on No. 2, the next day on No. 3, and the next day on No. 4, one could maintain the strain on unfavorable animals by alternating from one unfavorable animal to another. The lice did not thrive, but it could be done.

II. B. CONVENTIONAL LOUSE CONTROL METHODS

CONTROL OF LICE BY CHEMICALS

D. E. Weidhaas¹

I would like to make some general comments as an introduction to the discussion that will follow. Dr. Busvine will have some additional remarks, emphasizing particularly the formulations now in use.

It is a pleasure to introduce this panel on chemical compounds for the control of lice. The habits of lice associated with or restricted to human hosts (with a limited but significant survival in bedding and clothing of the host, for instance) have led to an emphasis on the approaches to be covered by the panel speakers. This panel will consider insecticidal powders (commonly known insecticides generally grouped as pyrethrums, chlorinated hydrocarbons, organophosphorus compounds, and carbamate compounds), impregnants (compounds generally similar to those in the powders), systemic insecticides, fumigants (such as methyl bromide, HCN, and, more recently, the organophosphorus compound dichlorvos, or DDVP), synergists (including those for pyrethroid-type compounds, and even organophosphorus and carbamate-type compounds), ovicides, and physical agents.

The numbers and types of compounds evaluated against lice represent most of the insecticidal and synergistic compounds known to entomologists. The occurrence of

insecticide resistance has played an important role in the evaluation and development of chemical compounds.

Two general comments will suffice to lead into the presentations and discussion. I would first like to present some historical information on the development of the louse powders, and then make a few comments about the development of control chemicals.

DDT has been mentioned several times as a louse powder. In reviewing the general literature, one finds references before World War II to certain concoctions that were used against body lice, but it was difficult to identify the components of the preparations. The general conclusion was that they were generally less than completely satisfactory. During World War II, an extensive evaluation program was undertaken in several areas, particularly in the development of louse powders. Just before the development of DDT louse powder, a powder of 0.2 per cent pyrethrins, 2 per cent dinitroanisole, 2 per cent n-isobutylundecyleneamid, and 1 per cent of phenol-s (isopropyl cresols) was developed which was highly effective.

This MYL powder was quickly replaced by DDT. Work in England emphasized the development of lindane and BHC. The Germans worked to develop a compound called Lauseta Neu (chloromethyl p-chlorophenyl sulfone). The occurrence of resistance led to increased work on lindane, and then organophosphorus compounds like mala-

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thion and the carbamates. Various types of chemical compounds were evaluated to overcome the problem of cross-resistance.

Since World War II, many thousands of compounds have been tested at our laboratory for activity against lice in a joint development program between the U.S. Departments of Defense and Agriculture. This evaluation program is concerned with obtaining compounds and determining their insecticidal activity. The compounds come from any source available and the largest source has been industry. Many come from synthetic chemists, universities, and other sources, however.

The body louse serves as a screening mechanism to identify those with potentially good insecticidal activity. In addition, it is useful in the initial development of body louse toxicants.

Since this evaluation program has been well reviewed, I would like only to emphasize that many thousands of materials have been

evaluated through it. The results about the compounds evaluated through 1964—about 19,700 compounds—are available in U.S. Department of Agriculture publications (1, 2). Because compounds have continued to be evaluated since 1964, over 20,000 compounds have now been evaluated. Results with insects other than body lice are also included in the two publications.

In addition to this program, the World Health Organization has an insecticide development scheme in which my own laboratory and seven others collaborate. (Two of those laboratories are represented at this meeting, my own and Dr. Schoof's laboratory in Savannah, Georgia.) More than 1,400 compounds have gone through the WHO screening program, whose results for the period 1962-72 are available in a WHO publication. Testing techniques, results, and the criteria for selection of the more promising compounds in the scheme are also given in the publication.

REFERENCES

1. KING, W. V. (ed.). Chemicals evaluated as insecticides and repellents at Orlando, Florida. Washington, D.C., Agricultural Research Service, Department of Agriculture, 1964. (USDA Handbook No. 69.)

2. UNITED STATES. DEPARTMENT OF AGRICULTURE. Materials evaluated as insecticides, repellents, and chemosterilants at Orlando and Gainesville, Florida, 1952-64. Washington, D.C., Agricultural Research Service, Department of Agriculture, 1967. (USDA Handbook No. 340.)