Bedbugs in Relation to Transmission of Human Diseases

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THE possible role of bedbugs in transmission of human diseases has been the subject of many studies during the past 55 years (*1*). Most of these studies were made on either the cosmopolitan bedbug, *Cimex lectularius* Linnaeus, or on *Cimex hemipterus* Fabricius (*C. rotundatus Signeret*), which is known as the tropical, subtropical, or Indian bedbug. A few studies on transmission of Chagas disease were concerned with other species, such as *Cimex* (*Leptocimex*) *boueti* Joyeux, a tropical bedbug of Africa and South America which normally infests bats; *Oeciacus* (*Cimex*) *hirundinis* (Jenyns), the European barn swallow bug; and *Haematosiphon inodora* (Duges), which infests poultry, the California condor, and the great horned owl.

Bedbugs have been suspected in the transmission of 41 human diseases, the agents or causes of which coincide with 10 categories, as shown below.

Bacteria. Anthrax, brucellosis, epidemic cerebrospinal meningitis, leprosy, paratyphoid fever, plague, pneumonia, septicemia, tuberculosis, tularemia, and typhoid fever.

Rickettsiae. Boutonneuse fever, epidemic typhus, exanthematous typhus of Minas Geraes, murine (endemic) typhus, Q fever, Rocky Mountain spotted fever (São Paulo typhus and Mexican typhus fever), Siberian tick-bite typhus, and South African tick-bite fever.

Spirochetes. Relapsing fevers, Weil's disease (leptospiral jaundice), and epidemic infectious jaundice of Brazzaville.

Viruses. Encephalomyelitis, influenza, lymphocytic choriomeningitis, poliomyelitis, smallpox, and yellow fever.

Protozoa. Chagas disease, kala-azar, malaria, oriental sore, and sleeping sickness.

Helminths. Filariasis (elephantiasis), mansonelliasis, and onchocerciasis.

Vitamin deficiencies. Beriberi and pellagra.

Allergies. Bullous erythema.

Iron deficiency. Hypochromic anemia.

Cause unknown. Cancer (in mice).

To determine the types of studies conducted on the bedbug, 116 sources were investigated. Summaries of 93 of these studies appear on pp. 516–521. Although in many cases actual experiments were carried out by feeding bedbugs on disease agents, some of the diseases, such as epidemic cerebrospinal meningitis, beriberi, pellagra, tuberculosis, influenza, sleeping sickness, epidemic typhus, malaria, and typhoid fever, were associated with bedbugs by inference, deductive reasoning, or conjecture. Several successful experimental studies have led to the dubious conclusion that bedbugs transmit leprosy (*44*), oriental sore (*12*), kala-azar (*11, 18,20*) Q fever (*59*), relapsing fever (*77*), and brucellosis (*55*) in nature.

Many of the causative agents of disease have developed or remained alive within the bedbug after experimental infection for varying numbers of days. This does not necessarily mean that these organisms are transmitted by bed-

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Survival times of various disease organisms in Cimex lectularius and Cimex hemipterus

Disease	Disease organism	Bedbug species and location of organism in body	Maximum survival time (days) [1]
		C. lectularius	
Filariasis (*3*)	*Wuchereria bancrofti*	Throughout body	8
Mansonelliasis (*7*)	*Mansonella ozzardi*	Gut	14+
Kala-azar (*17*)	*Leishmania donovani*	Gut	35
Oriental sore (*17*)	*Leishmania tropica*	Gut	35
Espundia (*17*)	*Leishmania braziliensis*	Gut	35
Leprosy (*42*)	*Mycobacterium leprae*	Head, proboscis, and hemocoel.	16
Septicemia (*46*)	*Staphylococcus aureus*	Salivary glands	14–15
Anthrax (*47*)	*Bacillus anthracis*	Throughout body and feces.	4
Pneumonia, type 2 (*49*)	*Diplococcus pneumoniae*	Gut and malpighian tubules (infected by injection).	2–30
Pneumonia, type 2 (*49*)	*Diplococcus pneumoniae*	Gut and malpighian tubules (infected by biting).	30–85
Tularemia (*52*)	*Pasteurella tularensis*	Feces	136+
Tularemia (*53*)	*Pasteurella tularensis*	Feces	250+
Brucellosis (*55*)	*Brucella abortus, B. melitensis, B. suis.*	Gut	90+
Paratyphoid fever (*57, 58*)	*Salmonella paratyphi*	Gut and feces	21
Plague (*51*)	*Pasteurella pestis*	Gut	147
Rocky Mountain spotted fever (*60*)	*Rickettsia rickettsii*	Gut	7–21
Mexican strain (*59*)	*Rickettsia rickettsii*	Gut	24
São Paulo strain (*59*)	*Rickettsia rickettsii*	Gut	27
São Paulo typhus (*61*)	*Rickettsia rickettsii*	Gut	1
Mexican typhus (*62, 63*)	*Rickettsia rickettsii*	Gut	10
Epidemic typhus (*59*)	*Rickettsia prowazekii*	Gut	30
Murine typhus (*69*)	*Rickettsia typhi*	Gut	6
Q fever (*70, 59*)	*Coxiella burnetii*	Feces	285, 134+
Relapsing fever (*77*)	*Borrelia recurrentis*	Not specified	1+
Relapsing fever (*79*)	*Borrelia duttoni*	Not specified	150+
Relapsing fever (*80*)	*Borrelia duttoni*	Stomach	2+
Relapsing fever (*79*)	*Spirochaeta merionesi*	Not specified	200
Infectious jaundice of Brazzaville (*82*).	*Leptospira icterohaemorrhagiae*	Not specified	38
Poliomyelitis (*83*)	Poliomyelitis virus	Not specified	7
Yellow fever (*86*)	Yellow fever virus	Feces	15
Smallpox (*88*)	Smallpox virus	Salivary glands and hemolymph.	12
Lymphocytic choriomeningitis (*89*)	Lymphocytic choriomeningitis virus.	Feces	85+
		C. hemipterus	
Filariasis (*2*)	*Wuchereria bancrofti*	Throughout body	1–20+
Oriental sore (*11*)	*Leishmania tropica*	Midgut	3
Oriental sore (*14–16*)	*Leishmania tropica*	Stomach (re-fed adult)	34
Oriental sore (*14–16*)	*Leishmania tropica*	Hind intestine (re-fed adult).	44
Oriental sore (*14–16*)	*Leishmania tropica*	Rectum (re-fed adult)	34
Oriental sore (*14–16*)	*Leishmania tropica*	Alimentary tract (once-fed adult).	23
Oriental sore (*14–16*)	*Leishmania tropica*	Midgut (re-fed nymph)	31
Oriental sore (*14–16*)	*Leishmania tropica*	Hind intestine (re-fed nymph).	36
Kala-azar (*11,18,20*)	*Leishmania donovani*	Midgut (after 6 uninfected blood meals).	41
Relapsing fever (*11*)	*Borrelia recurrentis*	Gut	7
Relapsing fever (*74*)	*Borrelia recurrentis*	Gut	5+
Relapsing fever (*75*)	*Borrelia recurrentis*	Coelomic fluid and legs	1–12
Relapsing fever (*76*)	*Borrelia recurrentis*	Not specified	28+
Yellow fever (*85*)	Yellow fever virus	Feces	1–2

[1] Does not necessarily refer to all of the bedbugs used. Survival times varied because of individual differences among the bugs.

bugs under natural conditions. Also, individual differences, or differences in strains of bedbugs or parasites, may account for any development or multiplication in nature that may occur following mechanical ingestion through feeding activities. Bedbugs have been found infected in nature with *Wuchereria bancrofti* (*1,2*), *Brugia malayi* (*1*), *Trypanosoma cruzi* (*36*), *Brucella melitensis* (*55*), *Coxiella burnetii* (*69*), and rickettsiae causing exanthematous typhus (*63,64*).

After finding infections in nature, the importance of the find can be ascertained by experimental feeding of bedbugs on the parasites or other agents involved. For instance, Burton (*1,2*) found small numbers of infective larvae, second instar larvae, and sausage stages of *W. bancrofti* and *B. malayi* in the bedbug. These findings indicated that the bedbug might play some role in transmission of filariasis; however, in detailed feeding experiments development of *W. bancrofti* larvae was very erratic and sporadic, with heavy general mortality of parasites, leading to the conclusion that individual differences were responsible for whatever development had occurred.

The differences in strains are apparent from the diverse results reported by various investigators from different countries or areas working with the same species of parasites and bedbugs and the same experimental animals (facing page). In experimental transmission of poliomyelitis to monkeys, by injecting emulsions of infected bedbugs, Howard and Clark (*83*) obtained positive results, but Kling and Levaditi (*84*) obtained negative results. Kum and Frobisher (*85*) obtained mostly negative results with yellow fever virus in monkeys, but Monteiro (*86*) obtained positive results. Monteiro (*61*) found that the São Paulo strain of *Rickettsia rickettsii* could not survive longer than 1 day in the bedbug, but Weyer (*59*) found it surviving up to 27 days. Wenyon (*11*) found that *Leishmania tropica* lived only 3 days in *C. hemipterus*, but Patton (*14–16*) found that it survived for 23 days after one feeding, and up to 44 days after a second feeding. A summary of survival times of various disease agents in bedbugs is shown in the table.

Another peculiarity is that the disease agent often fails to develop in the bedbug after it feeds on an infected animal; yet when a suspension of the disease agent is injected into the rectum of the bedbug, the parasite develops in its gut. This has occurred in the case of Rocky Mountain spotted fever, the organisms having remained virulent in the bedbug's gut for 24 to 27 days (*59*). In boutonneuse fever, the infections remained virulent in the guts of bedbugs up to 9 days (*59*); in epidemic typhus, up to 30 days (*59*); and in murine typhus for 6 days (*69*).

Bedbug feces have been found to contain disease agents and to be infective to laboratory animals in oriental sore (*17*), kala-azar (*11,18, 20,25,26*), Chagas disease (*29,32,37*), anthrax (*48*), tularemia (*52–54*), brucellosis (*55*), paratyphoid fever (*57,58*), yellow fever (*85,86*), smallpox (*88*), and lymphocytic choriomeningitis (*89*).

In only two of the studies reported here have disease organisms themselves caused deaths of bedbugs; pneumonia (*49*) and plague (*50*). Transovarian transmission of the disease agent has been reported in three studies: in Q fever (*70*), transmission of *Coxiella burnetii* in eggs occurred in 20 of 30 trials with *C. lectularius;* in exanthematous typhus of Minas Geraes (*64*), the rickettsiae were found in eggs and first instar nymphs of *C. lectularius;* and the nonpathogenic rickettsiae *R. lectularia* and *R. hirundinis* have been found in ovaries of *C. lectularius* (*71*) as hereditary infections.

Summary

A review of 93 studies concerned with the possible role of bedbugs in transmission of human diseases revealed that several successful laboratory experiments led certain investigators to the conclusion that bedbugs transmit leprosy, oriental sore, kala-azar, Q fever, relapsing fever, and brucellosis in nature. Actual transmission, however, has not been scientifically proved.

Although bedbugs have been found infected in nature with *Wuchereria bancrofti*, *Brugia malayi*, *Trypanosoma cruzi*, *Brucella melitensis*, *Coxiella burnetii*, and rickettsiae causing exanthematous typhus, transmission of the associated diseases is not necessarily effected by bedbugs. Individual differences due to physiological makeup or strains of bedbugs or para-

sites, or other disease agents, may result in viability or development of the organisms in bedbugs for varying numbers of days. Differences in strains were evidenced by the diverse results reported by various investigators from different countries or areas working with the same species of parasites and bedbugs and the same experimental animals.

NOTE: *References appear on pages 521–524.*

Summaries of 93 studies on the relation of various human disease organisms to bedbug species, by category and disease

HELMINTHS

Filariasis (elephantiasis)

Brugia malayi; Wuchereria bancrofti

Cimex lectularius. *B. malayi* sausage larvae found in tibiae and tarsi of five nymphs; infective larvae in antennae, abdomen, and prothorax of four nymphs. Two nymphs yielded three sausage and one infective larvae of *W. bancrofti.* No larvae found in adult bedbugs (*1*).

Wuchereria bancrofti

Cimex hemipterus. Early first instar larvae found in abdomen, thorax, legs, and antennae. Seven bugs had one sausage each, and another had two. Two sluggish second instar and three infective larvae (2 nonmotile, 1 motile) were found. Four experimental infections in older nymphs and adults yielded no infective larvae after 20 days, but several sausage and one second instar larvae developed. Frequent blood meals sustained larvae for longer periods. Parasite mortality indicated that bedbug is not good host for development of *W. bancrofti* (*2*).

Cimex lectularius. Microfilariae moved freely throughout body of bug until killed on eighth day. No further development seen (*3*).

Sheathed, dead microfilariae found in bugs collected from beds of infected persons (*4*).

Wuchereria bancrofti; Brugia malayi

Cimex hemipterus. No larval development found in adult bugs fed on human *W. bancrofti* carrier and on cats with *B. malayi* infection (*5*).

Wuchereria bancrofti; Brugia patei

Cimex lectularius. A few first- and second-stage *W. bancrofti* larvae were found in nymphs fed 20–25 days before. No infective larvae were found. With *B. patei* many microfilariae accumulated in bugs' legs and a few microfilariae were still alive 24 days after feeding. No *B. patei* larvae developed (*6*).

Mansonelliasis

Mansonella ozzardi

Cimex lectularius. Microfilariae still alive 14 days after ingestion; all dead by 18 days; none outside gut. No further development seen (*7*).

Onchocerciasis

Onchocerca volvulus

Cimex lectularius. Microfilariae none or rare in bugs' guts after feeding on persons with onchocerciasis (*8*).

Four days after feeding artificially on blood containing microfilariae extracted from nodules, 23 bugs showed no development beyond microfilaria stage (*9*).

Microfilariae found in only one of five bugs fed on infected persons and dissected ½–4 hours later (*10*).

PROTOZOA

Oriental sore

Leishmania tropica

Cimex hemipterus. Parasite passed into herpetomonad phase in bugs' midguts. Of 105 bugs fed on a sore, only those dissected on second and third days after feeding were positive; all dissected after third day were negative (*11*).

Flagellated forms developed from leishmanias which had been ingested from sores; conclusion therefore reached that bedbugs transmit disease in Baghdad (*12*).

Confirmed Wenyon's findings (*12*), and stated that bedbug was vector of oriental sore in Cambay (*13*).

Parasites remained alive 23 days in alimentary tracts of starved bugs; in re-fed bugs they were alive 34 days in stomach, 44 days in hind intestine, 34 days in rectum; in re-fed nymphs they were alive 31 days in midgut, 36 days in hind intestine and rectum, and at least 9 days in midgut of first instar nymph. Developed into flagellated stage when temperature was below 25° C. No transmission occurred during feeding of bedbugs (*14–16*).

Oriental sore; kala-azar; espundia; uta

Leishmania tropica; Leishmania donovani; Leishmania braziliensis

Cimex lectularius. Parasites passed up to 35 days in feces of artificially infected bedbugs (*17*).

Kala-azar

Leishmania donovani

Cimex hemipterus. Bedbugs infected by feeding on diseased persons. Herpetomonad phase in midgut and *Leishmania* phase in hindgut. Survived 41 days after infectious meal, despite six succeeding uninfected blood meals. Transmission to man occurred not by bite, but by rubbing infected feces or crushed insect into abraded skin (*11, 18–20*).

Bedbugs artificially infected heavily and cycle studied. Concluded that bedbug is only a casual host, harboring development only up to a certain point (*21*).

Bodies in salivary glands and ducts of bugs, found in nature, were thought to be *L. donovani*. Christophers determined them a new species of *Nosema* and named the organism *Nosema adiei* (*22–24*).

Cimex lectularius; Cimex hemipterus. C. lectularius, although a better host for the parasite than *C. hemipterus*, was absent from endemic areas. All transmission experiments by rubbing infected feces into puncture wound were negative. No transmission to monkeys occurred after they ate infected bedbug feces. No bedbugs were found infected in nature. Gave final proof that bedbug cannot transmit kala-azar from man to man by either bite or bug feces (*25,26*).

Cimex lectularius. Of 300 bedbugs fed on heavily infected cultures, only 6 were positive, with rare parasites. No bugs were found positive in nature. Concluded that parasite could not live in digestive tube of bedbug (*27*).

In bedbugs' stomachs, merozoites divided by fission, later changed to flagellated forms which entered cells and rounded up. No forms seen in salivary glands (*28*).

Chagas disease

Trypanosoma cruzi

Cimex lectularius; Cimex (*Leptocimex*) *bouetti*. Twenty-four hours after biting infected rats, bugs had no trypanosomes in their intestines. Only crithidia in hind intestines, which changed to trypanosomes 10 days later. Young rats became infected after injection of bedbugs' infected feces (*29*).

Cimex hirundinis. Parasite developed to infectious metacyclic forms (*30*).

Cimex stadleri. Parasite developed to infectious metacyclic forms (*31*).

Cimex hemipterus. Eight of every 10 bugs became infected after feeding on infected mice. Partial immunity developed in the mice because the parasites apparently had become weakened by passage of many generations through bedbugs. Bug feces had highly penetrating metacyclic trypanosomes (*32*).

Haematosiphon inodora. Fifteen bedbugs fed on lightly infected mouse showed metacyclic forms in intestines 15 days later. Suspensions of intestinal contents were infectious to healthy mice 21 days after bedbugs' infectious meal (*33*).

Cimex lectularius; Cimex hemipterus. Bedbugs infected with *T. cruzi* remained infected during their entire lives (*34*).

Clerada apicicornis. Parasite developed completely within this species (*35*).

Cimex lectularius. Natural infection of *T. cruzi* found in bedbugs from bed of child with Chagas disease in Argentina. Three and four months after acute period of child's infection, no more infected bugs were found (*36*).

Parasite capable of multiplying in bug, but not readily transmissible by feeding. It could, however, be transmitted in feces extracts (*37*).

No natural infections found in numerous bedbugs collected in nature. No hereditary transmission was found (*38*).

Sleeping sickness

Trypanosoma gambiense

Cimex hemipterus; Cimex lectularius. Royal Society's Commission on Sleeping Sickness considered bedbug a possible transmitting agent of this disease (*34*).

Malaria

Plasmodium sp.

Cimex lectularius; Cimex hemipterus. Author's experiences in Russia in World War I suggest that where *Anopheles* mosquitoes were not present and troops developed malaria, the disease was spread by bedbugs because of proximity of beds. Not verified by dissection of bugs, but conjecture based on epidemiologic studies (*39*).

BACTERIA

Tuberculosis

Mycobacterium tuberculosis

Cimex lectularius. Bedbugs incriminated in transmission of tuberculosis by inference, because Smith and associates had positive results with a related species, *M. leprae* (*40*).

Leprosy

Mycobacterium leprae

Cimex lectularius. Shaved skin of rat was stretched over a jar containing blood agar with many leprosy bacilli. In bedbugs which fed on this medium, leprosy bacilli were found in intestines and salivary glands (*40*).

Bedbugs were fed on leprous lesions and dissected up to 100 hours after feeding. All bacilli were absorbed, with no multiplication (*41*).

Of 75 bugs fed on lepers, 20 contained acid-resistant bacilli which 1 bug harbored for 16 days in head, proboscis, and hemocoel. Bugs could not transmit bacilli to animals or people by bite (*42*).

In Sainte Marie, Danish Antilles, 53 bedbugs were fed on leprous lesions and dissected for different periods up to 34 days. All were negative except three with doubtful results (at 1 hour, 6 hours, and 20 days) (*43*).

Acid-resistant bacilli found in all bedbugs fed on a facial lesion of a leper. Disease said to be transmitted in nature by bedbug bites (*44*).

Cimex hemipterus. Bacilli not taken up from skin of lepromatous patient during a blood meal; hence could not be transmitted to another person at a later meal (*45*).

Hemolytic staphylococcus (septicemia)

Staphylococcus aureus

Cimex lectularius. Bedbugs successful agents in

transmission of hemolytic staphylococcus in rabbits, mice, and guinea pigs. Only salivary glands of bugs infected, up to 14–15 days (*46*).

Anthrax

Bacillus anthracis

Cimex lectularius. No infections occurred in mice by bedbugs, but bedbugs' feces and crushed bodies may be infectious for up to 4 days (*47*).

Attempts to transmit anthrax in guinea pigs by bedbugs were unsuccessful when 100 bedbugs fed on infectious meal were put on healthy pigs 30 seconds after feeding (*48*).

Epidemic cerebrospinal meningitis

Neisseria meningitidis

Cimex lectularius. Beldwin suggested bedbug as possible vector of epidemic cerebrospinal meningitis based on epidemiologic studies where bedbugs were present in overcrowded sleeping quarters (*40*).

Pneumonia

Diplococcus pneumoniae

Cimex lectularius. Type 1 pneumonia virus multiplied more abundantly than type 2 in bedbug's stomach, epithelium of stomach, intestines, and malpighian tubules. For type 2 the incubation period was 2–5 days when animals were infected by injection, and 10–67 days when infection was by biting. Death occurred 2–30 and 30–85 days afterward. The infection appeared pathogenic to bedbugs themselves. Diplococci were seen in proctodaeum of nearly all bugs after second feeding on infected guinea pigs, and in all the bugs after five feedings. Transmission from infected to healthy animals occurred by biting of bedbugs (*49*).

Plague

Pasteurella pestis

Cimex lectularius. Most bedbugs fed on infected mouse died. Those which survived were able to reinfect mice with *P. pestis* after the bugs starved for 48 hours (*50*).

Retained plague bacilli up to 147 days after feeding on infected rats, and in one case the bite of an infected bug transmitted the disease in Russia (*51*).

Tularemia

Pasteurella tularensis

Cimex lectularius. Infected bedbugs fed on healthy guinea pigs or mice two or three times per month. Fatal tularemia was produced by bedbug bites only if bugs fed immediately or within 15 hours after their infectious meal. Injections into mice from cultures of feces showed that bacterium remained virulent for at least 136 days (*52*).

Bedbugs transmitted lethal tularemia by bite from infected to healthy mice. Fifty-five of 72 mice died of tularemia in an average of 4½ days after eating dead or dying infected bedbugs. Fresh feces of infected bedbugs were virulent and lethal for at least 250 days to entire life of bug, when injected subcutaneously. Suspension of dried feces, when injected, killed mice within 8 days (*53*).

Infected bedbugs produced disease in only 1 of 28 guinea pigs after 16 days of repeated biting. Crushed infected bedbugs on abraded skin of guinea pigs produced infection, as did inoculated bedbug feces. Bedbug nymphs became contaminated by contact with infected bedbug feces (*54*).

Brucellosis

Brucella abortus; B. melitensis; B. suis

Cimex lectularius. Natural infections of two strains of *B. melitensis* were found in bedbugs. Bugs fed on infected guinea pigs and mice were positive from sixth day to more than 3 months. *B. suis* was the most infectious species. No transmission occurred by biting. Bedbug feces could infect humans through skin, mucous membranes, digestive tract, or respiratory tract (*55*).

Typhoid fever

Salmonella typhosa

Cimex lectularius. Five cases of typhoid fever, on an island hitherto immune, were traced to a prisoner who had come recently from an endemic typhoid area. It was assumed that bedbugs were transmitting the disease. After fumigation of the jail, the disease disappeared (*56*).

Paratyphoid fever

Salmonella paratyphi

Cimex lectularius. Paratyphoid organisms remained in body and feces of bedbugs for 3 weeks after bugs fed on infected mice. No transmission occurred by bite of bedbugs. First instar nymphs became infected by contact with infectious bedbug feces. Bacilli disseminated by crushing bugs or by their excreta (*57, 58*).

RICKETTSIAE

Rocky Mountain spotted fever (exanthematous tick typhus of São Paulo; Mexican typhus fever)

Rickettsia rickettsii

Cimex lectularius. Bedbugs negative after being fed on infected mice, guinea pigs, or humans. After concentrated suspensions of rickettsiae were inoculated into bedbugs' rectums, the organisms remained virulent in guts for 24 days (Mexican strain) and up to 27 days (São Paulo strain) (*59*).

Rickettsiae survived 1–3 weeks in bedbugs (*60*).

São Paulo typhus

Rickettsia rickettsii (*Rickettsia braziliensis*)

Cimex lectularius. Organism active immediately after ingestion by bugs, but lost activity after 24 hours. Tests after 2, 3, 5, 10, 13, 16, and 35 days were negative regardless of means used to infect the bedbugs (*61*).

Mexican typhus fever

Rickettsia rickettsii (*Rickettsia braziliensis*)

Cimex lectularius. Rickettsiae survived 10 days in

bedbugs' guts. No transmission occurred by bite or feces. Emulsions of bugs prepared 1 and 10 days after infectious meal produced the infection in healthy guinea pigs (*62, 63*).

Exanthematous typhus of Minas Geraes (South American tick typhus)

Rickettsia nov. sp. (?)

Cimex lectularius. Naturally infected bedbugs were found in beds used by persons with the disease. Organisms were found in eggs of infected females and in first instar nymphs. The disease was transmitted when infected bugs fed on healthy guinea pigs, or when the guinea pigs were inoculated with suspensions of infected bedbugs. The bedbug was said to transmit the infection in nature, but the chief vector was the tick, *Amblyomma cajennense* (*64*).

Exanthematous typhus

Rickettsia sp.

Cimex lectularius. Bedbugs collected in dwelling near Ucacha, Argentina, had natural infections of rickettsiae which caused exanthematous typhus (*65*).

Boutonneuse fever

Rickettsia (*Dermacentroxenus*) *conorii*

Cimex lectularius. No infections occurred in bedbugs fed on infected mice, guinea pigs, or humans. After bugs were inoculated in rectums with concentrated suspensions of rickettsiae, the infections remained virulent in guts up to 9 days (*59*).

South African tick-bite fever

Rickettsia conorii var. *pijperi*

Cimex lectularius. All attempts failed to transmit organisms of tick-bite fever by bedbugs (*66*).

Siberian tick-bite typhus (Far Eastern typhus)

Rickettsia sp.

Cimex lectularius. Bedbugs could not be infected after feeding on infected mice, guinea pigs, or humans (*59*).

Epidemic typhus

Rickettsia prowazekii

Cimex hemipterus; Cimex lectularius. Based on case histories and epidemiologic studies, author surmised that the disease is transmitted by bloodsucking insects, such as bedbugs, body lice, and fleas. No experiments were conducted to verify this conclusion (*67*).

Bedbugs said to transmit the disease in Indian jails, based on epidemiologic studies (*68*).

Cimex lectularius. No infections occurred in bedbugs fed on infected mice, guinea pigs, or humans. When concentrated suspensions of rickettsiae were inoculated into bugs' rectums, infection remained virulent up to 30 days (*59*).

Murine or endemic typhus

Rickettsia typhi (*R. mooseri*)

Cimex lectularius. Bedbugs injected with brain and peritoneum suspensions of mice which had been infected with a Mexican strain of murine typhus retained the infection for 6 days, but no transmission occurred. No transmission occurred by crushing infected bugs on shaved skin of mice or by feeding bugs on infected mice and then transferring them to healthy mice. Feces of the bugs were not infected (*69*).

No infections occurred in bugs fed on infected mice, guinea pigs, or humans (*59*).

Q fever

Coxiella (*Rickettsia*) *burnetii*

Cimex lectularius. In 46 of 60 experiments, after feeding on infected guinea pigs, bedbugs had persistent rickettsiae in excreta up to 285 days regardless of temperature. In 20 of 30 trials, transovarian transmission occurred. Two bugs were infected with *C. burnetii* in nature. Bedbugs were considered potential carriers of Q fever (*70*).

After bedbugs fed on infected mice the infections persisted for at least 134 days, with virulent rickettsiae passed out in feces. Concluded that bedbugs may be carriers and reservoirs of the agent causing Q fever (*59*).

Trench fever

Rickettsia quintana

Cimex lectularius. No infections occurred in bedbugs fed on infected mice, guinea pigs, or humans (*59*).

Nonpathogenic rickettsiae

Rickettsia lectularia; Rickettsia hirundinis

Cimex lectularius. Natural hereditary infections were found intracellularly in malpighian tubules, intestines, and ovaries of bedbugs (*71*).

SPIROCHETES

Relapsing fever

Borrelia recurrentis

Cimex hemipterus. Parasites remained alive in bedbugs' guts up to 7 days. After 3 weeks in guts, organisms appeared normal in stained smears (*11*).

After feeding on infected monkeys, bedbugs infected healthy monkeys by biting. Bugs were allowed to feed partially on an infected monkey and to finish their blood meal on healthy monkey (*72*).

No transmission occurred when bugs fed on infected men or animals 1 to 36 days previously were allowed to feed on healthy men and animals. Rats were infected by inoculation with macerated bugs 32 days after bugs had become infected by feeding (*73*).

Healthy mouse was infected by transferring to it 35 bedbugs which had just fed partially on an infected mouse. Spirochetes remained virulent for at least 5 days (*74*).

Bedbugs fed on infected squirrel. Most parasites were killed by bugs' gastric juice in 24 hours, but some survived for 2 days. Spirochetes were found in legs and coelomic fluid within 1½ hours after infectious meal. Longest survival period was 12 days. Salivary glands and malpighian tubules remained negative (*75*).

Bedbugs recovered from a latrine transmitted relapsing fever for 28 days after the infectious meal (*76*).

Cimex lectularius. Organism was still alive and virulent in Chinese squirrels after being in bedbugs for 25½ hours. Bedbugs were thought to be natural vectors, infected in nature (*77*).

Borrelia recurrentis; Spirella morsus-muris (*Spirillum minus*)

Cimex lectularius. Four of every eight mice which ate infected bedbugs became infected with *B. recurrentis.* Results were negative with *S. morsus-muris* (*Spirillum minus*) (*78*).

Borrelia duttoni; Spirochaeta merionesi; Spirochaeta hispanica; Spirochaeta persica; Borrelia recurrentis

Cimex lectularius. *B. duttoni* survived in bedbugs at least 150 days; *S. merionesi* survived 200 days; *S. hispanica* and *S. persica* died early; no infection occurred by bites. *B. recurrentis* survived, but data were inconclusive (*79*).

Borrelia duttoni

Cimex lectularius. Suspensions of a few nymphs of bedbugs, infected 1 to 2 days previously, injected into healthy mice produced infection. Injections of bugs fed 3 or more days previously gave negative results at 62.6° F.; parasites were still mobile in stomach after 48 hours (*80*).

Weil's disease (leptospiral jaundice)

Leptospira icterohaemorrhagiae

Cimex lectularius. Organism was unable to survive in bedbugs, according to experiments of Reiter (*81*).

Infectious jaundice of Brazzaville

Leptospira icterohaemorrhagiae (?)

Cimex lectularius. Disease resembled icterohaemorrhagic fever of Brazzaville, and perhaps was the same. Sixteen bedbugs were macerated, emulsified, and injected into a guinea pig which died after 11 days with conjunctival icteric fever. Blood smears were negative for disease organisms. Liver of dead animal was emulsified and injected into another guinea pig, which died after 26 days. Parasites were found in blood, liver, and kidney. Bedbugs were fed on infected guinea pig, and fed on a healthy animal 5 days later. The healthy guinea pig died after 24 days and parasites were found in its blood, liver, and lungs. Bedbugs retained their infection for 38 days (*82*).

VIRUSES

Poliomyelitis

Poliomyelitis virus

Cimex lectularius. Fifteen of 16 monkeys injected with emulsions of infected bedbugs were negative. One monkey developed poliomyelitis after intracerebral injection with an emulsion of infected bedbugs which had retained the virus in their bodies for 7 days. Glycerinated nerve cord of the positive monkey, when suspended and injected into a healthy monkey, caused the healthy animal to acquire poliomyelitis (*83*).

Bedbugs fed on poliomyelitis patients were emulsified and injected into nervous system, peritoneum, and general circulation of healthy monkeys. These and biting experiments gave negative results (*84*).

Yellow fever

Yellow fever virus

Cimex hemipterus. Virus died so rapidly in bedbugs that disease was not transmissible to monkeys by injections of emulsified bugs later than second day after infectious meal. Possibly active virus was eliminated in bugs' feces on first and second days, but not later (*85*).

Cimex lectularius. Bedbugs were allowed to bite *Macacus rhesus* during febrile period. Bugs were kept in tube for 2 to 12 days afterward. Feces, emulsified and injected subcutaneously into healthy monkeys, produced clinical and postmortem appearance of yellow fever. Active virus was eliminated in bugs' feces until 15th day after infectious meal. Author inferred that person bitten by bedbugs might inoculate himself with yellow fever by scratching and rubbing in infected feces (*86*).

Cimex lectularius; Cimex hemipterus. Virus persisted only a short time in bedbugs (*87*).

Smallpox

Smallpox virus

Cimex lectularius. Smallpox virus was localized primarily in salivary glands and hemolymph where it was active for 12 days. Multiplication occurred in salivary glands. Infected bedbugs transmitted infection through bites and possibly through feces. Bugs were infected by feeding on infected rabbits (*88*).

Lymphocytic choriomeningitis

Lymphocytic choriomeningitis virus

Cimex lectularius. At 22–25° C., bedbugs transmitted disease from infected to normal guinea pigs in 11 of 18 attempts, at intervals from 10 minutes to 85 days. No transmission occurred by bite alone, only when defecation occurred in bitten area. Virus was detected in dried feces 85 days after infection, and it was transmitted by rubbing infected feces on slightly scarified skin of healthy guinea pigs (*89*).

CAUSE UNKNOWN

Cancer (in mice)

Cimex lectularius. Author quoted paper by Pierlot entitled, "Les cages à cancer dans l'élévage des souris," which concluded that bedbugs (*Cimex*) were responsible for spread of cancer in mice in those cages which showed an abnormally high percentage of the disease. This may agree with the quotation from an article, "Journées d'études biologiques du cancer," by Romary, Bordeaux, 1933, who hypothesized that the causal organism of cancer is *Entamoeba blattae* which infests cockroaches, and that the nematode *Gongylonema* causes the lesions through which *E. blattae* gains entrance to the body (*90*).

ALLERGIES

Bullous erythema

Bedbug bite

Cimex lectularius. Bedbug bites caused recurring blisters up to size of pigeon's egg across back of both calves above maximum circumference. Occurred only in women who had been bitten by bugs which inhabited grooves on lower edges of tram-car seat slats. Diagnosed as bullous erythema. Did not occur in men because of their protective trousers. After bedbugs were eliminated from tram cars, the attacks stopped (*91*).

VITAMIN DEFICIENCIES

Beriberi

Cimex lectularius; Cimex hemipterus. Based on inference, bedbugs may spread beriberi (*92*).

IRON DEFICIENCY

Hypochromic anemia

Cimex lectularius; Cimex hemipterus. The mean iron content of 100 large bedbugs was 0.73 mg., and of 100 mixed-size bugs 0.37 mg. Repeated small blood meals resulting in some blood loss may be significant in causing iron deficiency among infants and young children in the Hyderabad area in South India (*93*).

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Water Pollution Control Policies

A policy statement issued by the Board of Control of the Water Pollution Control Federation in 1962 reaffirms the federation's original policies set up in 1960. The 13-point statement also includes additions which stress the continued national need for effective and greater pollution abatement.

Among the revised articles are the following declarations:

• That decisions on the type and degree of treatment and control of wastes, and the disposal and utilization of adequately treated waste water, must be based on thorough consideration of all the technical and related factors involved in each portion of each drainage basin.

• That administration of State and interstate pollution control should remain in the hands of State and interstate water pollution control agencies which must be supported by increased budgets and adequately staffed by well-trained and compensated engineers, scientists, and other personnel. The rights of State and interstate agencies to control and protect water resources must be accompanied by equal responsibilities to perform their functions effectively.

• That Federal activity in water pollution control should be administered by the Public Health Service, which has demonstrated that it is best fitted to perform these functions by virtue of its long experience and close cooperation with State health departments and State and interstate water pollution control agencies.

• That while the primary objective of pollution control must be the protection of the public health, other objectives add impelling reasons for protecting the nation's water resources, such as the need for the use and re-use of surface and ground waters which receive and dilute liquid wastes.

Other articles of the statement affirm that the responsibility for water pollution control must be shared individually and jointly by industry and local, State, and Federal governments; that basic and applied research by competent personnel must be encouraged; and that administration of pollution control must be firm, effective, and equitable.

The statement further calls for a public information program as to the hazards of pollution, mandatory certification or licensing of operating personnel, standards for radiation hazards which are primarily directed toward protection of public health, control of toxic and exotic chemicals at the source, and establishment of fiscal laws and practices toward economical and effective means for financing construction, operation, and upgrading of waste-water treatment works.

Copies of the policy statement or other information on organization activities may be obtained from the Water Pollution Control Federation, 4435 Wisconsin Avenue, Washington 16, D.C.