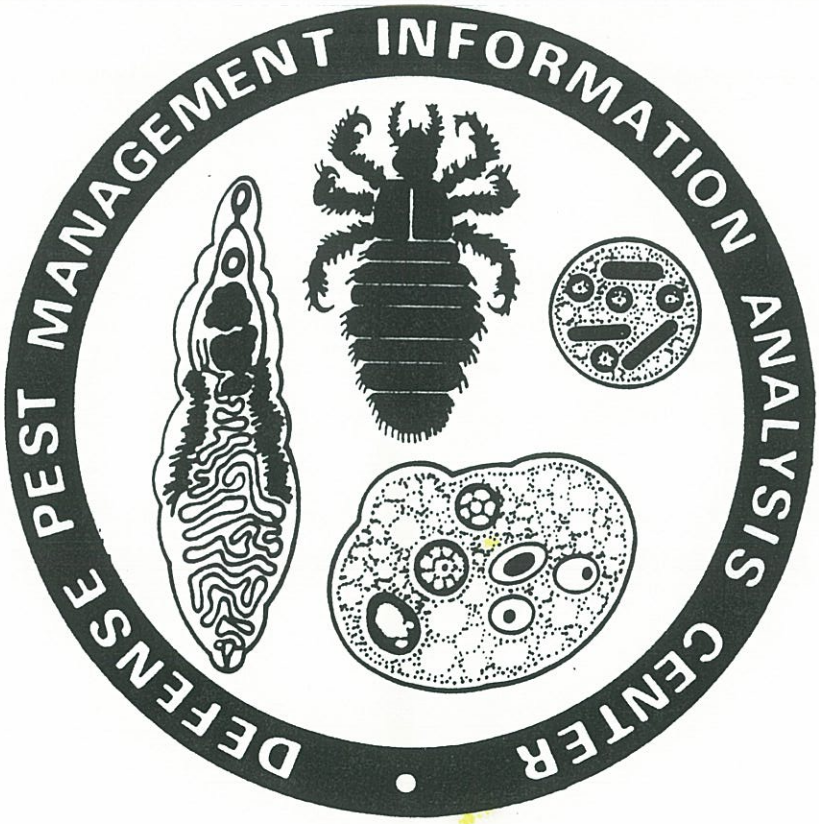
DISEASE VECTOR ECOLOGY PROFILE

SAUDI ARABIA



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LOUSE-BORNE DISEASES
OF SAUDI ARABIA

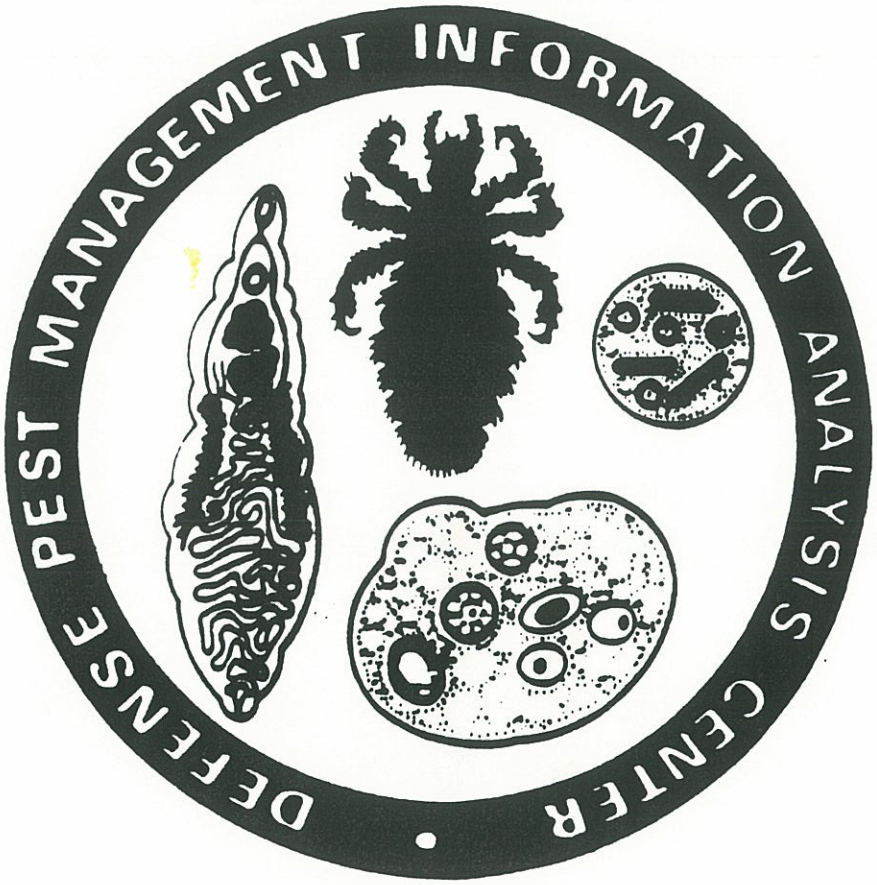
DISEASES AND EPIDEMIOLOGY	VECTOR, TRANSMISSION AND IMPORTANCE	VECTOR BIONOMICS
RELAPSING FEVER (Louse-borne) Borrelia recurrentis (Spirochaete) Disease is endemic throughout Saudi Arabia. Transmission occurs year-round. Poor sanitary and personal hygiene contribute to louse-borne transmission. Historically, epidemics are associated with refugee, famine or military conflict situations. Outbreaks occurred through the 1970s. Symptoms include recurring high fever lasting 5-7 days, nausea, body aches, enlarged spleen and liver, and jaundice.	Man is the only host. Transmitted by crushing the bodies of infected lice or by rubbing louse feces into lacerations or abrasions of the skin. Scratching the site of a louse bite is a frequent mode of entry. Louse feces do not contain infective Borrelia. VECTOR: Pediculus humanus humanus (Human body louse)	See EPIDEMIC TYPHUS

LOUSE-BORNE DISEASES OF SAUDI ARABIA

DISEASES AND EPIDEMIOLOGY	VECTOR, TRANSMISSION AND IMPORTANCE	VECTOR BIONOMICS
EPIDEMIC TYPHUS Rick..sia prowazeki Saudi Arabia is at the southern extreme of the distribution range of this disease. If present, it is confined to the northern provinces. The disease is commonly associated with overcrowding, transient populations of people and poor sanitary conditions. Symptoms include high fever, chills, toxemia and macular rash. An outbreak was reported among nomadic Saudi tribes in 1961 resulting in 49 cases and 2 deaths.	Transmitted by feces or gut contents of an infected louse being inoculated into a skin abrasion. Scratching the site of a louse bite is a common mode of entry. Pathogen may remain viable in louse feces up to 2 months, and aerosol transmission may occur. PRIMARY VECTOR: Pediculus humanus humanus (Human body louse)	Lice live on host clothing except when feeding. Eggs are deposited in clothing often along seams. Eggs hatch in a few days and immatures commence blood-feeding at once. Maturity is reached about 2-3 weeks after hatching. Adults live about one month. Infected lice usually die in about 10 days, but survivors are infective for life. Lice are very sensitive to temperature changes. They will leave a patient with a high fever. Lice are normally acquired by contact with louse-infested persons or infested clothing or bedding.

DISEASE VECTOR ECOLOGY PROFILE

IRAQ



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LOUSE-BORNE DISEASES
OF IRAQ

Relapsing Fever?

DISEASES AND EPIDEMIOLOGY	VECTOR, TRANSMISSION, AND IMPORTANCE	VECTOR BIONOMICS

EPIDEMIC TYPHUS

Rickettsia prowazekii

Epidemic typhus occurs mostly in the colder north and northeastern part of Iraq. The infection is conveyed from human to human through louse feces which are rubbed into abrasions when the louse is crushed during scratching. Epidemics can occur whenever a population becomes louse infested (which often occurs when people live in colder areas under poor hygienic conditions). The onset of the disease is often sudden, marked by headache, chills, prostration, fever, and general pains. In the absence of specific therapy, the case fatality rate increases with age and varies from 10-40%.

Humans are the reservoir and are responsible for maintaining the infection during interepidemic periods. The body louse is infected by feeding on the blood of an infected person. The infected louse then feeds on another person and usually defecates while feeding. Humans are infected by rubbing rickettsiae contaminated louse feces or a crushed louse into the bite or into superficial abrasions.

PRIMARY VECTOR:

Pediculus humanus
(Human body louse)

Different terminology in profile for Saudi Arabia

Lice are parasitic on animals. The human body louse is found on the body of people who do not keep clean or change their under garments frequently. Female lice lay up to 300 eggs during their lives which last up to a month under optimal conditions. At 30°C the eggs hatch in eight or nine days. Lice prefer a temperature of 29°-30°C, and they avoid, when possible, any change in humidity. Primary defense against human lice involves personal cleanliness of body and clothing.

Pediatr Emerg Care (UNITED STATES) Dec 1989, 5 (4) p241-2
Journal Code: PAU
Languages: ENGLISH
Pediculosis has many cutaneous manifestations. A previously unreported presentation is documented in this narrative. Maculae ceruleae associated with the head louse was initially confused with traumatic bruising and child abuse.

<DIALOG File 155: >
06528491 88173491
Pediculosis capitis among primary schoolchildren in urban and rural areas of Kwara State, Nigeria.
Ebomoyi E
Dept. of Epidemiology and Community Health, University of Ilorin, Kwara State, Nigeria.
J Sch Health Mar 1988, 58 (3) p101-3, ISSN 0022-4391 Journal Code: K13
Languages: ENGLISH
The prevalence of head lice (pediculosis humanus capitis) was investigated among urban and rural schoolchildren in Ilorin, capital of Kwara State, Nigeria, and two neighboring rural communities. Among the pupils, 57 (3.1%) of 1,842 urban schoolchildren examined were infested, compared with only one (0.1%) of 1.056 rural school pupils. More female pupils had infestations. Children less than age five, primary I pupils, and pupils in primary VI were not infested. Urban schoolchildren with 41-45 lice per head constituted 7.7% of those infested with nits and lice. Most infected children had one-five and 11-15 lice per head. The school health component of the national primary health care scheme should be intensified to screen schoolchildren regularly for pediculosis and other childhood diseases.

<DIALOG File 155: >
06303048 87277048
Pilot study of the prevalence of head lice infestation in a population of Saudi Arabian children.
Boyle P
Fam Pract Jun 1987, 4 (2) p138-42, ISSN 0263-2136 Journal Code: FAM
Languages: ENGLISH
The prevalence of infestation with the head louse, Pediculus capitis, was assessed among the child population, from birth to 10 years old inclusive, of the rapidly expanding Saudi Arabian city of Jeddah. Over a period of two months, 300 consecutive children attending the general practitioner for any reason were examined specifically to identify those infested with head lice: 37 cases of active infestation were found, which is an overall prevalence of 12%. An interesting distribution, however, was noted in respect of age, ranging from less than 2% in the first year of life, rising rapidly to around 30% in ages six to eight years, thereafter declining steadily to about 16% by age 10 years. The distribution of infestation among males and females was broadly similar. These results show a high head lice infestation, particularly in the early school years, where presumably interpersonal contacts are most frequent, facilitating contagious spread. As the head lice is known to spread several viral and rickettsial diseases, such as relapsing fever and typhus, greater efforts should be made towards patient

(cont. next page)

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Jackie I will be forwarding this article in its entirety. It is especially pertinent since the sample was taken from families of military personnel.

education in hygiene, and towards identifying and treating the disease when found.

<DIALOG File 155: >
06135974 87109974
Structural abnormalities of the hair shaft.
Whiting DA
J Am Acad Dermatol Jan 1987, 16 (1 Pt 1) p1-25, ISSN 0190-9622
Journal Code: HVG
Languages: ENGLISH
Abnormalities of the hair shaft are varied and often confusing. They do require accurate recognition, which can be helpful in the diagnosis and management of a hair disorder, or in the detection of underlying disease. A reliable evaluation of hair shaft abnormality is dependent on familiarity with the vast structural variations in normal hair, the hair growth cycle, and the changes produced by weathering. These structural abnormalities can be congenital or acquired, and some are associated with hair fragility. The classification used here has the advantage of simplicity and is keyed to easy recognition of the abnormalities under the light microscope. The four major types of abnormality are fractures, irregularities, twisting, and extraneous matter affecting the hair shaft. The diagnostic features of the different hair shaft abnormalities are outlined.

<DIALOG File 155: >
05936777 86237777
Resurgent yaws and other skin diseases in the Western Province of the Solomon Islands.
Eason RJ; Tasman-Jones T
Helena Goldie Hospital, Munda, Solomon Islands.
P N G Med J Dec 1985, 28 (4) p247-50, ISSN 0031-1480 Journal Code: YEU
Languages: ENGLISH
A clinical study of the extent and nature of skin disease was undertaken among 10,224 Melanesians in the Western Province of the Solomon Islands. It was performed concurrently with a survey and selective mass treatment campaign for yaws which has reappeared in the area for the first time in 20 years. For children under 15 years old, prevalence rates for pyoderma and infectious yaws were 52% and 8.5%, respectively. Tinea versicolor was the commonest superficial dermatomycosis affecting nearly half of all adults seen. Glabrous skin (16% of all cases) and nails (25% of all cases) were the principle sites infected by the dermatophytes. Tinea imbricata, whilst uncommon, was restricted to small endemic foci. Pediculosis capitis was universal but scabies was present in only 4% of young children. Unfavourable environmental conditions and misconceptions about personal hygiene are important aetiological determinants. Education and motivation at a village level will be the mainstay of future control with specific therapy generally reserved for treponematoses, extensive dermatophytoses and scabies.

<DIALOG File 155: >
05701122 86002122
Pediculosis capitis (head lice). Infectious Diseases and Immunization Committee, Canadian Paediatric Society.
Can Med Assoc J Oct 15 1985, 133 (8) p741-2, ISSN 0008-4409 Journal Code: CKWe:
Languages: ENGLISH

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IM	=	intramuscular
IU	=	international unit
IV	=	intravenous
kb	=	kilobase
kg	=	kilogram
LA	=	latex agglutination
lbs	=	pounds
m	=	meter
meq	=	milliequivalents
mg	=	milligram
µg	=	microgram
ml	=	milliliter
mm	=	millimeter
µm	=	micrometer
nm	=	nanometer
ppm	=	parts per million
RBC	=	red blood cell
RIA	=	radioimmunoassay
sp. or spp.	=	species
UNDP	=	United Nations Development Programme
USA	=	United States of America
USDA	=	US Department of Agriculture
UK	=	United Kingdom
URI	=	upper respiratory infection
USPHS	=	US Public Health Service
USSR	=	Union of Soviet Socialist Republic
UV	=	ultraviolet
v.	=	versus
WHO	=	World Health Organization
WBC	=	white blood cell

Control of Communicable Diseases in Man

Abram S. Benenson, editor

Fifteenth Edition 1990

An official report of the American Public Health Association

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1015 Fifteenth Street, NW
Washington, DC 20005

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02/21/92

2) Stress thorough cooking of crustacea.
3) Dispose of sputum and feces in a sanitary manner.
4) Control snails by molluscicides where feasible.

B. *Control of patient, contacts and the immediate environment:*

1) Report to local health authority: Official report not ordinarily justifiable, Class 5 (see Preface).
2) Isolation: None.
3) Concurrent disinfection: Of sputum and feces.
4) Quarantine: None.
5) Immunization of contacts: None.
6) Investigation of contacts and source of infection: None.
7) Specific treatment: Praziquantel (Biltricide®).

C. *Epidemic measures:* In an endemic area, occurrence of small clusters of cases, or even sporadic infections, is an important signal for examination of local waters for infected snails, crabs and crayfish, and determination of reservoir mammalian hosts to establish appropriate controls.

D. *Disaster implications:* None.

E. *International measures:* WHO Collaborating Centres at Tulane University and in Beijing.

PEDICULOSIS

ICD-9 132

1. **Identification**—Infestation of the head, the hairy parts of the body and clothing (especially along the seams of inner surfaces), with adult lice, larvae and nits (eggs), which results in severe itching and excoriation of the scalp or body. Secondary infection may occur with ensuing regional lymphadenitis (especially cervical). Crab lice usually infest the pubic area; they may also infest hair of the face (including eye lashes), axillae and body surfaces.

2. **Infesting agents**—*Pediculus capitis*, the head louse; *P. humanus*, the body louse; and *Phthirus pubis*, the crab louse. Only the body louse is of major medical importance as the vector of epidemic typhus, trench fever and louse-borne relapsing fever. Lice of lower animals do not infest man, although they may be present transiently.

3. **Occurrence**—Worldwide. Outbreaks of head lice are common among children in schools and institutions.

4. **Reservoir**—People.

5. **Mode of transmission**—For head and body lice, direct contact with an infested person; for body lice and to a lesser extent for head lice, indirect contact with their personal belongings, especially shared clothing and headgear. While other means are possible, crab lice are most frequently transmitted through sexual contact. Lice leave a febrile host; fever and overcrowding increase transfer from person to person.

6. **Incubation period**—Under optimal conditions the eggs of lice hatch in a week, and sexual maturity is reached approximately 8-10 days after hatching.

7. **Period of communicability**—As long as lice or eggs remain alive on the infested person or in clothing.

8. **Susceptibility and resistance**—Any person may become louse-infested under suitable conditions of exposure. Repeated infestations often result in dermal hypersensitivity.

9. **Methods of control**—

A. *Preventive measures:*

1) Avoid physical contact with infested individuals and their belongings, especially clothing and bedding.
2) Health education of the public on the value of laundering clothing and bedding in hot water (55°C or 131°F for 20 min.) or dry cleaning to destroy nits and lice.
3) Regular direct inspection of all primary school children for head lice and, when indicated, of body and clothing; this applies to children in schools, institutions, nursing homes and summer camps.

B. *Control of patient, contacts and the immediate environment:*

1) Report to local health authority: Official report not ordinarily justifiable; school authorities should be informed, Class 5 (see Preface).
2) Isolation: Contact isolation until 24 hours after application of effective insecticide.
3) Concurrent disinfection: With body lice among members of a family or group: Clothing, bedding and other appropriate vehicles of transmission (e.g., cosmetic articles) should be treated by laundering in hot water, dry cleaning or application of an effective chemical insecticide and ovicide (see 9B7, below). After chemical treatment has been completed, clothes and laundry facilities should be rinsed.
4) Quarantine: None.
5) Immunization of contacts: Does not apply.
6) Investigation of contacts and source [illegible]ection: Exami-

02/21/92 10:33 TROPICAL PUBLIC HEALTH 007

nation of household and other close personal contacts, with concurrent treatment as indicated.

7) Specific treatment: For head and pubic lice: 1% permethrin (a synthetic pyrethroid) creme rinse (NIX®) is highly effective for control of both head lice and nits. It binds to the hair and remains effective for several weeks, so retreatment is not necessary. Other effective agents include pyrethrins synergized with piperonyl butoxide (A-200 Pyrinate®, RID® and XXX®), 1% gamma benzene hexachloride lotions (lindane, Kwell®; not recommended for infants, young children, and pregnant or lactating women), carbaryl and benzyl benzoate. With these agents, retreatment after 7-10 days is recommended to assure that no eggs have survived. The removal of all nits is recommended as a prerequisite for return to school of children previously found infested and treated.

For body lice: Clothing and bedding should be washed with the hot water cycle of an automatic washing machine or dusted with powders containing 1% lindane (if washer not available), or preferably 1% malathion or pyrethrins with piperonyl butoxide or carbaryl (in view of widespread resistance to lindane), and then laundered before using. Abate® (temephos) as a 2% dusting powder is also effective and is recommended by WHO for use in areas where strains of body lice are resistant to malathion.

C. ***Epidemic measures:*** Mass treatment as recommended in paragraph 9B7, above.

D. ***Disaster implications:*** Diseases for which *P. humanus* is a vector are particularly prone to occur at times of social upheaval (see Typhus fever, Epidemic).

E. ***International measures:*** None.

PERTUSSIS ICD-9 033.0
PARAPERTUSSIS ICD-9 033.1
(Whooping Cough)

1. **Identification**—An acute bacterial disease involving the respiratory tract. The initial catarrhal stage has an insidious onset with an irritating cough which gradually becomes paroxysmal, usually within 1-2 weeks, and lasts for 1-2 months or longer. Paroxysms characterized by

repeated violent coughs; each series of paroxysms has many coughs without intervening inhalation and may be followed by a characteristic crowing or high pitched inspiratory whoop. Paroxysms frequently end with the expulsion of clear, tenacious mucus, often followed by vomiting. Infants less than 6 months old and adults often do not have the typical whoop or cough paroxysm. The number of fatalities in the USA is currently low; approximately 90% of deaths are among children under 1 year of age, and 75% are under 6 months. Case fatality rate is 0.5% in infants less than 6 months old in the USA. Morbidity and mortality are higher in females than males. In unimmunized populations, especially those with underlying malnutrition and multiple enteric and respiratory infections, pertussis is among the most lethal diseases of infants and young children. Pneumonia is the most common cause of death; fatal encephalopathy, probably hypoxic, and inanition from repeated vomiting occasionally occur.

In recent years in the USA, pertussis in adolescents and young adults, varying in severity from a mild, atypical respiratory illness to the full-blown syndrome, has been recognized with increasing frequency. Many of these cases occur in previously immunized persons, undoubtedly as a consequence of waning immunity.

Parapertussis is a similar but usually milder disease clinically indistinguishable from pertussis. It is usually seen in school-age children, and occurs relatively infrequently. Differentiation between *Bordetella parapertussis* and *B. pertussis* is based on culture, biochemical and immunologic differences. A similar clinical syndrome has been reported in association with viruses, especially adenoviruses.

Diagnosis is based on the recovery of the etiologic organism from nasopharyngeal swabs obtained during the catarrhal and early paroxysmal stages, or directly on cough plates. Direct FA staining of nasopharyngeal secretions may provide rapid presumptive diagnosis, but requires an experienced laboratory; false-positive and false-negative results can occur. Strikingly high total WBC counts with a strong preponderance of lymphocytes are found as the whooping stage develops; this may not occur in young infants.

2. **Infectious agents**—*Bordetella pertussis*, the pertussis bacillus; *B. parapertussis* causes parapertussis.

3. **Occurrence**—A disease common to children everywhere, regardless of race, climate, or geographic location. There has been a marked decline in incidence and mortality rates during the past four decades, chiefly in communities fostering active immunization and where good nutrition and medical care are available. From 1979-82, an average of 1,750 cases was reported annually in the USA. During 1983-87, reported cases increased to nearly 3,000 annually. In recent years, incidence rates have increased in countries where immunization levels have fallen (e.g., England, Japan and Sweden).

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C. *Epidemic measures:*

1) Search intensively for the case or carrier who is the source of infection and for the vehicle (water or food) by which infection was transmitted.
2) Exclude suspected food.
3) Pasteurize or boil milk, or exclude milk supplies and other foods suspected on epidemiologic evidence, until safety is assured.
4) Chlorinate suspected water supplies adequately under competent supervision or do not use them. All drinking water must be chlorinated, treated with iodine or boiled before use.
5) Routine use of vaccine is not recommended.

D. *Disaster implications:* With disruption of usual water supply and sewage disposal, and of controls on food and water, transmission of typhoid fever may occur if there are active cases or carriers in a displaced population. Efforts to restore safe drinking water supplies and excreta disposal facilities are more appropriate than mass typhoid vaccination. Vaccination of such populations is generally not recommended.

E. *International measures:*

1) For typhoid fever: Immunization is advised for international travelers to endemic areas, especially if travel will likely involve exposure to unsafe food and water, or close contact with rural areas and indigenous populations. Not a legal requirement for entry into any country.
2) For both typhoid and paratyphoid fevers, WHO Collaborating Centres (see Preface).

TYPHUS FEVER

I. EPIDEMIC LOUSE-BORNE TYPHUS FEVER

ICD-9 080

(Louse-borne typhus, Typhus exanthematicus, Classic typhus fever)

1. **Identification**—A rickettsial disease with variable onset; often sudden and marked by headache, chills, prostration, fever and general pains. A macular eruption appears on the 5th-6th day, initially on the upper trunk, followed by spread to the entire body, but usually not to the face, palms or soles. Toxemia is usually pronounced and the disease terminates by rapid lysis after about two weeks of fever. In the absence of specific therapy, the case fatality rate increases with age and varies from

10 to 40%. Mild infections may occur without eruption, especially in children and persons partially protected by prior immunization. The disease may recrudesce years after the primary attack (Brill-Zinsser disease, ICD-9 081.1); this is milder, has fewer complications, need not be associated with lice, and has a lower case fatality rate.

The IFA test is most commonly used, but it may not discriminate between louse-borne and murine typhus unless the sera are differentially absorbed with the respective rickettsial antigen prior to testing. Other diagnostic methods are CF with group-specific or washed type-specific rickettsial antigens, toxin-neutralization test, and Weil-Felix reaction with *Proteus* OX-19. Antibody tests usually become positive in the second week. In Brill-Zinsser disease, the initial antibody is IgG and the Weil-Felix test may not be positive.

2. **Infectious agent**—*Rickettsia prowazekii.*

3. **Occurrence**—In colder areas where people may live under unhygienic conditions and are louse-infested; historically a concomitant of war and famine. Endemic foci exist in mountainous regions of Mexico, Central and S America, in central Africa and numerous countries of Asia. In the USA, the last outbreak of louse-borne typhus occurred in 1921. In the USA, this rickettsia exists as a zoonosis of flying squirrels (*Glaucomys volans*); there is serologic evidence that at least 33 humans have been infected from this source, possibly by the squirrel flea. Most of these have been in the East Coast states, but two cases were reported from Indiana, and one each from California, Illinois, Ohio, Tennessee and W Virginia.

4. **Reservoir**—Man is the reservoir and is responsible for maintaining the infection during interepidemic periods. The importance of the flying squirrel as a reservoir has not been determined.

5. **Mode of transmission**—The body louse, *Pediculus humanus*, is infected by feeding on the blood of a patient with acute typhus fever. Patients with recrudescent typhus (Brill-Zinsser disease) can infect lice and may serve as foci for new outbreaks in louse-infested communities. Infected lice excrete rickettsiae in their feces and usually defecate at the time of feeding. Man is infected by rubbing feces or crushed lice into the bite or into superficial abrasions. Inhalation of infective louse feces as dust may account for some infections. Transmission from the flying squirrel is presumed to be by the bite of the squirrel flea, but this has not been documented.

6. **Incubation period**—From 1 to 2 weeks, commonly 12 days.

7. **Period of communicability**—The disease is not directly transmitted from person to person. Patients are infective for lice during the febrile illness and possibly for 2-3 days after the temperature returns to normal. The louse is infective by passing rickettsiae in its feces within 2-6 days after the infected blood meal; it is infective earlier if crushed. The

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louse invariably dies within two weeks after infection; rickettsiae may remain viable in the dead louse for weeks.

8. Susceptibility and resistance—Susceptibility is general. One attack usually confers long-lasting immunity.

9. Methods of control—

A. Preventive measures:

1) Apply an effective residual insecticide powder at appropriate intervals by hand or power blower to clothes and persons of populations living under conditions favoring lousiness. A lousicide should be used which has been shown to be effective on local lice.
2) Improve living conditions with provisions for bathing and washing clothes.
3) Treat prophylactically people who are subject to unusual risk, by application of residual insecticide to clothing by dusting or impregnation.
4) Immunize susceptible persons or groups of persons entering typhus areas, particularly military or labor forces. However, no commercially prepared vaccine is now available in the USA or Canada. A live vaccine prepared from the attenuated strain E of *R. prowazekii* has shown promise.

B. Control of patient, contacts and the immediate environment:

1) Report to local health authority: Report of louse-borne typhus fever required as a Disease under Surveillance by WHO, Class 1A (see Preface).
2) Isolation: Not required after proper delousing of patient, clothing, living quarters and household contacts.
3) Concurrent disinfection: Appropriate insecticide powder applied to clothing and bedding of patient and contacts; launder clothing and bedclothes; treat hair for louse eggs (nits) with effective chemical agents. Lice tend to leave abnormally hot or cold bodies in search of a normothermic, clothed body (see 9A1, above). If death from louse-borne typhus occurs before delousing, delouse the body and clothing by thorough application of an insecticide.
4) Quarantine: Louse-infested susceptibles exposed to typhus fever ordinarily should be quarantined for 15 days after application of an insecticide with [illegible]idual effect.
5) Management of contacts: All immed[illegible]ntacts should [illegible]

6) Investigation of contacts and source of infection: Every effort should be made to trace the infection to the immediate source.
7) Specific treatment: Tetracyclines or chloramphenicol orally in a loading dose of 2-3 g, followed by daily doses of 1-2 g/day in 4 divided doses until the patient becomes afebrile (usually 2 days) plus 1 day. A single dose of doxycycline (5 mg/kg) is also curative. When faced with a seriously ill patient with possible typhus, suitable therapy should be started without waiting for laboratory confirmation.

C. Epidemic measures: The imperative measure for rapid control of typhus is application to all contacts of an insecticide with residual effect. Where infestation is known to be widespread, systematic application of residual insecticide to all persons in the community is indicated. If a vaccine is available (see 9A4, above), it should be administered.

D. Disaster implications: Typhus can be expected to be a significant problem in endemic areas if social upheavals and crowding occur in louse-infested populations.

E. International measures:

1) Telegraphic notification by governments to WHO and to adjacent countries of the occurrence of a case or an outbreak of louse-borne typhus fever in an area previously free of the disease.
2) International travelers: No country currently requires immunization against typhus for entry.
3) Louse-borne typhus is a Disease under Surveillance by WHO. WHO Collaborating Centres (see Preface).

II. MURINE TYPHUS FEVER ICD-9 081.0

(Flea-borne typhus, Endemic typhus fever, Shop typhus)

1. Identification—A rickettsial disease whose course resembles that of louse-borne typhus, but is milder. The case fatality rate for all ages is less than 1%; it increases with age.

Absence of louse infestation, seasonal distribution and sporadic occurrence of the disease help to differentiate it from louse-borne typhus. For laboratory diagnosis, see 1, section 1, above.

2. Infectious agent—*Rickettsia typhi (Rickettsia mooseri).*

3. Occurrence—Worldwide. Found in areas where people and rats occupy the same buildings and where large numb[illegible]f mice live. In the USA, fewer than 80 cases are reported annually. [illegible]nal peak is in late

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SPIRILLOSIS

ICD-9 026.0

(Spirillary fever, Sodoku, Rat-bite fever due to *Spirillum minus*)

Rat-bite fever caused by *Spirillum minus (S. minor)* is the common form of sporadic rat-bite fever in Japan and Asia. Untreated, the case fatality rate is approximately 10%. Clinically, *Spirillum minus* disease differs from streptobacillary fever in the rarity of arthritic symptoms and the distinctive rash of reddish or purplish plaques. The incubation period is 1-3 weeks, and the previously healed bite wound reactivates when symptoms appear. Laboratory methods are essential for differentiation; animal inoculation is used for isolation of the spirillum.

RELAPSING FEVER

ICD-9 087

1. **Identification**—A systemic spirochetal disease in which periods of fever lasting 2-9 days alternate with afebrile periods of 2-4 days; the number of relapses varies from 1 to 10 or more. Each febrile period terminates by crisis. The total duration of the louse-borne disease averages 13-16 days; the tick-borne disease usually lasts longer. Transitory petechial rashes are common during the initial febrile period. The overall case fatality rate in untreated cases is between 2-10%; it has exceeded 50% in epidemic louse-borne disease.

Diagnosis is made by demonstration of the infectious agent in darkfield preparations of fresh blood or stained thick or thin blood films, or by intraperitoneal inoculation of laboratory rats or mice with blood taken during the febrile period.

2. **Infectious agents**—In louse-borne disease, *Borrelia recurrentis*. In tick-borne disease, many different strains have been distinguished by area of first isolation and/or vector rather than inherent biologic differences. Strains isolated during a relapse often show antigenic differences from those obtained during the immediately preceding paroxysm.

3. **Occurrence**—Characteristically, epidemic where it is spread by lice; endemic where it is spread by ticks. Louse-borne relapsing fever occurs in limited areas in Asia, eastern Africa (Ethiopia and the Sudan), northern and central Africa, and S America. The endemic tick-borne disease is widespread throughout tropical Africa; foci exist in Spain, northern Africa, Saudi Arabia, Iran, India, and parts of central Asia, as well as in N and S America. Epidemic louse-borne relapsing fever has not been reported in the USA for many years; human cases and occasional outbreaks of tick-borne disease occur in limited a[illegible]f several western states and western Canada.

4. **Reservoir**—For louse-borne disease, man; for tick-borne relapsing fevers, wild rodents and ticks through transovarian transmission.

5. **Mode of transmission**—Vector-borne; not directly transmitted from person to person. Epidemic relapsing fever is acquired by crushing an infective louse, *Pediculus humanus*, so as to contaminate the bite wound or an abrasion of the skin. Man also is infected by the bite or coxal fluid of an argasid tick, principally *Ornithodoros turicata* and *O. hermsi* in the USA, *O. rudis* and *O. talaje* in Central and S America, *O. moubata* and *O. hispanica* in Africa, and *O. tholozani* in the Near and Middle East. These ticks usually feed at night, rapidly engorge and leave the host; they have a longevity of 2-5 years and remain infective for their lifespan.

6. **Incubation period**—Five to 15 days; usually 8 days.

7. **Period of communicability**—The louse becomes infective 4-5 days after ingestion of blood from an infected person and remains so for life (20-40 days). Infected ticks can live for several years without feeding, remain infective during this period and pass the infection transovarially to their progeny.

8. **Susceptibility and resistance**—Susceptibility is general. Duration of immunity after clinical attack is unknown.

9. **Methods of control**—

A. Preventive measures:

1) Control lice by measures prescribed for louse-borne typhus fever (see Typhus fever, Epidemic Louse-Borne, 9A).
2) Control ticks by measures prescribed for Rocky Mountain spotted fever, 9A. Tick-infested human habitations present difficult problems and eradication is difficult or impossible. Spraying with approved acaricides such as diazinon, chlorpyrifos, propoxur or permethrin may be tried.
3) Use personal protection measures, including repellents and permethrin on clothing and bedding for persons with exposure in endemic foci.
4) Antibiotic chemoprophylaxis with tetracyclines may be taken after exposure (arthropod bites) when risk of acquiring the infection is high.

B. Control of patient, contacts and the immediate environment:

1) Report to local health authority: Report of louse-borne relapsing fever required as a Disease under Surveillance by WHO, Class 1A; tick-borne disease, in selected areas, Class 3B (see Preface).

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2) Isolation: Blood/body fluid precautions. The patient, his clothing, all household contacts and the immediate environment should be deloused or freed of ticks.
3) Concurrent disinfection: None, if proper disinfestation has been carried out.
4) Quarantine: None.
5) Immunization of contacts: None.
6) Investigation of contacts and source of infection: For the individual tick-borne case, search for sources of infection; for louse-borne disease, application of appropriate lousicidal preparation to infested contacts (see Pediculosis, 9B6).
7) Specific treatment: Tetracyclines.

C. *Epidemic measures:* When reporting has been good and cases are localized, apply 1% permethrin dust or spray (an insecticide with residual effect) to contacts and their clothing, and permethrin spray at 0.003-0.3 kg/hectare (2.47 acres) to the immediate environment of all reported cases. Where infection is known to be widespread, apply permethrin systematically to all persons in the community, or to outdoor target areas where ticks are prevalent. For sustained control, a treatment cycle of one month is recommended during the transmission season.

D. *Disaster implications:* A serious potential hazard among louse-infested populations. Epidemics are common in wars, famine, and other situations where the prevalence of pediculosis is enhanced, as among overcrowded, malnourished populations with poor personal hygiene.

E. *International measures:*

1) Telegraphic notification by governments to WHO and adjacent countries of the occurrence of an outbreak of louse-borne relapsing fever in an area previously free of the disease.
2) Louse-borne relapsing fever is not a disease subject to the International Health Regulations, but the measures outlined in 9E1, above, should be followed since it is a Disease under Surveillance by WHO.

RESPIRATORY DISEASE, ACUTE VIRAL (EXCLUDING INFLUENZA)

(Acute viral rhinitis, A.v. pharyngitis, A.v. laryngitis, etc.)

Numerous acute respiratory illnesses of known and presumed viral etiology are grouped here under the general title of Respiratory Disease, Acute Viral. Clinically, and by CIOMS taxonomy, infections of the upper respiratory tract can be designated as acute viral rhinitis (upper respiratory infections, URI), acute viral pharyngitis, and acute viral laryngitis; and infections involving the lower respiratory tract can be designated as acute viral tracheobronchitis, bronchitis, bronchiolitis or acute viral pneumonia. These respiratory syndromes are associated with a large number of viruses, each of which is capable of producing a wide spectrum of acute respiratory illnesses. The illnesses caused by known agents have important epidemiologic attributes in common, such as reservoir and mode of transmission. Many of the viruses invade any part of the respiratory tract; others show a predilection for certain anatomic sites. Some predispose to bacterial complications. Morbidity and mortality from acute respiratory diseases are especially significant in pediatric practice; in adults, the relatively high incidence and resulting disability, with consequent economic loss, make diseases of this group a major health problem worldwide.

Several other nonbacterial infections of the respiratory tract are recognized as disease entities and are presented as separate chapters because they are sufficiently uniform in their clinical and epidemiologic manifestations and occur in such regular association with specific infectious agents: influenza, ornithosis, enteroviral vesicular pharyngitis (herpangina) and epidemic myalgia (pleurodynia) are examples. Particularly in pediatric practice, influenza must be considered in cases of acute respiratory tract disease.

Symptoms of upper respiratory tract infection, mainly pharyngotonsillitis, can be produced by bacterial agents, of which group A streptococcus is the most common. Practical management of acute respiratory disease depends on the differentiation of viral infections from disease entities for which specific antimicrobial measures are available; thus, it is important to rule out group A streptococcal infection, especially in children over 2 years, by appropriate culture even though more illnesses are caused by viruses. In addition, in outbreaks or epidemics of continuing high incidence not due to streptococci, it is important to identify the cause in a representative sample of typical cases by appropriate clinical and laboratory methods to rule out other diseases, e.g., mycoplasmal pneumonia and Q fever, for which specific treatment may be effective.

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surgical closure of the wounds should be avoided. Antimicrobial prophylaxis with penicillin or the combination of amoxicillin and clavulanic acid may be given, but efficacy of prophylactic regimens has not been proven.

Pediculosis

Clinical Manifestations: Itching is the most common symptom of head lice infestation, but most children with light infestations (1 to 5 lice) do not complain. Itching is usually intense in persons with body or pubic lice infestations. Excoriation can result in a pyoderma with impetigo or regional lymphadenopathy. A characteristic sign of heavy pubic lice infestation is the bluish or slate-colored maculae caeruleae on the chest, abdomen, and thighs. Pubic lice can also infest eyelashes, eyebrows, and body and facial hair.

Parents may become aware of infestation by finding lice or eggs (nits) in their child's hair usually near the nape of the neck. In temperate climates, head lice deposit their eggs on the hair shaft near the scalp. Thus, the duration of infestation can be estimated by the distance of the nit from the scalp; nits 10 or more millimeters from the scalp have been present for 2 weeks or more and are unlikely to be viable.

Etiology: Three species of lice infest humans: *Pediculus humanus capitis*, the head louse; *P humanus corporis*, the body louse; and *Pthirus pubis*, the pubic or crab louse. Ova hatch in a week. Both nymphs and adult lice feed on human blood.

Epidemiology: Head lice infestation in day care and school-aged children, and pubic lice infestations in adolescents and young adults are common in the United States. Head lice occur in all socioeconomic groups, but in the United States, black persons are less commonly infested than white persons. Hair length does not influence infestation. Head or pubic lice infestation is not a sign of uncleanliness. However, body lice generally are found on persons with poor hygiene.

Transmission of *P capitis* occurs by direct contact with infested individuals or indirectly by contact with their personal belongings such as combs, brushes, and hats. Fomites play a major role in body lice transmission, a minor role in head lice transmission, and practically no role in pubic lice transmission. Lice generally cannot survive away from the host for more than 48 hours. Although body lice lay eggs in clothing, eggs generally do not survive away from the scalp at room temperatures for more than 7 days. Pubic lice usually are transmitted through sexual contact. Only body lice have been implicated as vectors of disease (epidemic typhus, trench fever, and relapsing fever). Head lice are not a major health hazard.

The **incubation period** is not known.

Diagnostic Tests: Identification of eggs, nymphs, and lice with the naked eye is possible; the diagnosis can be confirmed by using a hand lens or microscope.

Treatment: Permethrin, a synthetic pyrethroid (10-minute hair rinse); natural pyrethrin-based products (10-minute shampoos); lindane 1% (4-minute shampoo); and malathion 0.5% (8- to 12-hour lotion) are each effective in treating pediculosis of the scalp (see Table 44 for trade names). Lindane-resistant lice have not been reported in the United States. Pyrethrin products are available without prescription. Permethrin, pyrethrin products, and malathion have lower potential toxicity than lindane, but no serious adverse effects have been associated with any of these products when used according to package instructions. Toxicity with lindane has been reported only with misuse, such as ingestion or prolonged administration. When pyrethrin products or lindane is used, many experts recommend a second treatment 7 to 10 days later to kill newly hatched lice. A single treatment of permethrin or malathion appears adequate because these products persist in the hair for at least 2 weeks. Some experts advise a second application 7 days after the first irrespective of the choice of therapy.

Use of a fine-toothed comb aids in the mechanical removal of nits. Applying a damp towel to the scalp for 30 to 60 minutes, soaking the hair with white vinegar (3% to 5% acetic acid) followed by application of a damp towel soaked in the same solution for 30 to 60 minutes, or using a commercial rinse (Step 2*) containing 8% formic acid can facilitate the removal of nits by combing.

For infestation of eyelashes by the crab lice, petrolatum ointment applied twice daily for 8 to 10 days is effective. Nits should be removed mechanically from the eyelashes.

Isolation of the Hospitalized Patient: Contact isolation until therapy has been started is indicated.

Control Measures (for Pediculosis of the Scalp):

- Contacts should be examined and treated if they are infested. Differentiation of nits from benign hair casts (a layer of follicular cells that easily slide off the hair shaft) can be difficult. Bedmates should be treated prophylactically.

*GenDerm Corporation, Northbrook, IL.

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Isolation of the Hospitalized Patient: Drainage/secretion precautions should be used until the lesions stop draining. Respiratory isolation should be considered for patients with pneumonic tularemia.

Control Measures:

- Persons at risk should minimize opportunities for insect bites by wearing protective clothing and by frequent inspection and removal of ticks from the skin and scalp; insect repellents may be of some value (see Control Measures for Prevention of Tick-Borne Infections, page 112).
- Children should be discouraged from handling sick or dead rabbits and rodents.
- Rubber gloves should be worn when handling wild rabbits and other potentially infected animals.
- Wild rabbit meat should be thoroughly cooked.
- Face masks and rubber gloves should be worn by those working with cultures or infective material in the laboratory.
- Drainage/secretion precautions should be used for handling contaminated articles.
- Interstate or interarea shipment of infected animals should be prohibited.
- A live attenuated vaccine (available from the Centers for Disease Control) is recommended for those repeatedly exposed to the organism, such as laboratory research technicians.

Endemic Typhus

(Murine Typhus)

Clinical Manifestations: Endemic typhus resembles epidemic typhus but is usually milder, with less acute onset and less severe systemic symptoms. In young children, the disease is very mild. Fever can be accompanied by persistent headache and myalgias. The rash is typically macular or maculopapular, appears during days 3 to 5 of illness, lasts 4 to 8 days, and tends to remain discrete, with sparse lesions and no hemorrhage. The disease seldom lasts longer than 2 weeks, and visceral involvement does not usually occur. The disease is rarely fatal.

Etiology: Endemic typhus is caused by *Rickettsia typhi* (formerly *R mooseri*), an organism antigenically similar to *R prowazekii*.

Epidemiology: Rats, in which infection is inapparent, are the natural hosts. The vector for transmission to rats and occasionally and accidentally to humans is the rat flea (usually *Xenopsylla cheopis*). The disease is worldwide in distribution, affects all races, tends t more commonly in adults and in males, and is most comm April to October. The disease is infrequent in the United State cases occur in focal areas in southern California, the south Gulf Coast and southern border states, and Hawaii. Exposure and their fleas is the major risk factor, although a history exposure is frequently absent in infected patients.

The **incubation period** is 6 to 14 days.

Diagnostic Tests: Serum agglutinins against *Proteus* OX-19 peak weeks after the onset of disease, but these tests lack sensitiv specificity. Indirect fluorescent antibody, latex agglutination, c plement fixation antibody concentrations peak at a similar or s later time. A fourfold titer rise between acute and convalescent specimens is diagnostic but can also occur in patients with ep typhus. Serologic differentiation between these diseases is p by cross-absorption of the patient's serum. Isolation of the org in culture is possible but hazardous, and it requires speci facilities and experienced personnel.

Treatment: A single dose of tetracycline (5 mg/kg; maximum 200 the treatment of choice. Although tetracyclines should not be to children younger than 9 years, the risk of dental staining a single dose of doxycline is minimal. Chloramphenicol is a effective drug.

Isolation of the Hospitalized Patient: No special precautior recommended.

Control Measures: Rat fleas should be controlled by appropriate i ticides, preferably before the use of rodenticides, because th will seek alternate hosts when rats are not available. Rat popula should then be controlled by appropriate means. Endemic ty vaccine is no longer available. No treatment is recommended fo posed persons. This disease is reportable in a few states.

Epidemic Typhus

(Louse-Borne Typhus)

Clinical Manifestations: In epidemic typhus, the onset of high f chills, and diffuse aching accompanied by severe headache malaise is usually abrupt. Influenza-like illness is freque suspected. The rash appears 4 to 7 days later, beginning on the t

and spreading to the limbs. A concentrated eruption is present in the axillae. The rash is maculopapular, becomes petechial or hemorrhagic, then develops into brownish, pigmented areas. The face, palms, and soles are usually not affected. Mental changes are common, and delirium or coma may occur. Myocardial and renal failure occur when the disease is severe. Illness varies from moderately severe to fatal (10% to 40% mortality rate); when untreated it typically lasts 2 weeks and ends by lysis of fever and subsidence of symptoms. In untreated cases, mortality is uncommon in children, ranges from 10% to 40% in adults, and increases with increasing age. Brill-Zinsser disease is a relapse of louse-borne typhus that occurs years after the initial episode. Stress or an unknown factor serves to reactivate the rickettsiae. The recrudescent illness is similar to the primary infection, but is generally milder and of shorter duration.

Etiology: *Rickettsia prowazekii* is the cause.

Epidemiology: Humans are the major source of the organism, which is transmitted from person to person by the body louse *Pediculus humanus* subspecies *corporis*. All ages and races and both sexes are affected. Poverty, crowding, poor sanitary conditions, lack of bathing, and poor personal hygiene contribute to the spread of lice, and hence the disease. Currently, cases of typhus are rarely reported but have occurred throughout the world, including Asia, Africa, some parts of Europe, and Central and South America. Typhus was common during winter when conditions favor person-to-person transmission of the vector, the body louse. Rickettsiae are present in the blood and tissues of patients during the early febrile phase but not in secretions. Direct person-to-person spread of the disease does not occur in the absence of the vector.

Serologic evidence of epidemic typhus in flying squirrels in the United States has been reported, and cases in humans have been associated with contact with squirrels, their nests, or their ectoparasites. Squirrel-related epidemic typhus appears to be a milder illness than louse-borne epidemic typhus.

The **incubation period** is 1 to 2 weeks.

Diagnostic Tests: *R prowazekii* can be isolated from the blood by inoculation into guinea pigs and mice or the yolk sac of embryonated hens' eggs, but because isolation is dangerous, it is rarely attempted. Serum agglutinins against *Proteus* OX-19 reach peak titers 2 to 3 weeks after the onset of disease, but an indirect fluorescent antibody or complement fixation antibody test is preferred. A fourfold increase in antibody titer between acute and convalescent serum specimens is diagnostic of either epidemic or endemic typhus (see Endemic Typhus, page 515). An antibody absorption test can often differentiate the two diseases.

Treatment: Tetracycline or chloramphenicol, given intravenously or orally, is the antibiotic of choice. Tetracycline should not be given to children younger than 9 years unless the benefits are clearly greater than the risks of dental staining. Therapy is given until the patient is afebrile for at least 7 days; usual duration is 7 days. Cream and gel pediculocides containing pyrethrins (0.16% to 0.33%) and piperonyl butoxide (2% to 4%), crotamiton (10%), or lindane (1%) can be used for delousing. Convulsions have been reported in children receiving excessive doses of topical lindane.

Isolation of the Hospitalized Patient: No special precautions are recommended.

Control Measures: Thorough delousing in epidemic situations, particularly among exposed contacts of cases, is recommended. Several applications may be needed because the lice eggs are resistant to most insecticides. Washing clothes in hot water kills lice and eggs. During epidemics, insecticides dusted onto the clothes of louse-infested populations are effective in louse control efforts. In some circumstances, preventing flying squirrels from living in human dwellings by sealing their access ports is recommended. Epidemic typhus vaccine is no longer available in the United States. Cases should be reported to public health departments.

Varicella-Zoster Infections

Clinical Manifestations: Primary infection with varicella-zoster virus (VZV) results in chickenpox. Chickenpox is manifest by a generalized, pruritic, vesicular rash, with a mild fever and mild systemic symptoms. A variety of complications occur, including bacterial superinfection, thrombocytopenia, arthritis, hepatitis, encephalitis or meningitis, and glomerulonephritis. Reye syndrome can follow some cases of chickenpox. In immunocompromised children, progressive varicella characterized by continuing eruption of lesions and a high fever into the second week of the illness can occur. Encephalitis, pancreatitis, hepatitis, or pneumonia can develop. Children with AIDS can develop chronic chickenpox with new lesions appearing during a period of months. Chickenpox is often more severe in adults. Pneumonia, although rare in normal children, is the most common complication in older individuals.

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HEAD LICE

Detection, Treatment, and Prevention

A program of instruction for parents, teachers, and children

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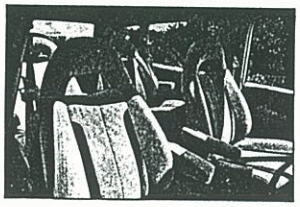

slide 17: Don't forget the car. It's just as important to vacuum car seats as it is sofas and carpets inside the house.

slide 18: A parent's first experience with head lice--even if it's just hearing that someone else's child has them--can be unnerving. But there's no cause for fear, guilt, or anger. Head lice have never been shown to transmit disease. They can cause the discomfort of itching. Vigorous scratching can open the way for skin infection. But by and large, head lice are a nuisance and nothing more.

slide 19: Effective treatments are widely available without a prescription. Follow the instructions carefully. It's best to use a medication that kills lice and nits and offers protection against reinfestation. It is important that you follow up treatment by combing out the nits. Otherwise, egg cases are taken as evidence of reinfestation and could prevent your child from being readmitted to school.

slide 20: If your child gets head lice and you need more information or a recommendation of treatment, ask the school nurse or your local pharmacist. And remember...head lice are a nuisance, not a health crisis, and they can be relatively easy to eliminate.

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Report: Head Lice

submitted by Gilbert Simon, M.D., Sacramento, CA (Pediatrician)

Send Debbie Auwaerter to comment back Gs

No-nits Policy: Is it relevant in the nineties?

Unlike the body and pubic louse, the head louse does not transmit bacteria or viruses. Essentially, this is an "infestation without a disease," whose impact is mainly financial and educational. Some estimate the cost at $65 per case, an annual national bill of $195 to $650 million. In terms of days lost from school, the average infested student who has access to health care will miss one to three days . . . those without can miss weeks to months.

Schools typically manage outbreaks of head lice by sending the child home to be treated. This is seldom much of a problem, since many shampoos, of the lindane, malathion, or pyrethrin class, are extremely effective louse killers. Many schools also insist that the child be kept at home until all the nits have been removed. That is a problem for a large and rapidly increasing number of families.

Common Sense Pest Control recommends an eighteen step process for complete nit removal that requires a metal nit comb, an 8-10X hand lens, and/or a fluorescent light to distinguish viable unhatched from dead or hatched nits, a deep bowl of hot detergent water, a box of tissue paper, a strong goose-neck lamp, many bobby pins or clips, a large clean towel, two comfortable seats, a washing machine to machine wash all washable clothing and bed linens which have been in contact with the infested child in the past three days, and a vacuum cleaner for all non-washable clothing, rugs, furniture, mattresses and other personal items.

Whomever is responsible for nit removal must also possess the visual acuity to identify a 0.8 x 0.3 mm nit, a steady hand, considerable patience, a sense of responsibility, concern and commitment for the child's welfare, a desire for the child to return to school as quickly as possible, a bathroom or sink for a sustained period of time, access to the child's contacts and the energy and desire to carry out the painstaking task of nit picking.

The National Pediculosis Association, an advocate for the no-nits policy, recognizes that "this process takes time and patience on the part of both parents and children," and that it "can sometimes be difficult." Nevertheless, they state that "nit removal is not an unrealistic goal, especially when parents are educated." Further, according to the NPA, the no-nits policy "returns lice control to the parents, who rightfully own it in the first place."

While these statements arguably apply to most American middle class children, their relevance must be questioned when it comes to the millions of children living in poverty. Can nit removal be a realistic goal when the child's caregiver is preoccupied with concerns for food and shelter and the struggle to survive, and has no time or energy for nit picking?

Prospects for successful nit removal are also slim for children whose caregivers attach little importance to education and assign a low priority to time consuming, monotonous activities required to get the child back to school; or have drugs and alcohol as the only focus to their lives; or are young, dependent and immature, and preoccupied with meeting their own needs; or are elderly and lack visual acuity, hand dexterity and physical stamina; or have several other louse infested children to treat at the same time; or are without access to a sink or must share a bathroom with several other families; or have low self-esteem, see themselves and their children as inferior, anticipate failure at every turn and therefore see little reason to take on a task providing no immediate gratification, that in all likelihood is going to fail; or are single parents sharing responsibility for the child and unable to control events when the child is under the control of the other parent; or are temporary, interim guardians having no ability to follow through with any treatment plans.

At best, the task of complete nit removal is easier said than done. For many disadvantaged children, it is impossible. What is more, total nit removal as an instrument in epidemic control has never been substantiated by published peer-reviewed research derived data. Additionally, many advocates for the poor, homeless and near-homeless see the no-nits policy as punitive, and finally, the recent availability of ovicidal agents having 99-100% effectiveness reduces nit removal to cosmetic importance only.

The scant research on nit removal has focused on developing better systems of nit removal. A recent study reported that, after five minutes of combing the "control" two inch square section of hair, 72.4% of nits still remained.[1] The authors did not speculate on the length of time required to remove all the nits from all the scalp, but conservatively estimated that it would "certainly exceed 5 minutes." Most state that, depending on the length of hair and the degree of infestation, between one and three hours of continuous nit picking are required. Significantly, there is no body of research that documents whether or not any of this effort makes any difference.

The no-nits policy is most costly to those who can least afford it. Advantaged children with head lice seldom

[1] De Felice, J., et al. *Int'l Journ. Derm.* 28:7, p. 468-70.

(see *No-Nits*, page 8)

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PRINCIPLES AND PRACTICE OF INFECTIOUS DISEASES

1990

THIRD EDITION

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TABLE 2. Selected Diseases Transmitted by Arthropods

Infectious Disease	Vector
A…s (including yellow fever, dengue fever, …ephalitis)	Mosquitoes and ticks
Babesiosis	Hard ticks
Brugia malayi (*Wuchereria bancrofti*)	Mosquitoes
Endemic relapsing fever (*Borrelia duttoni*)	Soft ticks
Epidemic relapsing fever (*Borrelia recurrentis*)	Human body lice
Epidemic typhus (*Rickettsia prowazekii*)	Human body lice
Filariasis	Mosquitoes
Leishmaniasis (*Leishmania* spp.)	Phlebotomid flies
Lyme disease (*Borrelia burgdorferi*)	Hard ticks
Malaria (*Plasmodium* spp.)	Mosquitoes
Murine typhus (*Rickettsia mooseri*)	Rat fleas, lice
Onchocerciasis (*Onchocerea volvulus*)	Black flies
Plague (*Yersinia pestis*)	Rat fleas
Q fever (*Coxiella burnetii*)	Hard ticks
Rickettsial pox (*Rickettsia akari*)	Mouse mites
Rocky Mountain spotted fever (*Rickettsia rickettsii*)	Hard ticks
Scrub typhus (*Rickettsia tsutsugamushi*)	Mites
Trench fever (*Rickettsia quintana*)	Human body lice
Trypanosomiasis (African)	Tse tse flies
Trypanosomiasis (American; *Trypanosoma cruzi*)	Triatomid drugs
Tularemia (*Francisella tularensis*)	Flies, hard ticks

tion. Methods used to control arthropod vectors include the use of insecticides or environmental manipulations that would limit breeding and spread of the undesirable arthropod species. Unfortunately, the widespread use of insecticides may induce the development of resistance in the arthropods, thereby requiring the development of new and different toxic agents. Extensive use of insecticides may create anxiety and controversy because of the possible hazardous effects they may have on the env…ment.

…ses spread by arthropods may be limited not only by controlling the arthropod vector but also by preventing access of the arthropod to its host. When possible, fine screening should be used on windows and doors to prevent entrance of flying arthropods into the dwelling. Insect repellents that are applied to the skin or clothes are also an effective means for protecting humans from arthropods. The active ingredient in most of the insect repellents available in the United States contain either *N,N*-diethyl-*m*-toluamide (deet) or ethyl hexanediol.

There is a great need to more clearly define the factors that are responsible for attracting ectoparasites to the skin of humans, and important work is underway in this area. When we understand these basic epidemiologic facts more precisely, better insect repellents can be produced, which would greatly improve the ability of humans to prevent parasitism. As with other organisms, cycles of increased pathogenicity occur among parasitic organisms, as attested to by epidemics of scabies. Better knowledge of the mechanisms whereby this occurs may also help in controlling these disorders.

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269. LICE (PEDICULOSIS)

BARBARA BRAUNSTEIN WILSON
PEYTON E. WEARY

THE ORGANISMS

The order Anoplura contains more than 200 species, of which only members of the family Pediculidae are parasitic for humans. The species of importance are *Pediculus humanus* var. *corporis*, the human body louse, *Pediculus humanus* var. *capitis*, the human head louse, and *Phthirus pubis*, the pubic or crab louse. (For the complete classification see Table 1, Chapter 266.)

Eggs laid by the fertilized adult female are firmly glued to body hairs or fibers of clothing and appear as small globoid or oval protrusions called nits (Fig. 1). Approximately 7–10 days after deposition small voracious nymphs emerge that must feed within 24 hours to survive. After 2–3 weeks and three successive molts, the mature adults mate. The fertilized females produce 250–300 eggs over the next 20–30 days before death.

The body louse and the head louse are virtually identical—small (2–4 mm), grayish white, flattened, wingless, and elongated parasites with pointed heads. From each segment of the fused triple-segmented thorax a pair of jointed legs protrude that end in clawlike projections (Fig. 2). The pubic louse is distinctively different in shape, being much wider and shorter than its cousins are and resembling a crab, whence its nickname.

FIG. 1. Nits (ova) of *Pediculus humanus* var. *capitis* attached to scalp hairs.

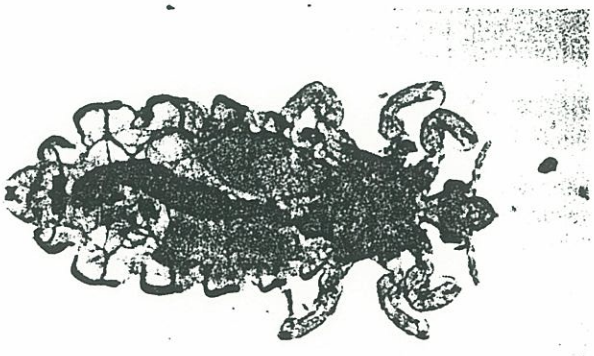

FIG. 2. *Pediculus humanus* var. *capitis*.

MIOLOGY

ce infestations have been observed in virtually every inhabited area of the world. At times of war, overcrowding, or widespread inattention to personal hygiene, major epidemics have occ[illegible]d.

PEDICULOSIS CAPITIS

Persons from all social and economic backgrounds can become infested with head lice, and infestations can reach epidemic proportions, especially among schoolchildren. The disease is uncommon in blacks and is seen most frequently in young females, presumably because of their longer hair styles. Lice are transferred by close personal contact and the sharing of hats, combs, and brushes.

PEDICULOSIS CORPORIS

Pediculosis corporis (body lice) is seen primarily where there is overcrowding and poor sanitation. The body louse lays its eggs and resides in the seams of the clothing rather than on the skin of its host. The body louse leaves the clothing only to obtain a blood meal from its host. Nits present in the clothing are viable for up to 1 month. Besides causing significant cutaneous disease, the human body louse is a vector for epidemic typhus, trench fever, and louse-borne relapsing fever.

PHTHIRUS PUBIS

Phthirus pubis (pubic lice) infestation is transmitted by sexual or close body contact and less often by the sharing of personal clothing or bedding. The pubic louse resides primarily in the p[illegible] hair but can also be seen in the eyebrows, eyelashes, a[illegible]y hair, and coarse hair on the back and chest of males. The pubic louse has also been found to infest scalp hair (unpublished case seen by author Barbara B. Wilson, M.D.).

CLINICAL MANIFESTATIONS

Pediculosis Capitis

Adult head lice and nits localize primarily in the temporal and occipital areas of the scalp; however, the entire scalp as well as the beard area may be involved. The adult lice may be difficult to observe, but the nits that are attached to the base of the hairshaft are easily seen. The major complaint of persons afflicted with head lice is pruritus of the scalp. Scratching leads to excoriations and secondary bacterial infection that is manifested by weeping and crusting of the scalp as well as tender occipital and cervical adenopathy. A pruritic, symmetric, morbilliform eruption may develop on the body, especially on the upper part of the trunk and arms. This eruption is felt to be an "id" reaction, which is hypothesized to be a hypersensitivity reaction to the inflammation and infection in the scalp.

Pediculosis Corporis

Except in cases of very severe infestations, the adult louse is not usually seen on the skin but rather in the seams of the clothing. Patients complain of pruritus and develop small erythematous macules, papules, and excoriations that are located pri[illegible]y on the trunk. Secondary impetiginization as well as an [illegible] reaction may occur. Persons with long-standing untreated pediculosis corporis may develop generalized hyperpigmentation and thickening of the skin with evidence of numerous healed excoriations, an entity known as "vagabonds' disease."

Phthirus Pubis

The primary complaint of perons infested with *Phthirus pubis* is marked pruritus of all affected areas, which may include axillary and coarse truncal hairs and eyelashes as well as pubic hair. One may or may not see erythematous macules and papules with excoriations and secondary infection, and if it occurs, it is usually less severe than that seen in pediculosis capitis and corporis. The nits and occasionally the adult pubic lice can be seen attached to the base of the hairs.

Small gray to bluish macules measuring less than 1 cm in diameter may be seen on the trunk, thighs, and upper parts of the arms. These lesions, known as "maculae cerulae," are felt to be caused by an anticoagulant that is injected into the skin by the biting louse. Infestation of the eyelashes by pubic lice can cause crusting of the lid margins. In such cases, the nits are readily seen at the base of the lashes.

THERAPY

Symptomatic treatment of pruritus in all three types of infestation consists of adequate doses of antihistamines such as hydroxyzine, 25 to 50 mg three to four times daily. Medium- to high-potency topical corticosteroids such as triamcinolone or fluocinolone cream should be applied to affected areas two to three times daily. When secondary bacterial infection is present, *Staphylococcus aureus* is a frequent cause, and patients should be treated with a systemic antibiotic such as erythromycin or dicloxacillin, 250 mg four times a day for 10 days.

Pediculosis Capitis

There are several different pediculicides that can be used to effectively treat head lice. These include 1% lindane or gamma benzene hexachloride shampoo (Kwell), pyrethrin liquid with piperonyl butoxide (RID, A-200 pyrinate liquid), 1% permethrin creme rinse (Nix) and 0.5% malathion lotion (Prioderm).

A recent study has shown that, when compared with lindane and the pyrethrins, malathion lotion was most effective and was the only product that showed excellent ovicidal activity.[1] Objections to malathion include an unpleasant odor and a treatment time of 8–10 hours as compared with the 10-minute therapy required by the other agents. Unfortunately, malathion is no longer available in the United States.

All of the other available pediculicides are probably comparable in efficacy. They are all cosmetically acceptable and easy to use, each requiring only a 10-minute application to the scalp. This treatment is repeated in 1 week.

Lindane is the only pediculicide that requires a prescription, but it offers no advantage over the other agents. There have been toxic side effects reported with lindane including seizures and even death; however, toxicity is not a problem when treating pediculosis because of the short periods of skin contact required and therefore minimal systemic absorption of the agent.

Nits should be removed from the hairs by applying a solution of equal parts of vinegar and water and then combing the hair with a fine-toothed comb that has been dipped in vinegar. Combs and brushes should be soaked in the pediculicide for 1 hour. Articles of clothing that have been in contact with an infested individual within the previous 3 days should be laundered in the hot cycle of the washing machine or dry cleaned.

Pediculosis Corporis

Body lice can be eradicated by either discarding the clothing, when practical, or by laundering clothes in the hot cycle and then carefully ironing the seams of clothing. Body lice can also be eliminated by dusting the clothing with either 1% malathion powder or 10% DDT powder.

irus Pubis

For pediculosis capitis, *Phthirus pubis* may be treated with lindane, permethrin, pyrethrin, or malathion as described above. The pediculicides should be applied to all affected areas except the eyelids. Eyelid infestation can be effectively treated by applying a thick layer of petrolatum to the eyelid margins twice a day for 8 days. Clothes and bedding of an infested individual should be laundered as described for pediculosis capitis.

PREVENTION

The spread of pediculosis capitis can be minimized by improving living conditions and personal hygiene and by avoiding the sharing of hats, combs, and hair brushes. Classroom epidemics are best prevented by frequent examination of the scalps of the students. Body lice are rarely seen in those with good personal hygiene who change their clothes frequently. Pubic lice are best prevented by avoiding sexual or close body contact with infested individuals.

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270. SCABIES

BARBARA BRAUNSTEIN WILSON
PEYTON E. WEARY

Scabies is a disease of great antiquity and possibly the cause for the "7-year itch" known to humanity for centuries. Napoleon's troops during the Russian campaign were thought to have had rampant scabies. Periodic epidemics of scabies are thought to occur at approximately 30-year intervals and persist for approximately 15 years.[1]

THE ORGANISMS

The organisms that cause scabies belong to the order Acarina, the family Sarcoptidae, and the genus *Sarcoptes*. Human scabies is caused by the itch mite *Sarcoptes scabiei* var. *hominis*. The scabies mite is one of the cutaneous parasites that burrow into the skin of its host, and therefore, some experts prefer to call it an "endoparasite" rather than an "ectoparasite."

Two to three eggs are laid daily by the fertilized female in burrows several millimeters in length created at the base of the stratum corneum of the epidermis. After 72–84 hours larvae emerge and, after several moults, become adult mites and mate after about 17 days. The males die shortly, but the gravid females proceed to burrow and complete the life cycle.

The full-grown adult female is only about 0.35 mm in length, rounded, with three pairs of short stubby legs. The organisms may be demonstrated in the burrows by an application of mineral oil to the skin overlying a burrow followed by vigorous scraping to the point where minimal bleeding occurs (Fig. 1). Under the

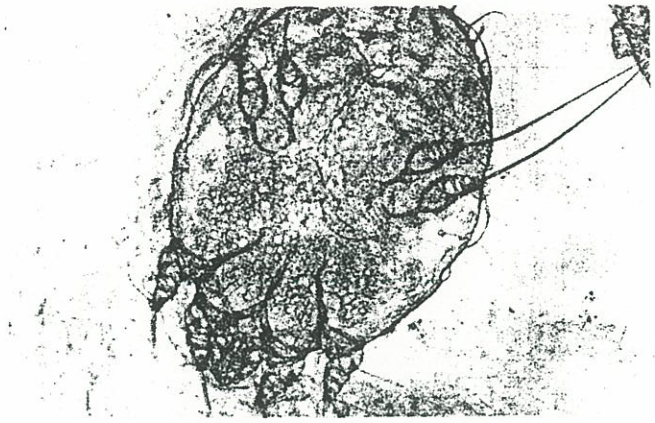

FIG. 1. Organism of scabies in a wet mount preparation.

mites, mite feces, or eggs. It is often difficult to demonstrate the organisms in older lesions or in nodular lesions.

EPIDEMIOLOGY

The disorder is worldwide in distribution, but the actual prevalence is unknown. There has been a substantial increase in the incidence of scabies in the United States since 1973. Epidemics have been associated with both world wars, so conditions of poverty, poor hygiene, overcrowding, malnutrition, and sexual promiscuity probably are contributory factors. The cyclic pattern may reflect the development of immunologic resistance of certain population groups at risk. Although the scabies mite can cause significant cutaneous disease, it is not a vector for infectious diseases.

Scabies is transmitted by intimate personal contact, often sexual in nature, but casual contact, including that of nursing attendants, may be adequate for transmission, and institutional epidemics can occur. The clinical picture of scabies is usually fairly characteristic but is extremely variable depending on the degree and duration of the infestation. Fastidious individuals who wash frequently may have fewer and more subtle lesions, while those who neglect themselves are more likely to have extensive cutaneous disease.

CLINICAL MANIFESTATIONS

Human Scabies

Most individuals infested with the scabies mite complain of intense itching that is usually more severe at night. Erythematous papules and excoriations are noted in areas of predilection such as the interdigital web spaces, wrists, elbows, anterior axillary folds, periumbilical skin, pelvic girdle, buttocks, penis, knees, and sides of the feet. In infants and small children the palms, soles, face, and scalp may be affected. One should look carefully for classic linear burrows, particularly in the interdigital spaces and on the penis (Fig. 2). Assistance in demonstrating the burrows can be provided by the application of blue or black ink to the skin overlying a suspected burrow. By capillary action the ink will be pulled into the burrow. Removal of the excess external ink with an alcohol swab will leave the ink in the burrow, thus demonstrating its presence. It is the burrow containing the organism and its eggs and feces that will yield a positive scraping. At times, scabies in infants may be vesicular or even bullous, and secondary pyoderma may obscure the underlying disorder. A background eczematous eruption may be present and is probably related to the development of hypersensitivity by the host to the scabies mite. Treatment with topical or systemic corticosteroids may alter the clinical picture such that the disease remains unrecognized, an entity known as "scabies in-

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Do Not Take From This Room

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controlled. An alternative drug is crotamiton (Eurax) cream or lotion, which may be applied in the same way as lindane (gamma benzene hexachloride). The old-fashioned medication consisting of 5% or 10% sulfur in petrolatum may still be used, applying it nightly from the collarbones down, for 3 nights, but one must be prepared to treat irritant dermatitis. Benzyl benzoate may be compounded as a lotion or emulsion in strengths from 20% to 35% and used as generalized (from collarbones down) applications overnight for 2 treatments 1 week apart. The NF XIV formula is 275 mL benzyl benzoate (containing 5 g of triethanolamine and 20 g of oleic acid) in water to make 1000 mL. It is cosmetically acceptable, clean, and not overly irritating. Persistent pruritic postscabietic papules may be painted with undiluted crude coal tar or Estargel.

Unless treatment is aimed at all infected persons in a family or institutionalized group, reinfestations will probably occur.

Resistant forms requiring multiple forms of treatment are appearing.

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PEDICULOSIS

Essentials of Diagnosis

- Pruritus with excoriation.
- Nits on hair shafts; lice on skin or clothes.
- Occasionally, sky-blue macules (maculae ceruleae) on the inner thighs or lower abdomen in pubic louse infestation.

General Considerations

Pediculosis is a parasitic infestation of the skin of the scalp, trunk, or pubic areas. It usually occurs among people who live in overcrowded dwellings with inadequate hygiene facilities, although pubic lice may be acquired by anyone sitting on an infested toilet seat—and, more commonly, by sexual transmission. There are 3 different varieties: (1) pediculosis pubis, caused by *Pthirus pubis* (pubic louse, "crabs"); (2) pediculosis corporis, by *Pediculus humanus* var *corporis* (body louse); and (3) pediculosis capitis, by *Pediculus humanus* var *capitis* (head louse).

Head and body lice are similar in appearance and are 3–4 mm long. Head louse infestations may be transmitted by shared use of hats or combs. The body louse can seldom be found on the body, because the insect comes onto the skin only to feed and must be looked for in the seams of the underclothing. Trench fever, relapsing fever, and typhus may be transmitted by the body louse, but this would be an extremely rare event in the USA.

Clinical Findings

Itching may be very intense in body louse infestations, and scratching may result in deep excoriations over the affected area. The clinical appearance is of gross excoriation. Pyoderma may be present and may be the presenting sign in any of these infestations. Head lice can be found on the scalp or may be manifested as small nits resembling pussy-willow buds on the scalp hairs close to the skin. They are easiest to see above the ears and at the nape of the neck. Body lice may deposit visible nits on the vellus hair of the body. Pubic louse infestations are occasionally generalized, particularly in a hairy individual; the lice may even be found on the eyelashes and in the scalp.

Differential Diagnosis

Distinguish head louse infestation from seborrheic dermatitis, body louse infestation from scabies, and pubic louse infestation from anogenital pruritus and eczema.

Treatment

For all types of pediculosis, lindane lotion (Kwell, Scabene) is used extensively. A thin layer is applied to the infested and adjacent hairy areas. It is removed after 12 hours by thorough washing. Remaining nits may be removed with a fine-toothed comb or forceps. Sexual contacts should be treated. Permethrin (Nix), 1% cream rinse, is a topical pediculocide and ovicide for the treatment of head lice and eggs. It is applied to the scalp and hair and left on for 10 minutes before being rinsed off with water. Synergized pyrethrins (A-200 Pyrinate, Pyrinyl, Rid) are over-the-counter products that are applied undiluted until the infested areas are entirely wet. After 10 minutes, the areas are washed thoroughly with warm water and soap and then dried. Nits may be treated as indicated above. For involvement of eyelashes, petrolatum is applied thickly twice daily for 8 days, and remaining nits are then plucked off. There is controversy about whether lice and the acarus of scabies can develop resistance to lindane.

Malathion lotion, 0.5% (Prioderm), compared with A-200 Pyrinate shampoo, R&C shampoo, Rid, Kwell shampoo (lindane), and A-200 Pyrinate liquid, is the only product for pediculosis capitis that shows excellent ovicidal activity. Hatching of eggs following treatment with the other agents leads to recurrence of the infestation.

Prognosis

Pediculosis responds to topical treatment.

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SKIN LESIONS DUE ARTHROPODS

Essentials of Diagn

- Localized rash with
- Furunclelike lesion
- Tender erythematou migrans").
- Generalized urticari

General Considerat

Some arthropods (eg ing flies) are readily others are not, eg, beca there is no immediate during sleep. Reactio hours; many severe r are most apt to consul are multiple and prur may be accompanied b and faintness or even c cover the body.

Many persons will r contacts with an arth lesions when travelin etc. Body lice, fleas, should be borne in mi believed to be the so humans, although the *Loxosceles reclusa*) n tions and death due t the black widow spi cause severe systemi

In addition to arth lesions are venomou ants, scorpions) or bi to vesicating furuncl or sand fleas in the sk due to a migrating la

Clinical Findings

The diagnosis ma has not noticed the ir reaction. Individual and tend to occur eit and gnats) or under waist or at flexures bedding or clothing) for 1–24 hours or r present and may be a starts to scratch. Sec serious consequence gic manifestations, common. Papules m sis is aided by searc and by considering t activities. The prin

HEAD LICE INFESTATION

BIOLOGY DIAGNOSIS MANAGEMENT

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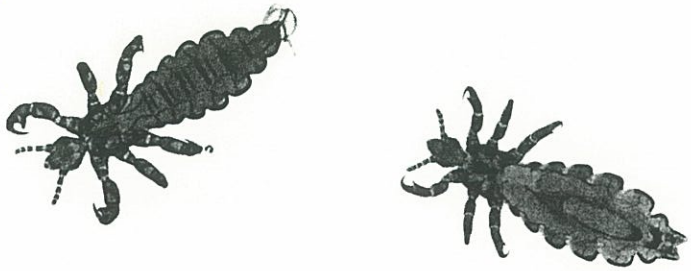

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The Head Louse
Management
Prevention
Self-Assessment Test

Pediculosis Capitis:
Description, Epidemiology, Transmission

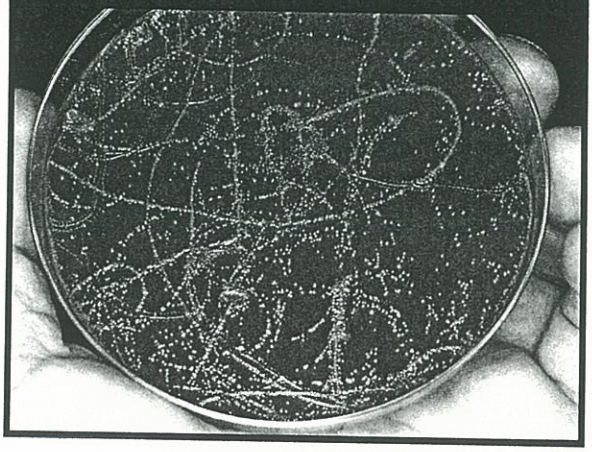
Description

The feeding bite of the louse is painless; the anticoagulant saliva, however, causes an allergic reaction with itching at the bite site. Initial infestation may produce no signs or symptoms, since the individual may not become chemically "allergic" to the bites for several weeks or until multiple exposures. Therefore, severe itching or skin irritation usually indicates an infestation that has already been present several weeks. Irritation leads to repeated scratching and inflammation. Excoriations also create an entryway for germs and lice feces, often resulting in secondary infections such as impetigo (pyoderma) and swollen glands in the neck. With a large number of lice bites, the infested individual may be feverish and feel tired and irritable.

Disease

Although the body louse is notorious as a carrier of typhus, research has not shown the head louse to be a vector of serious disease. Some health professionals feel, however, that the head louse has not been studied long or closely enough to rule out its potential for transmitting disease. Its actions would make it a potential carrier, since it characteristically leaves the sick or dead to find a healthy host, depositing its fecal matter containing blood from the sick individual onto the scalp of the healthy one. With the current concern about transmissable diseases carried in the blood, head lice are being considered as potentially more dangerous than they have been in the past. The louse has also been shown to carry pathogenic bacteria on its feet that could easily be transferred from host to host (Fig 10).

Fig 10—Bacteria growing on agar plate deposited when a louse collected from a human subject walked across the plate. Reproduced with permission.



Epidemiology

Essentially everyone with hair on his or her scalp is a possible victim of head lice. There are, however, groups more susceptible than others.

School-aged children are the most susceptible to louse infestation, particularly those aged 3 to 10.[7] In one study, peak incidence for boys was age 8 to 9, for girls 6 to 7.[10] Infestation rates decrease with age in both sexes.[12] In a study conducted by the Centers for Disease Control in 1973-74 among kindergarten-to-eighth-grade schoolchildren in New York, Florida and Georgia,[13] 10% of the girls and 7% of the boys examined had pediculosis.

Girls are generally more susceptible than boys, and women are more susceptible than men. The reason for both the age and sexual differentiation is believed to be related to the amount of direct physical contact and contact with objects or surfaces to which the louse can cling, e.g. brushes, combs, clothing and upholstery. Young children exhibit more physical contact and sharing of such objects than older children—girls more than boys—and women have more physical contact both with young children and among themselves than men do with women, children or other men.

Race has been shown to be a factor in infestation in only one instance—head lice are so uncommon in the black popula-

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Scabies, Lice, and Fungal Infections

David Taplin, and Terri Lynn Meinking†*

SCABIES

History and Description of Causative Agent

Scabies is a highly pruritic infestation of the skin caused by the human itch mite, *Sarcoptes scabiei* (Fig. 1). The clinical disease has been known for over 2,500 years,[1] but the mite was not described until 1687 when Giovan Cosimo Bonomo wrote his famous letter to the naturalist Francisco Redi.[1] The mite, which is about 400 μm in diameter, or just barely visible to the naked eye, is pearly white, with brownish legs and mouth parts, and is seen to be actively mobile when removed from the host. Unlike pediculosis capitis, no evidence exists for the presence of scabies in the Americas prior to the arrival of Columbus. There is ample evidence that the disease was rampant among the early settlers of Jamestown, Plymouth, and Philadelphia.[8]

Epidemiology

"The *Acarus scabiei* is notorious for its lack of respect for person, age, sex or race, whether it be in the epidermis of an emperor or a slave, a centurian or a nursling, it makes itself perfectly at home with undiscriminating impudence and equal obnoxiousness."[8]

Although scabies is more common in persons subjected to crowded living and sleeping conditions, including refugees, victims of natural disasters, and poverty, primary care providers should recognize that this infestation occurs in all races worldwide, and is not limited to the impoverished or those with poor hygiene.

In our observations of many thousands of cases in several countries, we cannot equate the incidence with levels of personal hygiene, nor even with the availability of running water. In 1986 we found the prevalence of scabies in a village in Central America to be 74 per cent of all children under 2 years of age, in spite of scrupulous attention to personal hygiene and washing of clothes.

Similarly, in 1987 we found a prevalence of 47 per cent among elderly residents in a Florida nursing home where patients were frequently bathed, and environmental cleanliness was very good. The most important factor con-

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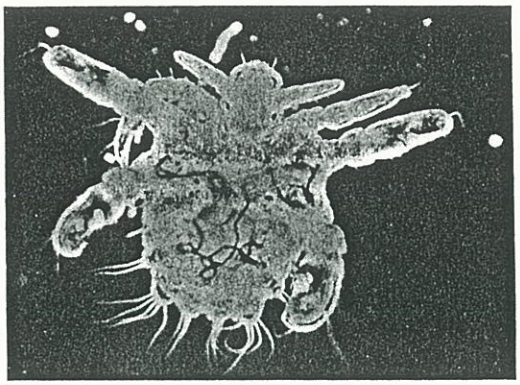

Figure 5. *Pthirus pubis*, the crab louse. Dark field. Note intestinal tract.

conditions of overcrowding, filth, and famine are capable of transmitting diseases which have changed the course of history.[24] These include typhus, trench fever, and murine typhus transmitted by the rickettsiae, *Rickettsia prowazekii*, *Rochaliamea* (*Rickettsia*) *quintana*, and *Rickettsia typhi* (*mooseri*), respectively. The fourth disease is relapsing fever which can be transmitted by body lice infected by the spirochete *Borrelia recurrentis*. The mechanism of transmission of these diseases is through infected louse feces, which enter the circulation of the victim through excoriations of the skin, including those associated with the pruritic louse bites (see Fig. 4). Early researchers believed that the source of infection was directly from the louse bite, and ignorance of the role of louse feces probably led to the deaths of such pioneers in the field as Bacot, Rickets, and Prowazek.

It has not been firmly established whether or not head lice and "crabs" can be vectors of blood-borne diseases, although this is due, in part, to the lack of research dedicated to this area.

Relapsing fever predominately by crushing of gut.

?

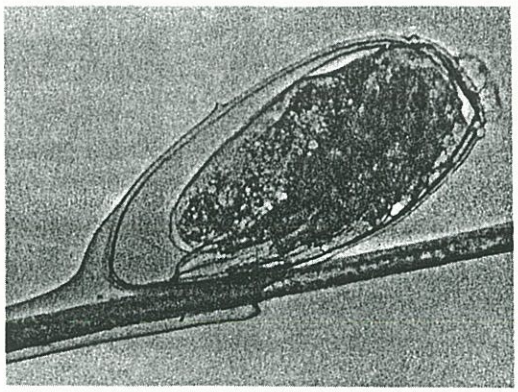

Figure 6. Egg of *Pediculus capitis*. Note developing embryo and cap or operculum at distal end.

Chapter in 1989 text "Common Dermatologic Problems"

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CLINICAL & DIAGNOSIS MANAGEMENT BY LABORATORY METHODS

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The itching results from a sensitization reaction to the parasites and their products and varies in severity from patient to patient. There may be itching in areas in which there are no mites. Lesions often become secondarily infected (Orkin, 1975). The diagnosis is established by dissecting organisms or eggs from the tunnels, placing them in 20 per cent potassium hydroxide or mineral oil for clearing, and examining them under the microscope. Eggs, six-legged larvae, eight-legged nymphs, or adults may be detected and are diagnostic. Unfortunately, these cannot be readily demonstrated in all patients.

When scabies has been detected in a place, such as a school, there are often numerous individuals who develop itching without evidence of disease, probably of psychological origin. Care must be taken to properly diagnose the disease to prevent such pseudoepidemics. A particularly severe form of the disease may occur, especially in institutionalized patients, and is called "Norwegian scabies."

Trombiculid mites infest grasses and bushes, and their six-legged larvae, chiggers (red bugs, harvest mites), may attack people. The larvae attach to the skin, usually in areas of tight clothing such as elastic bands or belts. In sensitive individuals there is reaction to the secretions of the larvae with swollen itching areas at the sites of attachment which persist for days. Excoriations may become secondarily infected. Diagnosis is established on clinical grounds. Other trombiculid mites may be vectors of scrub typhus caused by *Rickettsia tsutsugamushi*.

Ticks. Ticks belong to the superfamily Ixodoidea and are important as vectors in infectious diseases, but in addition may cause local damage from bites and may cause tick paralysis. There are two groups of ticks. Soft ticks belonging to the family Argasidae (Fig. 46–48) have a soft leathery body, and mouth parts are not visible from above. Hard ticks belonging to the family Ixodidae have a hard dorsal plate, and mouth parts are visible from above (Fig. 46–49). The dorsal plate covers the entire dorsum of the male, but only the anterior portion of the female, allowing the body to swell when engorged. Unengorged ticks are generally 3 to 4 mm long, but engorged ticks may be up to 1.5 cm long. The stages in development are egg, larva, nymph, and adult. Blood meals are essential for the development of ticks. Infestation is acquired in grassy or bushy areas where the ticks reside between blood meals on various mammals.

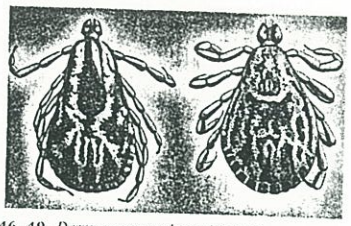
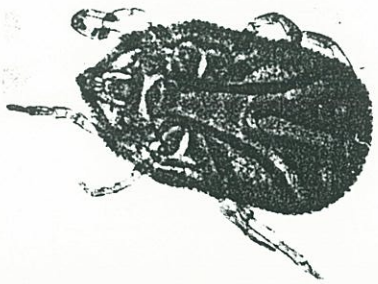


Figure 46–49. *Dermacentor andersoni* and *Dermacentor variabilis*: vectors of Rocky Mountain spotted fever rickettsiae. (Courtesy of Merck, Sharp & Dohme, Inc.)

Ticks may be vectors of a number of infectious diseases including Lyme disease or babesiosis *(Ixodes)* and Rocky Mountain spotted fever *(Dermacentor)*.

Tick paralysis is an ascending paralysis that develops in occasional patients, especially children, bitten by various ticks and is due to a toxic substance introduced by the tick. This disease may be confused with Guillain-Barré syndrome, poliomyelitis, botulism, and other paralytic diseases. Removal of ticks results in recovery.



Class Insecta

Members of the class Insecta are characterized by a body divided into head, thorax, and abdomen and having three pairs of legs. There are usually two pairs of wings. Insects of medical importance include lice, fleas, bugs, mosquitoes, and flies. Bees and wasps may cause severe reactions, particularly in sensitive individuals, but will not be further described.

Lice. Lice are flattened dorsoventrally and are wingless. There are three lice that infest humans and obtain nourishment by biting and sucking on the human host (Kim, 1986). They are named according to the region of the body that they usually (but not exclusively) inhabit. *Pediculus humanus capitis* is the head louse and is 1 to 2 mm long. *P. humanus corporis* is the body louse and is 2 to 4 mm long, and *Phthirus pubis* is the pubic or crab louse and is approximately 1 mm long (Fig. 46–50). The body louse is particularly important as the vector of epidemic typhus. Infestations with lice usually occur when people live in crowded conditions with little opportunity for bathing and laundering. Infestation may be spread by intimate contact, as in bed partners, or by contaminated hats, clothing, blankets, or furniture. Eggs of *P. humanus capitis* and *P. pubis* are attached to hairs and are known as "nits" (Fig. 46–51). Eggs of *P. humanus corporis* are attached to clothing. Diagnosis is suspected from finding bites but estab-

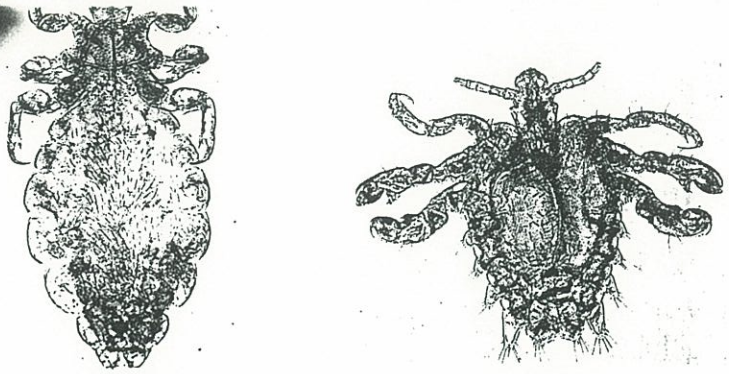


Figure 46–50. *Pediculus humanus* (*left*) and *Phthirus pubis* (*right*).

detecting "nits" attached to hairs *(P. humanus capitis* and *P. pubis)* or eggs in clothing *(P. humanus corporis)*. "Nits" should be examined microscopically to be certain they are truly eggs and not globs of hair spray or some other material. *P. humanus* may serve as a vector of typhus or trench fever. Outbreaks of infestation with head lice or pubic lice are common in the United States.

Fleas. Fleas are bloodsucking wingless insects that are laterally compressed and have large hind legs for jumping. They average 2 to 4 mm long and have bloodsucking mouth parts. The oriental rat flea *(Xenopsylla cheopis)* is the vector of plague, and fleas may be associated with other infections. Flea bites cause little trouble to some persons but are quite irritating to others, probably as a result of sensitization of the host. Humans may be infested by the human flea *(Pulex irritans)* (Fig. 46–52) or may be incidental hosts for fleas of other animals, particularly the dog flea *(Ctenocephalides canis)* and the cat flea *(C. felis)*. Eggs develop in dog and cat bedding and in carpets and furniture. They usually cause little difficulty for people unless the pet is no longer present, for then they will bite humans. This usually occurs after moving, boarding, or death of pets.

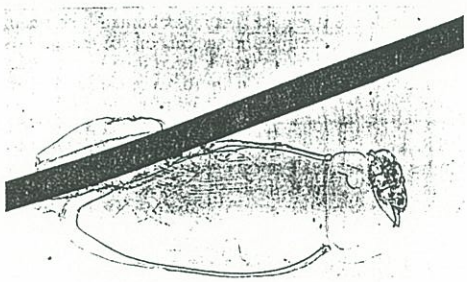


Figure 46–51. *Pediculus capitis*. Empty egg case (nit) attached to hair (× 60). (From Raphael, S. S.: Lynch's Medical Laboratory Technology. Philadelphia, W. B. Saunders Company, 1976).

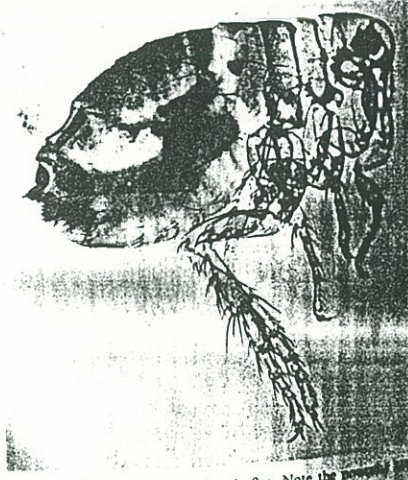


Figure 46–52. *Pulex irritans* female flea. Note the [illegible] legs.

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Rudolph's Pediatrics 19th edition

1991

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...harges to form a black eschar, at which time fever is ...bserved. Regional lymphadenopathy is a consistent ...ding, but the diagnosis is not usually suspected until the ...haracteristic rash appears 1 to 5 days after onset of fever. ...he rash begins as diffuse nonpruritic macules, progressing ... maculopapules and finally papulovesicles, resembling ...hicken pox. The palms, soles, and mucous membranes are ...ccasionally involved, but distribution of lesions is quite ...ariable. Fever, chills, and headache persist for about 5 days, ...nd rarely more than 10 days. Upper respiratory and gastro...ntestinal symptoms are common. Untreated, recovery is ...ill universal, but appropriate antibiotics may shorten dura...on of symptoms in more severe illness.

...iagnosis

...eil-Felix reactions are negative, so serologic confirmation ...quires specific complement-fixation or immunofluorescent ...*akari* antibody determination.

...fferential Diagnosis. Other etiologies producing febrile ...nesses accompanied by a papular or vesicular skin rash in...ude varicella-zoster, enteroviruses, Epstein-Barr virus, and ...anotti-Crosti syndrome. The presence of an eschar is very ...lpful for correctly identifying this rickettsiosis. .

...evention

...oper control or avoidance of rodent populations is the ob...ous method for eliminating disease in humans.

...IDEMIC (LOUSE-BORNE) TYPHUS

...storically, *R. prowazekii* has been a significant pathogen, ...using massive epidemics of disease during periods of war ...d famine and accounting for thousands of deaths in prison ...mps during and after World War II. More recently there ...ve been outbreaks in central Africa that could have been ...evented by simple public health measures. Major out...eaks have not occurred in the United States since 1893, but ...ly recently organisms have been identified in flying squir...s (*Glaucomys volans*) in the southeastern United States, ...ggesting a potential source for future outbreaks.

...idemiology and Transmission

...ease is transmitted to humans by the body louse, itself in...ted by feeding on another sick human or rodent animal ...ervoir. The rickettsial organism colonizes the louse gas...intestinal tract. Infected feces from the attached louse ...y be rubbed into a skin abrasion, mucous membranes, ...s, or aspirated into the respiratory tract of the host. Pa...nts who have recovered from epidemic typhus may retain ... organism for extended periods of time, relapsing years ...r with milder illness termed Brill-Zinsser disease. These ...ividuals represent an important reservoir for *R. pro...ekii* in interepidemic periods.

...lical Features

...nary epidemic typhus has an acute onset beginning 7 to ...ays after exposure to an infected louse. High fever (39 to ...C) and headache precede by 3 to 7 days a rash, which has ...ntral distribution, spreading to the extremities but usu...sparing the palms and soles. This rash progresses from ...aching macules to papules, petechiae, and occasionally ...ymoses. It is similar to the rash of RMSF but with a ...e central distribution. Elderly and previously debilitated ...alnourished patients may progress to severe sensorial ...ges, renal failure, and cardiovascular collapse. In such ...promised hosts, disease is frequently fatal.

...gnosis

...Weil-Felix reaction is very useful, particularly *Proteus* ...9, which is positive in 90% of patients during the second week of illness. Numerous tests are available for confirmation including the older complement-fixation and immunofluorescent assays and newer enzyme-linked immunosorbent assay (ELISA) and latex agglutination test.

Differential Diagnosis. Similar to RMSF, other diagnostic considerations include meningococcemia, enteroviruses, Epstein-Barr virus, leptospirosis, and measles.

Prevention

A formalin-killed vaccine is available for individuals who might travel to endemic regions or for more routine administration during an outbreak. This vaccine is more important in reducing the severity of subsequent disease than in offering absolute protection.

BRILL-ZINSSER DISEASE

This is the term applied to relapse of *R. prowazekii* disease, which occurs years after the primary attack. Organisms have apparently persisted in the reticuloendothelial system, reactivated during periods of physiologic stress in the host. Because these individuals possess rickettsial antibody and other specific immune reactivity, infection is greatly attenuated. The anamnestic antibody response to *R. prowazekii* is rapid, but the Weil-Felix reaction is almost always negative.

Rickettsial Diseases Not Endemic in the United States

MEDITERRANEAN SPOTTED FEVER

Disease caused by *R. conorii* has many different common names, primarily determined by geographic location (Table 14–59). However, there have been reported differences in clinical findings between regions, probably attributable to strain variations for this rickettsial pathogen. For example, the eschar at the site of tick attachment is seldom seen in Israel, where the rickettsiosis is called Israeli spotted fever. Fatalities are rare, even in locations observing more severe disease, and in general it is most convenient to remember Mediterranean spotted fever (MSF) as a mild form of RMSF but with an eschar at the site of a tick bite.

Epidemiology and Transmission

The reservoir and predominant vector of human disease is the dog tick, *Rhipicephalus sanguineus* (similar to *Ehrlichia canis*) prevalent in Mediterranean countries. In Europe most infections are reported from the south of France, Italy, and Spain. Recently there has been an increase of infection, particularly in Spain. As with other rickettsial infections, animal reservoirs play a major role in perpetuating endemicity; 80% of dogs in France and 86% in Sicily are antibody-positive for MSF. Fewer than 20% of reported human patients are children, as contrasted with RMSF, with which the majority of patients are younger than 20 years of age. More than 95% of infections are seen between April and October, reflecting activity of ticks and outdoor exposure of European residents during the summer months.

Clinical Features

Incubation ranges from 6 to 10 days. Classical clinical signs and symptoms occurring in virtually all patients include fever, a diffuse maculopapular rash, eschar at the site of a tick bite, headache, and myalgia. Other signs are hepato-

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Tropical and Geographical Medicine

SECOND EDITION

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TROPICAL AND GEOGRAPHICAL MEDICINE

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TREATMENT

In severely ill patients with RMSF or the typhus group, supp[o]rtive therapeutic regimens are based on the correction of oli[g]uria, hypotension, hypochloremia, hyponatremia, hypoalbuminemia, azotemia, edema, and coma. Careful use of serum albumin given intravenously will help support the circulation. Although it seems tempting to give intravenous fluids freely, such should be avoided in the presence of oliguria, edema, hypotension, and other circulatory changes. Dialysis may benefit some patients with renal shutdown.

Proper mouth care is needed. Frequent turning of the patient will help prevent aspiration pneumonia and pressure necroses of involved skin areas. When azotemia is minimal, nutritional support should include 2 to 3 g protein/kg. In confused or comatose patients without ileus or abdominal distension, tube feedings are helpful.

Chloramphenicol and the tetracyclines are specifically effective for the rickettsioses. These antibiotics, in spite of being only rickettsiostatic, lead to abatement of toxic and other clinical signs in 24 to 36 h and defervescence in 2 to 3 days. In scrub typhus, the response is more dramatic, whereas in Q fever clinical improvement may be less rapid. When therapy is delayed until the stage of diffuse hemorrhagic lesions with ecchymoses and skin necroses, the response is less dramatic but recovery will occur with proper management.

Generally the dosages of antibiotics are: chloramphenicol—an initial dose of 50 mg per kilogram of body weight and subsequent daily doses calculated on the same basis with treatment intervals of 6 to 8 h. For the tetracyclines, the same sc[he]dule is followed based on 25 mg per kilogram of body w[eight]. Antibiotic treatment is given until improvement is obvious and the patient has been afebrile for about 1 day (defervescence usually occurs in moderately ill patients in 2 to 3 days). Intravenous doses of either antibiotic are given when the patient is too ill to take oral medications [52–55]. As soon as possible, the oral route should be used. Under special conditions, a single large oral dose of chloramphenicol has been effective in scrub typhus and RMSF. Under primitive conditions, a single oral dose of 200 mg doxycycline was effective in epidemic typhus fever [56].

Critically ill patients may be helped by large doses of corticosteroids given for several days in conjunction with specific antibiotics and supportive care. They are not recommended or necessary for routine use in mild or moderately ill patients. Heparin has not been effective in correcting DIC phenomena.

POPULATION

ROCKY MOUNTAIN SPOTTED FEVER

Humans are accidentally infected when ticks transmit *R. rickettsii* either through tick attachment or through contamination of conjunctivas or abrasions of skin contaminated by infected tick feces or tissue juices. Four species of ixodid ticks are natural carriers of *R. rickettsii*—*Dermacentor andersoni* (Rocky Mountain wood tick), *Dermacentor variabilis* (the American dog tick), *Amblyomma americanum* (lone-star tick), and *Haemaphysalis leporus-palustris* (rabbit tick). *D. andersoni* and *D. variabilis* are the major vectors in the western and eastern United States, respectively. *Amblyomma americanum* infestation occurs in the south central and southeastern United States [57]. The rabbit tick rarely bites humans but it transmits spotted fever to other rabbits and helps maintain rickettsiae in human-infesting species of ticks. In Brazil and Colombia, the vector tick is *Amblyomma caijenneuse*.

The tick is a natural vector and reservoir since it survives the infection and transmits the agent readily to its offspring. Ticks remain infected for life. Infected small animals infect other ticks which feed on them.

Early in the season, ticks must usually remain attached for several hours before they cause infection. During warm months, the time of feeding and transmission is shorter. This phenomenon is called *reactivation* and represents a change from a nonvirulent resting phase (after overwintering and hibernation) to a virulent phase brought about by the ingestion of fresh blood, which changes the microbes' metabolism. Spotted fever occurs in the late spring and summer months because of hibernation of ticks during winter. Rickettsialpox which is antigenically related to RMSF occurs sporadically in the eastern United States and the U.S.S.R.

EPIDEMIC TYPHUS FEVER

Epidemic typhus is transmitted to humans by the body louse, which accounts for its epidemic tendencies given conditions of poverty and low health standards. Poor sanitary conditions favor lousiness during cold months. Persons of all ages are susceptible. Usually the illness is milder in children; higher mortality rates occur in adults and the elderly.

Lice, when they feed, drop infected feces onto the skin; infection occurs through minute abrasions or through contamination of conjunctivas and mucous membranes. *R. prowazekii* is quite resistant when dried and can be infectious for months. The human body louse dies of its infection; infected females do not transmit the agent to their offspring. Usually, outbreaks of typhus cease during warm months because of better bathing practices and decreased louse infestation.

Recently, flying squirrels in many eastern states have been implicated as infected hosts of *R. prowazekii* [58]; several dozen cases have been reported in humans who were in contact with such squirrels and their ectoparasites. The entire cycle of squirrel-vector-human infestation has not been fully elucidated but these findings presumably establish that humans are not the sole reservoir of *R. prowazekii* as originally surmised [59,60]. Person-to-person transmission through contact with saliva, sputum, urine, or human feces has not been shown.

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ments of Europe, Africa, Asia, and Latin and South America. Although it is largely controlled and of sporadic incidence, louseborne typhus remains an important cause of morbidity and mortality when socioeconomic and environmental conditions allow proper interplay between the microbe, vectors, and susceptible populations. The principal focuses are the highlands of central Africa, including Ethiopia, Burundi, Rwanda, southern Uganda, Nigeria, and Algeria; and Bolivia, Ecuador, and Peru in South America.

MURINE TYPHUS FEVER

The rat, *Rattus rattus* and *Rattus norvegicus*, is the natural reservoir of *Rickettsia mooseri;* mice are also susceptible. Infection in rodents is nonfatal and viable rickettsiae may persist in their brains for variable periods. The rat flea, *Xenopsylla cheopis*, is infected by feeding on rats during the acute stage of their infection. Once infected, fleas discharge pathogenic rickettsiae in their feces throughout their normal life cycle. Dried flea feces may infect humans via the conjunctivas or respiratory tract. *Pulex irritans* and human body lice are susceptible to experimentally induced murine typhus infection. Guinea pigs are readily susceptible to intraperitoneal inoculation of infected tissues or blood from patients during the active stages of illness. Humans are at risk when their activities bring them in contact with infected rats and their fleas near granaries and food depots or in congested, infested areas such as harbors.

Murine typhus occurs commonly in port cities and in densely crowded populations where rats and fleas are prevalent. Epidemic louseborne typhus fever, a clinically much more severe first cousin of murine typhus, has ravaged human populations during wars, famine, and poverty, or during periods of privation.

SCRUB TYPHUS FEVER

Scrub typhus infection is well established in nature as "typhus islands" in cycles involving mites and small rodents. Presumably there is transovarial transmission of the agent in mites, although with initial infection at the larval stages, this has not been conclusively elucidated. Humans invade these infected habitats, which may vary from semidesert, disturbed rain forests to river banks, seashores, and terrain undergoing secondary vegetative growth. The secondary hosts are rats, field mice, shrews, and voles. Larvae of several species of mites, especially *Leptotrombidium* (*Trombicula*) *akamushi* and *Leptotrombidium diliensis*, infect themselves by attaching to the skin of wild rodents and ultimately to humans as an accidental host [60]. Multiple serotypes of *R. tsutsugamushi* cause illness and produce effective homologous immunity but transient cross-protection. This unfortunate fact has precluded development of an effective vaccine.

Scrub typhus fever (tsutsugamushi disease) occurs in Japan, China, Australia, the Philippines, the South Pacific islands, Malaya, Burma, Thailand, Indonesia, and in the Asian subcontinent.

Q FEVER

Humans contract Q fever by inhaling infected dusts, by handling infected animal tissues, or by drinking milk contaminated with *C. burnetii*. The rickettsiae are widely distributed in nature in ticks, small wild animals, cattle, sheep, goat herds, and humans. Infection is transmitted in nature by ticks which may infect cattle. The animal-tick-animal cycle is a basic mechanism for perpetuation of this rickettsia in nature. Cattle hides contaminated with tick feces have caused aerosol infection in humans.

Species of ticks naturally infected are *D. andersoni* and *A. americanum*. Sheep and goats are naturally infected; *C. burnetii* has been recovered from the milk of these animals. Infected cows, sheep, and goats excrete *C. burnetii* during parturition since placental and birth fluids are grossly infected. Sheep herders, veterinarians, and others who are exposed to such infected aerosols are at risk, as are stockyard workers, wool processors, and laboratory investigators who are engaged in study of the agent. There is no evidence of direct transmission from person to person.

Q fever is globally distributed with the highest incidence of reported cases in Australia, New Zealand, England, Italy and other Mediterranean countries, the United States, and South Africa.

OTHER TICKBORNE RICKETTSIOSES

The agents which cause these mild illnesses are called by various names depending on their geographical localization—*Rickettsia conorii* (fièvre boutonneuse), *Rickettsia siberica* (North Asian tickborne rickettsioses), and *Rickettsia australis* (Queensland tick typhus). Other types are South African tick typhus, Kenya tick typhus, and Indian tick typhus.

R. conorii is the prototype for the group. Ixodid ticks, small wild animals, and dogs maintain the rickettsiae in nature. Humans accidentally invade this cycle and contribute nothing to its continuity. *Rhipicephalus sanguineus*, the brown dog tick, is the dominant vector in the Medterranean countries and South Africa. In Thailand, Malaysia, Europe, Israel, India, and Pakistan, there is serological evidence of spotted fever group rickettsiae.

PREVENTION AND CONTROL

Attempts to develop effective vaccines for the major rickettsioses have been partially successful. Epidemic typhus vaccines consisting of inactivated *R. prowazekii* prepared by the chick embryo technique protected humans against mortality and severe illness. Unfortunately, such vaccines are not generally available because of diminished demand and technical difficulties in preparation. A viable attenuated strain of *R.*

Volume II

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TEXTBOOK OF Pediatric Infectious Diseases

Second Edition

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may also be seen on the palms, soles, and mucous membranes. The number of lesions ranges from 5 or 6 to over 100. The characteristic papulovesicles distributed so haphazardly on the body make the rickettsialpox rash very similar in appearance to chickenpox rash in an adult.

Diagnosis

The diagnosis can be made serologically with either complement-fixation or immunofluorescent tests, using either RMSF or rickettsialpox antigens. Weil-Felix tests are useless since no *Proteus* agglutinins are produced. The major differential diagnostic problem is adult chickenpox. Infectious mononucleosis, gonococcemia, and infection with echovirus (types 9 and 16), coxsackievirus A (types 9 and 16), or coxsackievirus B (type 5) also should be considered.[58, 67, 68] Patients often give a history of having worked in basements or around incinerators or in similar areas that might be infested by house mice and their mites.

Treatment

Deaths have not been reported. Tetracycline is the drug of choice; chloramphenicol is an acceptable alternative.[57, 62] In infants and young children with mild illness, antibiotics may be withheld since the disease is self-limited.

TYPHUS GROUP

Three diseases—louse-borne typhus, Brill-Zinsser disease, and murine flea-borne typhus—make up the typhus group. Clinically and pathologically, these three illnesses are similar; epidemiologically they are very different, hence will be described under separate headings.

Primary Louse-borne Typhus Fever

Primary louse-borne typhus fever is an acute infectious disease transmitted to humans by the body louse. Louse-borne typhus has played a major role in the history of nations over the past five centuries. It has undoubtedly been more decisive than military campaigns, as Zinsser[80] has described convincingly in his book, *Rats, Lice and History*.

Typhus fever only occurs in the presence of the lice, which multiply to astronomic numbers during periods of war, famine, and social upheaval. During the last century epidemics occurred in Europe, Asia, Africa, and sporadically in the United States: the last recorded American epidemic occurred in Philadelphia in 1893. Following World War I, over 30 million people in eastern Europe were infected with typhus fever and an estimated 3 million died. During World War II, louse-borne typhus again infected millions in prison camps, the eastern European combat zone, and North Africa. In the 1970s, tens of thousands of louse-borne typhus cases occurred in uncontrolled epidemics in Burundi and Ruanda in central Africa.[82]

Since 1976, there have been at least 30 cases of disease due to *R. prowazekii* documented in the United States. These have occurred sporadically. The presumed source of infection is the flying squirrel (*Glaucomys volans*).[74, 77, 81]

The Organism

The etiologic agent is *Rickettsia prowazekii*. Its morphology, growth, metabolism, toxin production, and staining characteristics are similar to those described for the rickettsiae of the spotted fever group. Antigenically, the organisms of louse-borne and flea-borne (murine) typhus form a separate group, although they show some minor antigenic cross-over with the spotted fever group.

Epidemiology and Transmission

It has been assumed that the causative agent of epidemic typhus existed only in the human-louse-human cycle, and that patients who had recovered from typhus constituted the reservoir of *R. prowazekii* in interepidemic periods. If this were this case, eradication of the epidemic typhus would theoretically be possible, since few patients with Brill-Zinsser disease would be alive after long interepidemic periods. The findings of sporadic *R. prowazekii* infection, however, suggest that perpetuation of epidemic typhus is possible because it may persist in an animal reservoir.

The chain of typhus infection starts when *R. prowazekii* appears in a patient's blood during the acute febrile infection. A louse becomes infected during one of its frequent blood meals. After five to ten days of incubation in the louse, large numbers of rickettsiae appear in the louse feces. Transmission of rickettsiae from an infected louse to a new host can occur by several mechanisms. Since a louse defecates as it feeds, infected feces can be rubbed into the louse-bite wound. Additionally, dried louse feces also can gain access to the mucous membranes of the eye or respiratory tract. The epidemic spread of typhus throughout a community relates to temperature preferences of the louse. Lice prefer blood meals on people with a normal temperature, hence they tend to leave febrile patients (as well as the dead); crowding during wars and famine makes transfer to new hosts easy.

Pathology

It is similar to that described for the spotted fever group of diseases.

Clinical Manifestations

From one to two weeks after the bite of an infected louse, illness usually begins abruptly. The major clinical signs and symptoms are fever, headache, and a rash. Temperature usually rises rapidly to 40° C (104° F) or higher. In untreated patients it remains at this level with minor fluctuations until death or recovery ensues. The rash usually appears on the trunk by the fourth to seventh day to spread peripherally to the extremities,

...ally sparing the face, palms, and soles. At first the ...ash consists of macules that fade on pressure; they soon become fixed as maculopapules and later become petechial or hemorrhagic. A severe, intractable head-...e is characteristic. Severe untreated cases progress ...rostration, stupor, or delirium with terminal myocardial and renal failure. Complications, which are uncommon, include gangrene, parotitis, and otitis media.

Diagnosis

The various factors concerned with the diagnosis of louse-borne typhus are analogous to those discussed for the diagnosis of RMSF, with a few differences. The rash of louse-borne typhus begins centrally on the trunk and spreads peripherally to the extremities while the reverse is true for RMSF. Moreover, a rash on the palms and soles, common in RMSF, is rare in louse-borne typhus. Differentially, typhus usually occurs in epidemics under conditions of crowding and high louse populations.

Serologically, the Weil-Felix reaction is almost always positive with the *Proteus* OX19 strain and less commonly with the OX2 strain. Complement-fixation and immunofluorescent tests are technically the same as with the spotted fevers; however, with louse-borne typhus, *R. prowazekii* strains will be used as antigens. As noted under the spotted fever group, antigenic crossing between any of the members of the typhus and spotted fever groups of organisms occurs frequently.

Recently an enzyme-linked immunosorbent assay (ELISA) and latex agglutination test were evaluated and found to be sensitive and reproducible.[79, 80] Owing to the antigenic crossover between the typhus group of ...ttsiae, work is being done to isolate the species-...ific protein antigen of *R. prowazekii* for both immunodiagnosis and immunoprophylaxis.[76]

Therapy

This is analogous to that of the spotted fever group.

Prognosis

Case fatality rates in untreated cases correlate with age. Mortality, uncommon in children, is 10 per cent in young adults and may run as high as 60 to 70 per cent in those over 50 years of age. Recovery from an attack gives rise to an enduring immunity. (For exceptions, see Brill-Zinsser disease.)

Prevention

Two highly effective measures, vaccination and louse control, are available for controlling typhus epidemics. Potent killed vaccines produced from yolk sacs grown in chick embryos have proved highly effective in preventing mortality; these vaccines do not, however, regularly prevent infection. DDT and the newer insecticides lindane and malathion have proved highly effective in reducing louse infestation during typhus epidemics. The insecticides dusted into the clothes of louse-infested populations are effective in ridding the community of ... and curtailing louse-borne typhus epidemics.

Brill-Zinsser Disease[78]

Brill-Zinsser disease is a relapse or recrudescence of louse-borne typhus that occurs years after the primary attack. This relapsing form of typhus in many ways is analogous to a relapse of malaria. After a primary attack, the typhus rickettsiae remain dormant somewhere in the body, probably most commonly in cells of the reticuloendothelial system. Years later they are reactivated by stress or some unknown factor to multiply and cause a second acute infection. Because of partial immunity remaining from the primary typhus attack, the recrudescent infection is almost always a milder, shorter, and less debilitating illness. The causative agent is the same as for primary louse-borne typhus; the symptoms, signs, and pathology are similar to those described under louse-borne typhus.

Tetracycline is the drug of choice. A single dose of doxycycline may lead to prompt resolution of clinical symptoms in selected cases.[84]

Murine Typhus[85]

Murine typhus is a disease of rats passed from rat to rat by the rat flea and only occasionally and accidentally transmitted to humans by an infected rat flea bite. The disease is worldwide and occurs primarily along coastal areas and around granaries where rats abound. During the first half of this century, it was highly prevalent along the Atlantic seaboard and Gulf coastal areas.

The Organism

The causative organism, *R. mooseri* (classified as *R. typhi* in *Bergey's Manual*) is similar to *R. prowazekii* in metabolism, growth, toxin production, and staining characteristics though it is slightly smaller and more uniform in size. Because they possess a large common antigenic moiety, *R. mooseri* and *R. prowazekii* are classed together in one group.

Epidemiology and Transmission[83]

Murine typhus usually is acquired by humans in the following manner: the rat flea, *Xenopsylla cheopis*, becomes infected when feeding on an acutely ill rat. The rickettsiae multiply in the flea without causing any ill effects, but the feces of the infected flea will teem with rickettsiae for the rest of the flea's life. Rat fleas prefer to feed on rats but will feed on people if rats are not available. When an infected flea sucks blood, its dejecta is teeming with rickettsiae. If the flea bites a person, the infected feces may be rubbed into the bite wound or be transferred in a dried aerosol to the conjunctivae or respiratory tract. Humans obviously are not related to the maintenance of *R. mooseri* in nature. In California, sporadic cases have been related to transmission by fleas of *R. mooseri* from opossums to humans.[73]

In the early 1940s, 2000 to 5000 cases of murine typhus were reported annually in the United States. Most occurred in the southeastern and Gulf coast states. Currently murine typhus is not reported in most states, and only 60 to 80 cases are reported annually. Approximately 80 per cent of the cases are reported from Texas.[75]

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Carrier State. There are two groups of carrier state patients: (a) the convalescent carrier (patients that continue eliminating *S. typhi* for weeks or months) with self-limited condition and do not need treatment; and (b) chronic carriers (patients who continue eliminating *S. typhi* after 1 year). There are two approaches to chronic carriage of *S. typhi:* (1) prolonged antibiotic therapy, usually with ampicillin 4 to 6 grams per day plus 2 grams per day of probenecid, both divided in four oral doses for 6 weeks; carriage is eliminated in about 95% of patients without gallbladder disease; and (2) surgical removal of the gallbladder, cholecystectomy, is the treatment of choice in patients with gallbladder disease (gallstones and gallbladder dysfunction).

If drug therapy alone fails, antibiotic treatment in combination with cholecystectomy usually results in a good cure rate.

Ofloxacin concentrates in the gallbladder walls and bile acids, while cefoperazone concentrates mainly in bile acids; these properties make both drugs useful in the treatment of chronic carriers. Clinical studies proving their effectiveness in the carrier state have not been performed.

PREVENTION

Control of typhoid fever depends on high standards of environmental sanitary measures, maintenance of a supply of uncontaminated water, and a good control of chronic carriers. In epidemic areas vaccination is necessary. Some trials with parenteral typhoid vaccines showed that an acetone-inactivated *S. typhi* vaccine provided 79 to 93% protection; phenol-inactivated preparations were not as effective in providing immunity. Among subjects vaccinated with parenteral inactivated typhoid vaccine, 25% developed severe systemic and local reactions.

Two large-scale trials have been performed with live oral vaccine (*S. typhi* Ty 21a) in endemic areas: the first of these was performed in Egypt, with three doses containing 1 to 8 $\times$ 10^9 viable organisms at 2-day intervals; the rate of vaccine efficacy was estimated to be 95% in a 3-year follow-up. The second trial was performed in Chile; the vaccine provided 67% protective efficacy for up to 3 years. In both trials, minimal adverse reactions were observed.

Second-generation typhoid vaccines have been prepared by modern genetic techniques. Strains 541Ty and 543Ty have been tested in humans. When doses as high as 2 $\times$ 10^{10} organism were given orally, no detectable adverse clinical reactions were observed. Significant serum and intestinal antibody responses occurred in only a small number of volunteers. At this moment, there is not an ideal vaccine for prevention of typhoid fever.

TYPHUS FEVERS

method of
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Two species of *Rickettsia* are responsible for the diseases of the typhus fever group. These are *Rickettsia prowazekii,* the cause of epidemic (louse-borne) typhus and recrudescent typhus (Brill-Zinsser disease), and *Rickettsia typhi,* the cause of murine (endemic) typhus. The three diseases present similarly but differ in severity and in epidemiology.

Certain other rickettsial diseases are sometimes known as a form of typhus. Scrub typhus, caused by *Rickettsia tsutsugamushi,* differs clinically and epidemiologically from the typhus fevers. The spotted fever group of diseases, sometimes called tick typhus, results from infection by different rickettsial species in different parts of the world. The most severe form, Rocky Mountain spotted fever, which is caused by *Rickettsia rickettsii,* is found in the United States.

Although they are not called typhus and are quite different clinically, two other rickettsial infections are considered here because they respond to the same therapy: Q fever, caused by *Coxiella burnetii,* and rickettsial pox, caused by *Rickettsia akari.*

The *Rickettsia* species are widely distributed in nature, are transmitted primarily by arthropod vectors, and have various mammalian reservoirs. Except for epidemic typhus and Brill-Zinsser disease, human infection is incidental to the perpetuation of the organisms in nature. The global distribution of the rickettsial diseases depends on the ecology of the arthropod vectors and the mammalian reservoirs. Epidemic typhus, for example, is transmitted primarily from human to human by lice. Conditions such as war and disaster promote such transmission and can lead to major outbreaks of disease.

The common rickettsial diseases in the United States are Rocky Mountain spotted fever, endemic typhus, and Q fever. Epidemic typhus, Brill-Zinsser disease, and rickettsial pox occur only rarely. Rocky Mountain spotted fever, transmitted by ticks, occurs throughout the country, but most often in the south Atlantic and Gulf states. The reservoir is ticks and a variety of wild mammals. The peak period of the disease is in the spring and summer when ticks are most active. The incidence is highest in rural and suburban areas where humans are exposed to ticks. Endemic typhus is an infection of rats and other rodents that is transmitted to humans by fleas. Rickettsialpox is an infection of the house mouse and other rodents that is transmitted to humans by mites. Cases of both diseases occur when there is interaction of the requisite species. Q fever is an infection of small mammals, cattle, sheep, and goats that is transmitted both by ticks and by inhalation of dried infected material from mammals with the disease.

In humans the rickettsiae may multiply locally at

the site of inoculation and, in some instances, produce a local lesion. The organisms spread throughout the body and multiply within and injure the endothelial cells of the small blood vessels. The injury and inflammation lead to leakage and extravasation of blood elements into the tissues and also lead to thrombosis with resultant damage to tissues. The specific manifestations of each disease relate to the location of the dominant organ involved and the consequences of the angiitis.

The key features of the diseases are an acute, febrile illness that develops after an incubation period of a few days to 3 weeks, a rash (except for Q fever), and the consequences of the angiitis such as decreased effective circulating blood volume, azotemia, electrolyte imbalance, delirium, stupor, coma, myocarditis, hepatitis, a consumptive coagulopathy, and shock. The clinical manifestations and epidemiologic setting are the basis of initial diagnosis. Specific serologic tests provide confirmation. The nonspecific Weil-Felix reaction may help during the second week of illness. Rocky Mountain spotted fever is the most severe rickettsial disease. It may be fulminant and fatal before the results of serologic tests can be obtained. Antirickettsial therapy must not be delayed in cases of clinically suspected Rocky Mountain spotted fever until a diagnosis is made.

THERAPY

Prompt antirickettsial therapy is the principal concern in the treatment of rickettsial diseases. Patients who are cooperative and mildly ill can be treated as outpatients. Those who may be unreliable or who are moderately or severely ill should be hospitalized to ensure proper antimicrobial treatment and to attend to complications that tend to occur in the second week of untreated disease.

Supportive care is vital for the severely ill patient and must be directed to the complications, for example, azotemia, hypovolemia, and hypoproteinemia. Corticosteroids in large doses for up to 3 days may be tried in severely toxic patients.

General therapeutic measures include provision of a nutritious diet, protection of the agitated patient from injury, and skin and mouth care. The severe headache that is common in these diseases is most often intractable and not eased by the usual drugs.

Specific Therapy

Both tetracycline (or an equivalent congener) and chloramphenicol are highly effective in the treatment of the typhus group of infections and the other rickettsial diseases. Other common antibiotics are ineffective. Chloramphenicol is preferred in cases in which typhoid fever is included among the possible causes of illness and in children younger than the age of 8 years. Clinical response begins quickly. Fever remits and dramatic overall improvement typically occurs in the first 2 days of antimicrobial therapy. In Rocky Mountain spotted fever, the vascular lesions may be so extensive that permanent tissue damage occurs despite effective antibiotic treatment.

Patients who can tolerate oral medication should receive one of the following drugs:

1. Tetracycline
   a. Initial dose: 25 mg per kg
   b. Daily dose: 25 mg per kg per day in four equal doses every 6 hours
2. Chloramphenicol
   a. Initial dose: 50 mg per kg
   b. Daily dose: 50 mg per kg per day in four equal doses every 6 hours

Intravenous preparations should be administered to patients who are unable to tolerate oral treatment. Warnings on package inserts should be observed. Oral therapy should replace intravenous administration as soon as possible. Adults and children should receive one of the following drugs:

1. Tetracycline
   a. Initial dose: 15 mg per kg infused in 30 to 45 minutes
   b. Daily dose: 25 mg per kg per day infused in equal doses every 6 hours
2. Chloramphenicol sodium succinate
   a. Initial dose: 20 mg per kg infused in 30 to 45 minutes
   b. Daily dose: 50 mg per kg per day infused in equal doses every 6 hours

Treatment should continue until the patient is improved and has been afebrile for 24 to 48 hours. Treatment with these drugs stops the proliferation of organisms and hence treats the disease. The antibiotics do not, however, eradicate the organisms. Eradication depends on the host's immune response, which is relatively slow to develop. A relapse may follow cessation of therapy, especially if the treatment was instituted early in the course of the disease and was short. Such a relapse responds quickly to a new course of the same antibiotic.

Doxycycline is a long-acting derivative of tetracycline. A single oral dose is effective in the treatment of typhus and is both convenient and safe. This is probably the treatment of choice for the typhus fever group, especially in situations such as refugee camps or disasters when medical service is limited. A single dose of doxycycline is not, however, reliable therapy for Rocky Mountain spotted fever. For this disease, doxycycline should be continued until the patient has im-

ed and been afebrile for 24 to 48 hours. ycycline should be given as follows:

Doxycycline
Single dose: 100 or 200 mg

PREVENTION

Prevention of rickettsial disease relies primarily on reducing human contact with the arthropod vectors, for example, delousing and using insect repellents, and on reducing the number of reservoir hosts and vectors. A commercial vaccine for epidemic typhus is available. It consists of killed rickettsiae prepared as a formalin-treated suspension of infected yolk sac. The vaccine prevents or ameliorates the disease.

Local health authorities should be informed of the occurrence of a case of rickettsial disease.

WHOOPING COUGH

(Pertussis)

method of
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rtussis is a prolonged infection strictly localized e ciliated respiratory epithelium. The only host is man and virtually 100% of contacts acquire the disease, the hallmark of which is a chronic severe cough usually lasting more than 6 weeks. Approximately 5000 cases per year are reported in the United States (despite vaccination), but this total probably represents only 10% of all disease. Half of these cases occur in children under 1 year of age, while 20% are in those over 15 years old. This bimodal age distribution is a reflection of immunization that protects infants after three doses (i.e., after 6 months of age), but is not offered to children over 6 years of age, leading to waning immunity (postimmune pertussis).

The clinical features of whooping cough differ markedly according to the age of the patient. Severe infections occur in infants who commonly progress through three stages of illness. A prodromal catarrhal illness, indistinguishable from the common cold, persists for up to 2 weeks. Stage 2 is marked by greatly enhanced mucous secretion and the onset of paroxysmal cough, often ended by a gasping, inspiratory whoop. Paroxysms may be associated with cyanosis, apnea, and attendant cerebral anoxia. Chronic cough and extreme leukocytosis (often in excess of 15,000 cells per mm^3 with more than 70% lymphocytes) are the diagnostic features of the infection and are recognizable only in Stage 2. However, the period of infectivity and positive nasopharyngeal cultures begins in the catarrhal stage and extends only briefly into the paroxysmal phase, making confirmation of the clinical diagnosis and interruption of the spread of infection difficult. The convalescent stage extends for 3 to 4 weeks. The appearance of fever and persistent respiratory distress at this stage is an ominous sign, suggesting secondary bacterial pneumonia, which complicates the 10% of fatal cases. No long-term effects on pulmonary function have been documented. In contrast to this course, adults experience the catarrhal phase followed by a chronic cough, simulating bronchitis. The inability to prevent or recognize and treat the disease in adults is a major contributor to the persistence of the infection in the population. Children with immunodeficiency disorders, such as human immunodeficiency virus (HIV) infection, experience a prolonged course of infection.

The infection is caused by the fastidious gram-negative bacterium *Bordetella pertussis*, which adheres specifically to human cilia. After colonization, the bacteria produce at least five toxins, which are responsible for the systemic manifestations of disease, such as lymphocytosis and hypoglycemia. Ciliated cells are completely denuded by the mid-paroxysmal phase, and the continued infection may result from bacteria surviving within alveolar macrophages. Diagnosis rests on culture of the bacterium on Bordet-Gengou agar, with colonies appearing only after 3 to 4 days. Ancillary tests include immunofluorescent staining of nasopharyngeal secretions or detection of antibody to pertussis toxin or other bacterial components by ELISA (Table 1). However, because cellular immunity plays a major role in this infection, there does not appear to be a good serologic correlate of infection or vaccine-induced protection.

TABLE 1. Approach to the Patient with Whooping Cough

Diagnosis	Sensitivity
Nasopharyngeal culture	50%
IFA of secretions	60%
ELISA titer of toxin IgG at 4 wk	80%
Treatment	
1. Erythromycin estolate (Ilosone) 50 mg/kg/day orally in 4 doses for 14 days (maximum, 2 gm) Alternative if intolerant: TMP/SMZ (Bactrim, Septra) 8 mg per kg and 40 mg/kg/day in 2 oral doses	
2. If secondary pneumonia supervenes: ceftriaxone (Rocephin) 75 mg/kg/day intravenously in 2 doses or nafcillin (Unipen) 200 mg/kg/day intravenously in 4 doses and ampicillin 200 mg/kg/day intravenously in 4 doses	
3. For life-threatening paroxysms: dexamethasone 1 mg/kg/day intravenously in 4 doses	
4. Prophylaxis of family and close contacts: Erythromycin as per No. 1	

TREATMENT

Antimicrobial Treatment

Three features of this disease combine to make antibiotic therapy ineffective in changing the

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running, the alarmed termite leaves a scent trail (similar to the foraging trail) of pheromone that communicates direction and serves to recruit workers and soldiers to the point of disturbance.

EVOLUTION AND PALEONTOLOGY

Termites are related to the roaches and probably have evolved from a primitive roachlike ancestor. The most primitive living roach, the subsocial, wood-eating *Cryptocercus punctulatus*, which lives in rotten logs, has affinities with the termites. *Cryptocercus* harbours symbiotic, cellulose-digesting protozoans of the same genera as those found in the hindgut of primitive termites. The genitalia and certain internal structures of *Cryptocercus* have basic anatomical resemblances to those of the most primitive living termite, *Mastotermes darwiniensis*, from Australia. *Mastotermes* has further affinities with other roaches: its hindwing has a folded anal lobe, and its eggs are not laid singly as those of other termites but in clusters held together by a gelatinous material resembling the egg case of roaches.

Evidence of the relationship to primitive roaches suggests that termites evolved in the Late Permian (approximately 230,000,000 years ago), although the known fossil termites date only from the Early Cretaceous (about 130,000,000 years ago). The termite society may be older than any other society; the ant society is 100,000,000 years old.

CLASSIFICATION

Termites of the order Isoptera are small to medium-sized insects that live in social groups, or colonies, and are characterized by their highly developed caste system. The mouthparts are modified for chewing. Antennae are moniliform (beadlike) or filiform (threadlike). Isopterans are very soft bodied insects, usually light in colour. Head structures and the presence or absence of individual caste members are used to distinguish termite families.

Termites, often called white ants, differ from hymenopterans (bees, ants, and wasps) in several ways. Termites have a hemimetabolous (gradual) metamorphosis and pass through a series of nymphal stages. Hymenopterans have the more common holometabolous metamorphosis, with distinct larval, pupal, and adult stages. Termite social castes (reproductives, sterile workers, and sterile soldiers) usually contain members of both sexes in equal numbers, and both males and females develop from fertilized eggs. In the hymenopteran colony, however, the sterile castes contain females only; both sterile and reproductive females develop from fertilized eggs, while reproductive males develop by parthenogenesis from unfertilized eggs. The thorax in termites is joined broadly to the abdomen, without the "waist" characteristic of bees, ants, and wasps. Termites have two pairs of membranous wings, nearly equal in size, that break along a suture when shed, leaving only the wing base, or "scale," attached to the thorax—probably the most distinguishing characteristic of isopterans.

Annotated classification.

ORDER ISOPTERA (termites)
Highly developed caste system, may contain reproductives, soldiers, and workers; reproductives shed wings after mating; distribution worldwide, mostly in tropical rain forests; about 1,900 living, 60 fossil species; may inhabit moist subterranean or hot, dry locations; foods include plant cellulose, often digested by symbiotic protozoans in termite hindgut; all families (except Termitidae) known collectively as "lower termites" contain symbiotic protozoans in hindgut.

Family Mastotermitidae
Primitive; 1 living species (*Mastotermes darwiniensis*) in Australia; 13 Tertiary fossil species worldwide.

Family Kalotermitidae (dry-wood termites)
Wood dwelling, wood eating; survive dry conditions; 292 living, 11 fossil species (some from Baltic amber).

Family Hodotermitidae
Thirty living, 13 fossil species (1, the earliest known termite fossil, from Lower Cretaceous, Labrador); includes rotten-wood termites and harvester termites that forage and store food in nests; *Zootermopsis*, largest termite in North America, found in Rocky Mountains at altitudes of 2,000 to 2,500 metres; *Archotermopsis*, found in Himalayas; *Hodotermes* species, serious pests of African grasslands.

Family Rhinotermitidae (subterranean termites)
Lives under damp conditions; 158 living, 13 fossil species; *Reticulitermes*, widely distributed in North America and other temperate and subtemperate regions and a serious pest; *Coptotermes*, a serious pest in tropic... and subtropical regions.

Family Serritermitidae
One living species in South America; specialized family evolved from Rhinotermitidae.

Family Termitidae (higher termites)
Largest termite family (about 75 percent of all termites), 1,413 living, 3 fossil species; 4 subfamilies variable in morphology, social organization, and nesting habits. (K.K.)

Phthiraptera (lice)

Lice (order Phthiraptera) are small, wingless, parasitic insects divisible into two main groups: the Mallophaga, or chewing or biting lice, which are parasites of birds and mammals, and the Anoplura, or sucking lice, parasites of mammals only. One of the biting lice, the human louse, is the carrier of typhus and louse-borne relapsing fever; it thrives in conditions of filth and overcrowding. Outbreaks of louse-borne diseases were frequent by-products of famine, war, and other disasters before the advent of powerful insecticides (see INFECTIOUS DISEASES). Heavy infestations of lice may cause intense skin irritation, and scratching for relief may lead to secondary infections. In domestic animals rubbing and damage to hides and wool may also occur, and meat and egg production may be affected; in badly infested birds the feathers may be severely damaged. One of the dog lice is the intermediate host of the dog tapeworm, and a rat louse is a transmitter of murine typhus among rats.

GENERAL FEATURES

The flattened bodies of lice range from 0.33 millimetre to 11 millimetres in length and are whitish, yellow, brown, or black. Probably all species of birds have chewing lice, and most mammals have either chewing or sucking lice, or both. There are about 2,900 known species of Mallophaga, with many others still undescribed, and about 400 species of Anoplura. No lice have been taken from the duckbilled platypus or from anteaters and armadillos; and none are known from bats or whales. The size of louse populations varies enormously on different individuals, sometimes seasonally. Sick animals and especially birds with damaged bills, probably because of the absence of grooming and preening, may have abnormally large numbers: over 14,000 on a sick fox and over 7,000 on a cormorant with a damaged bill; the numbers found on healthy hosts are usually considerably smaller. Apart from grooming and preening by the host, lice and their eggs may be controlled by predatory mites, dust baths, intense sunlight, and continuous wetting.

NATURAL HISTORY

Life cycle. With the exception of the human body louse, lice spend their entire life cycle, from egg to adult, on the host. The females are usually larger than the males and often outnumber them on any one host; in some species males are rarely found, and reproduction is by unfertilized eggs (parthenogenetic). The eggs are laid singly or in clumps, usually cemented to a feather or hair; the human body louse lays its eggs on clothing next to the skin. The eggs may be simple ovoid structures glistening white among the feathers or hairs or may be heavily sculptured or ornamented with projections that assist in the attachment of the egg or serve in gas exchange. When the nymph within the egg is ready to hatch, it sucks in air through its mouth; this passes down the alimentary canal and accumulates behind the nymph until sufficient pressure is built up to force off the cap (operculum), helped by an armed, platelike structure, the hatching organ, at the upper end of the prelarval skin. The emergent nymph is similar to the adult but issmaller and uncoloured, has fewer hairs, and differs in certain other morphological details.

Metamorphosis in the lice is simple, the nymphs molting three times, each of the three stages between molts (instars) becoming larger and more like the adult (see Figure 20).

tions of the eye, central nervous system, and skin. Serious infections leading to death may occur in immunocompromised persons.

Herpetic venereal diseases

HSV-2 is associated most often with herpetic lesions of the genital area (venereal disease). HSV-1, however, can be recovered from 15 percent of genital lesions. The frequency of asymptomatic venereal infection is not known. Recurrences are the rule, especially with HSV-2.

In herpetic venereal diseases, the lesions are small, red, painful spots that become filled with fluid and then rupture, leaving eroded areas that eventually become scabbed. These primary lesions occur from two to eight days after exposure and may be present for up to three weeks. Viral shedding and pain usually resolve in two weeks. When infections recur, the duration of the pain, lesions, and viral shedding is approximately 10 days. The involved area includes the vagina, cervix, vulva, and, occasionally, the urethra in females and the head of the penis in males. The disease is usually transmitted by sexual contact.

Diagnosis of herpetic venereal disease is important because newborn infants born vaginally to mothers with active HSV-2 infection are at increased risk for generalized infection, including that of the central nervous system. HSV-2 can cause death in 60 percent of infants so affected and severe mental retardation in 20 percent of the surviving infants.

Examination of the fluid from the lesions shows viral particles within the nuclei of tissue cells and the presence of multinucleated giant cells. Diagnosis can also be made by examination of the blood for a change in the serum antibody levels as evidence of active infection. The elevation of antibody levels is not as striking in recurrent infections as it is in primary infections.

Early detection and prevention of the spread of herpetic venereal disease is important. In patients with the primary herpetic infection, an antiviral agent called acyclovir may be effective in diminishing the duration of symptoms and the period of time during which the virus may be recovered from the lesions. It is only effective before the latency state is established, however. During latency, when the virus lives in tissues without causing symptoms, it is protected against destruction. Subsequent reactivation of the virus can occur in response to a multitude of stimuli.

CHLAMYDIA

Chlamydia are intracellular parasites found in many vertebrates, including birds and human beings and other mammals. The diseases they cause were first recognized in the early 20th century.

Clinical illnesses are caused by two species, *Chlamydia trachomatis* and *C. psittaci.* The former is a frequent cause of genital infections in women and, if an infant passes through an infected birth canal, it can produce disease of the eye and pneumonia in the newborn. Young children sometimes develop ear infections, laryngitis, and upper respiratory tract disease from *Chlamydia.* Such infections can be treated with erythromycin.

Chlamydia psittaci produces psittacosis, a disease in human beings that results from exposure to the discharges of infected birds. The illness is characterized by high fever with chills, a slow pulse rate, pneumonia, headache, weakness, fatigue, muscle pains, anorexia, nausea, and vomiting. The diagnosis is usually suspected if the patient has a history of exposure to birds and is confirmed by blood tests. Mortality is rare and specific antibiotic treatment is available. (R.D.F./Re.G.)

RICKETTSIAS

The rickettsias are a family of microorganisms named for a U.S. pathologist, Howard T. Ricketts, who died of typhus in 1910 while investigating the spread of the disease. The rickettsias cause a group of diseases in human beings characterized by fever and a rash, and all depend for survival on passing at some stage through the body of a louse, mite, tick, or flea.

The great epidemic form of the disease, louse-borne typhus, is caused by *Rickettsia prowazekii,* which differs from most other rickettsias, except for that which causes trench fever, in that human beings and lice are its only hosts. It needs no other environment than that which human beings, when their hygiene is poor, provide.

Human beings get the other diseases only when they break into a cycle in nature in which the rickettsias live. In murine typhus, for example, *Rickettsia mooseri* is a parasite of rats conveyed from rat to rat by the rat flea, *Xenopsylla cheopis;* it bites human beings if they intrude into its environment. Scrub typhus is caused by *R. tsutsugamushi,* but it normally parasitizes only rats and mice and other rodents, being carried from one to the other by a small mite, *Leptotrombidium* (previously known as *Trombicula*). This mite is fastidious in matters of temperature, humidity, and food and finds everything suitable in restricted areas, or "mite islands," in South Asia and the western Pacific. It rarely bites human beings in their normal environment, but if people invade its territory en masse it attacks, and outbreaks of scrub typhus follow.

Scrub typhus

The spotted fevers are caused by rickettsias that spend their normal life cycles in a variety of small animals, spreading from one to the other inside ticks; these bite human intruders and cause African, North Asian, and Queensland tick typhus, as well as Rocky Mountain spotted fever. One other spotted fever, rickettsialpox, is caused by *Rickettsia akari,* which lives in the body of the ordinary house mouse, *Mus musculus,* and spreads from one to another inside the house mite *Allodermanyssus sanguineus.* This rickettsia is probably a parasite of wild field mice, and it is perhaps only when cities push out into the countryside that house mice catch the infection.

Typhus. Epidemic typhus has also been called jail fever, war fever, and camp fever, names that suggest overcrowding, underwashing, and lowered standards of living. The body louse, which spreads the disease, has a powerful sucking mouth. As it sucks the blood of a typhus victim, rickettsias pass into the louse's gut, where they invade the intestinal cells. They multiply and the cells burst after a few days, releasing hordes of rickettsias into the louse's intestinal canal. These either reinfect other cells or are passed out in the louse's feces. Lice leave a body when it gets too hot from fever or too cold from death and crawl to another human host. The clothing of a heavily infested typhus patient is contaminated with louse feces, and careless removal of it may raise a cloud of infected dust in the air and, in this way, spread typhus to others, especially physicians and nurses.

About 10 days after being bitten, a person falls ill. Intense headache and fever are always present, and after a few days a rash begins. The sick person is flushed, and his eyes are bleary. If the disease is untreated, circulation becomes sluggish and there may be spots of gangrene on the fingers, genitals, nose, and ears. Signs of pneumonia or kidney failure are common. Soon the person becomes stuporous and then may lapse into coma and die. Much depends on age—the young tend to recover, the aged to die.

Tetracycline and chloramphenicol have a dramatic curative effect, however, and, if treated early enough, few die. The disease can be diagnosed clinically during epidemics and by laboratory tests.

The prevention of epidemic typhus requires the elimination of human body lice. Antityphus vaccine is also effective; two doses are given about one month apart and a third is given after three months. Thereafter, doses at intervals of several months are given to people who are exposed to the disease. The vaccine provides significant protection against attack and almost complete protection against death.

Rocky Mountain spotted fever. Rocky Mountain spotted fever is the form of tick-borne typhus that occurs in the Western hemisphere. It is caused by *Rickettsia rickettsii,* which is spread to human beings by the wood tick (*Dermacentor andersoni*), dog tick (*D. variabilis*), lone star tick (*Amblyomma americanum*), and several others in different parts of the Americas. Despite its name, Rocky Mountain spotted fever is most common on the eastern coast of the United States and, in fact, has been found in every state. It is a disease of the summer and early fall, for only then are ticks active.

The illness begins with headache, fever, and chills, soon followed by pains in the bones and joints and great weak-

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I. LICE

by

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Since 1970 the Vector Biology and Control Division of WHO has prepared, with the assistance of collaborators outside the Organization, a number of papers on vector control. The Expert Committee on Insecticides held in October 1974 (Technical Report Series No. 561) recommended that these documents - general reviews of the ecology and control of individual vector groups - should be continued and revised from time to time to provide workers with up-to-date, practical information on the particular subject. It was also recommended that there should be a feedback to the Organization: readers are therefore requested to write to Vector Biology and Control giving comments on their experience of the subject reviewed.

The following is the second revision of the paper on lice (WHO/VBC/70.212); first revision (WHO/VBC/75.520).

INTRODUCTION

Of the many kinds of blood feeding insects and arthropods which bite people, cause annoyance and skin reactions, and transmit disease, lice have become the most closely associated with humans. The various forms and species of human lice have been dependent for survival on the environment of the human body since before prehistoric times; they will not normally feed on other animals. In addition to the fact that all human lice are disagreeable vermin, one form - the human body louse - acts as a vector of two serious epidemic diseases: typhus and relapsing fever both of which may give rise to a high mortality if not treated. It is also responsible for the transmission of trench fever. Other forms - head lice and crab or pubic lice are not associated with disease transmission, but can be problems. The obligatory association of these lice with the human body suggests that these pests should be controlled, reduced or eliminated. However, the human body louse persists in many parts of the world where normal hygienic measures of washing, bathing and changing clothes are not possible or not practised and it may be a serious potential problem during wars or natural disaster. Both head and crab lice persist and are annoying problems in virtually every country. Resistance to the insecticides used to control human lice has been documented in many parts of the world, further complicating the ability to control or reduce the problem of these infestations.

There are three species of human lice: (i) the human body louse, Pediculus humanus L. (Fig. 1a); (ii) the head louse, Pediculus capitis De Geer (Fig. 1b, c) and (iii) the crab louse or pubic louse, Phthirus pubis (Fig. 1d). The first two (body and head louse) are very closely related and many persons have considered them as no more than subspecies or varieties (Ferris, 1951). They can, in fact, be successfully interbred. The principal and most important differences between these two species or forms are in living habits and disease

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Development times and stages of the immatures as well as blood feeding and mating mechanisms are similar to the body louse. The head louse is reported to lay fewer eggs than the body louse, i.e. about 50-150 during the life time of a female or at the rate of 4-5 eggs per day. A high biotic potential is suggested by these life history data, but until information on survival of immatures and adults on the host are known the actual growth rates cannot be determined.

Phthirus pubis

The life cycle is not greatly different from that of the two species of Pediculus, e.g. there are three nymphal stages and feeding and reproduction are similar. All stages are much more sedentary than head or body lice. The insects grasp body hairs with their specially adapted claws. Eggs (Fig. 2B) are cemented to body hair particularly in the pubic region. In heavy infestations other body hairs may be involved. Egg laying is at a lower rate than Pediculus, around three eggs per day.

Dissemination of lice

Normal transmission of lice occurs under conditions involving close proximity or contact, e.g. in crowded sleeping quarters or when people are forced to live crowded together. It is easy to understand, therefore, why widespread body louse infestations are often a result of war or natural disasters in groups of prisoners or refugees. As regards the head louse or crab louse, the intimate contacts of family life are ideal for transmission and crab louse transmission is generally by sexual contact.

Accidental transmission may also occur in a variety of ways, e.g. by direct contact in crowded public resorts or conveyances, or indirectly by means of cushions, upholstery or seats. Common use of bedding, towels, clothing or hair brushes and combs by a number of people may also serve to spread lice from one person to another. Stray hairs and combings may convey lice or their eggs and it is likely that this is a means of transmission of pubic lice. A possible source of infestation is the seats of ill-kept public water closets.

It must be borne in mind that all forms of lice fare badly away from their normal environment and soon starve to death. Accordingly, the risks of transmission decrease steadily, from the intimate contacts of family life to the close association of children at play and finally to the remote risks of infestation via fomites (furniture and other articles used by the infested person).

PUBLIC HEALTH IMPORTANCE OF LICE

The direct effect of louse bites, probably due to the foreign proteins in the saliva injected is more or less intense irritation. Infested people, therefore, tend to scratch themselves and this may lead to dermatitis, impetigo and similar conditions, especially staphylococcal infections. While it is evident that some individuals are much more sensitive to louse bites than others, virtually everybody suffers to some extent. Nor is it possible to become immune (as to some blood-sucking insects) though a slight tolerance may develop as one learns not to scratch. One of the curious effects of prolonged infestation by body lice is a hardening and yellowing of the skin, sometimes described as melanoderma or "vagabond's disease".

Of the three forms of human lice, only body lice are involved as disease vectors. In the laboratory head lice can be made to act as vectors, but they have not been responsible for typhus or relapsing fever under natural conditions in the absence of body louse infestations. Since man is the only reservoir of louse-borne diseases, the importance of body lice as disease vectors depends upon the existence of a disease focus in the area and the prevalence of lice in the human community.

In developed countries, body lice are now rare, because regular laundering of underwear makes it impossible for them to survive. Accordingly in these countries, infestations are virtually restricted to people with low standards of hygiene, such as vagrants who rarely change their underwear and commonly sleep in their clothes. Such people tend to associate together,

Relapsing fever

Louse-borne relapsing fever is due to Borrelia recurrentis. The disease now occurs mainly in Ethiopia and the Sudan; there are small foci elsewhere in East Africa with occasional cases in South and Central America. Body lice take up spirochaetes from the blood of infected persons in which they are especially prevalent during bouts of fever. They can be found in the louse's stomach for a few hours, but nearly all are digested. Sometimes, however, a few persist and appear about a week later in the insect's body cavity. This is an enclosed sac, so that commonly they remain in the louse throughout its life; they appear to do it no harm.

Infection can only occur if a man crushes an infected louse and releases the spirochaetes into a scratch or mucous membrane. This happens frequently among chronically lousy individuals who may destroy some of their lice by bursting them between finger nails, or even between the teeth.

Trench fever

The causative agent of trench fever is Rochalimaea quintana. This rickettsial disease is characterized by intermittent fever, generalized aches and pains, negligible mortality and multiple relapses. The body louse transmits the disease from individual to individual (humans are the reservoir). Transmission takes place by contamination of abraded skin with infective louse faeces. Infected people generally experience long-term rickettsaemia; R. quintana has been recovered from the blood up to eight years after acute illness.

Measures against louse-borne diseases

Certain antibiotics are valuable for treatment of either typhus or relapsing fever; and some measure of protection of individuals may be obtained by prophylactic immunization. To deal effectively with epidemics, however, it is essential to reduce general lousiness as rapidly as possible. This will be discussed in a later section.

Hygienic importance of head lice and crab lice

The direct effects of head and crab lice, like those of body lice, are to cause irritation and consequent scratching. Secondary infection, especially with impetigo, is a frequent consequence of neglected heavy infestations.

Head lice are much more prevalent than body lice both in the developed and developing countries. During and immediately after World War II, vigorous countermeasures supplemented by the new insecticides DDT and HCH (lindane) substantially reduced their incidence; however, in recent years there is disturbing evidence of an upsurge (Gratz, 1977). Thorough and extensive investigations in Britain have revealed an incidence of as much as 15% among children in some cities and it is likely that similar figures would be discovered by careful searches in other countries.

In contrast to the body louse which is today often more common on elderly people with poor personal hygiene, the head louse tends to infest young people, especially children, more than older ones, and the infested children are often clean in other respects. Persons with an abundant growth of hair on the scalp are generally more liable to infestations of head lice than those with short hair. It used to be thought that this was the sole explanation of the higher incidence in girls and women; but the fashion for longer hair in boys and youths has not yet made them quite so liable to infestation as girls among whom infestation rates are generally higher.

Although head lice tend to be discouraged by normal hair brushing, combing and washing, one cannot guarantee that such operations will eliminate an infestation unless very thoroughly done. For this reason it is perhaps not surprising that among the poorer children in an industrial city, it was shown that the liability to infestation increased with the size of the family (at least up to five). No doubt this reflects the declining opportunities for close attention by the more harassed mother and the greater crowding of the children, often in a single bed, increasing direct contact and ease of transmission of the lice.

The most satisfactory formulation for mass disinfestation is a dusting powder. This is easily shipped, readily stored and easily applied by any type of dusting apparatus from compressed air dusters to hand-operated dusters and even hand application. Dusts leave obvious signs on the clothing and may be resisted by people unless the purpose is adequately explained.

Where populations are susceptible to DDT, 10% DDT dusting powder is the insecticide of choice. It has a low acute and chronic toxicity to humans and as a dusting powder against lice is not likely to contaminate the environment. Where resistance has occurred the compounds in Table 1 may be used.

For individual treatment about 30 g per person should be applied evenly over the inner surface of garments touching the skin with special attention to seams, folds and tops of socks. For mass treatment of large groups of people, clothing need not be removed and hand-operated dusters or motor-driven air compressors with as many as 10 duster heads can be used. About 50 g of powder is shaken or blown into the clothing through the neck openings, up sleeves and from all sides of the loosened waist or trousers. In delousing women an extra quantity may be introduced down the neck of the dress and application at the waistline omitted. The socks, head covering, the inner surfaces of extra garments and bedding should also be treated.

One thorough treatment of infested clothing with insecticides such as DDT and malathion should be sufficient. Retreatments may be required at intervals of 3-4 weeks if infestations persist or reinfestation is expected.

Insecticides - head lice

Treatment is directed against the infested parts - in heavy infestations the head louse may be found on the hairs of other parts of the body. Liquid formulations are more acceptable than powders or dust. An aqueous suspension containing 1% lindane or a lotion containing 0.5% deodorized malathion in isopropyl alcohol can be used or 2% temephos in an appropriate solvent. Bioallethrin is in use in lotion and shampoos (0.3-0.4%) and in aerosols (0.6%). Deltamethrin (0.03%) and permethrin (1%) lotion and shampoos are being developed.

Liquid formulations should be applied so that the hair is thoroughly wet to the skin. Treated persons should not bathe or shampoo again for at least 24 hours. Ten to 20 ml of emulsion or 5-10 ml of solution per head is the amount to apply. Since these formulations should kill eggs as well as the active stages, one treatment is usually sufficient. Persistent infestations should be retreated at 1-2 week intervals. ?

Insecticides - pubic lice

Although crab lice are found most often in the pubic hair and in the perianal and axillary areas, they may occasionally infest hair of the trunk, thigh and beard, eyebrows and eyelashes. Powders or emulsions used for body or head lice control are effective. An isopropyl alcohol lotion containing 2% DDT, 1% lindane or 0.5% malathion can be used (see Table 1). For eliminating crab lice from eyelashes a vaseline ointment containing pyrethrins is effective.

Powders, emulsions or lotions are applied by rubbing into the hairs. Treated parts should not be washed for at least 24 hours after application. If one application is not sufficient, retreatment may be carried out at 4-7 day intervals. ?

Resistance

Hurlbut et al. (1952) were the first to report DDT resistance in body lice. Following further demonstrations of DDT resistance, insecticide dusts containing other compounds were developed: other organochlorines (Eddy & Bushland, 1943), organophosphates (Cole & Burden, 1956) and carbamates (Cole & Clark, 1962). Resistance to several louse powders including lindane and malathion has been reported. Wright & Brown (1957), Brown (1958) and Wright & Pal (1965) conducted global surveys and summarized the resistance problem.

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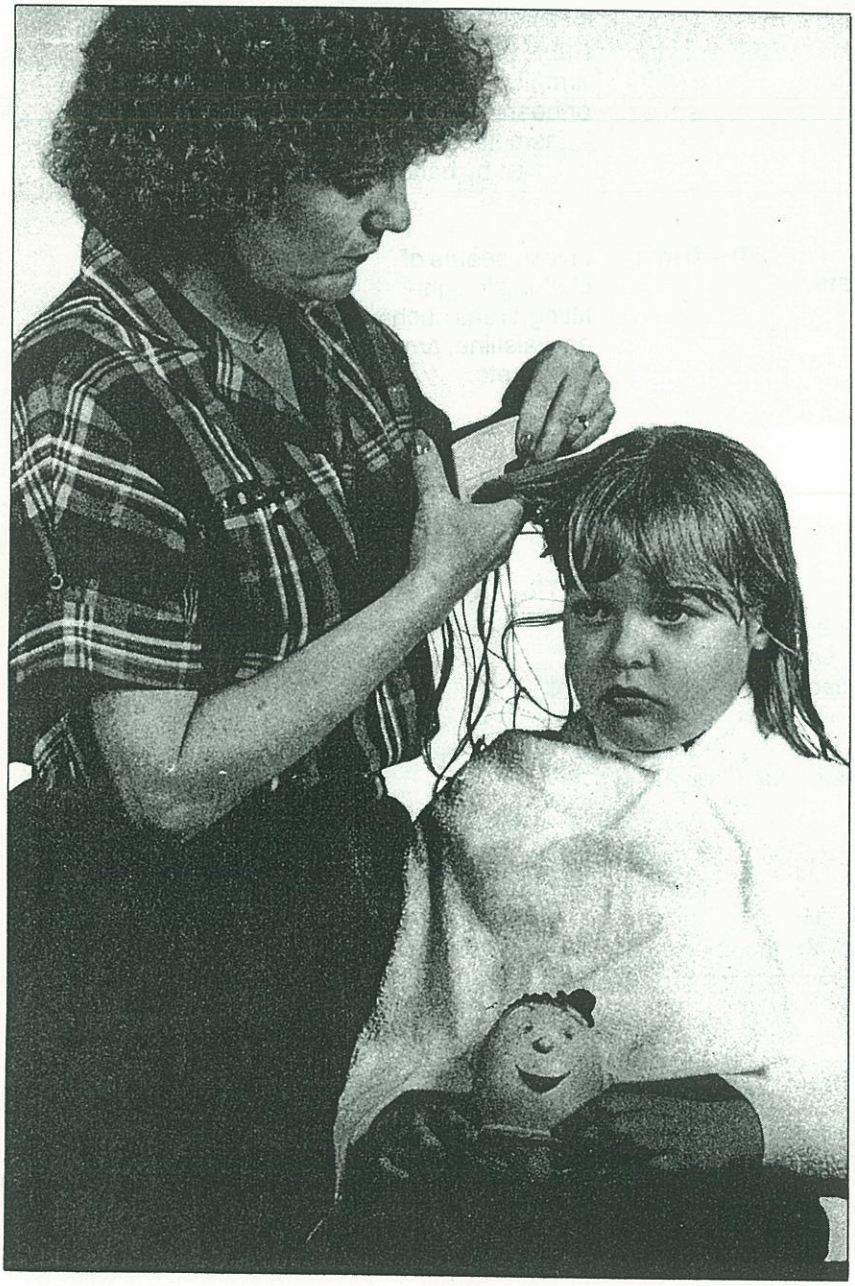
Treating head lice

PUBLIC HEALTH AND PHARMACY

Spread the word on pediculosis



With louse infestation a growing problem, pharmacists should make sure patients can come to them for knowledgeable and discreet counsel.

By JOHN J. WROBLEWSKI, R.Ph.

Would you recommend that a patient shave his head or dust his scalp with DDT? While these treatments have been relegated to the history books, along with the practice of dousing one's hair with kerosene, they were once serious approaches to the problem of louse infestation.

Although complications resulting from head, pubic, and body lice are rare in the United States, these conditions are embarrassing to the patient and his/her family. What's more, according to Dr. Charles Bainbridge, associate director of scientific services for the Pfipharmecs division of Pfizer Inc., New York City, the incidence of louse infestation appears to be spreading; it's estimated that two to 10 million Americans are infested with lice every year.

Many pharmacists, in concert with pediculicide manufacturers, are joining in a public education campaign to help foster awareness and stop the spread of lice, says Bainbridge.

A louse is an insect that's barely large enough to see, especially since it's rather transparent. The head louse, *Pediculus humanis capitis*, ranges from 1.0 to 2.0 mm in length and is found most frequently in the

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SIGNS AND SYMPTOMS OF LOUSE INFESTATION

1. Itching—severe and persistent, especially in the back of the head or nape of the neck.
2. Scratch marks or "rash."
3. Presence of "nits"—small, silvery eggs firmly attached to individual hairs.
4. Occasionally, swollen glands in the neck or under the arms.

MYTHS ABOUT LICE

1. Only dirty or unclean people get lice. (Anyone can have lice.)
2. You have to shave your head to get rid of lice. (Not necessary with today's effective products.)
3. Lice can be caught from plants or trees. (Lice are caught from other people, directly or indirectly.)
4. Only children are susceptible to lice. (Children are the most common victims, but adults can also be infested.)
5. Lice can jump or fly from one person to another. (Lice have no wings. Direct contact is necessary.)
6. Lice occur only during the summer. (Lice can occur during any season.)
7. Lice can be caught from house pets. (Human lice cannot live on animals.)

IMPORTANT CONSIDERATIONS IN TREATING HEAD LICE

1. Fine-toothed combs should be used to remove dead lice and nits from the hair—currently marketed pediculicide products do not dissolve dead lice or their eggs. Any nits that are not killed may hatch and reinfest the hair in seven to nine days.
2. Itching may persist for a while, even after the lice have been eradicated.
3. Reapplications of pediculicides may be necessary if lice or nits reappear.
4. Treat the patient's family and close associates if they too are infested.
5. Clothes, bedding, etc. may harbor lice and should be washed in hot water or dry-cleaned. Carpets should be vacuumed thoroughly. Alternatively, items may be isolated in plastic bags, which kills all lice in 30 to 35 days.
6. Use a pyrethrin-containing aerosol spray on carpets, mattresses, and upholstery that cannot be washed or laundered.

DrugTopics

hair of the scalp where it produces an intense, maddening pruritus. The itching is the result of saliva injected into the skin when the louse feeds.

"All lice are bloodsuckers," says Bainbridge, "and they can be rather irritating. Sometimes secondary infections can occur when people scratch and abrade the skin."

Because children are less concerned with hygiene and more apt to share combs and hats or engage in "rough and tumble" play, they are the most frequent victims of pediculosis. The lice are transmitted by direct contact or by contact with fomites such as clothing or brushes.

Diagnosis is relatively easy. The female attaches an egg or "nit" to the base of a hair fiber; a single head louse may deposit 140 nits in her 30-day life cycle. The nits are small, pearly, and oval-shaped and look something like dandruff flakes, but, unlike dandruff, they are extremely difficult to remove. "The female glues her nits to the hair fibers with a kind of cement that would defy Godzilla," says Bainbridge.

Pubic or "crab" lice deposit nits in the pubic hairs. Infestation with this variety of louse, *Phthirus pubis,* frequently extends to the perianal region but may also be found in axillary, beard, and mustache hairs. The lice themselves are quite different anatomically from head lice, being shorter and more rounded, and having larger claws—a crab-like appearance.

"Pubic lice can be considered one of the sexually transmitted diseases," says Bainbridge, and "tend to be prominent among male homosexuals who have multiple partners."

Body lice, or *Pediculus humanis corporis,* are very rare in the United States but are seen in war zones or when people are subjected to overcrowding or poor sanitation. They are larger than head lice but somewhat similar in shape. "Body lice live in the seams of clothing and move to the patient's body to feed," says Bainbridge. "They are the only lice that transmit serious diseases such as typhus and trench fever."

The only OTC pediculicides recommended by the Food & Drug Administration's Miscellaneous External Products Panel as safe and effective (Category 1) for the treatment of lice contain pyrethrins (0.17% to

Pfizer info 7/18/83

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To the memory of
Philip Hanson Hiss, Jr.,
Hans Zinsser,
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and David T. Smith

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Preface

With each passing year the term *microbiology* becomes a less satisfactory umbrella for the many disciplines that it attempts to cover. Bacteriology, immunology, virology, mycology, and parasitology have each long since become separate and independent disciplines. They are treated together in a single text simply because they deal with the agents that cause infectious diseases and with the mechanisms that hosts elaborate for defense against them.

In spite of the undeniable triumphs of antimicrobial chemotherapy, which has revolutionized the practice of medicine and very likely represents the greatest single triumph of biomedical science, "microbes" are by no means "conquered"; they continue to cause infections that demand a large amount of the physician's time. In fact, knowledge concerning new infectious agents, unsuspected properties of known agents, additional mechanisms for the genesis and persistence of infections, and the behavior of infectious agents at the molecular, cellular, and organismal levels is accumulating at an ever increasing pace. As a result, the scope and complexity of the material presented to students is expanding rapidly, and the compilation of a comprehensive textbook of manageable size is becoming ever more difficult.

This new edition of *Zinsser Microbiology*, the nineteenth, is designed for medical students experiencing their first exposure to medical microbiology. To that end, we not only describe the pathogenic infectious agents and the diseases that they cause, but also discuss the basic principles of bacterial physiology and genetics, of molecular and cellular immunology, and of molecular virology, our purpose being to provide a firm basis for growth with the field throughout the student's professional career. The book is also designed as a reference source for instructors; to that end each chapter is supplemented with a selection of both reviews and important original papers that provide a rapid entrée to any specialized topic.

The nineteenth edition represents a very extensive revision of the eighteenth edition. Many portions of the text have been completely rewritten and the remainder have been thoroughly updated. In the Basic Bacteriology section very important chapters on the molecular basis of genetics and genetic variation and exchange have been completely rewritten by a new contributor, Dr. Ken Kreuzer; he has taken the place of Dr. Richard Burns who died in 1984. The Clinical Virology section also has several new contributors who provide completely new chapters on arboviruses, rhabdoviruses, arenaviruses, and the human immunodeficiency viruses including the virus that causes the acquired immune deficiency syndrome (AIDS); and the chapter on recent and current developments in molecular pathogenesis has also been completely rewritten. In the Basic Virology section there is much new material, particularly in the chapters on the molecular aspects of virus multiplication cycles and on tumor viruses; these are areas in which a wealth of very important new information is coming to hand, information embodying new principles that are modifying drastically our views of the nature of genetic material and of the mechanisms that regulate its expression. Clearly these are areas of vital importance to medical practitioners. The same applies to the Immunology section where new chapters on the cellular basis of the immune system, immunopathology, and on the immune responses to infection have been provided. This section provides a comprehensive account of both basic and clinical immunology, organized so as to highlight topics currently deemed of maximum relevance to medical students. Finally, all chapters in the Medical Bacteriology, Medical Mycology, and Parasitology sections have been thoroughly updated, with new material added on recently recognized diseases such as Legionnaire's disease, the toxic shock syndrome, and Lyme disease. Increased emphasis has also been placed on the various organisms commonly associated with opportunistic infections which develop in immunocompromised patients or in patients with prosthetic device implants. In these sections, which like all other sections have been carefully edited by a single editor so as to ensure uniformity of format, emphasis is again placed on correlating the basic and clinical aspects of each infectious agent so that the student may acquire an appreciation of how fundamental research unravels the complexities of host–parasite relationships. Each chapter consists of (1) an introduction to the important biologic properties of the organism, (2) a description of the clinical infection in humans, including a discussion of the mechanisms of pathogenicity, (3) a section on laboratory diagnosis that provides information on modern culture and immunologic procedures, and (4) a discussion of the currently recommended treatment.

With regard to the bibliography, we have again elected not to reference specific statements in the text but to append to each chapter a list of recent reviews and key original papers. The former will quickly guide the reader to any specific aspect of microbiology and immunology that he or she wishes to pursue; the latter provide the detailed considerations and circumstances that have gone into the genesis of the most important discoveries. Many of the papers that are cited already are, or no doubt will soon become, "classics."

We have tried not to increase the size of the book—no easy task in view of the enormous amount of new information that has accumulated since publication of the last edition in 1984. Obviously, this has entailed the omission of a certain amount of older material; however, we are confident that there are no major gaps and that in our presentation of the newest advances we have not sacrificed careful and logical explanations of fundamental principles.

The list of individuals who have helped to produce this volume extends far beyond the circle of our colleagues who contributed textual material and to whom we are profoundly indebted. We would especially like to thank our many colleagues who permitted us to use illustrative material and who

almost invariably supplied us with original photographs, and the many publishers who allowed us to reproduce previously published material. We would also like to thank the artists who did a superb job in drawing the innumerable charts and diagrams, and the many secretaries who cheerfully massaged the text on their word processors again and again. Finally, we wish to express our appreciation to the staff of Appleton & Lange for their efficient cooperation in producing this new edition.

Wolfgang K. Joklik
Hilda P. Willett
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    - Treatment
    - Control

rian passage to occur. The infected tick may survive for years without food in environments of low humidity. The infection is transmitted by ticks both by contamination of the bite with coxal fluid and by the salivary fluid. The life span of the tick is not shortened by carrying borrelia. Many *Ornithodoros* species will feed on a variety of hosts.

B. hermsii and its vector are found primarily at elevations above 3000 feet and are associated with tree squirrels and chipmunks, which may carry the ticks into cabins where they become established. *O. turicatae* parasitizes goats, sheep, and rodents and is found in caves and animal burrows mainly in Florida and Mexico. *O. parkeri* inhabits the homes of ground squirrels and prairie dogs at lower elevations than *O. hermsi* and is widely distributed geographically. Numerous other small mammals also serve as reservoirs for tick-borne *Borrelia*, including rats, mice, rabbits, opossums, and hedgehogs. Birds have not been implicated. Once infected, the tick may harbor the disease for many years. However, tick-borne disease is not rapidly spread and, in the United States, is responsible only for sporadic cases.

In the United States, relapsing fever is a disease limited to persons who have come in contact with infected ticks. This most commonly results from vacationing in a tick-infested summer cottage. In one recent outbreak, 11 of 42 members of a Boy Scout troop contracted relapsing fever. Most of the infected had slept in a rodent-infested cabin, whereas the scouts who were younger had slept in tents and did not become infected. The spring and summer distribution of disease coincides with the season of maximal tick activity and the avocational invasion of humans into tick-infested areas. In the United States, foci of tick-borne borreliosis occur mainly in the western states, particularly Oklahoma, California, New Mexico, Colorado, Washington, Texas, and Kansas, which may reflect the distribution of *Ornithodoros* ticks.

In some areas of Africa, inhabitation of the home by *Ornithodoros moubata* is considered to be good luck and has resulted in introduction of disease.

Louse-borne Disease. The human body and head lice, *Pediculus humanus corporis* and *Pediculus humanus capitis*, are the vectors of epidemic relapsing fever caused by *B. recurrentis*, although there is some evidence that bedbugs may occasionally also transmit the disease. After the louse ingests *Borrelia*, the organisms pass exclusively into the hemolymph and central ganglion. Because other organs are not invaded, there occurs neither transovarian transmission nor direct infection during the feeding by an intact louse. *Borrelia* escape the louse to infect the host only when the louse is injured, as may occur during scratching. A single louse can, therefore, infect only one person. The infected louse remains infectious for its life span, which is approximately 10 to 60 days. Lice may rapidly and widely disseminate disease. Epidemics usually occur in the cold seasons, among the crowded and poor, and in homes with inadequate hygiene. Because of a narrow temperature preference, the louse typically leaves a febrile patient in search of a new host, thereby potentiating rapid spread of an epidemic. Although *B. recurrentis* is considered to be the louse-borne species, tickborne *Borrelia* may also be transmitted by lice. No natural animal reservoir of *B. recurrentis* is known. Currently, *B. recurrentis* is endemic, primarily in areas of Ethiopia.

Relapsing fever may occasionally be acquired by means other than louse or tick infestation. For example, transplacental transmission has caused congenital disease, and infected blood may be the cause of laboratory accidents leading to infection.

Pathogenesis. During the entire course of borreliosis, there is a constant spirochetemia, which worsens during febrile periods and wanes between recurrences. Specific pathogenic factors are ill-defined. However, the organisms appear to contain a heat-stable pyrogen that is not endotoxin. Skin biopsy specimens of infected persons have shown that there is no inflammatory response around the spirochetes that are within the dermis and that the dermal vessels show no thrombosis or other evidence of vasculitis, as would be expected if endotoxin were produced.

Borrelia are actively phagocytized by PMNs of humans. Immune serum both enhances phagocytosis and exerts a direct effect on *B. hermsii* causing decreased motility and viability and increased agglutination.

Clinical Manifestations. Prior to the development of effective antimicrobial agents, fever induction was used in the therapy for tertiary syphilis. Induced infection with *Borrelia* was often selected for this purpose, and much of our present knowledge concerning prodromata, incubation period, natural history, and complications stems from these experiences.

The symptoms and severity of relapsing fever depend on the immune status of the host, geographic location, strain of *Borrelia*, and phase of the epidemic. There may also be consistent differences between some characteristics of louse-borne disease and tick-borne disease, but both forms will be described together (Table 47–6).

The natural history of a course of relapsing fever includes the incubation period, the primary attack, the afebrile interval,

TABLE 47–6. CLINICAL MANIFESTATIONS IN RELAPSING FEVER

Manifestation	Mean Value of Incidence	
	Tick-borne Disease[a]	Louse-borne Disease[b]
Incubation period (days)	~7	
Duration of primary febrile attack (days)	3.1	5.5
Duration of afebrile interval (days)	6.8	9.25
Duration of relapses (days)	2.5	1.9
Number of relapses	3	1
Maximum temperature (primary attack)	~105F (40.5C)	
Splenomegaly (%)	41	77
Hepatomegaly (%)	17–18	66

and subsequent attacks. In epidemic, endemic, and therapeutically induced disease, few prodromata have been noted. The incubation period is approximately 6 days, with a range of 2 to 14 days. Late in the incubation period the patient may experience chills. The onset is usually very sudden and accompanied by fever, headache, tachycardia, and muscle pain. The initial attack usually lasts 3 to 7 days, may be longer for louse-borne than for tick-borne disease, and ends by crisis. The fever is usually continuous.

A macular rash is seen in varying numbers of patients and usually appears near the end of the first paroxysm. Hepatosplenomegaly, jaundice, nausea, and vomiting are common. Bronchitis and bronchopneumonia are frequent in the United States. Meningeal signs with and without encephalitic disease may affect up to 30 percent of some groups of patients, and ocular disease is common.

The crisis is coincidental with the immune response. Occasionally, the crisis is associated with shock. Usually, the temperature returns to normal, and the patient is asymptomatic until the subsequent attack. The interval between initial and subsequent attacks is usually shorter with louse-borne disease, 5 to 9 days, than with tick-borne disease, which is approximately 14 days.

Data from the period 1921 to 1941 described the course of untreated tick-borne disease as follows: no relapse, 16 percent; one relapse, 20 percent; two relapses, 27 percent; three relapses, 17 percent; four or more relapses, 18 percent of cases. A similar distribution of relapses has occurred in some outbreaks of louse-borne disease.

Subsequent attacks are usually shorter in duration, less severe, and with increasingly shorter apyrexial periods between attacks but are otherwise clinically similar to the initial episode. Most physicians fail to diagnose relapsing fever until one or more relapses have occured.

Treatment and Prevention. Treatment of relapsing fever includes general supportive measures such as fluid and electrolyte therapy. Evaluation of the efficacy of antimicrobial therapy has been inhibited by the lack of information on in vitro sensitivity. The most clinically effective antimicrobial agents appear to be tetracyclines and chloramphenicol. Streptomycin has been found to modify the disease, although it may fail to prevent relapses.

Prevention of relapsing fever is dependent on control of exposure to the arthropod vectors. In tick-borne borreliosis, this includes wearing protective clothing, careful cleaning of rodent-infested cabins, followed by spraying with appropriate insecticides such as aldrin, benzene hexachloride, or malathion. Louse-borne relapsing fever is controlled by the application of good personal and public standards of hygiene.

LYME DISEASE

The discovery or emergence of a newly recognized illness is a particularly exciting event in the history of infectious diseases. In 1975 a newly recognized syndrome consisting of an illness that was associated with a unique skin lesion, erythema chronicum migrans (ECM), was recognized in a cluster of rural children in Lyme, Connecticut. This illness, now known as Lyme disease, is also referred to as ECM, Lyme arthritis, and Bannwarth's syndrome. The causative agent of Lyme disease is a new species of *Borrelia, B. burgdorferi,* named for Dr. Burgdorf, the microbiologist who isolated and identified the agent. The rapid elucidation of the etiology, diagnosis, sequelae, and treatment of this major disease is one of the recent and exciting events in clinical microbiology and epidemiology.

its general morphology. It is similar in size to *Treponema* species. It has seven periplasmic flagella per cell end, which is less than the number for other *Borrelia* species, which contain 15 to 20 (Table 47–5).

Attributes of *B. burgdorferi* that led to its classification as a *Borrelia* species include its transmission by ticks, in vitro culture in a modified Kelly's medium as are other *Borrelia* species, and microaerophilic metabolism. Also, the chromosomal DNA nucleotides of *B. burgdorferi* show mole percent guanine-plus-cytosine ranges that are similar to those of other North American borreliae and that differ from those of *Leptospira* and *Treponema* species. DNA homology studies similarly indicate considerable homology between *B. burgdorferi* strains of other North American borrelia.

European forms of the disease caused by *B. burgdorferi* have a lower incidence of arthritis and a higher incidence of neurologic disease when compared with disease in the United States. To examine possible reasons for these dissimilarities, antigenic components of strains from a wide variety of sources have been examined. Analysis by polyacrylamide gel electrophoresis of the major exposed proteins of the spirochetal surface, the OspA proteins, has revealed differences between the U.S. and most European strains. It is not yet known whether *B. burgdorferi* resembles other *Borrelia* species in its capacity for antigenic variation.

Clinical Infection

Epidemiology. *B. burgdorferi* is transmitted through the bite of a tick. Ticks that are known to harbor and transmit Lyme disease to humans include *Ixodes pacificus* on the West Coast, *Ixodes dammini* on the East Coast and Midwest, *Ixodes ricinus* in Europe, and *Ambylomma americanum,* the Lone Star tick that is also a vector of tularemia and Rocky Mountain spotted fever. The preferred host for nymphal *Ixodes* ticks is the deer mouse, and for adult ticks it is deer. The human disease is mainly transmitted by nymphal ticks, which are very small and aggressive.

In the United States there are three major foci of recognized cases of Lyme disease; the midwestern coast, Minnesota and Wisconsin, and the northeast coast. However, almost 30 states have reported cases, as well as Europe and Asia.

Clinical Manifestations. The clinical disease commonly presents as three consecutive stages of illness. In the initial stage there is a highly characteristic expanding skin lesion that usually shows a papule at the site of the tick bite, has sharply demarcated borders, often shows relatively minimal involvement of the skin centrally, and is often accompanied by various constitutional symptoms such as malaise, fever, headache, and stiff neck. The lesion may reach a large size, fade over several months, may periodically recur, and may be associated with multiple other annular lesions at other sites.

The subsequent stage of the disease is estimated to occur in about 5 to 15 percent of patients and is characterized by the onset of neurologic or cardiac involvement, usually within a few months or less of the initial lesion. Headache, Bell's palsy, radiculoneuropathy, myocarditis, and arrhythmias are common presentations.

The third recognized stage of disease primarily involves migrating episodes of arthritis and occurs weeks to months after the tick bite. Each event may last as long as several months, remits, and may be followed by another attack in another joint. This may continue to be associated with fever and last for several years. The arthritis is not destructive, and joints are relatively normal after resolution.

period and severity of the disease may be related to the size of the inoculum of *R. rickettsii*.

Host Defenses

The relative importance of various host defense mechanisms in humans infected with rickettsiae is uncertain. It is apparent, however, that host defense against rickettsial infections involves both humoral and cell-mediated immunity. Delayed hypersensitivity to typhus-group antigens develops after human *R. prowazekii* infection, and lymphocyte-mediated hypersensitivity also can be demonstrated after vaccination or infection with *C. burnetii*. Lymphocytes collected from humans previously infected with *R. rickettsii* undergo blast transformation after in vitro exposure to spotted fever group antigens. An interaction between humoral antibody (opsonins) and macrophages is required for effective killing of *R. prowazekii* by human macrophages. Similarly, antibody-treated *R. rickettsii* are phagocytized and destroyed by guinea pig peritoneal macrophages, whereas untreated rickettsiae replicate and destroy peritoneal phagocytic cells. Although resistance to *R. typhi* infection is not transferred by immune serum, resistance to intradermal challenge with *R. typhi* is transferred by immune spleen cells. In addition, even unopsonized rickettsiae are destroyed by macrophages that have been treated with lymphokines from rickettsial antigen-treated mouse spleen cells. The lymphokine activity has been attributed to interferon-γ. It has been postulated that interferon-γ, a soluble product of T lymphocytes, is probably the mechanism for the effective control of rickettsiae within infected cells. In concert with antibody and macrophages that deal with extracellular rickettsiae, a complete system would be available for the immune elimination of *R. prowazekii*.

CLINICAL INFECTION

Among the diverse clinical illnesses produced by rickettsiae are primary pneumonia (Q fever), fulminant vasculitis (Rocky Mountain spotted fever), a febrile illness associated with a vesicular rash (rickettsialpox), asymptomatic infection (trench fever), a recrudescent infection appearing many years after primary infection (Brill–Zinsser disease), and endocarditis (Q fever) (Table 51–3). Endothelial damage secondary to angiitis is a common finding in rickettsial infections, particularly in spotted fever and typhus.

THE SPOTTED FEVER GROUP

The basic pathologic process in the spotted fever group is a widespread vasculitis involving the skin, with production of a rash. More severe disease can lead to disseminated intravascular coagulopathy with petechial and purpuric skin manifestations.

Rocky Mountain Spotted Fever (Tick-borne Typhus)

Epidemiology

Prevalence. Rocky Mountain spotted fever has been recognized as a distinct clinical entity for almost a century. Although the disease got its name from the Rocky Mountain region where it was first recognized, subsequent studies have shown that it is, instead, much more common in the Piedmont region of the southeastern United States (Fig. 51–2). Cases continue to be reported, however, in small numbers from almost every state in the United States. During 1984, 838 cases of Rocky Mountain spotted fever in the United States were reported to the Centers for Disease Control, and many other cases undoubtedly were either not reported or not diagnosed (Fig. 51–3). Rocky Mountain spotted fever accounts for over 95 percent of the reported rickettsial disease in humans in the United States.

Most cases of Rocky Mountain spotted fever in the eastern and the southern United States occur in children and adolescents, whereas in the Rocky Mountain region, adult men are more commonly affected. The seasonal distribution of Rocky Mountain spotted fever is related to the activities of the tick vector, with the result that the disease makes its appearance in April and continues through August. This seasonal distribution should be stressed because any febrile illness occurring during these months in endemic areas is suspect for Rocky Mountain spotted fever. In addition, cases of this disease have

TABLE 51–3. EPIDEMIOLOGIC FEATURES OF SELECTED RICKETTSIOSES

Disease	Etiologic Agent	Geographic Distribution	Arthropod Vector	Animal Reservoir	Weil–Felix		
					OX-19	OX-2	OX-K
Spotted fever group							
Rocky Mountain spotted fever	*R. rickettsii*	North and South America	Tick	Wild rodents, dogs	+	+	–
Rickettsialpox	*R. akari*	Worldwide	Mite	Mouse	–	–	–
Boutonneuse fever	*R. conorii*	Mediterranean countries, Africa, India	Tick	Wild rodents, dogs	+	+	–
Queensland tick typhus	*R. australis*	Australia	Tick	Wild rodents, marsupials	+	+	–
North Asian tick typhus	*R. siberica*	Siberia, Mongolia	Tick	Wild rodents	+	+	–
Typhus group							
Epidemic typhus	*R. prowazekii*	Worldwide					

mite bite. The first sign of disease is a local erythematous papule that evolves first into a vesicle and then into an eschar. Approximately 3 to 7 days after the appearance of the eschar, chills and fever begin abruptly and may be associated with headache, malaise, and myalgia. Within 72 hours of the appearance of fever, a generalized maculopapular rash becomes apparent and soon evolves into a vesicular eruption. Differentiation of the rash of rickettsialpox from chickenpox is important and is based on the following observations concerning the rash of rickettsialpox: (1) it occurs more often in adults, (2) it is associated with a primary eschar, and (3) the cutaneous vesicles are surrounded by papular rings. In contrast, the rash of chickenpox occurs most often in children, is entirely vesicular, and lacks a primary lesion. Smallpox does not have a primary eschar, is associated with an eruption that evolves into pustular lesions, and usually is a more severe illness.

No fatalities attributable to rickettsialpox have been reported. Although a small scar may occur at the site of the primary lesion, the vesiculopapular eruption heals without scarring.

Laboratory Diagnosis. Weil–Felix antibodies do not appear after infection with *R. akari*. However, complement-fixing antibodies can be detected 1 to 2 months after the onset of illness.

R. akari can be isolated from the blood and the vesicular fluid from lesions of infected persons. Such isolations require the technical facilities of specially equipped laboratories and are accomplished by the inoculation of infected specimens into laboratory animals or embryonated hens' eggs.

Treatment and Prevention. Both tetracycline and chloramphenicol produce rapid defervescence and clinical improvement.

Measures aimed at controlling both rodent populations and their mite ectoparasites will prevent transmission of *R. akari* to humans.

Other Tick-borne Diseases

Other species of *Rickettsia* cause tick-borne diseases that in many respects resemble Rocky Mountain spotted fever (Table 51-2). They are found on several continents and cause sporadic cases of a mild clinical pattern. *Rickettsia sibirica*, the agent of North Asian tick typhus, *Rickettsia australis*, the agent of Queensland tick typhus, and *Rickettsia conorii*, the agent of boutonneuse fever, are very similar but, by the use of cross-immunity and mouse-toxin neutralization tests, have been shown to be separate organisms. North Asian tick typhus occurs in central Asia, Mongolia, and the Siberian region of the USSR. Queensland tick typhus occurs in Australia, and boutonneuse fever occurs in the Mediterranean region, Africa, and India. Boutonneuse fever has also been called South African tick bite fever, Kenya tick typhus, and Indian tick typhus.

All three of these rickettsiae are maintained in nature in both ixodid ticks and wild animals. Humans only accidentally enter their natural cycle of infection and are not important in the maintenance of the rickettsiae in nature.

Diseases caused by these rickettsiae are characterized by local eschars or skin lesions at the site of tick attachment. All reactivity with other members of the spotted fever group occurs. All three rickettsiae are sensitive to both chloramphenicol and tetracycline.

Rickettsia Not Associated with Human Disease

Rickettsia parkeri, *Rickettsia montana*, and *Rickettsia rhipicephali* are species serologically related to the spotted fever group but whose disease potential in humans is currently unknown. *R. parkeri*, originally referred to as the "maculatum agent," was first isolated from Gulf Coast ticks (*Amblyomma maculatum*) removed from cattle in eastern Texas. Subsequent isolations of this rickettsia were made from the same tick species in Mississippi and Georgia. The southeastern and south central region of the United States is also an enzootic area of *R. rickettsii*, which is found primarily in *D. variabilis* but also in several other species of ticks. *R. parkeri* has not been isolated from any of these ticks, nor has *R. rickettsii* been isolated from *A. maculatum*. To date, *R. parkeri* has not been isolated from any other ticks or from any other part of the world. The reason for this host specificity is unclear, but an interference phenomenon has been invoked as an explanation. If a large proportion of *A. maculatum* are naturally infected with *R. parkeri*, any other rickettsia would be excluded from that particular niche. This could also explain the absence of *R. parkeri* in the natural acarine hosts of *R. rickettsii*.

R. montana was initially isolated from ticks in eastern Montana, but subsequent isolations in other states suggest that it is widely distributed in natural cycles involving small rodents and ticks. *R. montana* is a natural parasite of the major tick hosts of *R. rickettsii* but is antigenically distinct from any of the tick typhus rickettsiae. Guinea pigs immunized with *R. montana* are almost completely protected against challenge with *R. rickettsii*.

R. rhipicephali, another member of the spotted fever group, has been isolated from brown dog ticks (*Rhipicephalus sanguineus*) in several southeastern states. It is widespread and common in occurrence. *R. rhipicephali* differs biologically and antigenically from *R. rickettsiae*. There is no evidence of pathogenicity for the dog or for humans, but it has been hypothesized that this species as well as *R. montana* may provide partial protection of dogs against *R. rickettsii*.

THE TYPHUS GROUP

The typhus group of rickettsiae cause epidemic typhus (and its recrudescent infection, Brill–Zinsser disease) and murine typhus. Typhus-group organisms are characterized by intracytoplasmic growth and a common, soluble, group-specific, complement-fixing antigen.

Epidemic Typhus (Louse-borne Typhus)

Epidemic typhus is a louse-borne disease caused by *R. prowazekii*, a rickettsia named after the Polish investigator von Prowazek who died of typhus contracted in the course of his studies. The disease has had a tremendous impact on the history of humans. According to Zinsser, Napoleon's retreat from Moscow "was started by a louse." In World War I, typhus was

Ecology and Transmission. *R. prowazekii*, the etiologic agent of epidemic typhus, can infect both the human body louse (*Pediculus humanus corporis*) and the head louse (*Pediculus humanus capitis*), the former being the more significant vector. The body louse feeds only on humans, and all three stages of its life cycle (egg, nymph, and adult) can occur on the same host.

Lice become infected after taking a blood meal from a rickettsemic human. Several days later the ingested rickettsiae have multiplied sufficiently in the louse, and infective rickettsiae appear in the arthropod's feces. If the louse encounters a susceptible human at this point, transmission of *R. prowazekii* may occur. During each blood meal the louse defecates. The feeding process is irritating, and scratching by the host produces minor excoriations that function as portals of entry for the rickettsiae in the louse feces. Lice do not transmit *R. prowazekii* to their progeny but succumb to their infection within 1 to 3 weeks.

Because louse–human–louse transmission thrives under conditions in which individuals wear the same clothes continuously in crowded environments, it is not surprising that major epidemics have occurred in association with war, poverty, and famine. Persons in cold climates are more likely to acquire typhus infections, particularly if they are forced by poverty or unusually hard circumstances to wear the same clothes for long periods of time. Lice actively seek out locations where the temperature is approximately 20C, a temperature often found in the folds of clothing. Lice will abandon a host with a body temperature of 40C or greater as well as the body of a dead person.

The finding of an association of sporadic cases of epidemic typhus in the United States associated with the flying squirrel has raised a question as to the existence of another reservoir of this rickettsial agent. The exact mechanism of transmission between the rodent and the human is poorly understood.

Clinical Manifestations. The incubation period typically ranges from 10 to 14 days. Prodromal symptoms of headache, malaise, and minimal temperature elevations sometimes occur, but usually the onset is abrupt, with generalized myalgias, chills or chilliness, fever, and headache. Headache is characteristically frontal, severe, and unremitting. Other less specific symptoms are often present, including gastrointestinal complaints, weakness, and cough. Splenomegaly may be present, as may meningismus. The spinal fluid is typically normal.

A skin rash usually occurs from 4 to 7 days after the onset of illness. It may first appear as a patchy cutaneous erythema and progress to maculopapular, petechial, or hemorrhagic forms. In contrast to Rocky Mountain spotted fever, the rash in typhus usually spares the palms, soles, and face and characteristically appears first on the trunk and later spreads to the extremities. A wide variety of complications may occur in severe cases, including mental changes (stupor and delirium), hypotension, oliguria and azotemia, and even gangrene of the skin, genitalia, and digits.

Untreated, the disease may last up to 3 weeks. Mortality has varied from 10 to 40 percent in different outbreaks. Case fatality ratios have been shown to increase with increasing age. Survivors of epidemic typhus are generally immune for years after their primary infection although mild recurrences of illness (Brill–Zinsser disease) may occur years later.

Laboratory Diagnosis. Once a clinical diagnosis is made, treatment should be instituted before laboratory confirmation. Substantiation of a clinical diagnosis can be obtained either by isolation of *R. prowazekii* or by serologic means. The former is difficult, potentially dangerous, expensive, and involves specialized personnel and equipment.

As in Rocky Mountain [illegible] in the second week after onset. Generally, agglutination is maximal with OX-19 strains, although strongly positive reactions with OX-2 antigens sometimes occur. Serial serum specimens should be tested rather than a single convalescent sample. A fourfold rise in agglutinating titer is suggestive of recent infection.

Antibodies against group-specific, complement-fixing antigens (prepared from yolk-sac-grown rickettsiae) typically appear in the third week after onset. Microagglutination and fluorescent antibody tests are also available through specialized laboratories.

Treatment. Both chloramphenicol and tetracycline produce prompt defervescence and clinical improvement when given early in the course of the illness. Patients who develop circulatory and renal complications before receiving either antibiotic may die despite therapy.

Prevention and Control. It is possible to interrupt epidemic louse–human–louse transmission of *R. prowazekii* by mass application of insecticides to humans and their clothing. Some populations of lice, especially in Africa, have become increasingly resistant to insecticides (including DDT and malathion). Once free of lice, patients with typhus are not infectious. Typhus vaccine prepared from infected yolk sacs is also an effective control measure. Although controlled studies in humans are lacking, it is generally accepted that typhus vaccine lessens the severity and shortens the course of clinical disease. Two doses of vaccine 4 weeks apart are necessary for primary immunization. Booster doses are recommended every 6 to 12 months during periods of exposure.

Brill–Zinsser Disease

Individuals who previously have had epidemic typhus may develop recrudescent infection many years later. This illness was named for Nathan Brill, who first recognized and described the clinical features, and Hans Zinsser, who first suggested in 1934 that the disease was a relapse of a prior epidemic typhus infection. Epidemiologic, clinical, and experimental evidence has since been published that confirms Zinsser's hypothesis.

Epidemiology. In the United States, recrudescent typhus occurs primarily in immigrants from previously endemic areas such as eastern Europe. The disease may occur in an individual living in a louse-free environment, and many years may have passed since the patient's initial infection with *R. prowazekii*. However, lice that feed on a patient with recrudescent typhus can become infected, and if local conditions are favorable for louse–human–louse transmission, an outbreak of epidemic typhus may result. Latent human infection thus is an interepidemic reservoir for *R. prowazekii*.

Clinical Manifestations. Brill–Zinsser disease is a milder illness than classic epidemic typhus; skin rash is rarely seen, and the duration of disease is shorter (less than 2 weeks). Fever may be erratic instead of sustained. As in epidemic typhus and other rickettsial diseases, headache, malaise, and myalgias are common symptoms. Complications and fatalities are rare.

Laboratory Diagnosis. In the United States, Brill–Zinsser disease should be suspected when fever of obscure origin occurs in a foreign-born person from an area where epidemic typhus has occurred and who complains of an intense headache and develops a maculopapular skin rash on the fourth to sixth day of illness.

Weil–Felix agglutinins often do not develop in patients

fixing antibodies may be detected during the second week after onset, which is earlier than in patients with epidemic typhus. Because some typhus patients may have detectable complement-fixing antibodies many years after the primary infection, an isolated convalescent serum sample may yield confusing results. Therefore, a fourfold complement-fixing antibody titer rise should be sought in patients suspected of having recrudescent typhus.

Consistent with Zinsser's hypothesis, it has been found that patients with epidemic typhus initially have an IgM followed by an IgG antibody response whereas patients with Brill–Zinsser disease initially have an anamnestic IgG antibody response. This microimmunofluorescence test is the most sensitive and reliable method to differentiate between Brill–Zinsser disease and primary epidemic typhus.

The clinical history, epidemiologic setting, and dynamics of the antibody response are helpful in distinguishing among epidemic, recrudescent, and murine typhus.

Treatment and Prevention. As in epidemic typhus, tetracycline and chloramphenicol are both effective in treatment. The ultimate prevention of Brill–Zinsser disease necessarily is dependent on the prevention of epidemic typhus. If recrudescent typhus occurs in an environment where lice rarely infest humans, no special precautionary public health measures are required. In areas where the potential for louse–human–louse transmission is high, delousing of the patient and his or her contacts may be necessary to prevent an outbreak of epidemic typhus.

Murine Typhus (Endemic Typhus, Flea-borne Typhus, Rat Typhus)

Murine typhus is a flea-borne illness caused by *R. typhi*, a member of the typhus group. Typically, murine typhus is a mild illness characterized by fever, headache, and often by a generalized skin rash.

Epidemiology

Prevalence. Murine typhus is endemic in many countries, including the United States where it occurs primarily in the Southeast and Gulf Coast region. It is also endemic in parts of Central America and Mexico. Investigations of murine typhus in Alabama and Florida completed over 45 years ago revealed that most cases occurred in individuals who worked in rat-infested shipyards and harbors. More recently, cases have been reported from inland rural locations, presumably because infected rats and mice may occur in large numbers in areas where grains and feeds are stored. In the past decade, over half of all reported cases in the United States have occurred in Texas.

Although murine typhus is a reportable disease in the United States, considerable numbers of cases may be neither diagnosed nor reported. Despite this problem, it appears that the incidence of murine typhus has gradually decreased in the past 2 decades.

Ecology. *R. typhi* is cycled in nature by the rat and two of its ectoparasites, the rat flea (*Xenopsylla cheopis*) and the rat louse (*Polyplax spinulosus*). The former is the more important vector. As in Rocky Mountain spotted fever, humans enter this arthropod–vertebrate–arthropod cycle only accidentally. *X. cheopis* acquires *R. typhi* infection by feeding upon a rickettsemic mouse or rat. Once infected, the flea may infect other susceptible rodents, and thus a natural cycle of flea–rodent–flea infection may become established. Rodents infected with *R. typhi* do not succumb to their infection, despite the presence of viable rickettsiae in rodent brains for periods up to several months. Fleas do not transmit *R. typhi* transovarially.

Transmission. Transmission of *R. typhi* to humans occasionally occurs. When infected fleas taking a blood meal defecate on the host, the host rubs the infected feces into small excoriations during scratching. Flea feces are also infective if accidentally transmitted to mucosal surfaces such as the conjunctiva.

Clinical Manifestations. Murine typhus is usually a mild illness with a death rate of less than 2 percent. The incubation period ranges from 1 to 2 weeks. The hallmarks of the disease are an abrupt onset of fever, headache, malaise, and myalgias and, in most cases, a macular to maculopapular, nonpruritic skin rash that begins on the third to fifth day on the trunk and spreads to the extremities. As in epidemic typhus, involvement of the palms, soles, and face is rare. The rash may be fleeting or absent in some cases and may be inapparent in blacks without careful inspection. Chills or chilliness, cough, nausea, vomiting, arthralgias, weakness, and extreme prostration may be associated symptoms. Untreated, the illness may last up to 2 weeks. Defervescence may occur by either abrupt crisis or gradual lysis. Fatalities are more likely to occur in the old and infirm. Such fatality may be heralded by peripheral vascular collapse and evidence of central nervous system involvement, such as stupor and coma.

Confusion between murine typhus and Rocky Mountain spotted fever may occur because both diseases are associated with rising titers against *Proteus* OX-19 (and sometimes OX-2) antigens. Spotted fever is usually a more severe illness and is often associated with an antecedent tick bite. The rash of murine typhus begins first on the trunk and spreads to the extremities, whereas the opposite evolution occurs in Rocky Mountain spotted fever. Because precise information on the evolution of the rash is often unavailable, the differential point is often not helpful. In older immigrant patients, confusion between murine typhus and Brill–Zinnser disease may also occur (p. 600).

Laboratory Diagnosis. Agglutinins to *Proteus* OX-19 and, less commonly, to OX-2 appear in the second week of infection. Complement-fixing antibodies against *R. typhi* appear slightly later. With the complement-fixation test, serologic cross-reactions among members of the typhus group are common, but in patients with murine typhus much higher antibody titers are obtained against *R. typhi* than against *R. prowazekii*. The use of specific antigens in the complement-fixation test also permits differentiation of *R. typhi* and *R. prowazekii*. An indirect fluorescent antibody test using IgM and IgG fluorescent-labeled conjugates has been developed.

Intraperitoneal inoculation of blood from patients with endemic typhus into a male guinea pig produces severe testicular lesions and scrotal swelling in contrast to the very mild disease produced by the inoculation of blood from epidemic typhus patients.

Treatment and Prevention. Both tetracycline and chloramphenicol are effective rickettsiostatic agents. Patients with laboratory-acquired murine typhus treated with chloramphenicol 2 to 4 days after its onset have been known to experience clinical relapses despite the presence of antirickettsial antibodies. These relapses responded to reinstitution of the same antimicrobials. Insecticides and rodenticides are both effective in reducing rat–flea–human transmission in endemic areas.

Rickettsia canada Infections

R. canada was first isolated from rabbit ticks collected in Ontario, Canada, in 1967. Antigenically, *R. canada* belongs to the typhus-group biotype. However, it grows in both the cytoplasm and the nuclei of infected cells, a characteristic of the spotted fever group.

The clinical spectrum of human infection with *R. canada* is still largely unknown. The organism has not been isolated

CONTROLLING

HEAD LICE

Note the change in tone from CDC in 75 to CDC in 89.

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PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL
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I. Biology

The head louse, *Pediculus humanus* var *capitis*, is a bloodsucking insect parasite found on the heads of humans. It does not successfully live on any other animal.

The head louse is restricted to the hair growing on the surface of the scalp, preferring the area behind the ears and the nape of the neck. It seldom occurs on eyebrows or eyelashes; lice in those sites are another species, *Phthirus pubis*, the pubic louse. Head lice are small (1 mm to 2 mm long) and vary in color according to the complexion of the human host. A louse on a person with dark skin and hair is usually darker than one on a person with fair skin and hair. Head lice grasp hairs with their hook-like claws and opposing digits located at the end of each of their 6 legs.

Little information exists on the life span of adult lice under natural conditions, but under optimum conditions in the laboratory, they have been reported to live about 1 month. Head lice can live an average of 2.3-3.5 days at 74 °F (23 °C) and 1-2 days at 86 °F (30 °C) when separated from a human host (3) but they do not naturally leave the human host. During her lifetime, a female head louse will deposit about 90 eggs—about 3 to 4 per day.

Live eggs—commonly called nits—are just visible to the unaided eye, and are yellowish or opalescent, oval, and opaque (Figure 1). They measure about 0.8 mm by 0.3 mm and are firmly attached to a shaft of hair close to the scalp by a cement-like substance. Eggs hatch in about 1 week. The resulting immature louse (nymph) becomes adult and reproduces in about 8 to 9 days. Adult head lice, eggs, and nymphs are illustrated in Figure 2.

Both immature and adult lice feed solely on human blood. To feed, the louse penetrates the skin with its mouthparts (which retract within a sac in the head when not in use), and injects saliva to prevent hemostasis. It then sucks the host's blood into its digestive tract. Blood-sucking may continue for long periods if the louse is not disturbed. While feeding, lice excrete dark red feces onto the scalp.

Itching, the major symptom of louse infestation, is caused by an inflammatory reaction by the host against saliva. Scratching by a sensitive patient may cause a secondary bacterial infection. Although head lice can

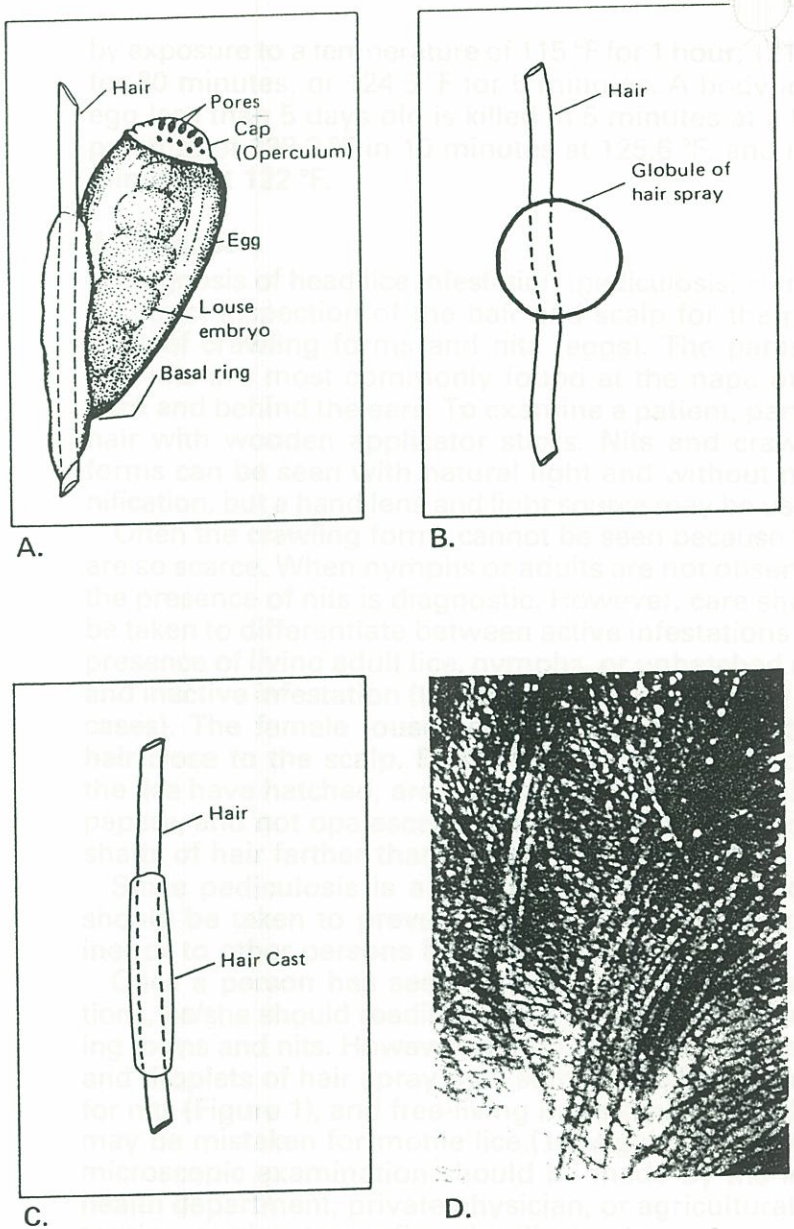

Figure 1. a. Egg of head louse; b. hair spray globule; c. hair cast; d. human hair with louse eggs (nits) attached.