CLINICAL MICROBIOLOGY REVIEWS, Oct. 1989, p. 354–359
0893-8512/89/040354-06$02.00/0
Copyright © 1989, American Society for Microbiology
Vol. 2, No. 4

Erysipelothrix rhusiopathiae: An Occupational Pathogen

ANNETTE C. REBOLI† AND W. EDMUND FARRAR*

Infectious Diseases Division, Medical University of South Carolina, Charleston, South Carolina 29425

INTRODUCTION

The genus *Erysipelothrix* consists of a single species, *Erysipelothrix rhusiopathiae*, formerly known as *Erysipelothrix insidiosa*. It is a thin, pleomorphic, nonsporulating, gram-positive rod first isolated by Koch in 1878 (31). In 1886, it was described by Loeffler (35) as the etiologic agent of swine erysipelas. In 1909, Rosenbach reported isolation of the organism from a patient with localized cutaneous lesions, thus establishing it as a human pathogen (52). He coined the term "erysipeloid" to avoid confusion with the lesions of human erysipelas. Long recognized as an important cause of infection in animals, it is also recognized as a serious pathogen in humans (15, 67). Erysipeloid, a cutaneous infection, is the most common manifestation of human disease, but rare cases of septicemia and endocarditis have been reported.

EPIDEMIOLOGY

E. rhusiopathiae, and infections due to this organism, are worldwide in distribution. It has been found as a commensal or a pathogen in a wide variety of vertebrate and invertebrate species, including "swine, sheep, cattle, horses, dogs, wild bears, kangaroos, reindeer, mice, wild rodents, seals, sea lions, cetaceans, mink, chipmunks, crustaceans, fresh- and saltwater fish, crocodiles, caymen, stable flies, house flies, ticks, mites, mouse lice, turkeys, chickens, ducks, geese, guinea fowl, pigeons, sparrows, starlings, eagles, parrots, pheasants, peacocks, quail, parakeets, mud hens, canaries, finches, siskins, thrushes, blackbirds, turtledoves, and white storks" (5, 6, 14, 21, 61, 67, 69). The major reservoir of *E. rhusiopathiae* is generally believed to be domestic swine, but rodents and birds are also frequently infected. The organism causes no known disease in fish but can grow and persist for long periods of time in the mucoid exterior slime of these animals (67). It now appears that, contrary to previous belief, the organism is not able to exist indefinitely in soil, but it may live long enough to cause infection weeks or months after initial soil contamination. The greatest commercial impact of *E. rhusiopathiae* infection is due to disease in swine, but infection of sheep, turkeys, and ducks is also of economic importance (5). The risk of human infection with *E. rhusiopathiae* is closely related to the opportunity for exposure to the organism (39). Relation to age, sex, race, and socioeconomic status appears to reflect only opportunity for exposure. Most human cases are related to occupational exposure. Individuals at greatest risk for infection include butchers, fishermen, fish handlers, abattoir workers, veterinarians, and housewives (2, 5, 15, 20, 21, 28, 40, 68, 69), but erysipelothrix infection has been associated with a wide variety of occupations, including butchers, meat cutters, meat-processing workers, poultry-processing workers, meat inspectors, rendering-plant workers, knackers, animal caretakers, farmers, fishermen (including lobster fishermen, fish and lobster handlers, fish-processing workers, crab and crayfish-processing workers, and clam openers), veterinarians (including veterinary students), cooks, bakers, housewives, kitchen workers, food handlers, caterers, button (bone) makers, game handlers, furriers, leather workers, soap makers, fertilizer workers, sewer workers, bacteriologists laboratory workers, and stockyard workers (67). Infection is especially common among individuals who handle fish. Hunter (21) noted the occurrence of fish-handlers' disease among "fishermen, fish cleaners, gutters and picklers, fish porters, fish-box repairers, fish-lorry drivers, fish-meal workers, smoke driers, fish mongers, cooks and housewives who infect themselves through abrasions of the skin caused by the spines, fins, and bones of fish, especially skate." During World War II, outbreaks of the disease occurred in factories in Norway where fish was dried and tinned and cod heads were made into fertilizer; the delay caused by the fishing boats having to sail in convoy apparently allowed the organisms to multiply, resulting in the high incidence of infections (21). Seal finger and whale finger occur in those who capture these animals and scratch their hands on the steel ropes used in their work

* Corresponding author.
† Present address: Infectious Diseases Division, Hahnemann University, Philadelphia, PA 19102.

(20). Anglers may be infected through puncture wounds made by fish hooks or the teeth of fish or by the claws of lobsters and crabs; in the series of 329 cases of erysipeloid described by Gilchrist, 323 were caused by injuries produced by crabs (13). Abattoir workers, meat porters, butchers, and poulterers may become infected through small cuts from the knives they use or through abrasions caused by splinters of bone (pork finger). Veterinary pathologists may become accidentally inoculated by injury from knives or bone splinters during necropsy of infected animals, particularly poultry. Workers who peel potatoes and other root vegetables may become infected by earth contaminated with the manure of infected animals.

Most cases in humans and other animals probably occur via scratches or puncture wounds of the skin, but in some cases it appears that the organism has penetrated intact skin (39). Human-to-human infection has not been documented. Although *E. rhusiopathiae* is killed by moist heat at 55°C for 15 min, it is resistant to many environmental influences, including salting, pickling, and smoking (5). Meat and bacon may contain the organism after pickling for 170 days or after 30 days in a mixture of salt and potassium nitrate, and the organism has been recovered from smoked ham. It may remain alive for 12 days in direct sunlight and for many months in carcasses left to decay on the surface of the ground or buried as deep as 7 feet (213.36 cm). It has also been found in city sewage containing drainage from abattoirs and stables.

CLINICAL MANIFESTATIONS OF DISEASE

Four clinical entities have been described in swine: (i) an acute septic form, (ii) a subacute urticarial form marked by reddish-purple rhomboid spots or "diamonds" in the skin, (iii) a joint or arthritic form, and (iv) a chronic cardiac form (endocarditis) (14, 29, 68, 69). The spectrum of disease seen in humans closely parallels that seen in swine (17, 27, 48, 57). There are three well-defined clinical categories of human disease: (i) a localized cutaneous form, erysipeloid; (ii) a generalized cutaneous form; and (iii) a septicemic form which is often associated with endocarditis (17).

Localized Cutaneous Form or Erysipeloid of Rosenbach

Erysipeloid is a localized skin infection which is actually a cellulitis. Because of its mode of acquisition (i.e., contact with infected animals, fish, or their products, with organisms gaining entrance via cuts or abrasions on the skin), lesions are usually confined to the fingers and hands. The patient complains of pain and swelling of the finger or part of the hand. The pain is often severe and may be described as a burning, throbbing, or itching sensation. A history may be elicited of a scratch or wound of the infected part by a bone or knife contaminated by animal secretions approximately 5 to 7 days or, at most, 2 weeks prior to the onset of symptoms (50). The infected area is swollen. The lesion consists of a well-defined, slightly elevated, violaceous zone which spreads peripherally as discoloration of the central area fades (26). Systemic effects are uncommon. Low-grade fever and arthralgias occur in approximately 10% of cases, and lymphangiitis and lymphadenopathy occur in approximately one-third (44). There may be arthritis of an adjacent joint. Vesicles may be present, but suppuration does not occur. The absence of suppuration along with the violaceous color, lack of pitting edema, and disproportionate pain help to distinguish erysipeloid from staphylococcal or streptococcal infection. Erysipeloid is a self-limited condition, the lesions usually resolving without therapy within 3 or 4 weeks.

Diffuse Cutaneous Form

Diffuse cutaneous form constitutes a rare situation in which the violaceous cutaneous lesion progresses proximally from the site of inoculation or appears at remote areas (8, 17). Bulla formation may occur. The patients often have systemic manifestations such as fever and joint pains, but blood cultures are negative. The clinical course is much more protracted than in the localized disease form, and recurrences are not uncommon. In one instance, a butcher who ate sausage from a pig slaughtered because of swine erysipelas developed widespread urticaria with the rhomboid pattern characteristic of swine erysipelas (21).

Septicemia and Endocarditis

Systemic *E. rhusiopathiae* infection is uncommon. It rarely develops from localized infection. No cases of systemic disease were seen among the 500 cases of erysipeloid described by Nelson (44) or among the 329 cases reported by Gilchrist (13). Fifty cases of systemic *E. rhusiopathiae* infection have been reported, with a very high incidence of endocarditis (90%) among them (W. D. Alexander and C. S. Goodwin, Letter, Br. Med. J. **1**:804, 1973; 1–4, 10, 16–18, 22, 30, 32–34, 36–38, 41, 42, 45–47, 49, 51, 53–55, 58, 59, 64). All reported cases of endocarditis, except one recent case of infection involving a Starr-Edwards prosthetic aortic valve (16), have involved native valves. Some 36% of patients had a history of an antecedent skin lesion or a concurrent characteristic skin lesion of erysipeloid (15). Gorby and Peacock (15) compared clinical features of *E. rhusiopathiae* endocarditis with those of endocarditis caused by other bacteria (25). They found a higher male/female ratio (which probably reflects occupational exposure), a greater propensity for involvement of the aortic valve, and a much higher (38%) mortality rate among patients with *E. rhusiopathiae* endocarditis. There was more prior heart disease among those with endocarditis caused by other organisms (25). In nearly 60% of patients, *E. rhusiopathiae* endocarditis apparently developed on previously normal heart valves (15). The clinical picture with respect to fever, peripheral skin stigmata of endocarditis, emboli, splenomegaly, hematuria, and mycotic aneurysm was similar for the two groups (15, 25). Very few cases of endocarditis have occurred in immunocompromised patients, but a history of alcohol abuse was present in 33%. The presentation is most often subacute but may be acute (15, 54, 59). Ognibene et al. reported the first case of septic shock associated with this organism (46). The most common complication of endocarditis, congestive heart failure, was present in approximately 80% of patients (10, 17, 18, 32, 38, 53). Myocardial abscesses and aortic valve perforation have been reported (11, 19, 32, 36, 41, 53). Over one-third of patients required valve replacement (15). Diffuse glomerular nephritis and meningitis have also been reported as complications (58, 59).

Other Infections

Osseous necrosis of the thumb has been reported in a patient who developed fatal endocarditis (30), and Torkildsen (63) described a case of intracranial abscess. Chronic arthritis has been reported in a few cases in Europe (8).

PATHOGENESIS AND PATHOLOGY

Very little is known about the pathogenesis of infection in humans. *E. rhusiopathiae* produces a hyaluronidase and a

neuraminidase. The level of activity of these enzymes may correlate with virulence. The neuraminidase may have a role in the pathogenesis of arteritis and thrombocytopenia in rats experimentally infected with *E. rhusiopathiae* (43, 56).

Intravenous injection of the organism into rabbits is fatal in 2 to 3 days. An erysipeloid rash develops in the injected ear. The lungs become hemorrhagic and a pericardial exudate develops. Congestion of the viscera is noted, with pinpoint focal necrosis in the liver and mononuclear cell infiltrates in the spleen (60). On histopathologic examination, bacilli are scarce. Inoculation of the conjunctiva produces conjuctivitis, which is often followed by fatal disseminated infection. Subcutaneous injections seldom cause death.

The rhomboidal skin lesions described in swine are the result of thrombotic vasculitis of end arterioles (17). Injection of the organism into swine produces an inflammatory polyarthritis, lymphadenopathy, endocarditis, peripheral monocytosis, and focal necrosis of the liver and myocardium (60). The endocarditis in swine usually involves the mitral valve, and there is a tendency for the vegetations to invade the mural endocardium (53). In human cases of septicemia and endocarditis, pathological changes are indistinguishable from changes caused by other bacterial organisms.

Swine arthritis bears some similarities to human rheumatoid arthritis (17, 48, 57). It is marked by pannus formation with destruction of cartilage at the site of pannus attachment, intra-articular fibrous adhesions, and subchondral cellular reaction (17, 57).

BACTERIOLOGY

Morphology and Growth

E. rhusiopathiae is a straight or slightly curved, thin, rod-shaped organism which is 0.2 to 0.4 μm in diameter and 0.8 to 2.5 μm in length. Organisms are arranged singly, in short chains, or in pairs in a "V" configuration or are grouped randomly. Filaments and long chains are sometimes seen. Nonencapsulated, nonsporulating, and nonmotile, it is gram positive but may appear gram negative because it decolorizes readily. It is not acid fast.

Growth occurs at temperatures ranging from 5 to 44°C, with an optimal temperature of 30 to 37°C, and at a pH of 7.2 to 7.6 (range, 6.8 to 8.2). It is a facultative anaerobe. Growth is improved by 5 to 10% carbon dioxide.

On blood agar it may be α-hemolytic but is never beta-hemolytic. There is a dual colonial and microscopic appearance. After growing for 24 h at 37°C, colonies are small, circular, and transparent, with a smooth glistening surface and edge. These are smooth or S forms. Larger flatter colonies with a matte surface and fimbriated edge are R-form or rough colonies. Both forms are usually light blue in color or sometimes green. Intermediate forms are also seen. S-form colonies dissociate to give rise to intermediate and R-form colonies. R-form colonies also give rise to S forms (23). In broth, S-form organisms cause a slight turbidity and a powdery deposit; R forms have a tangled hairlike appearance. Microscopically, S-form organisms are 0.3 to 0.6 μm by 0.8 to 2.5 μm, while R-form organisms form long nonbranching filaments which can be >60 μm in length.

Morphology varies with the medium, pH, and temperature of incubation. Acidic pH and temperature of 37°C favor R forms (17). Alkaline pH (7.6 to 8.2) and temperature of 30°C favor S forms. S-form organisms are seen in smears from blood and tissue in acute forms of illness such as sepsis; R forms are seen in more chronic conditions such as endocarditis or arthritis (9).

E. rhusiopathiae is catalase and oxidase negative. Growth is improved by blood or serum, tryptophan, and glucose. The best growth occurs in 0.1% glucose broth or 0.5% glucose agar. Larger amounts of glucose may be inhibitory (60). Glucose metabolism is via the Embden-Meyerhof pathway, with a small amount by the hexose monophosphate shunt. Fermentative activity is weak (60). In addition to lactic acid, small amounts of acetic acid, formic acid, and ethyl alcohol are produced. Acid without gas is produced within 48 h from glucose, lactose, fructose, and galactose. Maltose fermentation produces acid in 6 to 7 days. Xylose, mannitol, and sucrose are not fermented.

This genus is indole, Voges-Proskauer and methyl red negative (61). There is no discoloration of methylene blue milk and little or no change in litmus milk. The majority of strains produce hydrogen sulfide, but results can vary with the medium used. This is a very important reaction. It is best carried out on triple sugar iron agar slants, on which hydrogen sulfide causes a blackened butt. Gelatin stab cultures yield a very characteristic pattern of growth described as a "test tube brush" or a "pipe cleaner" (23, 65). After 24 h, growth is faint and hazy and limited to an area just below the surface. Within a few days, however, growth extends in a column to the bottom of the tube. There is no liquefaction of the gelatin. S forms produce fine horizontal outgrowths which extend only 2 to 3 mm from the stab. R forms extend further out. This test is not convenient for most laboratories to do since the gelatin must be incubated at 22 to 25°C to maintain its solid state. Furthermore, this test is not required for identification.

Specimen Collection, Transport, and Maintenance

Routine blood culture techniques are adequate for specimen collection and organism growth in suspected cases of sepsis or endocarditis. Because organisms are located only in deeper parts of the skin in cases of erysipeloid, aspirates or biopsy specimens from the edge of the lesion are needed to obtain the organism. Biopsies should be of the entire thickness of the dermis. Immediately after collection, the specimen should be put into an infusion broth of 1% glucose and kept at room temperature or refrigerated until it reaches the laboratory.

Cultures can be maintained for several months by stab inoculation into tubes of nutrient agar (pH 7.4) (23). Freeze-drying or freezing in glass beads at −70°C is appropriate for long-term maintenance.

Isolation and Identification

Commercially available blood culture media are satisfactory for primary isolation from blood since *E. rhusiopathiae* is not particularly fastidious. Biopsy specimens or tissue aspirates from the skin lesions should be put into an infusion broth of 1% glucose and incubated aerobically in 5 to 10% carbon dioxide at 35 to 37°C (65). At 24-h intervals, subcultures to blood agar plates should be made. Use of selective media is not necessary unless the specimen is heavily contaminated. Many selective media have been described, including a nutrient broth containing horse serum, kanamycin, neomycin, and vancomycin and a tryptose blood agar containing crystal violet and sodium azide (23, 66). Another selective liquid enrichment medium for *E. rhusiopathiae* contains kanamycin, crystal violet, sodium azide, and liquefied phenol (9). A solid medium which is a modification of this contains water blue and sucrose (9). Since *E. rhusio-*

pathiae does not ferment sucrose, colonies appear colorless on the water blue-sucrose agar. In cases of chronic infection in which the number of bacteria is small, enrichment by the addition of horse, calf, or swine serum in broth and incubation for longer than 10 days may be necessary. Identification of the organism is based on the results of Gram stain, lack of motility, hydrogen sulfide production, indole production, catalase activity, growth on agar containing potassium tellurite, and hemolysis on blood agar. The result of any one test is insufficient for identification. *E. rhusiopathiae* will need to be differentiated from other gram-positive bacilli, in particular, from *Actinomyces* (*Corynebacterium*) *pyogenes* and *Arcanobacterium* (*Corynebacterium*) *haemolyticum*, and from *Listeria monocytogenes*. The first two organisms are beta-hemolytic on blood agar and do not produce hydrogen sulfide in the butt of triple sugar iron agar slants. *L. monocytogenes* is catalase positive and motile. The neomycin susceptibility test can be used to distinguish *E. rhusiopathiae* from *L. monocytogenes*, the former being resistant to neomycin and the latter being susceptible (12). *E. rhusiopathiae* has occasionally been misidentified as a viridans streptococcus (15, 51). It has also been dismissed as a contaminant (22).

The "mouse protection test" is considered the best method for confirming an isolate as *E. rhusiopathiae* (23). In this test, a subcutaneous injection of organisms from an 18- to 24-h broth culture of the suspected *E. rhusiopathiae* is administered to mice along with a dose of equine hyperimmune *E. rhusiopathiae* antiserum. A control group of mice is injected with the broth culture but not the antiserum. If the organism is *E. rhusiopathie*, the mice that did not receive antiserum die in 5 to 6 days, but those receiving antiserum are protected (65). The test detects only those strains which are virulent for mice, but since most strains are virulent, it is a good confirmatory test. One may also inject suspect clinical material subcutaneously into mice and isolate the organism from the kidneys or spleen when they die a few days later (9).

E. rhusiopathiae has heat- and acid-stable, type-specific antigens and heat-labile, species-specific antigens (17, 23, 24). Strains may be identified serologically. The recommended method for serologic investigation is the double agar-gel diffusion precipitation test (23). Most virulent strains causing acute infection belong to serovar A. Strains of serovar B have been isolated primarily from chronic cases (9). Different serotyping schemes have been proposed, but the numbered system of Kucsera is preferred over the older alphabetical system (23). A and B correspond to 1 and 2. These are the most common of the 22 serovars (23).

Direct and indirect fluorescent-antibody tests are also available in lieu of the mouse protection test to confirm identification as *E. rhusiopathiae* (7, 65). In general, serological tests are not practical for routine use in a clinical laboratory for identification of the organism or detection of antibody in patient sera.

TREATMENT

The mainstay of treatment of infections caused by *E. rhusiopathiae* is antibiotic therapy. Although skin lesions usually heal spontaneously within 4 weeks, second attacks may occur and lesions may persist for months. Healing is hastened by antibiotic therapy. Susceptibility data are limited. Most strains are highly susceptible to penicillins, cephalosporins, erythromycin, and clindamycin (15, 19, 49, 61, 62). Minimal inhibitory concentrations of penicillins have been reported to range from 0.0025 to 0.06 μg/ml, with minimal bactericidal concentrations of 0.0025 to 0.75 μg/ml (15). Susceptibility to chloramphenicol and tetracycline is variable. Most strains are resistant to sulfonamides, trimethoprim-sulfamethoxazole, aminoglycosides, vancomycin, novobiocin, and polymyxins. They are also resistant to 0.2% phenol, 0.1% sodium azide, and crystal violet (23). Resistance to vancomycin is noteworthy because this agent is often used in empiric therapy of prosthetic valve endocarditis and in the treatment of native valve endocarditis due to gram-positive organisms in individuals who are allergic to penicillins.

Penicillin G, in doses of 12×10^6 to 20×10^6 U/day, is the drug of choice for serious infections caused by *E. rhusiopathiae* (49). Recommended duration of therapy for endocarditis is 4 to 6 weeks, although shorter courses consisting of 2 weeks of intravenous therapy followed by 2 to 4 weeks of oral therapy have been successful (1, 42, 46). There has been no reported experience in the treatment of penicillin-allergic patients. Cephalosporins are the most appropriate alternatives since both clindamycin and erythromycin are only bacteriostatic agents. Valve replacement has been necessary in about one-third of cases of endocarditis.

THE FUTURE

Reporting of infections due to *E. rhusiopathiae* is not required by health authorities, so it is difficult to know whether the incidence of these infections is increasing or decreasing. Some technologic changes in industries that use animal products have probably resulted in reduced contact between *E. rhusiopathiae* and humans. For example, nearly all buttons are now made of plastic, rather than bone. To the extent that such changes reduce occupational exposure to the organism, the future incidence of erysipeloid, and more serious forms of infection with *E. rhusiopathiae*, will decline.

LITERATURE CITED

1. **Baird, P. J., and R. Benn.** 1975. Reports of cases: erysipelothrix endocarditis. Med. J. Aust. **2**:743–745.
2. **Berg, R. A.** 1984. *Erysipelothrix rhusiopathiae*. South. Med. J. **77**:1614.
3. **Blount, J. G.** 1965. Bacterial endocarditis. Am. J. Med. **38**: 909–922.
4. **Borchardt, K. A., R. W. Sullivan, R. S. Blumberg, R. H. Gelber, V. Botch, S. Crull, and D. J. Ullyot.** 1977. *Erysipelothrix rhusiopathiae* endocarditis. West. J. Med. **127**:149–151.
5. **Conklin, R. H., and J. H. Steele.** 1979. Erysipelothrix infections, p. 327–337. *In* J. H. Steele (ed.), CRC handbook. Series in zoonoses, vol. 1, sect. A. CRC Press, Inc., Boca Raton, Fla.
6. **Creech, G. T.** 1921. The bacillus of swine erysipelas isolated from urticarial lesions of swine in the United States. J. Am. Vet. Med. Assoc. **59**:139–150.
7. **Dacres, W. G., and A. H. Groth, Jr.** 1959. Identification of *Erysipelothrix insidiosa* with fluorescent antibody. J. Bacteriol. **78**:298–299.
8. **Ehrlich, J. C.** 1946. *Erysipelothrix rhusiopathiae* infection in man. Arch. Intern. Med. **78**:565–577.
9. **Ewald, F. W.** 1981. The genus *Erysipelothrix*, p. 1688–1700. *In* M. P. Starr, H. Stolp, H. G. Trüper, A. Balows, and H. G. Schlegel (ed.), The prokaryotes: a handbook on habitats, isolation, and identification of bacteria. Springer-Verlag, New York.
10. **Fliegelman, R. M., R. S. Cohen, and B. Zakhireh.** 1985. *Erysipelothrix rhusiopathiae* endocarditis: report of a case and review of the literature. J. Am. Osteopath. Assoc. **85**:39–42.

11. **Fowler, N. O., M. H. Hamburger, and K. E. Bove.** 1967. Aortic valve perforation. Am. J. Med. **42**:539–546.
12. **Fuzi, M.** 1963. A neomycin sensitivity test for the rapid differentiation of *Listeria monocytogenes* from *Erysipelothrix rhusiopathiae*. J. Pathol. Bacteriol. **85**:524–525.
13. **Gilchrist, T. C.** 1904. Erysipeloid, with a record of 329 cases, of which 323 were caused by crab bites, or lesions produced by crabs. J. Cutan. Dis. **22**:507–519.
14. **Gledhill, A. W.** 1948. Discussion on swine erysipelas (*Erysipelothrix rhusiopathiae*) in man and animals. Proc. R. Soc. Med. **41**:330–332.
15. **Gorby, G. L., and J. E. Peacock, Jr.** 1988. *Erysipelothrix rhusiopathiae* endocarditis: microbiologic, epidemiologic, and clinical features of an occupational disease. Rev. Infect. Dis. **10**:317–325.
16. **Gransden, W. R., and S. J. Eykyn.** 1988. *Erysipelothrix rhusiopathiae* endocarditis. Rev. Infect. Dis. **10**:1228.
17. **Grieco, M. H., and C. Sheldon.** 1970. *Erysipelothrix rhusiopathiae*. Ann. N.Y. Acad. Sci. **174**:523–532.
18. **Heggers, J. P., R. S. Buddington, and H. A. McAllister.** 1974. Erysipelothrix endocarditis diagnosis by fluorescence microscopy: report of a case. Am. J. Clin. Pathol. **62**:803–806.
19. **Heilman, F. R., and W. E. Herrell.** 1944. Penicillin in the treatment of experimental infections due to *Erysipelothrix rhusiopathiae*. Proc. Staff Meet. Mayo. Clin. **19**:340–345.
20. **Hillenbrand, F. K. M.** 1953. Whale finger and seal finger: their relation to erysipeloid. Lancet **i**:680–681.
21. **Hunter, D.** 1974. The diseases of occupations, 5th ed., p. 709–712. The English Universities Press, Ltd., London.
22. **James, O., O. Morgan, M. Adam, and E. Schallibaum.** 1976. *Erysipelothrix insidiosa* endocarditis: a report of two cases in Jamaica. West Indian Med. J. **25**:265–268.
23. **Jones, D.** 1986. Genus *Erysipelothrix* Rosenbach 367[al], p. 1245–1249. *In* P. H. Sneath, N. S. Mair, and M. E. Sharpe (ed.), Bergey's manual of systematic bacteriology, vol. 2. The Williams and Wilkins Co., Baltimore.
24. **Kalf, G. F., and M. A. Grece.** 1964. The antigenic components of *Erysipelothrix rhusiopathiae*. III. Purification of B- and C-antigens. Arch. Biochem. Biophys. **107**:141–146.
25. **Kaye, D. (ed.).** 1976. Infective endocarditis. University Park Press, Baltimore.
26. **King, P. F.** 1946. Erysipeloid. Survey of 115 cases. Lancet **ii**:196–198.
27. **Klauder, J. V.** 1926. Erysipeloid and swine erysipelas in man. J. Am. Med. Assoc. **86**:536–541.
28. **Klauder, J. V.** 1938. Erysipeloid as an occupational disease. J. Am. Med. Assoc. **111**:1345–1348.
29. **Klauder, J. V.** 1944. *Erysipelothrix rhusiopathiae* infection in swine and in human beings: comparative study of the cutaneous lesions. Arch. Dermatol. Syphilol. **50**:151–159.
30. **Klauder, J. V., D. W. Kramer, and L. Nicholas.** 1943. *Erysipelothrix rhusiopathiae* septicemia; diagnosis and treatment. Report of fatal case of erysipeloid. J. Am. Med. Assoc. **122**:938–943.
31. **Koch, R.** 1880. Investigations into the etiology of traumatic infective diseases. New Sydenham Society, London.
32. **Kramer, M. R., M. E. Gombert, M. L. Corrado, M. A. Ertgin, V. Burnett, and J. Ganguly.** 1982. *Erysipelothrix rhusiopathiae* endocarditis. South. Med. J. **75**:892.
33. **Lerner, P. I., and L. Weinstein.** 1966. Infective endocarditis in the antibiotic era. N. Engl. J. Med. **274**:199–206.
34. **Lawes, F. A. E., E. G. Durie, N. E. Goldsworthy, and H. C. Spies.** 1952. Subacute bacterial endocarditis caused by *Erysipelothrix rhusiopathiae*. Med. J. Aust. **1**:330–331.
35. **Loeffler, F. A.** 1886. Experimentelle Untersuchungen uber Schweinerothlauf. Arb. Kais. Gesundheitsamte **1**:46–55.
36. **Mandal, B. N., and J. A. Malloch.** 1971. Endocarditis due to *Erysipelothrix rhusiopathiae*. N. Z. Med. J. **73**:355–357.
37. **McCarty, D., and S. Bornstein.** 1960. Erysipelothrix endocarditis: report on a septicemic form of the erysipeloid of Rosenbach. Am. J. Clin. Pathol. **33**:39–42.
38. **McCracken, A. W., C. U. Mauney, T. W. Huber, and R. V. McCloskey.** 1973. Endocarditis caused by *Erysipelothrix insidiosa*. Am. J. Clin. Pathol. **59**:219–222.
39. **McGinnes, G. F., and F. Spindle.** 1934. Erysipeloid condition among workers in a bone button factory due to the bacillus of swine erysipelas. Am. J. Public Health **24**:32–35.
40. **Morrill, C. C.** 1939. Erysipeloid; occurrence among veterinary students. J. Infect. Dis. **65**:322–324.
41. **Morris, C. A., H. Schwabacher, P. G. Lynch, C. D. Cross, and T. O. Dada.** 1965. Two fatal cases of septicaemia due to *Erysipelothrix insidiosa*. J. Clin. Pathol. **18**:614–617.
42. **Muirhead, N., and T. M. S. Reid.** 1980. *Erysipelothrix rhusiopathiae* endocarditis. J. Infect. **2**:83–85.
43. **Nakato, H., K. Shinomiya, and H. Mikawa.** 1986. Possible role of neuraminidase in the pathogenesis of arteritis and thrombocytopenia induced in rats by *Erysipelothrix rhusiopathiae*. Pathol. Res. Pract. **181**:311–319.
44. **Nelson, E.** 1955. Five hundred cases of erysipeloid. Rocky Mountain Med. J. **52**:40–42.
45. **Normann, B., and E. Kihlstrom.** 1985. *Erysipelothrix rhusiopathiae* septicaemia. Scand. J. Infect. Dis. **17**:123–124.
46. **Ognibene, F. P., R. E. Cunnion, V. Gill, J. Ambrus, A. S. Fauci, and J. E. Parrillo.** 1985. Erysipelothrix rhusiopathiae bacteremia presenting as septic shock. Am. J. Med. **78**:861–864.
47. **Park, C. H., D. M. Poretz, and R. Goldenberg.** 1976. *Erysipelothrix* endocarditis with cutaneous lesion. South. Med. J. **69**:1101–1103.
48. **Phillips, P. E.** 1986. Infectious agents in the pathogenesis of rheumatoid arthritis. Semin. Arthritis Rheum. **16**:1–10.
49. **Poretz, D. M.** 1985. *Erysipelothrix rhusiopathiae*, p. 1185–1186. *In* G. L. Mandell, R. G. Douglas, Jr., and J. E. Bennett (ed.), Principles and practice of infectious diseases, 2nd ed. J. Wiley & Sons, Inc., New York.
50. **Price, J. E. L., and W. E. J. Bennett.** 1951. The erysipeloid of Rosenbach. Br. Med. J. **2**:1060–1062.
51. **Procter, W. I.** 1965. Subacute bacterial endocarditis due to *Erysipelothrix rhusiopathiae*. Am. J. Med. **38**:820–824.
52. **Rosenbach, F. J.** 1909. Experimentalle morphologische und klinische Studie uber die krankheitserregenden Mikroorganismen des Schweinerotlaufs, des Erysipeloids und der Mausesepsis. Z. Hyg. Infektionskr. **63**:343–369.
53. **Russell, W. O., and M. E. Lamb.** 1940. Erysipelothrix endocarditis: a complication of erysipeloid. J. Am. Med. Assoc. **114**:1045–1050.
54. **Schiffman, W. L., and A. Black.** 1956. Acute bacterial endocarditis caused by *Erysipelothrix rhusiopathiae*. N. Engl. J. Med. **255**:1148–1150.
55. **Scully, R. E., J. J. Galdabini, and B. U. McNeely.** 1978. Weekly clinicopathological exercises. Case 16-1978. Case Records Massachusetts General Hospital. N. Engl. J. Med. **298**:957–962.
56. **Shinomiya, K., and H. Nakato.** 1985. An experimental model of arteritis: periarteritis induced by Erysipelothrix rhusiopathiae in young rats. Int. J. Tissue React. **7**:267–271.
57. **Sikes, D.** 1958. A comparison of rheumatoid-like arthritis in swine and rheumatoid arthritis in man. Ann. N.Y. Acad. Sci. **70**:717–725.
58. **Silberstein, E. B.** 1965. *Erysipelothrix* endocarditis: report of a case with cerebral manifestations. J. Am Med. Assoc. **191**:862–864.
59. **Simberkoff, M. S., and J. J. Rahal, Jr.** 1973. Acute and subacute endocarditis due to *Erysipelothrix rhusiopathiae*. Am. J. Med. Sci. **266**:53–57.
60. **Smith, G.** 1983. Erysipelothrix and Listeria, p. 50–59. *In* G. Wilson, A. Miles, and M. T. Parker (ed.), Topley and Wilson's principles of bacteriology, virology and immunity, 7th ed. The Williams & Wilkins Co., Baltimore.
61. **Sneath, P. H. A., J. D. Abbott, and A. C. Cunliffe.** 1951. The bacteriology of erysipeloid. Br. Med. J. **2**:1063–1066.
62. **Stiles, G. W.** 1947. Chronic erysipeloid (swine erysipelas) in a man. The effect of treatment with penicillin. J. Am. Med. Assoc. **134**:953–955.
63. **Torkildsen, A.** 1943. Intracranial erysipelothrix abscess. Bull. Hyg. **18**:1013–1015.
64. **Townshend, R. H., A. E. Jephcott, and M. H. Yekta.** 1973. Erysipelothrix septicaemia without endocarditis. Br. Med. J. **1**:464.

65. **Weaver, R. E.** 1985. Erysipelothrix, p. 209–210. *In* E. H. Lennette, A. Balows, W. J. Hausler, Jr., and H. J. Shadomy (ed.), Manual of clinical microbiology, 4th ed. American Society for Microbiology, Washington, D.C.
66. **Wood, R. L.** 1965. A selective liquid medium utilizing antibiotics for isolation of *Erysipelothrix insidiosa*. Am. J. Vet. Res. **26**:1303–1308.
67. **Wood, R. L.** 1975. Erysipelothrix infection, p. 271–281. *In* W. T. Hubbert, W. F. McCullough, and P. R. Schnurrenberger (ed.), Diseases transmitted from animals to man, 6th ed. Charles C Thomas, Publisher, Springfield, Ill.
68. **Wood, R. L., and R. D. Shuman.** 1974. Swine erysipelas, p. 565–620. *In* H. W. Dunne and A. D. Leman (ed.), Diseases of swine, 4th ed. Iowa State University Press, Ames.
69. **Woodbine, M.** 1950. *Erysipelothrix rhusiopathiae*. Bacteriology and chemotherapy. Bacteriol. Rev. **14**:161–178.