

Microbiology of Animal Bite Wound Infections

Fredrick M. Abrahamian^{1,2*} and Ellie J. C. Goldstein^{1,3*}

David Geffen School of Medicine at UCLA, Los Angeles, California¹; Department of Emergency Medicine, Olive View-UCLA Medical Center, Sylmar, California²; and R. M. Alden Research Laboratory, Culver City, California³

INTRODUCTION	231
MICROBIOLOGY	232
Mammals	232
Dogs	232
(i) Aerobes	232
(ii) Anaerobes	234
Cats	234
(i) Aerobes	235
(ii) Anaerobes	236
Rare infectious diseases in humans acquired from cats and dogs	236
(i) Cats	236
(ii) Dogs	236
(iii) Cats and dogs	236
Monkeys/simians	236
Bears	237
Pigs	238
Ferrets	238
Horses	238
Sheep	239
Tasmanian devils	239
Reptiles	239
Snakes	239
Komodo dragon	240
Monitor lizards	241
Iguana	241
Alligators/crocodiles	241
Rodents	241
Rats	241
Guinea pigs	241
Hamsters	241
Prairie dogs	241
Birds	241
Swans	241
Sharks	242
SUMMARY AND FUTURE DIRECTIONS	242
REFERENCES	242

INTRODUCTION

The sustained and worldwide epidemic of animal bite injuries and infections has been the subject of many scholarly reviews, and there has been a plethora of series and case reports but very few systematic studies. Since human-animal contact is a daily occurrence for most people worldwide in various settings, from farms to domestic pets to feral animals,

it is not surprising that as a result of this contact, bite injuries are caused by a wide variety of domestic and wild animals. Most of these wounds are minor injuries and go unreported, and patients self-administer first aid and often do not seek or require medical attention. In industrialized countries, most patients with moderate to severe bite injuries will seek some form of medical attention whether in an emergency department or in a physician's office. When these injuries are reported, the authors of the reports generally concentrate on unusual or resistant organisms or unusual complications and their management. These retrospective incidents form the basis for anecdotal medical decision-making that is employed worldwide.

Very few studies have been systematic and have attempted to define the presentation, epidemiology, bacteriology, and/or therapy of bite wounds. However, even these systematic studies

* Corresponding author. Mailing address for Fredrick M. Abrahamian: Department of Emergency Medicine, Olive View-UCLA Medical Center, 14445 Olive View Drive, North Annex, Sylmar, CA 91342. Phone: (818) 364-3112. Fax: (818) 364-3268. E-mail: fmasjc@ucla.edu. Mailing address for Ellie J. C. Goldstein: R. M. Alden Research Laboratory, 2021 Santa Monica Blvd., Suite 740 E, Santa Monica, CA 90404. Phone: (310) 315-1511. Fax: (310) 315-3662. E-mail: ejcgmd@aol.com.

are generally limited to dog or cat bites and involve relatively small numbers of patients, and one must often extrapolate the best form of antimicrobial therapy to employ. One principle that has emerged and is scientifically established is that the bacteria recovered from bite wounds are reflective of the oral flora of the biting animal. In a minority of cases the pathogenic bacteria come from the victim's own skin, often as secondary invaders, or from the physical environment at the time of injury. Thus, bites by aquatic animals have a bacteriology that is reflective of their water environment. A second established principle is that the oral flora of the biting animal not only contains the usual "normal flora" but also is influenced by the microbiome of their ingested prey and other foods. Some of these isolates may be transient, while others are persistent and remain. Most individual elements of bite wound care come from localized "standards of care" or general applications of wound care principles but have never been further validated.

In an attempt to make some sense of the status of the bacteriology of animal bite wounds in humans, excluding human bites, we have attempted to review the literature using computer-assisted database searches, references from cited articles, and personal experience. By no means have we found or cited every reference, and there must be a variety of other animal bite wounds reported that we have not included. Our hope is that readers will reply with correspondence to fill in any voids in our review.

MICROBIOLOGY

Mammals

Dogs. The microbiology of infected bite wounds from dogs is similar to that of the organisms that colonize the dog's oral cavity (7, 23, 85, 90, 91, 93, 96, 192, 195). Less frequently, isolates may also come from the environment and patients' skin. Both clinically infected and early-presenting (less than 8 h postinjury and/or not yet clinically infected) dog bite wounds are polymicrobial, with a broad combination of aerobic and anaerobic microorganisms (23, 91, 92, 96, 171, 206).

The clinical presentation and microbiology of clinically infected dog bite wounds in patients without prior antibiotic exposure were studied by Talan et al. (206). This was a multicenter, prospective trial of 50 dog bite wounds, of which 60% were punctures, 10% were lacerations, and 30% were a combination of both. A majority of the infections were purulent wounds without abscess formation (58%), followed by nonpurulent wounds with cellulitis, lymphangitis, or both (30%) and abscesses (12%). An abscess was defined as a fluctuant lesion that required incision and drainage for treatment.

The numbers of bacterial isolates varied depending on the type of wound. The highest numbers of bacterial isolates obtained from cultures were from abscesses (median, 7.5; range, 2 to 11; interquartile range, 3 to 10), followed by purulent wounds (median, 5.0; range, 0 to 16; interquartile range, 2 to 11) and nonpurulent wounds (median, 2.0; range, 0 to 9; interquartile range, 1 to 4). A majority (48%) of the dog bite wounds were polymicrobial, with a mix of aerobic and anaerobic organisms. Pure aerobic growth occurred in 42% of the cases, and only one case among the abscess group demonstrated pure anaerobic growth with *Bacteroides tectus* and *Por-*

TABLE 1. Common aerobic and anaerobic bacterial genera isolated from 50 infected dog bite wounds^a

Bacterial genus	Frequency (%)
Aerobic organisms	
<i>Pasteurella</i>	50
<i>Streptococcus</i>	46
<i>Staphylococcus</i>	46
<i>Neisseria</i> ^b	32
<i>Corynebacterium</i>	12
<i>Moraxella</i>	10
<i>Enterococcus</i>	10
<i>Bacillus</i>	8
Anaerobic organisms	
<i>Fusobacterium</i>	32
<i>Porphyromonas</i>	28
<i>Prevotella</i>	28
<i>Propionibacterium</i>	20
<i>Bacteroides</i> ^b	18
<i>Peptostreptococcus</i>	16

^a Based on data from reference 206.

^b The frequencies differ from the actual numbers cited in the reference due to the reclassification of some of the isolates since its publication.

phyromonas gulae (previously *Porphyromonas gingivalis*).

With respect to the type of wound, mixed aerobic and anaerobic infections were most common among abscesses (67%), followed by purulent wounds (62%) and nonpurulent wounds (13%). Pure aerobic growth was seen most commonly in nonpurulent wounds but with associated cellulitis/lymphangitis (67%), followed by purulent wounds (34%) and abscesses (17%). No growth on culture media occurred for only four patients (8%). For three of these patients, their wounds were described as being a nonpurulent wound with cellulitis, lymphangitis, or both, and the remaining one patient had a purulent wound. No cultures were done for mycobacteria, *Chlamydia*, *Mycoplasma*, or viruses.

(i) Aerobes. Table 1 shows common aerobic bacterial genera isolated from 50 infected dog bite wounds (206). In this multicenter study, the most common aerobic organisms isolated at a research laboratory were *Pasteurella* (50%), *Streptococcus* (46%), *Staphylococcus* (46%), *Neisseria* (32%), and *Corynebacterium* (12%) species. Other aerobic organisms, in order of decreasing frequency, included *Moraxella* species (10%), *Enterococcus* species (10%), *Bacillus* species (8%), *Pseudomonas* species (6%), *Actinomyces* species (6%), *Brevibacterium* species (6%), *Gemella morbillorum* (6%), *Escherichia coli* (6%), *Weeksella zoohelcum* (4%), *Klebsiella* species (4%), *Lactobacillus* species (4%), *Citrobacter* species (4%), *Flavobacterium* species (4%), *Micrococcus* species (4%), *Proteus mirabilis* (4%), *Stenotrophomonas maltophilia* (4%), *Capnocytophaga ochracea* (2%), *Eikenella corrodens* (2%), *Flavimonas oryzihabitans* (2%), *Dermabacter hominis* (2%), *Oerskovia* species (2%), *Pediococcus damnosus* (2%), and *Stomatococcus mucilaginosus* (2%).

Among members of the genus *Pasteurella*, *Pasteurella canis* was the most common species isolated from infected dog bite wounds (26%) (206). Others, in order of decreasing frequency, included *Pasteurella multocida* subsp. *multocida* (12%), *Pasteurella stomatis* (12%), *Pasteurella multocida* subsp. *septica* (10%), *Pasteurella dagmatis* (4%), and *Pasteurella multocida*

subsp. *gallicida* (2%). *Pasteurella multocida* subsp. *multocida* and subsp. *septica* have been implicated in more serious systemic infections, with *Pasteurella multocida* subsp. *septica* having a tendency to result in central nervous system infections (15, 91, 92, 115, 183). In comparison to staphylococci or streptococci, the isolation of *Pasteurella* species is commonly associated with a shorter latency period, which is defined as the time from the bite to the appearance of the first symptoms of infection (1, 206, 212, 230). Other observations have included finding *Pasteurella* more frequently in abscesses, nonpurulent wounds with lymphangitis, puncture wounds, and bites involving the upper extremities than from bites involving the lower extremities (206).

Although *Pasteurella* has been recovered from many other animals (10, 19, 25, 76, 87, 122, 136, 148, 237), including birds (32, 110), in a study of 146 human infections with *Pasteurella* species, *Pasteurella canis* (biotype 1) was associated exclusively with dog bites (115). The higher prevalence of *Pasteurella canis* in dogs was also observed in a study of *Pasteurella* strains involving various animal hosts (15). Some species of *Pasteurella* are capable of producing toxins, such as *Pasteurella multocida* subsp. *multocida* and *Pasteurella canis* (115, 167); however, the role of toxin production, the mechanism of action in the pathogenicity of human diseases, and its clinical relevance are not clearly understood. Toxin-producing *Pasteurella* strains have been reported for minor wound infections (115).

The second most common species isolated from dog bite wounds in equal frequencies were *Streptococcus* and *Staphylococcus* species (206). The prevalence of these two species was notably more common in nonpurulent wounds with cellulitis/lymphangitis than in abscesses or purulent wounds (206). Among members of the genus *Streptococcus*, the most common species isolated was *Streptococcus mitis* (22%). Other species, in order of decreasing frequency, included *Streptococcus mutans* (12%), *Streptococcus pyogenes* (12%), *Streptococcus sanguis* biotype II (8%), *Streptococcus intermedius* (6%), *Streptococcus constellatus* (4%), *Streptococcus equinus* (2%), *Streptococcus sanguis* biotype I (2%), *Streptococcus agalactiae* (2%), *Streptococcus sanguis* (2%), beta-hemolytic *Streptococcus* group G (2%), and *Streptococcus dysgalactiae* (2%).

Among staphylococci, *Staphylococcus aureus* was the most common species isolated (20%), followed by *Staphylococcus epidermidis* (18%) and *Staphylococcus warneri* (6%). Other less commonly isolated staphylococci included *Staphylococcus intermedius* (2%), *Staphylococcus hominis* (2%), *Staphylococcus auricularis* (2%), *Staphylococcus cohnii* (2%), and *Staphylococcus xylosum* (2%). β -Lactamase production is common among strains of staphylococci isolated from infected bites (206).

Canine gingival cultures were obtained from 135 healthy dogs at an urban veterinary clinic in Santa Monica, CA, 10% of which grew *Staphylococcus aureus* and were associated with dogs of working breeds with weights greater than 40 lb and outdoor habitats (209). In that same study, *Staphylococcus intermedius* was isolated in 39% of cultures and was associated with dogs from nonworking breeds with weights less than 40 lb and indoor habitats. *Staphylococcus aureus* and *Staphylococcus intermedius* were concomitantly isolated in only three dogs (2%) (209).

Staphylococcus intermedius may mistakenly be identified as *Staphylococcus aureus*, since both are coagulase positive (206, 208). Its features that distinguish it from *Staphylococcus aureus*

include its β -galactosidase activity and the lack of acetoin production (209). *Staphylococcus intermedius* also demonstrates a high level of phenotypic similarity to *Staphylococcus pseudintermedius* (8, 58). Genetically, it was shown that the *Staphylococcus intermedius* group consists of three distinct species, *Staphylococcus intermedius*, *Staphylococcus pseudintermedius*, and *Staphylococcus delphini* (8). The distinction among these species is difficult with routine diagnostic bacteriology, and confirmation requires molecular techniques (8). The discovery of the *Staphylococcus intermedius* group and its three distinct species has led to *Staphylococcus pseudintermedius*, not *Staphylococcus intermedius*, becoming a common cause of canine skin infections (8, 57, 75, 80).

In addition, *Staphylococcus intermedius* may also be incorrectly identified as methicillin-resistant *Staphylococcus aureus* (MRSA). Reports of such misidentification have occurred when the penicillin binding protein 2a latex agglutination test was used to detect methicillin resistance (179). *Staphylococcus intermedius* is often susceptible to oxacillin (209); however, reports of oxacillin-resistant strains have been made (111). Conclusive identification may require molecular techniques such as PCR and the demonstration of the absence of the *mecA* gene (179). MRSA-associated infections, commonly linked with orthopedic surgeries, have been reported for dogs and other animals (51, 169, 174, 182, 213). The transmission and carriage of identical strains of MRSA (including Pantone-Valentine leucocidin-positive strains) between humans and dogs have also been demonstrated through genetic and molecular techniques (9, 33, 154, 222, 223). We are not aware of any reports of MRSA isolated from an infected dog bite wound in a human.

Neisseria and *Corynebacterium* species were also commonly found in samples from dog bite wounds (4, 206). The most commonly found *Neisseria* species was *Neisseria weaveri* (14%) (formerly known as CDC group M-5), followed by *Neisseria zoodegmatidis* (10%) (formerly known as EF-4b), *Neisseria animaloris* (6%) (formerly known as EF-4a), and *Neisseria subflava* (2%). Among *Corynebacterium* species, the most commonly isolated species were *Corynebacterium* group G (6%) and *Corynebacterium minutissimum* (4%). Less frequently observed corynebacterial isolates were *Corynebacterium aquaticum* (also known as *Leifsonia aquaticum*), *Corynebacterium jeikeium*, *Corynebacterium afermentans*, *Corynebacterium* group E, and *Corynebacterium pseudodiphtheriticum*. In a recent case report, *Corynebacterium freiburgense* was isolated from a human wound inflicted by a dog (83).

Species that were present $\leq 10\%$ of the time included *Moraxella* isolates not identified to the species level (10%) (isolates could not be identified beyond the genus level), *Enterococcus faecalis* (6%), *Brevibacterium* (6%) (isolates could not be identified beyond the genus level), *Gemella morbillorum* (6%), *Escherichia coli* (6%), *Bacillus firmus* (4%), *Actinomyces viscosus* (4%), *Proteus mirabilis* (4%), *Stenotrophomonas maltophilia* (4%), and *Bergeyella zoohelcum* (4%) (previously known as *Weeksella zoohelcum* and prior to that known as CDC group IIj) (206). *Bergeyella zoohelcum* is likely to be an opportunistic pathogen (202) and has been reported in association with various clinical syndromes, including meningitis, bacteremia, and abscess formation after a dog bite (20, 161, 187).

Less commonly observed ($\leq 2\%$) organisms included

Moraxella catarrhalis, *Enterococcus avium*, *Enterococcus malodoratus*, *Bacillus circulans*, *Bacillus subtilis*, *Pseudomonas aeruginosa*, *Pseudomonas vesicularis*, *Pseudomonas diminuta*, *Actinomyces neuii* subsp. *anitratus*, *Klebsiella oxytoca*, *Klebsiella pneumoniae*, *Lactobacillus lactis*, *Citrobacter amalonaticus*, *Citrobacter koseri*, *Flavobacterium* group IIa, *Flavobacterium brevis*, *Micrococcus lylae*, *Capnocytophaga ochracea*, *Eikenella corrodens*, *Flavimonas oryzihabitans*, *Dermabacter hominis*, *Oerskovia* species, *Pediococcus damnosus*, and *Stomatococcus mucilaginosus* (206).

Capnocytophaga canimorsus has been implicated as a pathogenic agent in a variety of clinical conditions such as septicemia, purpura fulminans, peripheral gangrene, endocarditis, and meningitis following dog bites (116, 139, 158, 166, 178, 189). Although fulminant infections with *Capnocytophaga canimorsus* after a dog bite have been reported for immunocompetent patients (116), it appears that immunocompromised patients (e.g., those who have undergone splenectomy and those with liver disease, etc.) are most susceptible to this type of infection and its complications (139, 166, 178, 189). *Capnocytophaga canimorsus* (formerly known as CDC group DF-2) is a fastidious organism, and growth in blood cultures often takes days (mean, 6 days; range, 1 to 14 days) (139). Growth on blood or chocolate agar is significantly improved when culturing is performed in a CO₂-enriched atmosphere (22, 116). MacConkey or triple-sugar-iron agars do not support *Capnocytophaga canimorsus* growth (22). It has been recommended that horse blood agar plates with Difco blood agar base 2 support the growth of *Capnocytophaga canimorsus* better than chocolate agar (178). Examination of the Gram stain of buffy coat preparations or samples obtained from petechial lesions was suggested to be a useful rapid diagnostic test for the identification of *Capnocytophaga canimorsus* (109, 139, 158).

Less commonly reported than *Capnocytophaga canimorsus*, *Capnocytophaga cynodegmi* (formerly known as CDC group DF-2-like) was also reported to result in infections following dog bites (22, 197). Distinguishing these two organisms from one another is often difficult and requires species-specific molecular-based detection systems (205). Although both *Capnocytophaga canimorsus* and *Capnocytophaga cynodegmi* share several characteristics with *Capnocytophaga* species, they differ genetically from other members of *Capnocytophaga* species (e.g., *Capnocytophaga ochracea*, *Capnocytophaga gingivalis*, and *Capnocytophaga sputigena*) (22). Biochemical reactions useful in differentiating *Capnocytophaga canimorsus* and *Capnocytophaga cynodegmi* from other *Capnocytophaga* species include positive oxidase and catalase reactions with *Capnocytophaga canimorsus* and *Capnocytophaga cynodegmi* (22).

(ii) Anaerobes. Table 1 shows common anaerobic bacterial genera isolated from 50 infected dog bite wounds (206). The most common anaerobic organisms, in order of decreasing frequency, included *Fusobacterium nucleatum* (16%), *Bacteroides tectus* (14%), *Prevotella heparinolytica* (14%), *Propionibacterium acnes* (14%), *Prevotella intermedia* (8%), *Peptostreptococcus anaerobius* (8%), *Porphyromonas macacae* (6%), and *Porphyromonas cansulci* (6%) (206). Pure anaerobic growth is rare and is almost always accompanied by aerobic organisms (96, 206). β -Lactamase production is a common feature among anaerobes isolated from infected bites (23, 206). Anaerobes are commonly present in abscesses, puncture

wounds, and upper extremity wounds (206). The growth and identification of anaerobic organisms are frequently a difficult task. In comparison to research laboratories, local hospital laboratories are not often able to grow the majority of anaerobic organisms (206). This limitation may erroneously lead clinicians to believe that there is an absence of these organisms in infected wounds. In addition, some anaerobic isolates such as those belonging to the genera *Bacteroides* and *Prevotella* (e.g., *Bacteroides tectus* and *Prevotella bivia* or *Prevotella heparinolytica* and *Bacteroides uniformis*) have a close resemblance in colony morphology and biochemical activity, hence making their distinction from one another more difficult (3). Primary culture often requires longer incubation periods, good anaerobic media, and biochemical tests. In order to increase the yield of anaerobes, it has been recommended that laboratories retain inoculated anaerobic plates from bite wounds for at least 7 days (42).

Less commonly observed ($\leq 4\%$) anaerobic isolates include *Tannerella forsythia* (previously *Bacteroides forsythus*), *Campylobacter gracilis* (previously *Bacteroides gracilis*), *Campylobacter ureolyticus* (previously *Bacteroides ureolyticus*), *Porphyromonas canoris*, *Porphyromonas cangingivalis*, *Prevotella zoogloformans*, *Eubacterium* species (isolates were not identified beyond the genus level), *Fusobacterium russii*, *Fusobacterium gonidiformans*, *Fusobacterium alocis*, *Bacteroides tectus* group E, *Bacteroides fragilis*, *Bacteroides ovatus*, *Porphyromonas circumdentaria*, *Porphyromonas levii*-like, *Prevotella melaninogenica*, *Prevotella denticola*, *Propionibacterium acidipropionici*, *Propionibacterium freudenreichii*, *Peptostreptococcus asaccharolyticus*, and *Lactobacillus jensenii* (206, 220).

Other organisms isolated from infected dog bites have included *Veillonella parvula* (96), *Porphyromonas salivosa* (42, 118), *Prevotella bivia* (3), and the newly described *Fusobacterium canifelinum*, which are intrinsically resistant to fluoroquinolones due to the replacement of Ser79 with leucine and the replacement of Gly83 with arginine in the quinolone resistance-determining region (*gyrA*) (46, 47).

Cats. The microbiology of infected human cat bite wounds is similar to that of wounds inflicted by dogs in that both are polymicrobial in nature (23, 96, 206). In a multicenter prospective study of 57 clinically infected human cat bite wounds (206), 63% of the wounds were a mix of aerobic and anaerobic organisms. Pure aerobic growth occurred in 32% of the cases, and none demonstrated pure anaerobic growth. Only three (5%) of the cultures resulted in no growth (one from a patient with a purulent wound and two from patients with nonpurulent wounds but associated cellulitis/lymphangitis).

The most common type of infection was a nonpurulent wound with cellulitis, lymphangitis, or both (42%), followed by a purulent wound without abscess formation (39%) and abscesses (19%). Mixed aerobic and anaerobic infections were most common among abscesses (73%), followed by purulent wounds (64%) and nonpurulent wounds (58%) (206).

The number of bacterial isolates varied depending on the type of wound. The highest numbers of bacterial isolates were from abscesses (median, 7.0; range, 3 to 13; interquartile range, 5 to 11), followed by purulent wounds (median, 6.5; range, 0 to 13; interquartile range, 3 to 10) and nonpurulent wounds (median, 5.0; range, 0 to 12; interquartile range, 2 to 6). The majority (85%) of injuries were described as being

TABLE 2. Common aerobic and anaerobic bacterial genera isolated from 57 infected cat bite wounds^a

Bacterial genus	Frequency (%)
Aerobic organisms	
<i>Pasteurella</i>	75
<i>Streptococcus</i>	46
<i>Staphylococcus</i>	35
<i>Neisseria</i> ^b	35
<i>Moraxella</i>	35
<i>Corynebacterium</i>	28
<i>Enterococcus</i>	12
<i>Bacillus</i>	11
Anaerobic organisms	
<i>Fusobacterium</i>	33
<i>Porphyromonas</i>	30
<i>Bacteroides</i>	28
<i>Prevotella</i>	19
<i>Propionibacterium</i>	18

^a Based on data from reference 206.

^b The frequency differs from the actual number cited in the reference due to the reclassification of some of the isolates since its publication.

puncture wounds, while the rest were lacerations (3%) or mixes of both lacerations and puncture wounds (12%). In comparison to dog bites, the median time from the bite to the appearance of the first symptoms of infection was significantly shorter for cat bites (12 h; interquartile range, 7 to 18 versus 24 h; interquartile range, 12 to 48 h; $P < 0.001$) (206).

(i) Aerobes. Table 2 shows common aerobic bacterial genera isolated from 57 infected cat bite wounds (206). *Pasteurella* was the genus most commonly found for cat bite wounds, with *Pasteurella multocida* subsp. *multocida* (54%) and *Pasteurella multocida* subsp. *septica* (28%) being the predominant *Pasteurella* isolates. The predominance of these two *Pasteurella* strains in cat bites has also been observed by other studies (15, 115). Other less frequently found isolates included *Pasteurella dagmatis* (7%), *Pasteurella stomatis* (4%), and *Pasteurella canis* (2%) (206). *Pasteurella multocida* has also been reported for numerous human infections following bites of larger cats such as tigers (25, 122, 237), lions (25), and cougars (136).

Streptococci were the second most common species isolated from cat bite wounds (206). *Streptococcus mitis* was also the most common *Streptococcus* species isolated from both types of animal bite wounds (23% in cats and 22% in dogs) (206). Other species found in cat bite wounds included *Streptococcus sanguis* biotype II (12%), *Streptococcus mutans* (11%), *Streptococcus equinus* (5%), *Streptococcus sanguis* biotype I (5%), *Streptococcus intermedius* (4%), *Streptococcus constellatus* (4%), *Streptococcus agalactiae* (2%), *Streptococcus sanguis* (2%), and beta-hemolytic *Streptococcus* group F (2%). Among dog and cat bite wounds, the majority of *Streptococcus* isolates shared a similar frequency of occurrence, with the exception of *Streptococcus pyogenes*, which was not seen in cat bite wounds (12% prevalence for dog bites) (206).

In equal frequency, staphylococci, *Neisseria*, and *Moraxella* were the next most common genera found in cat bite wounds. The most common staphylococcal isolates were *Staphylococcus epidermidis* (18%) and *Staphylococcus warneri* (11%). Other less frequently found isolates were *Staphylococcus aureus* (4%), *Staphylococcus sciuri* subsp. *lentus* (4%), *Staphylococcus*

intermedius (2%), *Staphylococcus hominis* (2%), *Staphylococcus capitis* (2%), *Staphylococcus haemolyticus* (2%), *Staphylococcus hyicus* (2%), *Staphylococcus saprophyticus* (2%), and *Staphylococcus simulans* (2%). In comparison, *Staphylococcus aureus* was less frequently observed for cat bites than for dog bites (4% versus 20%) (206). Among *Neisseria* species, *Neisseria weaveri* (14%) (formerly known as CDC group M-5) was the most common *Neisseria* species, followed by *Neisseria zoodegmatidis* (9%) (formerly known as EF-4b), *Neisseria subflava* (2%), *Neisseria cinerea-Neisseria flavescens* (2%), and *Neisseria mucosa* (2%). There were no significant differences in the prevalences of the various *Neisseria* isolates between cat and dog bite wounds (206). Human infections after a cat bite associated with *Neisseria canis* have also been reported (104, 113). Among *Moraxella* species, *Moraxella catarrhalis* was the most commonly found identifiable isolate. Most others were not identified beyond the genus level. *Moraxella catarrhalis* was found more often in cat bite wounds than in dog bite wounds (11% versus 2%) (206).

For *Corynebacterium* species, the most commonly isolated species were *Corynebacterium (Leifsonia) aquaticum* (14%), *Corynebacterium minutissimum* (7%), and *Corynebacterium* group G (5%). Less frequently observed corynebacterium isolates included *Corynebacterium jeikeium*, *Corynebacterium* group B, *Corynebacterium* group F-1, *Corynebacterium kutscheri* (2%), *Corynebacterium propinquum* (2%), and *Corynebacterium striatum* (2%). *Corynebacterium (Leifsonia) aquaticum* was more prevalent in cat bites than in dog bites (14% versus 2%). *Enterococcus durans* (9%) (none isolated from dog bites) and *Enterococcus faecalis* (4%) were the most commonly found enterococcal isolates. *Bacillus firmus* (4%) and *Bacillus circulans* were the most commonly found identifiable bacillus isolates (206).

Less frequently encountered aerobic organisms, in order of decreasing frequency, included *Bergeyella zoohelcum* (7%) (previously known as *Weeksella zoohelcum* and prior to that known as CDC group IIj), *Capnocytophaga* species (7%), *Acinetobacter* species (7%), *Pseudomonas* species (5%), *Actinomyces* species (4%), *Brevibacterium* species (4%), *Gemella morbillorum* (4%), *Actinobacillus* species (4%), *Alcaligenes* species (4%), *Enterobacter cloacae* (4%), *Erysipelothrix rhusiopathiae* (4%), *Riemerella anatipetifer* (4%), *Rothia dentocariosa* (4%), *Klebsiella oxytoca* (2%), *Lactobacillus* species (2%), *Eikenella corrodens* (2%), *Flavimonas oryzihabitans* (2%), *Aeromonas hydrophila* (2%), *Pantoea agglomerans* (2%), *Rhodococcus* species (2%), and *Streptomyces* species (2%) (206).

Infection with *Bergeyella zoohelcum* was reported for a case of cellulitis after a cat bite (202). In that report, the strain was unusually fastidious and did not yield sufficient growth for identification using standard culture methods and required molecular techniques (PCR with rRNA gene sequencing) for identification. The use of the MicroSeq 500 16S rRNA gene-based bacterial identification system has resulted in the misidentification of *Bergeyella zoohelcum* as *Riemerella anatipetifer* (236). *Bergeyella zoohelcum* was also reported to result in tenosynovitis associated with *Pasteurella multocida* and other Gram-negative bacteria after a bite from a Siberian tiger (122). *Capnocytophaga canimorsus* (formerly known as CDC group DF-2) has been implicated in severe infections following cat bites or exposure (31, 217, 234). Newly discovered organisms in

cats include *Haemophilus felis*, initially identified as *Aggregatibacter (Haemophilus) paraphrophilus* (121), and a novel biotype of *Corynebacterium diphtheriae* (105). Their pathogenicity and potential for transmission to humans are unknown.

Cats are also the main reservoir of *Bartonella henselae*, the causative agent of cat scratch disease (190). Most patients with cat scratch disease report a history of a scratch or a bite by a cat (155, 240). The transmission of *Bartonella henselae* to cats is vector mediated through the cat flea, *Ctenocephalides felis* (41). Due to its fastidious nature, *Bartonella henselae* growth and isolation can be very difficult. Various culture media (e.g., rabbit blood and chocolate agars), the serological detection of *Bartonella henselae* antibodies by an indirect immunofluorescent assay, and other molecular techniques such as PCR are often utilized for its identification (54, 55, 56, 190). Another *Bartonella* species recently implicated in the development of cat scratch disease due to a cat bite is *Bartonella clarridgeiae* (138). This species should also be considered a potential agent of cat scratch disease, especially for patients who demonstrate negative seroreactivity against *Bartonella henselae*. The spectrum of cat scratch disease can range from regional tender lymphadenopathy to, less commonly, fulminant systemic infections, osteomyelitis, and encephalopathy (5, 153, 190, 235).

(ii) **Anaerobes.** Table 2 shows common anaerobic bacterial genera isolated from 57 infected cat bite wounds (206). In this case series, anaerobes were almost always isolated in mixed cultures along with aerobic organisms, and none of the wound specimens demonstrated pure anaerobic growth (206).

Fusobacterium nucleatum prevailed as the most commonly found *Fusobacterium* isolate from cat bites (25%). Other isolates found in cat bites included *Fusobacterium russii* (14%) and *Fusobacterium gonidiaformans* (2%). The prevalence of *Fusobacterium russii* was significantly higher in cat bites than in dog bites (14% versus 2%). The most common *Porphyromonas* isolate in cat bites was *Porphyromonas gulae* (11%). Other isolates included *Porphyromonas canoris* (9%), *Porphyromonas macacae* (7%), *Porphyromonas circumdentaria* (5%), *Porphyromonas cangingivalis* (4%), and *Porphyromonas cansulci* (2%). There were no significant prevalence differences among the various *Porphyromonas* species in dog and cat bite wounds. *Bacteroides tectus* was the main *Bacteroides* species isolated from both dog and cat bites (14% in dog bites and 28% in cat bites). Other *Bacteroides* isolates were *Bacteroides tectus* group E (4%) and *Bacteroides fragilis* (2%) (206).

Among *Prevotella* species, *Prevotella heparinolytica* was the most commonly found isolate in both dog and cat bite wounds (14% in dog bites and 9% in cat bites). Other *Prevotella* isolates less frequently encountered were *Prevotella zoogeleoformans* (2%) and *Prevotella melaninogenica* (2%). Within the genus *Propionibacterium*, *Propionibacterium acnes* dominated in both dog and cat bite wounds (14% in dog bites and 16% in cat bites). Other *Propionibacterium* isolates were *Propionibacterium avidum* (2%) and *Propionibacterium lymphophilum* (2%). Less frequently encountered anaerobes included *Peptostreptococcus anaerobius* (5%), *Filifactor villosus* (5%), *Eubacterium* species (2%) (could not be identified beyond the genus level), *Clostridium sordellii* (2%), and *Veillonella* species (2%) (were not identified beyond the genus level) (206).

Rare infectious diseases in humans acquired from cats and dogs.

(i) **Cats.** Cat and other feline bites and exposures have been associated with the human transmission of *Francisella tularensis* resulting in tularemia (6, 28, 73, 225, 231). The role of cats, especially those that are free-roaming, is increasingly being recognized as one probable mode of transmission of *Yersinia pestis* and a source of human plague (34, 38, 48, 60, 84, 99, 232, 233). Infections following bites, scratches, and contact with infectious materials as well as the inhalation of infectious aerosol while examining an infected cat have been reported (34, 84, 232, 233). The presentation of pet-associated human plague has included bubonic (34, 48, 84, 232), septicemic (48, 84), and pneumonic (34, 60, 84, 233) forms. In a review of human cases of plague in the United States from 1977 to 1994, the prevalence of primary pneumonic plague was higher among cat-associated cases (4 cases out of 15) than among other cases associated with mammals that transmit plague (1 out of 236) (34).

The transmission of *Sporothrix schenckii* to humans from cats has been widely reported (11, 53, 62, 77, 81, 144, 185, 186, 199, 239) and confirmed at the genetic level (186). Infections following a bite or scratch and even in instances without an obvious penetrating injury have been reported (53, 62, 77, 186, 199). Household outbreaks and epidemics of sporotrichosis involving cats and humans have been described (11, 53, 81, 199). Most infections in humans present as localized lymphocutaneous lesions. Sporotrichosis has also been reported for dogs and other animals (49, 199); however, in contrast to cats, the zoonotic transmission of sporotrichosis to humans from other animals has not been as widely reported.

(ii) **Dogs.** Blastomycosis in humans acquired from dogs has also been reported (152, 198). Reports of the mode of zoonotic transmission of *Blastomyces dermatitidis* from infected dogs have included bites and accidental exposure to infected material (89, 100, 124, 181). Various *Bartonella* species DNA, including that of *Bartonella henselae*, have been isolated from dog saliva (61). *Bartonella* infection such as endocarditis has also been reported for dogs (21, 170). However, in comparison to cats, the role of dogs as a source of human *Bartonella* infection is not as well understood and is restricted to only a few non-bite-related case reports (129, 130, 216).

(iii) **Cats and dogs.** Rabies in domestic animals such as cats and dogs is rare in the United States. In a 2008 U.S. rabies surveillance study, the prevalences of rabies in cats and dogs were 4.3% and 1.1%, respectively. In comparison to the previous year, the number of rabies cases reported in 2008 increased among cats and decreased among dogs (17). The dog-to-dog transmission of canine rabies virus variants has been eliminated in the United States since 2004 (17). Most human rabies cases in the United States have been associated with rabid bats, while in developing countries, dogs are the most common animals implicated in the transmission of rabies to humans (39).

Monkeys/simians. Bites from monkeys may occur in people who keep them as pets, those who use them for medical research, and those who travel to cities and countries that have a high prevalence of free-roaming monkeys (e.g., Gibraltar, Bali, and certain parts of India). In addition, persons who visit or

TABLE 3. Oral bacteriology of rhesus monkeys and isolates recovered from humans with simian bite wounds

Organism	Oral bacteriology of rhesus monkey (% of isolates) determined by:		No. of patients (n = 3) ^f
	Rayan et al. ^a (n = 17)	Eke et al. ^b (n = 9)	
<i>Actinomyces</i> species		85	
<i>Aerococcus</i> species	2.4		
<i>Aggregatibacter actinomycetemcomitans</i> ^d		73	
<i>Bacillus</i> species	7.4		
<i>Bacteroides fragilis</i> group		5	1
<i>Campylobacter</i> species		28	
<i>Capnocytophaga</i> species		30	
<i>Eikenella corrodens</i>	1.2	4.7	1
<i>Escherichia coli</i>	1.2		1
<i>Fusobacterium nucleatum</i>		90	1
<i>Haemophilus influenzae</i>	1.2		
<i>Haemophilus parainfluenzae</i>	17.2		
<i>Haemophilus</i> species		100	
<i>Klebsiella pneumoniae</i>			1
<i>Moraxella</i> species	2.4		
<i>Neisseria</i> species	19.8		1 (<i>N. subflava</i>)
<i>Peptostreptococcus micros</i>		89	
<i>Pseudomonas</i> species	2.4		
<i>Staphylococcus aureus</i>	3.6		
<i>Staphylococcus</i> species	1.2		2 (<i>S. epidermidis</i>)
<i>Staphylococcus</i> , beta-hemolytic	3.8		
Alpha-hemolytic streptococci	19.8		1 (unidentified), 1 (<i>S. mitis</i>), 1 (<i>S. sanguis</i>), 1 (<i>S. sanguis</i> biotype II), 1 (<i>S. anginosus</i>), 1 (<i>S. intermedius</i>)
Beta-hemolytic streptococci			1 (<i>S. agalactiae</i>)
Nonhemolytic streptococci	1.2		2 (<i>E. faecalis</i>)
<i>Streptococcus</i> species	1.2		
<i>Wolinella</i> species		66	

^a Based on data from reference 184.

^b Based on data from reference 67.

^c Based on data from reference 97.

^d Previously known as *Actinobacillus actinomycetemcomitans*.

work in wilderness areas and national parks worldwide where monkeys reside are also at a higher risk. These animals are often considered “mischievous” and will scour for food if hungry.

Although monkey bites are commonly reported, surprisingly very few studies have investigated the bacteriology of monkey bite wounds. Kizer (135) reported that 5 out of 332 (1.7%) patients who presented to the University of California—Los Angeles (UCLA) Emergency Department during 1975 for bite wounds were inflicted by monkeys. Rates of monkey bites in facilities seeing animal bite wounds have been reported from a number of countries and range from 3.2% in India to 0.7% in Israel (72, 102, 120). In the United Kingdom, an animal housing facility reported 85 monkey bites (67 incidents in men and 18 in women) in handlers over a 6-year period (215).

The oral flora of selected monkeys and isolates from human bites is shown in Table 3. In a study of the aerobic tongue flora of 17 rhesus monkeys, commonly isolated microorganisms included *Neisseria* species (19.5%), streptococcus species (19.5%), and *Haemophilus parainfluenzae* (17.2%) (184). Another study of rhesus monkeys involving the bacteriology of gingival sulci and associated plaques revealed the following

organisms colonizing the gingival sulci, in order of decreasing frequency: *Haemophilus* species (100%), *Fusobacterium nucleatum* (90%), *Peptostreptococcus micros* (89%), *Actinomyces* species (85%), oral streptococci (80%), black-pigmented anaerobic rods (80%), *Actinobacillus actinomycetemcomitans* (73%), *Wolinella* species (66%), *Capnocytophaga* species (30%), *Campylobacter* species (28%), *Eikenella corrodens* (4.7%), and spirochetes (4.7%) (67).

The spectrum of isolates from humans bitten by monkeys is similar to that of isolates from human bite wounds. There is a predominance of alpha-hemolytic streptococci, enterococci, *Staphylococcus epidermidis*, *Neisseria* and *Haemophilus* species, *Eikenella corrodens*, and anaerobes, including *Bacteroides* and *Fusobacterium* species (97).

A major concern with monkey bites is the transmission of viral diseases. Jones-Engel et al. studied the prevalence of selected enzootic primate-borne viruses among 39 rhesus monkeys in Katmandu, Nepal (126). Evidence of infection of these monkeys through positive antibody responses to simian foamy virus (97.4%), *Cercopithecine herpesvirus 8* (94.9%), simian virus 40 (89.7%), and *Cercopithecine herpesvirus 1* (64.1%) (also known as B virus) with various molecular techniques was noted.

Documented cases of B virus infection in humans have been attributed mostly to monkey bites. However, other less commonly reported modes of transmission have been due to scratches and percutaneous inoculation with infected materials (45). A fatal case of B virus infection following mucocutaneous exposure was reported (35). Other reports of bite-related primate-borne viral infections in humans have included simian foamy virus (especially from ape bites) and monkeypox virus infections (26, 163, 200).

Bears. Bear attacks have been reported worldwide (29, 52, 106, 164, 188, 214, 227). Human injuries from grizzly bears (*Ursus arctos horribilis*) in the national parks of North America have been reported, with an injury rate of 1 person per 2 million visitors (106). From 1900 to 1985, 162 bear-inflicted injuries (~2 attacks per year) were reported for the U.S. and Canadian national parks (107). Although bear-inflicted human injuries and death are uncommon (78), as the remote bear habitat decreases and humans enter wilderness areas for living and recreation, there are more chances of encounters between bears and humans.

Data for the oral bacteriology of bears as well as bear bite wounds in humans are limited to a few studies and case reports (79, 140, 145, 175, 193). Organisms isolated from grizzly or black bear oral cavities have included *Streptococcus* (61%), *Staphylococcus* (48%), *Escherichia* (40%), *Enterobacter* (25%), *Citrobacter* (10%), *Hafnia* (10%), *Proteus* (6%), and *Acinetobacter* (6%) species (175).

The aerobic oral flora of 12 black bears (*Ursus americanus*) with periodontal disease from the northern lower peninsula of Michigan has been investigated (79). All of the cultures yielded at least one organism. In order of decreasing frequency, isolates included *Micrococcus* species (83%), alpha-hemolytic *Streptococcus* (50%), *Staphylococcus aureus* (33%), *Staphylococcus epidermidis* (16%), beta-hemolytic *Streptococcus* (16%), *Acinetobacter* species (16%), nonhemolytic *Streptococcus* (8%),

TABLE 4. Bacteria isolated from bear bite wounds in humans^a

Bacterium isolated from bear bite wounds
<i>Aeromonas hydrophila</i>
<i>Bacillus cereus</i>
<i>Citrobacter diversus</i>
<i>Enterococcus durans</i>
<i>Escherichia coli</i>
<i>Mycobacterium fortuitum</i>
<i>Neisseria sicca</i>
<i>Proteus vulgaris</i>
<i>Serratia fonticola</i>
<i>Serratia marcescens</i>
<i>Staphylococcus aureus</i>
<i>Staphylococcus epidermidis</i>
<i>Streptococcus sanguis</i>

^a Based on data from references 140, 145, and 193.

Bacillus species (8%), *Enterobacter* species (8%), *Klebsiella pneumoniae* (8%), *Proteus mirabilis* (8%), *Proteus* species (8%), and *Actinomyces* species (8%).

The bacteria isolated from bear bite wounds come from a number of individual case reports (Table 4). Kunimoto et al. reported a 49-year-old hunter who was attacked by a grizzly bear and sustained multiple injuries, including a bite on his skull (140). Deep-wound cultures from the scalp lacerations grew *Serratia fonticola*, *Serratia marcescens*, *Aeromonas hydrophila*, *Bacillus cereus*, and *Enterococcus durans*. Anaerobic cultures did not grow any organisms. In this case report, microbiological results were limited by a delay in obtaining wound cultures (12 h after the injury) and a history of prior exposure to antibiotics (cefazolin and metronidazole). Other organisms grown from grizzly bear wounds in humans have included *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Proteus vulgaris*, *Citrobacter diversus*, and *Escherichia coli* (193). In this report, it is unclear if the injuries occurred as a result of a claw or a bite and if patients had any prior exposure to antibiotics.

Lehtinen et al. reported a case of a 56-year-old male who sustained several bite wounds from a brown bear (*Ursus arctos*) (145). Bacterial cultures from a deep thigh bite wound grew *Streptococcus sanguis*, *Neisseria sicca*, *Bacillus* species, and *Mycobacterium fortuitum*. It is unclear if anaerobic cultures were obtained from this patient or if antibiotic prophylaxis (ceftriaxone) was administered prior to obtaining wound cultures.

Rabies virus infection of bears has also been reported (36). However, to our knowledge there have not been any reports of rabies transmission from bears to humans.

Pigs. Pig bite injuries are a common occupational hazard in pig farmers and can also be seen in those who keep pigs as household pets. Injuries, often on extremities, commonly occur during the capture, transport, or immobilization of the pig (10, 168, 221). Human wound infections after a pig bite have been reported in a few case reports and series (10, 66, 70, 95).

Table 5 lists the bacteria isolated from pig bite wound infections in humans. Barnham reported the microbiology of six patients who developed localized wound infections after being bitten or gored by pigs (10). Both monobacterial and polymicrobial infections were encountered. One patient did not grow any organisms from his wound or blood cultures, likely due to prior antibiotic exposure. Organisms isolated from these patients included *Streptococcus agalactiae*, *Streptococcus dysgalac-*

TABLE 5. Bacteria isolated from pig bite wound infections in humans^a

Bacterium isolated from pig bite wound infections
<i>Actinobacillus suis</i>
<i>Bacteroides</i> species, including <i>Bacteroides fragilis</i>
<i>Escherichia coli</i>
<i>Flavobacterium</i> group IIB-like organism
<i>Pasteurella aerogenes</i>
<i>Pasteurella multocida</i>
<i>Proteus</i> species
<i>Staphylococcus aureus</i> (methicillin resistant)
<i>Staphylococcus</i> species (coagulase negative)
<i>Streptococcus agalactiae</i>
<i>Streptococcus dysgalactiae</i>

^a Based on data from references 10, 51, 66, 70, and 95.

tiae (likely subsp. *dysgalactiae*), *Streptococcus suis*, *Pasteurella aerogenes*, *Proteus* species, *Escherichia coli*, *Bacteroides* species including *Bacteroides fragilis*, *Pasteurella multocida*, and coagulase-negative *Staphylococcus*. In the same case series, that author also reported *Streptococcus milleri* bacteremia in a 20-year-old man who sustained a hand laceration while cutting teeth from piglets (10). Interestingly, in this patient, there were no localized signs of infection noted.

Pasteurella aerogenes, found in the digestive tract of pigs, has often been isolated from human wounds associated with pig bites or exposure (10, 66, 147). Other organisms associated with human infections after a pig bite have included *Actinobacillus suis* (70), a *Flavobacterium* group IIB-like organism (95), and MRSA (51).

Recent studies, mostly from the Netherlands, have shown a high prevalence of nasal MRSA colonization in people commonly in contact with live pigs (133, 137, 203, 219, 226, 238). Molecular characterization of MRSA found in pigs and humans commonly in contact with pigs has revealed that the MRSA isolate often belonged to *Staphylococcus aureus* protein A (*spa*) type t108 and sequence type 398 (ST398) (137, 218, 224, 226). The clonal spread and transmission of *spa* type t108 and ST398 MRSA isolates among family members of a pig farmer, his coworkers, and his pigs have been reported (119).

Rabies virus infection of a pig has been reported (63). However, to our knowledge there has not been any reports of rabies virus transmission from pigs to humans.

Ferrets. Ferrets are kept by many as exotic pets. Young children and infants seem to be at the greatest risk for severe bites. In a study of the oral microflora of five ferrets at the gingival sulcus and mucosal membrane, commonly isolated bacteria belonged to *Pasteurella*, *Corynebacterium*, and *Rothia* species (74). No anaerobic bacteria were isolated from the cultures. Anaerobes, specifically *Porphyromonas gulae* and *Fusobacterium* species, were found in ferrets with ligature-induced periodontitis (74). Jones et al. (125) reported a case of recurrent *Mycobacterium bovis* infection following a ferret bite. Rabies may also be transmitted by ferret bite.

Horses. Humans and horses have shared a close relationship with one another over thousands of years. Throughout the world, millions of people have contact with horses through recreation or sporting or for occupational reasons. In 1958, Carithers reported that 5 out of 157 (3%) animal bites seen in children in Jacksonville, FL, over a 20-month period were

TABLE 6. Bacteria isolated from horse bite wounds in humans^a

Bacterium isolated from horse bite wounds
<i>Actinobacillus equuli</i> -like bacterium
<i>Actinobacillus lignieresii</i>
<i>Actinobacillus suis</i>
<i>Bacteroides fragilis</i>
<i>Campylobacter ureolyticus</i> ^b
<i>Escherichia coli</i>
<i>Neisseria</i> species
<i>Pasteurella caballi</i>
<i>Prevotella melaninogenica</i>
<i>Staphylococcus aureus</i>
<i>Streptococcus anginosus</i>
<i>Streptococcus mutans</i>
<i>Yersinia</i> species

^a Based on data from references 12, 59, 71, 156, 165, 177, and 180.

^b Previously *Bacteroides ureolyticus*.

horse bites (30). In England, over a 2-year period, a local hospital reported 622 patients with horse-related injuries, 24 (3.8%) of which were bite wounds (65). In this case series, very few had extensive muscle damage, and most injuries healed uneventfully. In eastern Turkey, over a period of 2 years, horse bites accounted for 17% of animal bite injuries and were second only to dog bites (69%) (69). It has been estimated that roughly 3% to 4.5% of all horse-related injuries are from bites (143).

Most reports of the bacteriology of horse bite wounds in humans have revealed infections to be polymicrobial, with a mixture of aerobic and anaerobic organisms (59, 177). Table 6 lists the bacteria isolated from horse bite wounds in humans.

Actinobacillus lignieresii in infected wounds of humans bitten by horses has often been reported (12, 59, 177). *Actinobacillus* species, specifically *Actinobacillus suis*, has been found to be a part of normal horse oral and upper respiratory tract floras (16, 134). Peel et al. (177) reported two cases of *Actinobacillus* infections from a horse bite. For one patient who had an infected hand bite wound, the cultures grew *Actinobacillus lignieresii*, an *Actinobacillus equuli*-like bacterium, *Staphylococcus aureus*, and oral *Neisseria* and *Streptococcus* species. The wound cultures of the other patient, who sustained a bite and an open forearm fracture and who had repeated infections, grew *Actinobacillus suis*, *Staphylococcus aureus*, *Prevotella melaninogenica*, *Escherichia coli*, and *Pasteurella multocida*. The identification of *Actinobacillus lignieresii* can also be difficult, and this organism may be mistaken for a *Pasteurella pneumotropica* isolate (59, 146, 177). *Escherichia coli* and *Bacteroides* species have also been reported for infections with foul-smelling purulent drainage from horse bite wounds (59).

Other organisms isolated from human horse bite wounds have included *Streptococcus anginosus* and *Streptococcus mutans*, from an infected laceration (156); *Campylobacter ureolyticus*, *Prevotella melaninogenica*, and *Bacteroides fragilis*, from a purulent wound with a subsequent development of osteomyelitis (165); *Pasteurella caballi* (71); and *Yersinia* (180). Wound infection with *Staphylococcus hyicus* subsp. *hyicus* after a donkey bite was reported (173).

Sheep. *Actinobacillus lignieresii* has been recovered from a pure culture from an infected wound in a patient who sustained a bite injury from a sheep over his finger (177).

Tasmanian devils. Tasmanian devils are Australian marsupials. Georghiou et al. reported a case of a 5-year-old boy with a *Pasteurella multocida* infection following a Tasmanian devil bite (87).

Reptiles

Snakes. The true global incidence of human injuries inflicted by snake bites or envenomations and associated deaths is unknown (40, 128, 229). It was estimated that between 1.2 million and 5.5 million humans are bitten by snakes annually. Of these, at least 421,000 to 1,841,000 are envenomations and 20,000 to 94,000 lead to death (128). Most venomous snake bites occur in Southern and Southeastern Asia, sub-Saharan Africa, and Central and South America (128). In the United States, an estimated 7,000 venomous snake bites occur annually, with a mortality rate of less than 1% (see the Texas Department of State Health Services Zoonosis Control Branch website [http://www.dshs.state.tx.us/idcu/health/zoonosis/Animal/Bites/Information/venom/snake/]). Bites from nonnative venomous snakes, often involving cobras, have also been reported in the United States (159).

Investigations into the oral bacterial flora of various snakes and their venom have revealed a mixture of both aerobic and anaerobic bacterial species (94, 141, 201, 211). Studies of the oral flora bacteriology of Chinese cobras, bamboo pit vipers (201), and Malayan pit vipers (211) have yielded a plethora of aerobic and anaerobic organisms, such as coagulase-negative staphylococci, *Proteus* species, *Morganella morganii*, *Aeromonas hydrophila*, *Enterococcus faecalis*, and *Clostridium* species. Common bacterial isolates from venom of 15 captive rattlesnakes included *P. aeruginosa*, *Proteus* species, coagulase-negative staphylococci, *Clostridium* species, and *Bacteroides fragilis* (94).

Goldstein et al. studied the venom of four rattlesnakes with their fang sheaths retracted and decontaminated and found that 50% of the samples of the venom had no bacterial growth (94). It is likely that the isolated oral flora of rattlesnakes reflects the fecal flora of ingested prey. In addition, venom may also possess some antibacterial activity against some of the organisms isolated from snake oral flora (207, 211). In a study of venoms from three crotalid species, it was noted that the venom had some antibacterial activity against staphylococci, *Pseudomonas aeruginosa*, and *Enterobacter*, *Citrobacter*, *Proteus*, and *Morganella* species but not against anaerobic isolates (207). This potential antibacterial property of venom may have some antagonistic role in the overall pathogenesis of infection after envenomation injuries and may explain the observed low incidence of wound infection in snake bite victims (18, 44, 132, 150).

Polymicrobial infections are commonly observed for patients with infected wounds secondary to a snake bite (127, 131). Jorge et al. reported 40 patients with abscesses at the site of *Bothrops* bites, none of whom were previously treated with antimicrobials (127). Those authors found that *Morganella morganii* was the most frequently isolated organism (23 patients). Other organisms isolated, in order of decreasing frequency, included group D streptococcus, *Providencia rettgeri*, *Bacteroides* species, *Enterobacter* species, and *Escherichia coli*. A mixture of aerobic and anaerobic organisms with enteric,

coliform organisms accounted for the majority of isolates and was also observed for abscesses secondary to pit viper envenomation (131).

More recently, Garg et al., in a retrospective study, reported the bacteriology of 43 wound infections secondary to snake bite (86). Fifty-six percent of patients presented with a subcutaneous abscess, and the remainder had localized tissue necrosis. Interestingly, in that study, approximately 80% of the infections were monobacterial (likely due to a major limitation of the study in that anaerobic cultures were not performed). The most commonly found organisms isolated included *Staphylococcus aureus*, *Escherichia coli*, coagulase-negative staphylococcus, *Enterococcus faecalis*, and *Klebsiella pneumoniae*. Other organisms less frequently observed included *Proteus* species, *Morganella morganii*, *Pseudomonas aeruginosa*, *Acinetobacter* species, *Enterobacter* species, and *Streptococcus* species.

Human infections with *Mycobacterium ulcerans* (112), *Aeromonas jandaei* (196), *Enterobacter aerogenes* (formerly *Aerobacter aerogenes*) (149), *Serratia marcescens* (S. A. Minton and F. Russell, personal communication), *Clostridium perfringens* (123), and *Clostridium tetani* (204) following snake bites have been reported.

Komodo dragon. Komodo dragons are native of Komodo, Rinca, Flores, and Gili Motang in the Indonesian Islands and are the largest living lizards, reaching a length of approximately 10 feet and weighing 150 pounds. In addition, they are held in captivity in many zoos around the world. They are carnivores and eat mostly carrion but will prey on birds and mammals.

Montgomery et al. (162) took oral and saliva samples for aerobic cultures from 23 wild Komodo dragons in Komodo National Park, Indonesia, and from 13 captive animals at the Gembira Loka zoo in Yogyakarta, Java. Those authors reported that captive Komodo dragons had "significantly" fewer species isolated than did wild species. *Escherichia coli* was the most commonly found isolate, found in 39% (9 out of 23) of wild Komodo dragon mouths, while *Staphylococcus capitis* (5 out of 13 [38%]) and *Staphylococcus caseolyticus* (5 out of 13 [38%]) were the isolates most commonly found in captive Komodo dragon mouths. Table 7 lists the bacteria found in wild and captive Komodo dragons. *Pasteurella multocida* is not typically part of reptilian oral flora, and it is likely acquired transiently in Komodo dragons through the ingestion of prey animals (82).

A recent study by Citron et al. (43) reported the growth of 17 aerobes and 8 anaerobes from the mouths of two captive Komodo dragons. The male Komodo dragon had eight aerobic and three anaerobic species, while the female had nine and five, respectively. Both Komodo dragons grew 3+ to 4+ enteric bacilli, including *Escherichia coli*, *Klebsiella oxytoca*, *Enterobacter* species, and *Providencia rettgeri*. Both Komodo dragons had heavy growth of *Enterococcus faecalis*, and the female also had *Streptococcus dysgalactiae*. Both Komodo dragons had coagulase-negative *Staphylococcus* species, including *Staphylococcus sciuri*, and a variety of Gram-positive rods, including *Bacillus cereus*, *Rothia* species, and *Corynebacterium* species. The female Komodo dragon grew *Fusobacterium varium*, two other Gram-negative rods resembling *Bacteroides* and *Parabacteroides* species, *Clostridium perfringens*, and *Clostridium sardiniense*. The male grew *Bacteroides fragilis*, *Clostridium sordellii*,

TABLE 7. Oral bacteriology of wild and captive Komodo dragons

Organism	No. of isolates from Komodo dragon wounds determined by:		
	Montgomery et al. ^a		Citron et al. ^b for captive animals (n = 2)
	Wild (n = 23)	Captive (n = 13)	
Aerobic Gram-positive organisms			
<i>Aerococcus</i> species	2	0	
<i>Bacillus cereus</i>	3	0	2
<i>Bacillus coagulans</i>	1	0	
<i>Bacillus stearothermophilus</i>	1	0	
<i>Bacillus subtilis</i>	4	0	
<i>Bacillus</i> species	4	0	
<i>Brevundimonas diminuta</i>	2	0	
<i>Corynebacterium</i> species	7	0	2
<i>Enterococcus faecalis</i>	1	0	2
<i>Enterococcus casseliflavus</i>	1	0	
<i>Kurthia</i> species	0	2	
<i>Micrococcus</i> species	5	0	
<i>Rothia</i> species			2
<i>Staphylococcus aureus</i>	5	3	
<i>Staphylococcus auricularis</i>	1	0	
<i>Staphylococcus capitis</i>	0	5	
<i>Staphylococcus caseolyticus</i>	0	5	
<i>Staphylococcus cohnii</i>	0	1	
<i>Staphylococcus gallinarum</i>	1	0	
<i>Staphylococcus haemolyticus</i>	2	1	
<i>Staphylococcus hominis</i>	1	0	
<i>Staphylococcus kloosii</i>	1	0	
<i>Staphylococcus saprophyticus</i>	1	0	
<i>Staphylococcus sciuri</i>	2	0	2
<i>Staphylococcus warneri</i>	1	2	
<i>Staphylococcus xylosum</i>	1	1	
<i>Staphylococcus</i> species	10	0	
<i>Streptococcus bovis</i>	1	0	
<i>Streptococcus agalactiae</i>	1	0	
<i>Streptococcus dysgalactiae</i>			1
<i>Streptococcus</i> species	14	0	
Aerobic Gram-negative organisms			
<i>Acinetobacter calcoaceticus</i>	2	0	
<i>Aeromonas hydrophila</i>	2	1	
<i>Alcaligenes faecalis</i>	2	0	
<i>Burkholderia cepacia</i>	3	0	
<i>Enterobacter</i> species	6	3	2
<i>Escherichia coli</i>	9	1	2
<i>Klebsiella</i> species	9	1	2
<i>Pasteurella multocida</i>	2	0	
<i>Proteus mirabilis</i>	5	2	
<i>Providencia rettgeri</i>	1	0	2
<i>Pseudomonas</i> species	5	1	
<i>Serratia</i> species	4	1	
Anaerobic organisms			
<i>Fusobacterium varium</i>			1
<i>Bacteroides fragilis</i>			1
<i>Bacteroides</i> species			1
<i>Parabacteroides</i> species			1
<i>Clostridium perfringens</i>			1
<i>Clostridium sardiniense</i>			1
<i>Clostridium sordellii</i>			1
Swarming <i>Clostridium</i> species			1

^a Based on data from reference 162.

^b Based on data from reference 43.

and a swarming *Clostridium* species.

Komodo dragon attacks on humans are rare, with three having been reported in the press, but to our knowledge, there has not been any report of the bacteriology of infected human wounds from bites of this animal.

Monitor lizards. Monitor lizards, also known as biawak or goannas, belong to the genus *Varanus* and are related to Komodo dragons. A case of *Pseudomonas aeruginosa* septic arthritis after a bite from a monitor lizard was reported (210).

Iguana. Iguanas are popular exotic pets in the United States. In a recent report from poison centers in Texas between 1998 and 2008, 59 iguana bites were cited (79a). A majority of the injuries often involve the upper extremities (commonly fingers) and face (14, 79a). Data on the microbiology of infected human wounds from iguana bites are restricted to a few case reports (101, 117). An 8-year-old boy was bitten on his left index finger and developed purulent cellulitis that grew *Serratia marcescens* and *Staphylococcus aureus* (117). Two cases of *Serratia marcescens* bullous cellulitis in adults following iguana bites have also been reported (101). It is noteworthy that *Serratia* species is commonly resistant to aminopenicillins, an antibiotic class commonly prescribed for animal bites. It is often susceptible to fluoroquinolones, carbapenems, and aminoglycosides.

Alligators/crocodiles. Both the alligator population and human encounters with alligators have increased in the United States (142). The same situation exists for crocodiles in Australia (27, 103). From 1948 to 2004, there were 376 injuries and 15 deaths reported to be due to alligators in the United States (142). In 2009, 11 provoked and 8 unprovoked alligator attacks were reported in Florida (see the Florida Fish and Wildlife Conservation Commission website [http://myfwc.com/docs/WildlifeHabitats/Alligator_GatorBites.pdf]). Unprovoked bites were defined as bites on human beings from wild alligators, which were not provoked by handling or intentional harassment.

Flandry et al. (76) investigated the oral microbiology of 10 American alligators (*Alligator mississippiensis*) and found a plethora of aerobic and anaerobic bacteria, including *Aeromonas hydrophila*, in all 10 alligators. Other isolates found included *Citrobacter freundii*, *Klebsiella oxytoca*, *Pasteurella haemolytica*, *Pseudomonas pickettii*, *Bacteroides thetaiotaomicron*, *Bacteroides vulgatus*, other *Bacteroides* species, *Clostridium clostridioforme*, *Clostridium tetani*, *Fusobacterium nucleatum*, and *Peptostreptococcus* species.

Data on the microbiology of human wounds inflicted by alligators or crocodiles are limited to a few case reports (76, 228). In a report of a 22-year-old male with an infected thumb injury following an alligator bite, wound cultures grew *Aeromonas hydrophila*, *Enterobacter agglomerans*, *Citrobacter diversus*, *Enterococcus* species, and *Clostridium* species (76). Recently, Wamisho et al. (228) reviewed their experience with five Malawian patients bitten by crocodiles, in which one wound culture grew a *Citrobacter* species. In their review of the literature, they noted that other investigators isolated *Vibrio vulnificus*, *Citrobacter* species, *Burkholderia pseudomallei*, *Pantoea agglomerans*, *Bacteroides melanogenicus*, *Aeromonas hydrophila*, *Serratia fanticola*, *Clostridium perfringens*, *Pseudomonas aeruginosa*, and *Pro-*

teus vulgaris. Caiman bites in Brazil have been reported, and it was speculated that their oral flora might be similar to that of alligators in the United States (108).

Rodents

Rats. It is estimated that there are 20,000 rat bites in the United States annually. Children are commonly bite victims, as rats and other rodents have gained popularity as pets (68, 88). With respect to diseases associated with rats, most attention has been related to rat bite fever, an ancient disease caused by *Streptobacillus moniliformis*, a fastidious, highly pleomorphic, filamentous, Gram-negative rod, and *Spirillum minus*, a short, tightly coiled, Gram-negative rod (68). *Streptobacillus moniliformis* infection is more common in North America, while *Spirillum minus* is more common in Asia.

Ordog et al. conducted a prospective study of 50 patients with uninfected rat bite wounds (172). Bacterial isolates were cultured for only 30% of wounds, 43% of which were *Staphylococcus epidermidis* isolates and the remainders of which were *Bacillus subtilis*, alpha-hemolytic *Streptococcus*, and diphtheroid isolates. Prophylactic antibiotics were withheld, and only one patient developed infection. *Actinobacillus equuli* has been isolated from the nasopharynx of laboratory mice and rats (146). *Actinobacillus lignieresii* has also been isolated from the middle ear and nasopharynx of laboratory rats (146).

Cases of rat bite-associated infections of humans with *Corynebacterium kutscheri* (114) and *Leptospira* (98, 151) have been reported.

Guinea pigs. Guinea pigs are common household pets of children. *Actinobacillus equuli* has been isolated from the conjunctiva and posterior nasopharynx of laboratory guinea pigs (146). Case reports of *Haemophilus influenzae* (191) and *Pasteurella* species (148) infections after guinea pig bites have been published.

Hamsters. Tularemia in a 3-year-old boy following a hamster bite to his finger shortly before the hamster died has been reported (37). A left axillary lymph node biopsy specimen grew *Francisella tularensis*. A case of a glandular form of tularemia acquired during hamster hunting has been reported (160).

Prairie dogs. A case of a *Francisella tularensis* infection in a 3-year-old boy following a prairie dog bite was reported (2).

Birds

Human infections from bird bites are generally minor and rare, but a few case reports have been made. Berkowitz and Jacobs (13) reported a fatal case of a 16-month-old girl who was pecked on her head by a rooster and 4 days later presented with seizures. She died 6 days after admission, and her brain tissue grew *Streptococcus bovis*, *Clostridium tertium*, and *Aspergillus niger*. Those authors concluded that soil microorganisms contaminated by chicken feces were the most likely source of the organisms. Davis and Wenzel (50) reported a man attacked by a horned owl, who developed a scalp infection that grew two undetermined but different *Bacteroides* isolates.

Swans. A *Pseudomonas aeruginosa* infection in a 39-year-old woman who was vigorously pecked on a finger while feeding a swan was reported (64).

Sharks

The International Shark Attack File reported 455 shark attacks from 1999 to 2009 in the United States (<http://www.flmnh.ufl.edu/fish/sharks/isaf/isaf.htm>). The majority of attacks occurred in Florida (294 attacks), Hawaii (42 attacks), South Carolina (32 attacks), and California (30 attacks). Worldwide, during the same period, 700 shark attacks were reported, with 51 (7.3%) being fatal attacks. The highest numbers of attacks worldwide, in order of decreasing frequency, were reported from Florida, Australia, Hawaii, South Africa, and California.

Buck et al. (24) studied the oral aerobic flora of a male great white shark from Connecticut waters and recovered various isolates of *Vibrio* species, such as *Vibrio parahaemolyticus*, *Vibrio alginolyticus*, and *Vibrio fluvialis*. Other isolates included *Pseudomonas putrefaciens*, "gold-pigmented" *Staphylococcus* species, *Citrobacter* species, and *Micrococcus* species. *Vibrio carchariae*, initially mistaken for a *Clostridium* species, was isolated from an infected wound in a shark bite victim swimming off the South Carolina coast (176). Royle et al. (194) reported two cases of infections following shark bites in Australia. The wound cultures from one of the patients grew *Vibrio parahaemolyticus* and *Aeromonas caviae*, and the cultures from the other patient grew *Vibrio alginolyticus*, *Aeromonas hydrophila*, *Proteus* species, *Klebsiella oxytoca*, *Klebsiella pneumoniae*, *Clostridium freundii*, and an *Enterococcus* species.

SUMMARY AND FUTURE DIRECTIONS

Our review has focused on infected dog and cat bite wounds in humans and a variety of more exotic animal bite wound infections. As noted above, the bacteriology of even common dog and cat bite wounds is diverse, and despite the use of currently optimal aerobic and anaerobic culture methods, approximately 7% of infected wounds still do not yield bacterial growth. This suggests that other more fastidious pathogens, including *Chlamydia*, mycoplasmas, and perhaps even viruses, may be involved in these wounds. Additional systematic studies of these wounds employing new molecular diagnostic techniques are needed. Another issue is the lack of an understanding of the pathogenic significance of all these cultured organisms. Perhaps antimicrobial treatment studies with a differing spectrum of activity may shed some light in this area. In addition, systematic studies with strict infection criteria are needed. A majority of the data have come from case reports with no established criteria for an infected wound. Organizing a research network among zoos, veterinary practices, and rural clinics and hospitals may be helpful in gathering information in a more methodical fashion. One can hope for future interest in and research on these issues, but to paraphrase Robert Frost, we have miles to go before we sleep.

REFERENCES

- Aghababian, R. V., and J. E. Conte, Jr. 1980. Mammalian bite wounds. *Ann. Emerg. Med.* **9**:79–83.
- Alcalá Minagorbe, P. J., A. Fernández Bernal, A. Sánchez Bautista, and C. Loeda Ozores. 2004. *Francisella tularensis* infection transmitted by prairie dog. *An. Pediatr. (Barc.)* **60**:583–584. (In Spanish.)
- Alexander, C. J., et al. 1997. Characterization of saccharolytic *Bacteroides* and *Prevotella* isolates from infected dog and cat bite wounds in humans. *J. Clin. Microbiol.* **35**:406–411.
- Andersen, B. M., et al. 1993. *Neisseria weaveri* sp. nov., formerly CDC group M-5, a Gram-negative bacterium associated with dog bite wounds. *J. Clin. Microbiol.* **31**:2456–2466.
- Anderson, B. E., and M. A. Neuman. 1997. *Bartonella* spp. as emerging human pathogens. *Clin. Microbiol. Rev.* **10**:203–219.
- Arav-Boger, R. 2000. Cat-bite tularemia in a seventeen-year-old girl treated with ciprofloxacin. *Pediatr. Infect. Dis. J.* **19**:583–584.
- Baillie, W. E., E. C. Stowe, and A. M. Schmitt. 1978. Aerobic bacterial flora of oral and nasal fluids of canines with reference to bacteria associated with bites. *J. Clin. Microbiol.* **7**:223–231.
- Bannoehr, J., A. Franco, M. Iurescia, A. Battisti, and J. R. Fitzgerald. 2009. Molecular diagnostic identification of *Staphylococcus pseudintermedius*. *J. Clin. Microbiol.* **47**:469–471.
- Baptiste, K. E., et al. 2005. Methicillin-resistant staphylococci in companion animals. *Emerg. Infect. Dis.* **11**:1942–1944.
- Barnham, M. 1988. Pig bite injury and infection: report of seven human cases. *Epidemiol. Infect.* **101**:641–645.
- Barros, M. B., et al. 2004. Cat-transmitted sporotrichosis epidemic in Rio de Janeiro, Brazil: description of a series of cases. *Clin. Infect. Dis.* **38**:529–535.
- Benaoudia, F., F. Escande, and M. Simonet. 1994. Infection due to *Actinobacillus lignieresii* after a horse bite. *Eur. J. Clin. Microbiol. Infect. Dis.* **13**:439–440.
- Berkowitz, F. E., and D. W. Jacobs. 1987. Fatal case of brain abscess caused by rooster pecking. *Pediatr. Infect. Dis. J.* **6**:941–942.
- Bibbs, C. S., F. B. Willis, and R. L. Bratton. 2001. Iguana bites to the face. *J. Am. Board Fam. Pract.* **14**:152–154.
- Biberstein, E. L., S. S. Jang, P. H. Kass, and D. C. Hirsh. 1991. Distribution of indole-producing urease-negative pasteurellas in animals. *J. Vet. Diagn. Invest.* **3**:319–323.
- Bisgaard, M., K. Piechulla, Y. T. Ying, W. Frederiksen, and W. Mannheim. 1984. Prevalence of organisms described as *Actinobacillus suis* or haemolytic *Actinobacillus equuli* in the oral cavity of horses. Comparative investigations of strains obtained and porcine strains of *A. suis* sensu stricto. *Acta Pathol. Microbiol. Immunol. Scand. B* **92**:291–298.
- Blanton, J. D., K. Robertson, D. Palmer, and C. E. Rupprecht. 2009. Rabies surveillance in the United States during 2008. *J. Am. Vet. Med. Assoc.* **235**:676–689.
- Blaylock, R. S. 1999. Antibiotic use and infection in snakebite victims. *S. Afr. Med. J.* **89**:874–876.
- Boisvert, P. L., and M. D. Fousek. 1941. Human infection with *Pasteurella lepisepica* following a rabbit bite. *JAMA* **116**:1902–1903.
- Bracis, R., K. Seibers, and R. M. Julien. 1979. Meningitis caused by group II J following a dog bite. *West. J. Med.* **131**:438–440.
- Breitschwerdt, E. B., et al. 1995. Endocarditis in a dog due to infection with a novel *Bartonella* subspecies. *J. Clin. Microbiol.* **33**:154–160.
- Brenner, D. J., D. G. Hollis, G. R. Fanning, and R. E. Weaver. 1989. *Capnocytophaga canimorsus* sp. nov. (formerly CDC group DF-2), a cause of septicemia following dog bite, and *C. cynodegmi* sp. nov., a cause of localized wound infection following dog bite. *J. Clin. Microbiol.* **27**:231–235.
- Brook, I. 1987. Microbiology of human and animal bite wounds in children. *Pediatr. Infect. Dis. J.* **6**:29–32.
- Buck, J. D., S. Spotte, and J. J. Gadbow, Jr. 1984. Bacteriology of the teeth from a great white shark: potential medical implications for shark bite victims. *J. Clin. Microbiol.* **20**:849–851.
- Burde, D. R., D. Scheifele, and D. P. Speert. 1985. Serious *Pasteurella multocida* infections from lion and tiger bites. *JAMA* **253**:3296–3297.
- Calattini, S., et al. 2007. Simian foamy virus transmission from apes to humans, rural Cameroon. *Emerg. Infect. Dis.* **13**:1314–1320.
- Caldicott, D. G., D. Croser, C. Manolis, G. Webb, and A. Britton. 2005. Crocodile attack in Australia: an analysis of its incidence and review of the pathology and management of crocodilian attacks in general. *Wilderness Environ. Med.* **16**:143–159.
- Capellan, J., and I. W. Fong. 1993. Tularemia from a cat bite: case report and review of feline-associated tularemia. *Clin. Infect. Dis.* **16**:472–475.
- Cardall, T. Y., and P. Rosen. 2003. Grizzly bear attack. *J. Emerg. Med.* **24**:331–333.
- Carithers, H. A. 1958. Mammalian bites of children; a problem in accident prevention. *AMA J. Dis. Child.* **95**:150–156.
- Carpenter, P. D., B. T. Heppner, and J. W. Gnnann, Jr. 1987. DF-2 bacteremia following cat bites. Report of two cases. *Am. J. Med.* **82**:621–623.
- Carpenter, T. E., et al. 1989. *Pasteurella multocida* recovered from live turkeys: prevalence and virulence in turkeys. *Avian Dis.* **33**:12–17.
- Cefai, C., S. Ashurst, and C. Owens. 1994. Human carriage of methicillin-resistant *Staphylococcus aureus* linked with pet dog. *Lancet* **344**:539–540.
- Centers for Disease Control and Prevention. 1994. Human plague—United States, 1993–1994. *MMWR Morb. Mortal. Wkly. Rep.* **43**:242–246.
- Centers for Disease Control and Prevention. 1998. Fatal *Cercarial dermatitis* (B virus) infection following a mucocutaneous exposure and interim recommendations for worker protection. *MMWR Morb. Mortal. Wkly. Rep.* **47**:1073–1076.
- Centers for Disease Control and Prevention. 1999. Multiple human exposures to a rabid bear cub at a petting zoo and barnwarming—Iowa, August 1999. *MMWR Morb. Mortal. Wkly. Rep.* **48**:761.
- Centers for Disease Control and Prevention. 2005. Tularemia associated

- with hamster bite—Colorado 2004. MMWR Morb. Mortal. Wkly. Rep. 53:1202–1203.
38. Centers for Disease Control and Prevention. 2006. Human plague—four states, 2006. MMWR Morb. Mortal. Wkly. Rep. 55:940–943.
 39. Centers for Disease Control and Prevention. 2009. Imported human rabies—California, 2008. MMWR Morb. Mortal. Wkly. Rep. 58:713–716.
 40. Chippaux, J. P. 1998. Snake-bites: appraisal of the global situation. Bull. World Health Organ. 76:515–524.
 41. Chomel, B. B., et al. 1996. Experimental transmission of *Bartonella henselae* by the cat flea. J. Clin. Microbiol. 34:1952–1956.
 42. Citron, D. M., et al. 1996. Frequency of isolation of *Porphyromonas* species from infected dog and cat bite wounds in humans and their characterization by biochemical tests and arbitrary primed-polymerase chain reaction fingerprinting. Clin. Infect. Dis. 23(Suppl. 1):S78–S82.
 43. Citron, D. M., K. L. Tyrrell, and E. J. C. Goldstein. 2010. Anaerobic and aerobic bacteria in the saliva of Komodo dragons (*Varanus komodoensis*), abstr. 60. Abstr. Proc. 7th Bienn. Meet. Anaerobe Soc. Am., Philadelphia, PA.
 44. Clark, R. F., B. S. Selden, and B. Furbee. 1993. The incidence of wound infection following crotalid envenomation. J. Emerg. Med. 11:583–586.
 45. Cohen, J. I., et al. 2002. Recommendations for prevention of and therapy for exposure to B virus (*Cercopithecine Herpesvirus 1*). Clin. Infect. Dis. 35:1191–1203.
 46. Conrads, G., D. M. Citron, and E. J. Goldstein. 2005. Genetic determinant of intrinsic quinolone resistance in *Fusobacterium canifelinum*. Antimicrob. Agents Chemother. 49:434–437.
 47. Conrads, G., D. M. Citron, R. Mutters, S. Jang, and E. J. Goldstein. 2004. *Fusobacterium canifelinum* sp. nov., from the oral cavity of cats and dogs. Syst. Appl. Microbiol. 27:407–413.
 48. Craven, R. B., G. O. Maupin, M. L. Beard, T. J. Quan, and A. M. Barnes. 1993. Reported cases of human plague infections in the United States, 1970–1991. J. Med. Entomol. 30:758–761.
 49. Crothers, S. L., S. D. White, P. J. Ihrke, and V. K. Affolter. 2009. Sporotrichosis: a retrospective evaluation of 23 cases seen in northern California (1987–2007). Vet. Dermatol. 20:249–259.
 50. Davis, B., and R. P. Wenzel. 1992. Striges scalp: *Bacteroides* infection after an owl attack. J. Infect. Dis. 165:975–976.
 51. Declercq, P., D. Petré, B. Gordts, and A. Voss. 2008. Complicated community-acquired soft tissue infection by MRSA from porcine origin. Infection 36:590–592.
 52. De Giorgio, F., J. Rainio, V. Pascali, and K. Lalu. 2007. Bear attack—a unique fatality in Finland. Forensic Sci. Int. 173:64–67.
 53. de Lima Barros, M. B., A. de Oliveira Schubach, M. C. Galhardo, T. M. Schubach, R. S. dos Reis, M. J. Conceição, and A. C. do Valle. 2003. Sporotrichosis with widespread cutaneous lesions: report of 24 cases related to transmission by domestic cats in Rio de Janeiro, Brazil. Int. J. Dermatol. 42:677–681.
 54. Del Prete, R., et al. 1999. Prevalence of antibodies to *Bartonella henselae* in patients with suspected cat scratch disease (CSD) in Italy. Eur. J. Epidemiol. 15:583–587.
 55. Del Prete, R., D. Fumarola, L. Fumarola, and G. Miragliotta. 2000. Detection of *Bartonella henselae* and *Afpia felis* DNA by polymerase chain reaction in specimens from patient with cat scratch disease. Eur. J. Clin. Microbiol. Infect. Dis. 19:964–967.
 56. Del Prete, R., D. Fumarola, S. Ungari, L. Fumarola, and G. Miragliotta. 2000. Polymerase chain reaction detection of *Bartonella henselae* bacteraemia in an immunocompetent child with cat-scratch disease. Eur. J. Pediatr. 159:356–359.
 57. Devriese, L. A., K. Hermans, M. Baele, and F. Haesebrouck. 2009. *Staphylococcus pseudintermedius* versus *Staphylococcus intermedius*. Vet. Microbiol. 133:206–207.
 58. Devriese, L. A., et al. 2005. *Staphylococcus pseudintermedius* sp. nov., a coagulase-positive species from animals. Int. J. Syst. Evol. Microbiol. 55:1569–1573.
 59. Dibb, W. L., A. Digranes, and S. Tønnum. 1981. *Actinobacillus lignieresii* infection after a horse bite. Br. Med. J. (Clin. Res. Ed.) 283:583–584.
 60. Doll, J. M., et al. 1994. Cat-transmitted fatal pneumonic plague in a person who traveled from Colorado to Arizona. Am. J. Trop. Med. Hyg. 51:109–114.
 61. Duncan, A. W., R. G. Maggi, and E. B. Breitschwerdt. 2007. *Bartonella* DNA in dog saliva. Emerg. Infect. Dis. 13:1948–1950.
 62. Dunstan, R. W., R. F. Langham, K. A. Reimann, and P. S. Wakenell. 1986. Feline sporotrichosis: a report of five cases with transmission to humans. J. Am. Acad. Dermatol. 15:37–45.
 63. DuVernoy, T. S., K. C. Mitchell, R. A. Myers, L. W. Walinski, and M. O. Tinsley. 2008. The first laboratory-confirmed rabid pig in Maryland, 2003. Zoonoses Public Health 55:431–435.
 64. Ebrey, R. J., and L. J. Hayek. 1997. Antibiotic prophylaxis after swan bite. Lancet 350:340.
 65. Edixhoven, P., S. C. Sinha, and D. J. Dandy. 1981. Horse injuries. Injury 12:279–282.
 66. Ejlersen, T., B. Gahm-Hansen, P. Søgaard, O. Heltberg, and W. Frederiksen. 1996. *Pasteurella aerogenes* isolated from ulcers or wounds in humans with occupational exposure to pigs: a report of 7 Danish cases. Scand. J. Infect. Dis. 28:567–570.
 67. Eke, P. I., L. Braswell, R. Arnold, and M. Fritz. 1993. Sub-gingival microflora in Macaca mulatta species of rhesus monkeys. J. Periodontol. Res. 28:72–80.
 68. Elliott, S. P. 2007. Rat bite fever and *Streptobacillus moniliformis*. Clin. Microbiol. Rev. 20:13–22.
 69. Emet, M., et al. 2009. Animal-related injuries: epidemiological and meteorological features. Ann. Agric. Environ. Med. 16:87–92.
 70. Escande, F., A. Bailly, S. Bone, and J. Lemozy. 1996. *Actinobacillus suis* infection after a pig bite. Lancet 348:888.
 71. Escande, F., E. Vallee, and F. Aubart. 1997. *Pasteurella caballi* infection following a horse bite. Zentralbl. Bakteriol. 285:440–444.
 72. Eslamifard, A., et al. 2008. Animal bites in Teheran, Iran. Arch. Iran. Med. 11:200–202.
 73. Evans, M. E., Z. A. McGee, P. T. Hunter, and W. Schaffner. 1981. Tularemia and the tomcat. JAMA 246:1343.
 74. Fischer, R. G., S. Edwardsson, and B. Klinge. 1994. Oral microflora of the ferret at the gingival sulcus and mucosa membrane in relation to ligature-induced periodontitis. Oral Microbiol. Immunol. 9:40–49.
 75. Fitzgerald, J. R. 2009. The *Staphylococcus intermedius* group of bacterial pathogens: species re-classification, pathogenesis and the emergence of methicillin resistance. Vet. Dermatol. 20:490–495.
 76. Flandry, F., et al. 1989. Initial antibiotic therapy for alligator bites: characterization of the oral flora of *Alligator mississippiensis*. South. Med. J. 82:262–266.
 77. Fleury, R. N., et al. 2001. Zoonotic sporotrichosis. Transmission to humans by infected domestic cat scratching: report of four cases in São Paulo, Brazil. Int. J. Dermatol. 40:318–322.
 78. Floyd, T. 1999. Bear-inflicted human injury and fatality. Wilderness Environ. Med. 10:75–87.
 79. Floyd, T., A. M. Manville, and S. P. French. 1990. Normal oral flora in black bears: guidelines for antimicrobial prophylaxis following bear attacks. J. Wilderness Med. 1:47–49.
 - 79a. Forrester, M. B. 2010. Iguana bites reported to Texas poison centers. Am. J. Emerg. Med. 28:817–819.
 80. Frank, L. A., S. A. Kania, E. M. Kirzeder, L. C. Eberlein, and D. A. Bemis. 2009. Risk of colonization or gene transfer to owners of dogs with methicillin-resistant *Staphylococcus pseudintermedius*. Vet. Dermatol. 20:496–501.
 81. Freitas, D. F., A. C. do Valle, R. de Almeida Paes, F. I. Bastos, and M. C. Galhardo. 2010. Zoonotic sporotrichosis in Rio de Janeiro, Brazil: a protracted epidemic yet to be curbed. Clin. Infect. Dis. 50:453.
 82. Fry, B. G., et al. 2009. A central role for venom in predation by *Varanus komodoensis* (Komodo dragon) and the extinct giant *Veranus (Megalania) prisca*. Proc. Natl. Acad. Sci. U. S. A. 106:8969–8974. doi:10.1073/pnas.0810883106.
 83. Funke, G., R. Frodl, K. A. Bernard, and R. Englert. 2009. *Corynebacterium freiburgense* sp. nov., isolated from a wound obtained from a dog bite. Int. J. Syst. Evol. Microbiol. 59:2054–2057.
 84. Gage, K. L., et al. 2000. Cases of cat-associated human plague in the Western US, 1977–1998. Clin. Infect. Dis. 30:893–900.
 85. Ganiere, J. P., F. Escande, G. Andre, and M. Larrat. 1993. Characterization of *Pasteurella* from gingival scrapings of dogs and cats. Comp. Immunol. Microbiol. Infect. Dis. 16:77–85.
 86. Garg, A., S. Sujatha, J. Garg, N. S. Acharya, and S. C. Parija. 2009. Wound infections secondary to snakebite. J. Infect. Dev. Ctries. 3:221–223.
 87. Georghiou, P. R., T. F. Mollie, and M. H. Tilse. 1992. *Pasteurella multocida* infection after a Tasmanian devil bite. Clin. Infect. Dis. 14:1266–1267.
 88. Glaser, C., P. Lewis, and S. Wong. 2000. Pet-, animal- and vector-borne infections. Pediatr. Rev. 21:219–232.
 89. Gnann, J. W., Jr., G. S. Bressler, C. A. Bodet III, and C. K. Avent. 1983. Human blastomycosis after a dog bite. Ann. Intern. Med. 98:48–49.
 90. Goldstein, E. J. C. 1992. Bite wounds and infection. Clin. Infect. Dis. 14:633–640.
 91. Goldstein, E. J. C. 1998. New horizons in the bacteriology, antimicrobial susceptibility and therapy of animal bite wounds. J. Med. Microbiol. 47:95–97.
 92. Goldstein, E. J. C. 1999. Current concepts on animal bites: bacteriology and therapy. Curr. Clin. Top. Infect. Dis. 19:99–111.
 93. Goldstein, E. J. C., D. M. Citron, and S. M. Finegold. 1980. Dog bite wounds and infection: a prospective clinical study. Ann. Emerg. Med. 9:508–512.
 94. Goldstein, E. J., D. M. Citron, H. Gonzalez, F. E. Russell, and S. M. Finegold. 1979. Bacteriology of rattlesnake venom and implications for therapy. J. Infect. Dis. 140:818–821.
 95. Goldstein, E. J. C., D. M. Citron, T. E. Merkin, and M. J. Pickett. 1990. Recovery of an unusual *Flavobacterium* IIB-like isolate from a hand infection following pig bite. J. Clin. Microbiol. 28:1079–1081.
 96. Goldstein, E. J. C., et al. 1978. Bacteriology of human and animal bite wounds. J. Clin. Microbiol. 8:667–672.

97. Goldstein, E. J., E. P. Pryor III, and D. M. Citron. 1995. Simian bites and bacterial infection. *Clin. Infect. Dis.* **20**:1551–1552.
98. Gollop, J. H., A. R. Katz, R. C. Rudoy, and D. M. Sasaki. 1993. Rat-bite leptospirosis. *West. J. Med.* **159**:76–77.
99. Gould, L. H., J. Pape, P. Ettestad, K. S. Griffith, and P. S. Mead. 2008. Dog-associated risk factors for human plague. *Zoonoses Public Health* **55**:448–454.
100. Graham, W. R., Jr., and J. L. Callaway. 1982. Primary inoculation blastomycosis in a veterinarian. *J. Am. Acad. Dermatol.* **7**:785–786.
101. Grim, K. D., C. Doherty, and T. Rosen. 2010. *Serratia marcescens* bullous cellulitis after iguana bites. *J. Am. Acad. Dermatol.* **62**:1075–1076.
102. Gross, E. M., and V. Torok. 1984. Monkey-caused injuries in the region served by the Beer Sheva district health office, 1978–1982. *Isr. J. Med. Sci.* **20**:725–726.
103. Gruen, R. L. 2009. Crocodile attacks in Australia: challenges for injury prevention and trauma care. *World J. Surg.* **33**:1554–1561.
104. Guibourdenche, M., T. Lambert, and J. Y. Riou. 1989. Isolation of *Neisseria canis* in mixed culture from a patient after a cat bite. *J. Clin. Microbiol.* **27**:1673–1674.
105. Hall, A. J., et al. 2010. Novel *Corynebacterium diphtheriae* in domestic cats. *Emerg. Infect. Dis.* **16**:688–691.
106. Herrero, S. 1970. Human injury inflicted by grizzly bears. *Science* **170**:593–598.
107. Herrero, S., and S. Fleck. 1990. Injury to people inflicted by black, grizzly, and polar bears: recent trends and new insights. *Proc. Int. Conf. Bears Res. Manage.* **8**:25.
108. Hertner, G. 2006. Caiman bite. *Wilderness Environ. Med.* **17**:267–270.
109. Hicklin, H., A. Verghese, and S. Alvarez. 1987. Dysgonic fermenter 2 septicemia. *Rev. Infect. Dis.* **9**:884–890.
110. Hirsh, D. C., et al. 1990. Characteristics of *Pasteurella multocida* isolated from waterfowl and associated avian species in California. *J. Wildl. Dis.* **26**:204–209.
111. Hoekstra, K. A., and R. J. Paulton. 2002. Clinical prevalence and antimicrobial susceptibility of *Staphylococcus aureus* and *Staph. intermedius* in dogs. *J. Appl. Microbiol.* **93**:406–413.
112. Hofer, M., et al. 1993. Disseminated osteomyelitis from *Mycobacterium ulcerans* after a snakebite. *N. Engl. J. Med.* **328**:1007–1009.
113. Hoke, C., and N. A. Vedros. 1982. Characterization of atypical aerobic Gram-negative cocci isolated from humans. *J. Clin. Microbiol.* **15**:906–914.
114. Holmes, N. E., and T. M. Korman. 2007. *Corynebacterium kutscheri* infection of the skin and soft tissue following rat bite. *J. Clin. Microbiol.* **45**:3468–3469.
115. Holst, E., J. Roloff, L. Larsson, and J. P. Nielsen. 1992. Characterization and distribution of *Pasteurella* species recovered from infected humans. *J. Clin. Microbiol.* **30**:2984–2987.
116. Hovenga, S., et al. 1997. Dog-bite induced sepsis: a report of four cases. *Intensive Care Med.* **23**:1179–1180.
117. Hsieh, S., and F. E. Babl. 1999. *Serratia marcescens* cellulitis following an iguana bite. *Clin. Infect. Dis.* **28**:1181–1182.
118. Hudspeth, M. K., S. Hunt Gerardo, D. M. Citron, and E. J. Goldstein. 1997. Growth characteristics and a novel method for identification (the WEE-TAB system) of *Porphyromonas* species isolated from infected dog and cat bite wounds in humans. *J. Clin. Microbiol.* **35**:2450–2453.
119. Huijsdens, X. W., et al. 2006. Community-acquired MRSA and pig-farming. *Ann. Clin. Microbiol. Antimicrob.* **5**:26.
120. Ichhpujani, R. L., et al. 2008. Epidemiology of animal bites and rabies cases in India. A multicentric study. *J. Commun. Dis.* **40**:27–36.
121. Inzana, T. J., J. L. Johnson, L. Shell, K. Möller, and M. Kilian. 1992. Isolation and characterization of a newly identified *Haemophilus* species from cats: “*Haemophilus felis*.” *J. Clin. Microbiol.* **30**:2108–2112.
122. Isotalo, P. A., D. Edgar, and B. Toye. 2000. Polymicrobial tenosynovitis with *Pasteurella multocida* and other Gram negative bacilli after a Siberian tiger bite. *J. Clin. Pathol.* **53**:871–872.
123. Jackson, D. 1944. Management of snake bite, p. 882. In Z. T. Bercovitz (ed.), *Clinical tropical medicine*. P. Hoeber, Inc., New York, NY.
124. Jaspers, R. H. 1974. Transmission of Blastomyces from animals to man. *J. Am. Vet. Med. Assoc.* **164**:8.
125. Jones, J. W., J. V. Pether, H. A. Rainey, and C. R. Swinburn. 1993. Recurrent *Mycobacterium bovis* infection following a ferret bite. *J. Infect.* **26**:225–226.
126. Jones-Engel, L., et al. 2006. Temple monkeys and health implications of commensalism, Kathmandu, Nepal. *Emerg. Infect. Dis.* **12**:900–906.
127. Jorge, M. T., L. A. Ribeiro, M. L. da Silva, E. J. Kusano, and J. S. de Mendonça. 1994. Microbiological studies of abscesses complicating Bothrops snakebite in humans: a prospective study. *Toxicol.* **32**:743–748.
128. Kasturiratne, A., et al. 2008. The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. *PLoS Med.* **5**:e218. doi:10.1371/journal.pmed.0050218.
129. Keret, D., M. Giladi, Y. Kletter, and S. Wientroub. 1998. Cat-scratch disease osteomyelitis from a dog scratch. *J. Bone Joint Surg. Br.* **80**:766–767.
130. Kerkhoff, F. T., J. M. Ossewaarde, W. S. de Loos, and A. Rothova. 1999. Presumed ocular bartonellosis. *Br. J. Ophthalmol.* **83**:270–275.
131. Kerrigan, K. R. 1992. Bacteriology of snakebite abscess. *Trop. Doct.* **22**:158–160.
132. Kerrigan, K. R., B. L. Mertz, S. J. Nelson, and J. D. Dye. 1997. Antibiotic prophylaxis for pit viper envenomation: prospective, controlled trial. *World J. Surg.* **21**:369–372.
133. Khanna, T., R. Friendship, C. Dewey, and J. S. Weese. 2008. Methicillin resistant *Staphylococcus aureus* colonization in pigs and pig farmers. *Vet. Microbiol.* **128**:298–303.
134. Kim, B. H., J. E. Phillips, and J. G. Atherton. 1976. *Actinobacillus suis* in the horse. *Vet. Rec.* **98**:239.
135. Kizer, K. W. 1979. Epidemiological and clinical aspects of animal bite injuries. *JACEP* **8**:134–141.
136. Kizer, K. W. 1989. *Pasteurella multocida* infection from a cougar bite. A review of cougar attacks. *West. J. Med.* **150**:87–90.
137. Köck, R., et al. 2009. Prevalence and molecular characteristics of methicillin-resistant *Staphylococcus aureus* (MRSA) among pigs on German farms and import of livestock-related MRSA into hospitals. *Eur. J. Clin. Microbiol. Infect. Dis.* **28**:1375–1382.
138. Kordick, D. L., et al. 1997. *Bartonella clarridgeiae*, a newly recognized zoonotic pathogen causing inoculation papules, fever, and lymphadenopathy (cat scratch disease). *J. Clin. Microbiol.* **35**:1813–1818.
139. Kullberg, B. J., R. J. Westendorp, J. W. van’t Wout, and A. E. Meinders. 1991. Purpura fulminans and symmetrical peripheral gangrene caused by *Capnocytophaga canimorsus* (formerly DF-2) septicemia—a complication of dog bite. *Medicine* **70**:287–292.
140. Kunimoto, D., R. Rennie, D. M. Citron, and E. J. C. Goldstein. 2004. Bacteriology of a bear bite wound to a human: case report. *J. Clin. Microbiol.* **42**:3374–3376.
141. Lam, K. K., et al. 2011. A cross-sectional survey of snake oral bacterial flora from Hong Kong, SAR, China. *Emerg. Med. J.* **28**:107–114.
142. Langley, R. L. 2005. Alligator attacks on humans in the United States. *Wilderness Environ. Med.* **16**:119–124.
143. Langley, R., and T. Morris. 2009. That horse bit me: zoonotic infections of equines to consider after exposure through the bite or the oral/nasal secretions. *J. Agromedicine* **14**:370–381.
144. Lappin, M. R. 1993. Feline zoonotic diseases. *Vet. Clin. North Am. Small Anim. Pract.* **23**:57–78.
145. Lehtinen, V. A., et al. 2005. *Mycobacterium fortuitum* infection after a brown bear bite. *J. Clin. Microbiol.* **43**:1009.
146. Lentsch, R. H., and J. E. Wagner. 1980. Isolation of *Actinobacillus lignieresii* and *Actinobacillus equuli* from laboratory rodents. *J. Clin. Microbiol.* **12**:351–354.
147. Lindberg, J., W. Frederiksen, B. Gahm-Hansen, and B. Bruun. 1998. Problems of identification in clinical microbiology exemplified by pig bite wound infections. *Zentralbl. Bakteriol.* **288**:491–499.
148. Lion, C., M. C. Conroy, M. L. Dupuy, and F. Escande. 1995. *Pasteurella* “SP” group infection after a guinea pig bite. *Lancet* **346**:901–902.
149. Lockhart, W. E. 1965. Treatment of snakebite. *JAMA* **193**:36–38.
150. LoVecchio, F., J. Klemens, S. Welch, and R. Rodriguez. 2002. Antibiotics after rattlesnake envenomation. *J. Emerg. Med.* **23**:327–328.
151. Luzzi, G. A., L. M. Milne, and S. A. Waitkins. 1987. Rat-bite acquired leptospirosis. *J. Infect.* **15**:57–60.
152. MacDonald, P. D., R. L. Langley, S. R. Gerkin, M. R. Torok, and J. N. MacCormack. 2006. Human and canine pulmonary blastomycosis, North Carolina, 2001–2002. *Emerg. Infect. Dis.* **12**:1242–1244.
153. Maguina, C., and E. Gotuzzo. 2000. Bartonellosis: new and old. *Infect. Dis. Clin. North Am.* **14**:1–22.
154. Manian, F. A. 2003. Asymptomatic nasal carriage of mupirocin-resistant, methicillin-resistant *Staphylococcus aureus* (MRSA) in a pet dog associated with MRSA infection in household contacts. *Clin. Infect. Dis.* **36**:e26–e28.
155. Margileth, A. M. 1993. Cat scratch disease. *Adv. Pediatr. Infect. Dis.* **8**:1–21.
156. Marrie, T. J., J. M. Bent, A. B. West, T. M. Roberts, and E. V. Haldane. 1979. Extensive gas in tissues of the forearm after horse bite. *South. Med. J.* **72**:1473–1474.
157. Reference deleted.
158. Mellor, D. J., S. Bhandari, K. Kerr, and A. R. Bodenham. 1997. Man’s best friend: life threatening sepsis after minor dog bite. *BMJ* **314**:85–156.
159. Minton, S. A. 1996. Bites by non-native venomous snakes in the United States. *Wilderness Environ. Med.* **7**:297–303.
160. Mittermayer, T. 1972. Case of glandular form of tularemia acquired during hamster-hunting. *Cesk. Epidemiol. Mikrobiol. Immunol.* **21**:263–265. (In Czech.)
161. Montejo, M., et al. 2001. *Bergeyella zoohelcum* bacteremia after a dog bite. *Clin. Infect. Dis.* **33**:1608–1609.
162. Montgomery, J. M., D. Gillespie, P. Sastrawan, T. M. Fredekind, and G. L. Stewart. 2002. Aerobic salivary bacteria in wild and captive Komodo dragons. *J. Wildl. Dis.* **38**:545–551.
163. Mutombo, M., I. Arita, and Z. Jezek. 1983. Human monkeypox transmitted by a chimpanzee in a tropical rain-forest area of Zaire. *Lancet* **i**:735–737.
164. Nabi, D. G., S. R. Tak, K. A. Kangoo, and M. A. Halwai. 2009. Increasing

- incidence of injuries and fatalities inflicted by wild animals in Kashmir. *Injury* **40**:87–89.
165. Nahass, R. G., and T. Nordstrom. 1993. Anaerobic osteomyelitis following a horse bite. *Infect. Dis. Clin. Pract.* **2**:357–358.
 166. Ndon, J. A. 1992. *Capnocytophaga canimorsus* septicemia caused by a dog bite in a hairy cell leukemia patient. *J. Clin. Microbiol.* **30**:211–213.
 167. Nielsen, J. P., M. Bisgaard, and K. B. Pedersen. 1986. Production of toxin in strains previously classified as *Pasteurella multocida*. *Acta Pathol. Microbiol. Immunol. Scand. B* **94**:203–204.
 168. Nishioka, S. A., S. T. Handa, and R. S. Nunes. 1994. Pig bite in Brazil: a case series from a teaching hospital. *Rev. Soc. Bras. Med. Trop.* **27**:15–18.
 169. Oehler, R. L., A. P. Velez, M. Mizrachi, J. Lamarche, and S. Gompf. 2009. Bite-related and septic syndromes caused by cats and dogs. *Lancet Infect. Dis.* **9**:439–447.
 170. Ohad, D. G., D. Morick, B. Avidor, and S. Harrus. 2010. Molecular detection of *Bartonella henselae* and *Bartonella koehlerae* from aortic valves of Boxer dogs with infective endocarditis. *Vet. Microbiol.* **141**:182–185.
 171. Ordog, G. J. 1986. The bacteriology of dog bite wounds on initial presentation. *Ann. Emerg. Med.* **15**:1324–1329.
 172. Ordog, G. J., S. Balasubramaniam, and J. Wasserberger. 1985. Rat bites: fifty cases. *Ann. Emerg. Med.* **14**:126–130.
 173. Osterlund, A., and E. Nordlund. 1997. Wound infection caused by *Staphylococcus hyicus* subspecies *hyicus* after a donkey bite. *Scand. J. Infect. Dis.* **29**:95.
 174. Owen, M. R., A. P. Moores, and R. J. Coe. 2004. Management of MRSA septic arthritis in a dog using a gentamicin-impregnated collagen sponge. *J. Small Anim. Pract.* **45**:609–612.
 175. Parry, R. G., R. Ziemis, H. Reynolds, and S. Miller. 1983. Brown/grizzly bear mouth cultures in Alaska. *Alaska Med.* **25**:1–2.
 176. Pavia, A. T., J. A. Bryan, K. L. Maher, T. R. Hester, Jr., and J. J. Farmer. 1989. *Vibrio carchariae* infection after a shark bite. *Ann. Intern. Med.* **111**:85–86.
 177. Peel, M. M., K. A. Hornidge, M. Luppino, A. M. Stacpoole, and R. E. Weaver. 1991. *Actinobacillus* spp. and related bacteria in infected wounds of humans bitten by horses and sheep. *J. Clin. Microbiol.* **29**:2535–2538.
 178. Pers, C., B. Gahrn-Hansen, and W. Frederiksen. 1996. *Capnocytophaga canimorsus* septicemia in Denmark, 1982–1995: review of 39 cases. *Clin. Infect. Dis.* **23**:71–75.
 179. Pottumarthy, S., et al. 2004. Clinical isolates of *Staphylococcus intermedius* masquerading as methicillin-resistant *Staphylococcus aureus*. *J. Clin. Microbiol.* **42**:5881–5884.
 180. Räisänen, S., and A. Alavaikko. 1989. *Yersinia* infection following a horse bite. *Duodecim* **105**:1496–1497. (In Finnish.)
 181. Ramsey, D. T. 1994. Blastomycosis in a veterinarian. *J. Am. Vet. Med. Assoc.* **205**:968.
 182. Rankin, S., et al. 2005. Panton Valentine leukocidin (PVL) toxin positive MRSA strains isolated from companion animals. *Vet. Microbiol.* **108**:145–148.
 183. Rao, N., and S. Jain. 1999. *Pasteurella multocida* meningitis from animal exposure. *Infect. Dis. Clin. Pract.* **8**:307–309.
 184. Rayan, G. M., D. J. Flournoy, and S. L. Cahill. 1987. Aerobic mouth flora of the rhesus monkey. *J. Hand Surg. Am.* **12**:299–301.
 185. Read, S. I., and L. C. Sperling. 1982. Feline sporotrichosis. Transmission to man. *Arch. Dermatol.* **118**:429–431.
 186. Reed, K. D., F. M. Moore, G. E. Geiger, and M. E. Stemper. 1993. Zoonotic transmission of sporotrichosis: case report and review. *Clin. Infect. Dis.* **16**:384–387.
 187. Reina, J., and N. Borrell. 1992. Leg abscess caused by *Weeksella zoohelcum* following a dog bite. *Clin. Infect. Dis.* **14**:1162–1163.
 188. Risholt, T., E. Persen, and O. I. Solem. 1998. Man and polar bear in Svalbard: a solvable ecological conflict? *Int. J. Circumpolar Health* **57**(Suppl. 1):532–534.
 189. Roblot, P., M. Bazillou, G. Grollier, B. Becq-Giraudon, and J. L. Fauchère. 1993. Septicemia due to *Capnocytophaga canimorsus* after a dog bite in a cirrhotic patient. *Eur. J. Clin. Microbiol. Infect. Dis.* **12**:302–303.
 190. Rolain, J. M., et al. 2004. Recommendations for treatment of human infections caused by *Bartonella* species. *Antimicrob. Agents Chemother.* **48**:1921–1933.
 191. Rolfe, U. 2000. *Haemophilus influenzae* cellulitis after bite injuries in children. *J. Pediatr. Surg.* **35**:1408–1409.
 192. Rolloff, J., G. Nordin-Fredriksson, and E. Holst. 1989. *Pasteurella multocida* occurs in a high frequency in the saliva of pet dogs. *Scand. J. Infect. Dis.* **21**:583–584.
 193. Rose, S. C. 1982. Bear maulings in Alaska. *Alaska Med.* **24**:29–32.
 194. Royle, J. A., et al. 1997. Infections after shark attacks in Australia. *Pediatr. Infect. Dis. J.* **16**:531–532.
 195. Saphir, D. A., and G. R. Carter. 1976. Gingival flora of the dog with special reference to bacteria associated with bites. *J. Clin. Microbiol.* **3**:344–349.
 196. Sarma, P. S. 2002. *Aeromonas jandaei* cellulitis and bacteremia in a man with diabetes. *Am. J. Med.* **112**:325.
 197. Sarma, P. S., and S. Mohanty. 2001. *Capnocytophaga cynodegmi* cellulitis, bacteremia, and pneumonitis in a diabetic man. *J. Clin. Microbiol.* **39**:2028–2029.
 198. Sarosi, G. A., M. R. Eckman, S. F. Davies, and W. K. Laskey. 1979. Canine blastomycosis as a harbinger of human disease. *Ann. Intern. Med.* **91**:733–735.
 199. Schubach, A., M. B. Barros, and B. Wanke. 2008. Epidemic sporotrichosis. *Curr. Opin. Infect. Dis.* **21**:129–133.
 200. Schweizer, M., V. Falcone, J. Gänge, R. Turek, and D. Neumann-Haefelin. 1997. Simian foamy virus isolated from an accidentally infected human individual. *J. Virol.* **71**:4821–4824.
 201. Shek, K. C., et al. 2009. Oral bacterial flora of the Chinese cobra (*Naja atra*) and bamboo pit viper (*Trimeresurus albolabris*) in Hong Kong SAR, China. *Hong Kong Med. J.* **15**:183–190.
 202. Shukla, S. K., et al. 2004. Isolation of a fastidious *Bergeyella* species associated with cellulitis after a cat bite and a phylogenetic comparison with *Bergeyella zoohelcum* strains. *J. Clin. Microbiol.* **42**:290–293.
 203. Smith, T. C., et al. 2009. Methicillin-resistant *Staphylococcus aureus* (MRSA) strain ST398 is present in midwestern U.S. swine and swine workers. *PLoS One* **4**:e4258. doi:10.1371/journal.pone.0004258.
 204. Suankratay, C., H. Wilde, P. Nuntaphisud, and M. Khantipong. 2002. Tetanus after white-lipped green pit viper (*Trimeresurus albolabris*) bite. *Wilderness Environ. Med.* **13**:256–261.
 205. Suzuki, M., M. Kimura, K. Imaoka, and A. Yamada. 2010. Prevalence of *Capnocytophaga canimorsus* and *Capnocytophaga cynodegmi* in dogs and cats determined by using a newly established species-specific PCR. *Vet. Microbiol.* **144**:172–176.
 206. Talan, D. A., D. M. Citron, F. M. Abrahamian, G. J. Moran, and E. J. C. Goldstein. 1999. Bacteriologic analysis of infected dog and cat bites. *N. Engl. J. Med.* **340**:85–92.
 207. Talan, D. A., et al. 1991. Antibacterial activity of crotalid venoms against oral snake flora and other clinical bacteria. *J. Infect. Dis.* **164**:195–198.
 208. Talan, D. A., E. J. Goldstein, D. Staatz, and G. D. Overturf. 1989. *Staphylococcus intermedius*: clinical presentation of a new human dog bite pathogen. *Ann. Emerg. Med.* **18**:410–413.
 209. Talan, D. A., et al. 1989. *Staphylococcus intermedius* in canine gingiva and canine-inflicted human wound infections: laboratory characterization of a newly recognized zoonotic pathogen. *J. Clin. Microbiol.* **27**:78–81.
 210. Tehrani, H., R. Tejero-Trujeque, and S. K. Dhital. 2008. Septic arthritis due to a Savannah monitor lizard bite: a case report. *J. Hand Surg. Eur.* **33**:810.
 211. Theakston, R. D., et al. 1990. Bacteriological studies of the venom and mouth cavities of wild Malayan pit vipers (*Calloselasma rhodostoma*) in southern Thailand. *Trans. R. Soc. Trop. Med. Hyg.* **84**:875–879.
 212. Thomas, P. R., and J. A. Buntine. 1987. Man's best friend? A review of the Austin hospital's experience with dog bites. *Med. J. Aust.* **147**:536–540.
 213. Tomlin, J., et al. 1999. Methicillin-resistant *Staphylococcus aureus* infections in 11 dogs. *Vet. Rec.* **144**:60–64.
 214. Tough, S. C., and J. C. Butt. 1993. A review of fatal bear maulings in Alberta, Canada. *Am. J. Forensic Med. Pathol.* **14**:22–27.
 215. Tribe, G. W., and E. Noren. 1983. Incidence of bites from cynomolgus monkeys, in attending animal staff—1975–80. *Lab. Anim.* **17**:110.
 216. Tsukahara, M., H. Tsuneoka, H. Iino, K. Ohno, and I. Murano. 1998. *Bartonella henselae* infection from a dog. *Lancet* **352**:1682.
 217. Valtonen, M., et al. 1995. *Capnocytophaga canimorsus* septicemia: fifth report of a cat-associated infection and five other cases. *Eur. J. Clin. Microbiol. Infect. Dis.* **14**:520–523.
 218. Van Belkum, A., et al. 2008. Methicillin-resistant and -susceptible *Staphylococcus aureus* sequence type 398 in pigs and humans. *Emerg. Infect. Dis.* **14**:479–483.
 219. Van Cleef, B. A., et al. 2010. High prevalence of nasal MRSA carriage in slaughterhouse workers in contact with live pigs in the Netherlands. *Epidemiol. Infect.* **138**:756–763.
 220. Vandamme, P., L. Debruyne, E. De Brandt, and E. Falsen. 2009. Reclassification of *Bacteroides ureolyticus* as *Campylobacter ureolyticus* comb. nov., and emended description of the genus *Campylobacter*. *Int. J. Syst. Evol. Microbiol.* **60**:2016–2022.
 221. Van Demark, R. E., Sr., and R. E. Van Demark, Jr. 1991. Swine bites of the hand. *J. Hand Surg. Am.* **16**:136–138.
 222. van Duijkeren, E., et al. 2004. Human-to-dog transmission of methicillin-resistant *Staphylococcus aureus*. *Emerg. Infect. Dis.* **10**:2235–2237.
 223. van Duijkeren, E., M. J. Wolfhagen, M. E. Heck, and W. J. Wannet. 2005. Transmission of a Panton-Valentine leukocidin-positive, methicillin-resistant *Staphylococcus aureus* strain between humans and a dog. *J. Clin. Microbiol.* **43**:6209–6211.
 224. van Loo, I., et al. 2007. Emergence of methicillin-resistant *Staphylococcus aureus* of animal origin in humans. *Emerg. Infect. Dis.* **13**:1834–1839.
 225. von Schroeder, H. P., and E. P. McDougall. 1993. Ulceroglandular and pulmonary tularemia: a case resulting from a cat bite to the hand. *J. Hand Surg. Am.* **18**:132–134.
 226. Voss, A., F. Loeffen, J. Bakker, C. Klaassen, and M. Wulf. 2005. Methicillin-resistant *Staphylococcus aureus* in pig farming. *Emerg. Infect. Dis.* **11**:1965–1966.

227. **Vougiouklakis, T.** 2006. Fatal brown bear (*Ursus arctos*) attack: case report and literature review. *Am. J. Forensic Med. Pathol.* **27**:266–267.
228. **Wamisho, B. L., et al.** 2009. Ward round—crocodile bites in Malawi: microbiological and surgical management. *Malawi Med. J.* **21**:29–31.
229. **Warrell, D. A.** 2010. Snake bite. *Lancet* **375**:77–88.
230. **Weber, D. J., J. S. Wolfson, M. N. Swartz, and D. C. Hooper.** 1984. *Pasteurella multocida* infections: report of 34 cases and review of the literature. *Medicine (Baltimore)* **63**:133–154.
231. **Weinberg, A. N., and J. A. Branda.** 2010. Case 31—2010: a 29-year-old woman with fever after a cat bite. *N. Engl. J. Med.* **363**:1560–1568.
232. **Weniger, B. G., et al.** 1984. Human bubonic plague transmitted by a domestic cat scratch. *JAMA* **251**:927–928.
233. **Werner, S. B., et al.** 1984. Primary plague pneumonia contracted from a domestic cat at South Lake Tahoe, CA. *JAMA* **251**:929–931.
234. **Westwell, A. J., M. B. Spencer, and K. G. Kerr.** 1987. DF-2 bacteremia following cat bites. *Am. J. Med.* **83**:1170.
235. **Windsor, J. J.** 2001. Cat-scratch disease: epidemiology, aetiology, and treatment. *Br. J. Biomed. Sci.* **58**:101–110.
236. **Woo, P. C., et al.** 2003. Usefulness of the MicroSeq 500 16S ribosomal DNA-based system for identification of clinically significant bacterial isolates with ambiguous biochemical profiles. *J. Clin. Microbiol.* **41**:1996–2001.
237. **Woolfrey, B. F., C. O. Quall, and R. T. Lally.** 1985. *Pasteurella multocida* in an infected tiger bite. *Arch. Pathol. Lab. Med.* **109**:744–746.
238. **Wulf, M. W., et al.** 2008. MRSA carriage in healthcare personnel in contact with farm animals. *J. Hosp. Infect.* **70**:186–190.
239. **Yegneswaran, P. P., et al.** 2009. Zoonotic sporotrichosis of lymphocutaneous type in a man acquired from a domesticated feline source: report of a first case in southern Karnataka, India. *Int. J. Dermatol.* **48**:1198–1200.
240. **Zangwill, K. M., et al.** 1993. Cat scratch disease in Connecticut. Epidemiology, risk factors, and evaluation of a new diagnostic test. *N. Engl. J. Med.* **329**:8–13.

Fredrick M. Abrahamian, D.O., F.A.C.E.P., is board certified in emergency medicine (EM) and has completed a research fellowship in infectious diseases. He is the director of education at the Olive View-UCLA (OV-UCLA) EM Residency Program and co-chairman of the OV-UCLA National Conferences on Advances in EM. He has served as the president of the faculty council and Education and Research Institute at the OV-UCLA Medical Center. He has been honored with the prestigious American College of Emergency Physicians (ACEP) Faculty Teaching Award in 2005, Council of EM Residency Directors Faculty Teaching Award in 2006, ACEP Scientific Assembly Rookie Speaker of the Year Award in 2007, and Education Award by the ACEP State Chapter of California in 2009. Dr. Abrahamian has been involved in numerous infectious disease-related research projects with publications in the *New England Journal of Medicine*, *Clinical Infectious Diseases*, and *Annals of Emergency Medicine*. His interests include animal bites, skin infections, tetanus, pneumonia, and pyelonephritis.



Ellie J. C. Goldstein, M.D., F.I.D.S.A., is Clinical Professor of Medicine, the David Geffen School of Medicine, UCLA; Director, R. M. Alden Research Laboratory; and in private practice in Santa Monica, CA. He has received the IDSA-Clinician of the Year Award and has over 360 publications. His interests include the diagnosis, pathogenesis, and therapy of anaerobic infections, including intra-abdominal infections, diabetic foot infections, *C. difficile*, human and animal bites, and the *in vitro* susceptibility of anaerobic bacteria to new antimicrobial agents. He is active in the Anaerobe Society of the Americas, the IDSA, ASM, and the Surgical Infection Society. He founded, and served as President, of the Infectious Diseases Association of California and the Anaerobe Society of the Americas. He is currently a Section Editor for *Clinical Infectious Diseases* and chair of the publications committee of *Anaerobe*. In the past he has served as an Associate Editor for *Clinical Infectious Diseases* and the *Journal of Medical Microbiology*.

