**Piscine Mycobacteriosis**

Fish Tuberculosis, Piscine Tuberculosis, Swimming Pool Granuloma, Fish Tank Granuloma, Fish Handler’s Disease, Fish Handler’s Nodules

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**Importance**

The genus *Mycobacterium* contains more than 150 species, including the obligate pathogens that cause tuberculosis in mammals as well as environmental saprophytes that occasionally cause opportunistic infections. At least 20 species are known to cause mycobacteriosis in fish. They include *Mycobacterium marinum*, some of its close relatives (e.g., *M. shottsi*, *M. pseudoshottsi*), common environmental organisms such as *M. fortuitum*, *M. chelonae*, *M. abscessus* and *M. gordoneae*, and less well characterized species such as *M. salmoniphilum* and *M. haemophilum*, among others. Piscine mycobacteriosis, which has a range of outcomes from subclinical infection to death, affects a wide variety of freshwater and marine fish. It has often been reported from aquariums, research laboratories and fish farms, but outbreaks also occur in free-living fish. The same organisms sometimes affect other vertebrates including people. Human infections acquired from fish are most often characterized by skin lesions of varying severity, which occasionally spread to underlying joints and tendons. Some lesions may be difficult to cure, especially in those who are immunocompromised.

**Etiology**

Mycobacteriosis is caused by members of the genus *Mycobacterium*, which are Gram-positive, acid fast, pleomorphic rods in the family Mycobacteriaceae and order Actinomyccetales. This genus is traditionally divided into two groups: the members of the *Mycobacterium tuberculosis* complex (e.g., *M. tuberculosis*, *M. bovis*, *M. caprae*, *M. pinnipedi*), which cause tuberculosis in mammals, and the nontuberculous mycobacteria. The organisms in the latter group include environmental saprophytes, which sometimes cause opportunistic infections, and other species such as *M. marinum*, which are more frequent pathogens. Nontuberculous mycobacteria can be further separated into fast-growing mycobacteria, which can form visible colonies on solid agar within a week (e.g., *M. fortuitum*, *M. chelonae*, *M. abscessus*), and slow growers (e.g., *M. marinum*, *M. ulcerans*, *M. haemophilum*, *M. gordonae*), which may take weeks or even months to become visible.

At least 20 species of nontuberculous mycobacteria, including both fast and slow growers, can cause disease in fish. They include *Mycobacterium marinum*, some other members of the *M. marinum* complex (e.g., *M. shottsi*, *M. pseudoshottsi*, *M. ulcerans*), *M. haemophilum*, *M. gordonae*, some members of the *M. fortuitum* group (e.g., *M. fortuitum*, *M. peregrinum*, *M. saopaulense*), and the *M. chelonae* *M. abscessus* complex (e.g., *M. abscessus*, *M. chelonae*, *M. salmonophilum*), and a number of less frequently isolated species such as *M. chesapeake*, *M. montefiorens*, *M. neoaurum*, *M. simiae*, *M. scrofulaceum* and *M. stephanolepidis*. Some of these organisms, such as *M. marinum* and *M. haemophilum*, appear to be relatively pathogenic to fish; others are often carried subclinically.

**Species Affected**

**The Mycobacterium marinum complex**

*M. marinum* has been described in at least 150 species of captive or free-living freshwater and marine fish, including eels and syngnathid fish (seahorses, pipefish, seadragons). Outbreaks seem to be especially common in captive Anabantidae (betta and gouramis), Characidae (tetras and piranhas), and Cyprinidae (danios and barbs), though this might be due to their relatively long lives rather than unusual susceptibility.

Information on the other members of the *M. marinum* complex is limited. *M. shottsi* has been found in striped bass (*Morone saxatilis*), which are also hosts for *M. pseudoshottsi* and *M. Chesapeake*. Other marine fish known to be infected with *M. pseudoshottsi* include yellow tail (Seriola quinqueraadiata) sea bream (*Sparus aurata*), white perch (*Morone americana*), Atlantic menhaden (*Brevoortia tyrannus*) and bay anchovy (*Anchoa mitchilli*), among others. Most cases were reported from fish farms. *M. ulcerans* is generally associated with stagnant water, and although it has been found in fish, they do not seem to act as reservoirs.
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Information on the other members of the *M. marinum* complex is limited. *M. shottsii* has been found in striped bass (*Morone saxatilis*), which are also hosts for *M. pseudoshottsii* and *M. chesapeake*. Other marine fish known to be infected with *M. pseudoshottsii* include yellow tail (*Seriola quinquergadiata*) sea bream (*Sparus aurata*), white perch (*Morone americana*), Atlantic menhaden (*Brevoortia tyrannus*) and bay anchovy (*Anchoa michilli*), among others. Most cases were reported from fish farms. *M. ulcerans* is generally associated with stagnant water, and although it has been found in fish, they do not seem to act as reservoirs.

*M. marinum* is among the more commonly reported species of *Mycobacterium* in sick amphibians. It has caused clinical cases in captive bullfrogs (*Rana catesbeiana*), Japanese forest green tree frogs (*Rhacophorus arboreus*), leopard frogs (*Rana pipiens*), a red-eyed tree frog (*Agalychnis callidryas*) and Hong Kong warty newts (*Paramesotriton hongkongensis*). It has also been found in reptiles, including Egyptian spiny-tailed lizards (*Uromastyx aegyptius*), a fly river tortoise (*Caretochelys insculpta*) and a bearded dragon (*Pogona vitticeps*). There are rare reports of *M. marinum* in marine mammals, pigs, cattle, camels, a European hedgehog (*Erinaceus europaeus*), a bilby (*Macrotis lagotis*) and an armadillo, and one report of disease in a bird, a blue-fronted Amazon parrot (*Amazona aestiva*). Experimental infections have been established in mice and bats.

Other species that cause piscine mycobacteriosis

Many organisms found in fish, such as the members of the *M. fortuitum* group and the *M. chelonei* *M. abscessus* complex, are common environmental organisms in soil, water and vegetation. They are not particularly associated with fish, but are reported sporadically in various vertebrates including amphibians, reptiles, mammals and birds, with or without clinical signs. Some of them might preferentially affect certain piscine hosts. *M. chelonei* and *M. salmoniphilum*, for instance, have been reported primarily in coldwater fish, especially salmonids. *M. salmoniphilum* can also cause disease in burbot (*Lota lota*), a freshwater species, and experimentally infected Atlantic cod (*Gadus morhua*). *M. haemophilum* has been seen in zebrafish (*Danio rerio*) and various pet/aquarium fish (e.g., Cuch’s blue tetra, *Boehliae fredcochui*; peacock gudgeon, *Tateurdina ocellicauda*).

Zoonotic potential

Mycobacteria that have been found at least occasionally in fish and are also known to affect humans include *M. marinum*, *M. ulcerans*, *M. haemophilum*, *M. abscessus*, *M. chelonei*, *M. fortuitum*, *M. peregrinum*, *M. gordonei*, *M. iranicum*, *M. neoaurum*, *M. florentinum* and *M. arupense*. *M. senegalese*, a species normally associated with cattle, caused mycobacteriosis in cuts from a broken fish tank in a child with no apparent link to cattle. Other organisms capable of causing piscine mycobacteriosis should probably also be considered potentially zoonotic. *M. pseudoshottsii* and *M. shottsii*, in particular, are closely related to *M. marinum* and are difficult to distinguish from this organism.

Geographic Distribution

Most of the bacteria that cause mycobacteriosis seem to be widespread, though information on some species is limited. *M. marinum* has been found worldwide in bodies of fresh, brackish and salt water. *M. ulcerans* has also been found in fish in many locations including North America, Europe and the Middle East. However, the disease it causes in humans (Buruli ulcer) is most common in tropical and subtropical rural areas of Africa, Latin America and the western Pacific. The subspecies *M. ulcerans* ssp. *shinshuense* causes Buruli ulcer in Japan and possibly some other parts of Asia.

Transmission

Fish are thought to acquire *M. marinum* by ingesting contaminated feed, cannibalizing infected fish or aquatic detritus, or inoculating the organism into the skin via injuries or external parasites. Transovarial transmission has been reported in at least one species of viviparous fish, *Platypoecilus maculatus*. Other mycobacteria that affect fish are probably acquired similarly.

There are many possible sources of organisms. Mycobacteria can occur in external lesions on the skin or gills of fish, and are sometimes shed from the gastrointestinal tract. They are also common in water and may be transmitted on fomites. *M. marinum* can contaminate various food sources including fish meal and infected live feed (e.g., tubificid worms, mosquito larvae, brine shrimp, rotifers). This organism is reported to remain viable in the environment (soil and water) for two years or more, and in carcasses and organs for up to one year. Many other mycobacteria are opportunistic saprophytes that normally reside in soil, vegetation, water and other environmental sources. Snails and other invertebrates seem to play a role in spreading some organisms.

Other vertebrates are probably infected by similar routes. Some *M. marinum*-infected reptiles were housed in tanks that previously held fish, and others seem to acquire this organism by eating infected fish or amphibians. People usually become infected through breaks in the skin after contact with contaminated water or infected fish. One
respiratory infection was probably acquired while mouth siphoning water from a fish tank. *M. marinum* has been isolated from unpasteurized milk, though no human infections were associated with this source, and from wells. *M. ulcerans* is unusual in that it is also thought to be transmitted mechanically by mosquitoes or other biting arthropods. Person-to-person transmission of nontuberculous mycobacteria generally seems to be absent or rare; however, *M. abscessus* has been transmitted between cystic fibrosis patients in clinics.

**Disinfection**

Mycobacteria are reported to be susceptible to alcohols, aldehydes, some alkalis, sodium hypochlorite, freshly prepared iodine compounds with a high concentration of available iodine, some peroxygen compounds and some phenols. However, they tend to be more resistant than many bacteria, and some agents require higher concentrations and/or longer contact times than usual. Species of *Mycobacterium* may differ in their susceptibility to specific agents. For instance, *M. marinum* and *M. fortuitum* were reported to be susceptible to 2% alkaline glutaraldehyde, while *M. gordonae* and *M. chelonae* were resistant; and *M. peregrinum*, *M. chelonae* and *M. abscessus* were significantly less susceptible to bleach than *M. gordonae* in one study. Mycobacteria can also be inactivated by temperatures greater than 65°C (149°F) for 30 minutes or longer, and by UV light.

**Infections in Animals**

**Incubation Period**

Piscine mycobacteriosis usually takes several weeks to months to become apparent. Experimentally infected fish occasionally develop acute illness within a few weeks.

**Clinical Signs**

**Fish**

Piscine mycobacteriosis is usually a chronic disease, with affected fish surviving for several weeks to years. It is characterized by granulomatous inflammation in internal organs and muscles and sometimes the integument. The clinical signs are variable and often nonspecific, frequently resembling other fish diseases. Affected fish may be inapetant, emaciated or less active than normal; they may swim erratically and/or separate from other fish and seek out corners of the holding facility. A decreased growth rate or increased mortality may also be noted in the group. Some fish develop shallow, irregular, nonhealing skin ulcerations; nodular skin lesions (e.g. farmed Atlantic salmon infected with *M. chelonae*); cutaneous hemorrhages from the rupture of a lesion in the muscles; or nonspecific changes in cutaneous pigmentation, such as fading in tropical fish or bright colors in salmonids. Other visible lesions may include exophthalmos (bulging eyes), pale gills, excessive cutaneous mucus, raised scales, abdominal distention, skeletal deformities such as spinal curvature or stunting defects, and fin and tail rot. Fish with internal granulomas sometimes have no external signs of disease.

Acute cases have been documented primarily after experimental inoculation, and seem to be rare. Experimentally infected fish sometimes died within 2-3 weeks, with few clinical signs other than edema, erosions on the fins, and/or accumulations of fluid in the coelomic cavity.

**Other vertebrates**

Reptiles and amphibians with mycobacteriosis can also have nonspecific signs of illness, cutaneous lesions, and granulomas in the internal organs, which may be accompanied by abdominal distension. Reported skin lesions include solitary or multiple nodules, other granulomatous lesions and ulcers, as well as ulcerations on the pectoral or white foci on the carapace of chelonians. Granulomas or nodules in the oral cavity of reptiles, joint lesions, signs of osteoarthritis and respiratory signs (from chronic granulomatous lesions of the lung) have also been reported. One case in a captive marine toad was discovered when a granuloma caused a bone fracture. In another instance, lesions caused by *M. marinum* were detected in the internal organs of Hong Kong warty newts that developed opportunistic skin lesions from water mold (*Saprolegnia* sp.) and died despite treatment for the water mold.

Nontuberculous mycobacteria can also cause localized or disseminated cutaneous and internal lesions in mammals and birds. Descriptions of illnesses caused by *M. marinum* are rare. This organism caused a systemic infection in a European hedgehog and was detected rarely in tuberculosis-like lesions of camels at slaughter inspection. Intravenously inoculated mice housed at room temperature only had skin lesions; however, mice kept at 4°C developed a systemic disease that affected the lungs and spleen and resembled tuberculosis. The only external lesion in a parrot with disseminated disease was a granuloma involving the proximal rhinotheca of the beak and extending into the rostral sinuses. The bird became progressively emaciated and died during treatment, and granulomas were found in lung, liver, spleen and kidney as well as the oral mucus membranes, facial sinuses and facial bone.

**Post Mortem Lesions**

Fish with mycobacteriosis may have visible granulomas in the internal organs or only microscopic evidence of disease. The spleen, kidney and liver are involved most often, but many other organs can be affected in advanced cases. Gross lesions usually appear as gray to white or tan, hard to soft granulomas of various sizes that may be scattered, grouped or coalescing. Infection with *M. marinum* can result in caseating or noncaseating granulomas or both, depending on the species of fish. Diffuse
granulomatous lesions, without discrete granulomas, have also been seen in piscine mycobacteriosis, for instance in striped bass infected with *M. shottis* or *M. gordonae*, or salmonids infected with *M. chelonae* or *M. salmoniphilum*. Some fish may have edema or peritonitis, with the accumulation of fluid in the coelomic cavity, and in severe cases, the visceral organs may be swollen and fused by whitish membranes around the mesenteries.

Descriptions of lesions in amphibians and reptiles are often similar. However, only microscopic nongranulomatous necrotic foci were found in the liver, spleen and kidney of Hong Kong warty newts infected with *M. marinum*. The liver, which was the only organ visibly affected, was mottled gray and dark red.

**Diagnostic Tests**

Acid-fast bacilli may be found in smears or tissue sections of lesions stained with Ziehl-Neelsen (or its modifications). Fite’s acid fast stain or fluorochrome (auramine). Histopathology is also helpful.

A definitive diagnosis can be made by isolating mycobacteria from internal organs. These organisms may also be found in external lesions; however, the latter findings should be interpreted carefully as they may be contaminants. Fast-growing species (e.g., *M. chelonae*, *M. fortuitum* or *M. abscessus*) will grow on non-selective media and usually appear within 5 days. Slow growers such as *M. marinum*, *M. haemophilum* and *M. ulcerans* may be more difficult to recover and are usually isolated on selective mycobacterial media (e.g., Middlebrook 7H10 agar or Lowenstein-Jensen slants). *M. haemophilum* is unique in also requiring iron (heme supplementation) in its media. The optimal growth temperature varies with the species but is often around 20-30°C (68-86°F) for most of the organisms that affect fish. *M. marinum* grows best at about 30°C.

Primary isolation of *M. marinum* may take several weeks (though laboratory-adapted strains can form colonies faster), and cultures that may contain this organism should be kept for at least 6-8 weeks or more. *M. haemophilum* and *M. ulcerans* can take even longer to form visible colonies, and PCR may be more practical for detecting these organisms.

Mycobacterial species can be distinguished to a limited extent with biochemical tests; however, genetic techniques are now preferred for identification. PCR-based methods such as multiplex PCR assays can be useful in identifying mycobacteria but cannot distinguish some species, such as the individual members of the *M. marinum* complex. A loop-mediated isothermal amplification (LAMP) assay for the detection of the *M. marinum* complex has also been published. DNA sequencing, DNA fingerprinting, MALDI-TOF (mass spectral fingerprinting) and other techniques can be used for further species identification if necessary.

**Treatment**

Treatment is infrequently attempted, as it appears unlikely to eliminate *Mycobacterium* from affected fish colonies, and it is generally not practical in fish intended as food. However, there are a few reports documenting the treatment of valuable ornamental, zoo or research fish with medicated feed, water baths or injections. Drugs that have been used to treat *M. marinum* in humans include clarithromycin, doxycycline, minocycline, trimethoprim-sulfamethoxazole, certain fluoroquinolones, rifampin, rifabutin and ethambutol. Some of these agents, in various combinations, have been employed in fish and other vertebrates, with varying success. *M. marinum* is usually resistant to isoniazid, pyrazinamide and streptomycin. Other organisms that affect fish have different patterns of antibiotic susceptibility.

**Control**

**Disease reporting**

Veterinarians who encounter or suspect a mycobacterial infection should follow their national and/or local guidelines for reporting. State regulations should be consulted in the U.S.

**Prevention**

Preventive measures include quarantines of new stock (though many mycobacterial infections do not become clinically apparent within this time), disinfection of eggs, regular cleaning and disinfection of tanks and equipment, and treatment of water with a germicidal UV lamp. Fish and eggs can be screened by PCR or other methods; however, mycobacteria also occur in water and food and on various fomites, and may be difficult to completely exclude. Some facilities have sentinel programs to detect agents soon after they are introduced. If trash fish or tissues from fish (e.g., salmon viscera) are used as a source of protein in the feed, they should be pasteurized or heated at 76°C (169°F) for 30 minutes.

Any sick or dead fish should be removed quickly and destroyed. They should not be fed to reptiles or other animals that may be susceptible to mycobacteriosis. The usual response to the introduction of mycobacteriosis in an infected commercial facility is to cull the fish, thoroughly clean and disinfect the tanks and equipment, then restock. Depopulation may or may not be pursued in hobby aquaria or valuable fish in displays. If mycobacteria are not eliminated, husbandry measures such as minimizing stress and crowding and ensuring good water quality may help reduce the risk of illness.

**Morbidity and Mortality**

Mycobacteriosis is a common disease in fish and has been reported in aquaria, research laboratories, commercial fish farms and some wild populations. Published reports of its incidence are in the range of 10-15% or higher, and may
reach 100% in some closed facilities. Stressors such as poor water quality, poor diet and crowding increase the severity of outbreaks. Mycobacteriosis caused by *M. marinum* is usually a chronic disease, but sudden mass mortality is seen occasionally, such as when adding new fish to an aquarium. Advanced clinical cases in individual fish are usually fatal.

Most of the studies on mycobacteriosis have been done in zebrafish, a laboratory species that is frequently affected by this disease. *M. marinum* and *M. haemophilum* are the most important organisms in zebrafish, and can cause up to 30-100% mortality. Some outbreaks caused by *M. haemophilum* have been especially severe. Most of the environmental opportunists (e.g., *M. chelonae, M. abscessus, M. fortuitum* and *M. peregrinum*) tend to cause chronic illnesses with low mortality in zebrafish.

There are occasional reports of various mycobacteria causing high morality on commercial fish farms, and sporadic reports of mycobacteriosis in free-living fish. One ongoing outbreak, caused mainly by members of the *M. marinum* complex and particularly *M. shottsi*, is estimated to affect 50% or more of the striped bass in the Chesapeake Bay. Clinical cases have been observed in this location since at least the 1980s. Moderately and severely affected striped bass are estimated to have survival rates that are 84% and 54% that of healthy fish.

*M. marinum* is rarely reported in mammals or birds, but amphibians and reptiles seem to be more susceptible, most likely due to their lower body temperature. Immunosuppression may be a factor in some outbreaks in poikilotherms. Experimentally infected leopard frogs (*Rana pipiens*) can survive for a long time with well-encapsulated granulomas and no obvious signs of illness, but they developed acute, fatal, fulminant disease when they were immunosuppressed. *M. marinum* has persistently infected exhibits of snakes and bullfrogs.

**Infections in Humans**

**Incubation Period**

Clinical cases in people usually take weeks to months to become apparent. Skin lesions caused by *M. marinum* typically appear in about 2-8 weeks, but incubation periods as long as 6-9 months have been reported.

**Clinical Signs**

*M. marinum* usually causes slowly developing, superficial cutaneous granulomas in humans. The elbows, knees and feet are often affected after immersion in contaminated water, and the hands and fingers after handling fish or fish tanks. The initial lesion is typically a small erythematous papule, which develops into one or more erosive, ulcerative, crust or verrucous papules, nodules or plaques. Healthy people often have only a single lesion, but people who are immunocompromised tend to develop clusters that may spread proximaly along the line of the lymphatics. The latter is known as the sporotrichoid form. Rapid dissemination of nodules been reported, but a gradually spreading, chronic course is more typical.

There are also reports of unusual presentations, such as localized, mildly erythematous, eczema-like scaling and crusting. A variant with extensive skin ulcers, enlarging verrucous plaques, papules and erythema, which is sometimes called “Spam disease,” occurs in Pacific islanders. Cutaneous lesions caused by *M. marinum* sometimes heal within weeks to months in healthy people, but they can also persist for years or even indefinitely. “Spam disease” is often persistent. Cutaneous infections occasionally spread locally to deeper tissues, resulting in tenosynovitis, bursitis, arthritis or osteomyelitis, especially in immunocompromised individuals or those who are misdiagnosed and treated with immunosuppressive agents for a presumed inflammatory disease. Skin lesions may or may not be found concurrently in these cases. Extensive scarring and adhesions, especially of the hand and wrist, can lead to compromised function.

Other presentations are rare, and mainly seem to occur in people who are immunocompromised. Some infections have involved the lungs or other sites such as the bone marrow, nasal cavity (destructive nasal lesions mimicking lymphoma) and in one case the testes (epididymoorchitis). A pulmonary infection, with symptoms of lethargy, weight loss and a dry cough, occurred in an older patient who had mild preexisting lung disease but was otherwise healthy. He was thought to have become infected when siphoning fish tank water with his mouth. A few immunocompetent children developed chronic lymphadenitis or skin lesions with enlargement of the draining lymph nodes, resembling *M. avium* infections.

Similar skin diseases and other syndromes can be caused by other agents of piscine mycobacteriosis. One organism in the *M. marinum* complex, *M. ulcerans*, causes an ulcerative skin condition known as Buruli ulcer. Buruli ulcer has a distinct clinical presentation thought to be related to its ability to produce an immunosuppressive polyketide called mycolactone. It usually begins as a small painless nodule, or occasionally a papule, indurated plaque or diffuse swelling, and gradually evolves into an ulcer with indeterminate borders. There is usually no pain or inflammation. Untreated Buruli ulcer can develop into extensive skin ulcerations, which may result in scars, deformities and contractions. *M. ulcerans* may also invade deeper tissues, resulting in osteomyelitis, arthritis or even loss of the limb.

**Diagnostic Tests**

Piscine mycobacteriosis may be suspected with a suggestive clinical history, combined with consistent histopathology in a tissue biopsy and/or the detection of acid-fast bacteria in lesions. Organisms are more likely to be found in the earlier stages of the disease. Fluorochrome (auramine) staining, which is highly sensitive but has low
specificity, is often used as a screening test, and can be confirmed by Ziehl-Neelsen staining. A definitive diagnosis may be obtained by culture and/or PCR-based tests, as in animals. *M. ulcerans* is a particularly slow grower, and PCR may be more practical than culture in suspected Buruli ulcer. Tuberculosis skin tests are usually positive in patients with cutaneous lesions caused by piscine mycobacteria.

**Treatment**

Human infections are generally treated with antibiotics, which may be combined with surgery, especially in cases with involvement of tendons, joints or other deep tissues. Alternative treatments such as photodynamic therapy, local hyperthermia and cryotherapy have also been used, though not extensively evaluated. Immunosuppressive drugs may sometimes need to be temporarily stopped, if possible, for antibiotics to clear the infection.

The recommended antibiotics vary with the species of *Mycobacterium* and condition. A single drug is sometimes used to treat uncomplicated skin lesions caused by *M. marinum*. Combinations of two or more agents are generally recommended when the lesions are more extensive. Depending on the form and severity of the condition, underlying conditions and other factors, the duration of treatment can range from a few weeks to a year or more. Amputation was necessary in a few cases that did not respond to treatment and/or had extensive tissue damage.

**Prevention**

Broken skin should be protected from contact with fish or potentially contaminated water. Wearing gloves when with working with fish (including fish being prepared for food), water and equipment from fish tanks can decrease the risk of mycobacteriosis. A heavy glove may be necessary if contact with fish with sharp spines is anticipated. The hands should be washed thoroughly with soap and water afterward. Contact with mucous membranes (e.g., mouth aspirating water from fish tanks) should also be avoided. Care should be taken to prevent fish tanks from contaminating other sites (e.g., bathtubs) where mycobacteria may persist. Chlorination of swimming pools greatly reduces the risk of *M. marinum* from this source.

**Morbidity and Mortality**

Nontuberculous mycobacteria are often opportunists, and exposure is much more common than disease. *M. marinum* is an uncommon cause of illness in humans. In the U.S., the annual incidence was approximately 200 cases/year in 1993-1996. However, cases may become more common with the increased use of transplantation and the introduction of some new biologicals, such as agents that suppress TNF.

*M. marinum* infections are often linked to aquaria, but they also occur in people who handle farmed seafood or process it for cooking, or who have other waterborne or fish-related exposures such as swimming, boating or even exposure to coal mine water. Several outbreaks were linked to swimming pools in the past, but pool-associated cases have become uncommon since the advent of chlorination. Other mycobacteria are also found occasionally after fish-associated exposures. For instance, *M. abscessus* caused severe, chronic tenosynovitis of the hand in two healthy people who regularly handled fresh or frozen fish, and *M. iranicum* caused the sporotrichoid form of cutaneous mycobacteriosis in a healthy person exposed to a fish tank.

* M. marinum and other nontuberculous mycobacteria can affect healthy people, but clinical cases are usually more extensive and more difficult to treat in those who are immunocompromised. Cases limited to the skin and adjacent tissues are not usually life-threatening, but they can result in debility or disfigurement, especially when they affect joints, tendons and other deep tissues. Involvement of internal organs such as the lungs, though rare in people infected with *M. marinum*, may be fatal.

**Internet Resources**


The Merck Manual [https://www.merckmanuals.com/professional](https://www.merckmanuals.com/professional)


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References


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