

# Possible Mycobacterium Marinum Infection

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# 1 Executive Summary

In March-May 2014, we were traveling by private sailboat within the outer islands of the Federated States of Micronesia (FSM), a tropical wet environment. We like to hike, and frequently go on hikes in the jungle, looking for waterfalls, and World War II debris.

In mid-March, I got a boil-like sore on my right ankle, after a hike in the jungle, which has still not healed by May 1. I have also had 3 more similar sores pop up spontaneously on the right leg, on the knee and ankle.

I think I may have an infection caused by the bacterial *Mycobacterium marinum* (*M. marinum*), aka Fish Tank Granuloma.

# 2 Background

In March-May 2014, we were traveling by private sailboat within the outer islands of the Federated States of Micronesia (FSM), a tropical wet environment.

In mid-March, I got a boil-like sore on my right ankle, after a hike in the jungle at Satawon, which has still not healed by May 1. I have also had 3 more similar sores pop up on the right leg, on the knee and ankle.



What it looked like originally



What it looks like now

**Note: These are NOT pictures of my actual sore--I didn't take pictures at the outset. But "similar" pictures from literature to illustrate what it looked like originally, and what it looks like now.**

American doctors visiting Satowin in 2009 reported a "rare" disease on Satawan (see the Explorer's Club Report). We didn't know this at the time of our visit. And didn't take much notice of it, until the sore that started at Satawan (a) won't heal and (b) appears to be replicating itself in my body.

## **2.1 Area Background**

The Federated States of Micronesia lie between 7 degrees North and 10 degrees North, in the western Pacific (between the Marshall Islands and the Philippines, north of New Guinea). The temperature is generally 85 degrees year round, with frequent rainfall.

The FSM was part of contested Japanese territory during World War II, and many of the islands were heavily bombed by US forces.

One of the results of the bombing were large bomb craters left in the ground. Typically these craters are 10-15' in diameter, about 5-10' deep. They quite often filled with water. We have been specifically seeking out islands that had Japanese occupation (my husband is a WW2 buff).

We have been told by the islanders, that during the Japanese occupation, the Japanese imported freshwater fish from Japan, to put in the bomb craters and other bodies of fresh water on the occupied islands, to eat mosquito larvae.

We were told that this was the source of a disease found on Lekinioch (aka Lukunor) and Satawan, named "Spam Disease" locally, which was suspected to have been caused by *Mycobacterium marinum* (*M. marinum*). Originally thought to be isolated to Satawan, this could very well have been introduced by the Japanese fish on many similar islands. (see 2009 report by the Explorer's club that visited Satawan and Lekinioch).

We have had two other cruiser reports of "bug bites that got infected and wouldn't heal" that both came from Lamotrek.

None of the sores have been proven to be *mycobacterium* (it takes a PCR test which is difficult to do in the FSM/Palau).

Even in the "big cities" on Pohnpei, Chuuk, and Yap, the medical facilities are poor. In the outer islands, they are almost non-existent. Occasionally, they get visiting doctors from 1<sup>st</sup> World countries, but generally the outer island clinics are poorly stocked and attended by slightly-trained "medics".

There are no stores in the outer islands. All goods come from the more populated place by ship, which typically visits every 3 months. There is no internet or phone service on most islands. The islands communicate with the rest of FSM via Single Side Band radio.

## **2.2 Patient Background**

I am a white female aged 58 in fairly good health. A little overweight, with high blood pressure (under treatment, and controlled), and I drink about 3 alcoholic drinks a day. I am not allergic to anything, I rarely get sick--never serious, am rarely bitten by mosquitos (as opposed to my husband who gets bitten a lot). The small cuts and scratches I get in the normal course of living life generally heal well. I have a fairly high pain threshold, so I don't take much notice of small cuts/scratches.

Because of our unusual lifestyle and sometimes constrained diet, I take a number of OTC vitamins daily: Adult multivitamin, Calcium, Fish Oil, Glucosamine Chondroitin, Cinnamon. I

also take a baby aspirin, and the following prescription meds: Lotrel 5/10, Atenolol 50mg, Lipitor 40mg.

We get annual physicals in the U.S. and they confirm I am fairly healthy with the typical age-related and affluence-related diseases of a 58-year old. (high blood pressure, elevated cholesterol)

I think this infection was started in mid-March in Satawan, but since some of the literature I have read talks about 2-6 weeks before showing signs of infection, I have started a bit earlier than Satawan.

**Note:** From late February through 1 May, our only means of going ashore is via a "beach landing" in the dinghy, which means wading in salt water (usually less than 1-2 feet deep), and wallowing in sand as we drag our small boat up the beach.

In **mid-February 2014**, I participated in at least two long **day-hikes in the jungle** in Ponpei (Ponape), the capital of FSM. Footwear was open Teva-type sandals. On one hike, we waded for miles up freshwater stream beds to reach waterfalls in the jungle. I may or may not have had cuts or open sores on my feet at that time. It was a fairly grueling hike, over slippery rocks, and everyone was sore and bruised at the end.

In **early March**, we were at **Lekinioch** (aka Lukunor) atoll in the Mortlocks group of FSM. We did a **day-hike through the jungle**, mostly on a path, but also waded along the beach in salt water for 100 yards. We may have also stepped in small holes filled with fresh water and detritus.

On **March 18**, we were at **Satawan** atoll. We did a **long day-hike in the jungle**, specifically looking at World War II bombing remains. As on many of the islands we have visited during this period, there were a large number of WW2 bomb craters on Satawan. We were mostly on paths, but a few places we actually (following a guide) went bush-whacking into the dense jungle. I remember standing around on the edge of at least one bomb crater looking at a gun emplacement. Again, footwear was Teva-like sandals. At the end of the hike, I also distinctly remember wading into some soupy salt water to retrieve our dinghy, which had been anchored 10' offshore.

On the evening of **March 18**, I felt a pain and general swelling in my right ankle. I thought I had just sprained my ankle during the hike. But examining it closely, I found a **large boil-like (red-round-with pimple) bump on the right ankle** just below the ankle-bone. It looked something similar to the lower left picture in this article: [Atypical Mycobacterial Infection \(NZ\)](#), reproduced below.



This sore was maybe just smaller than a dime. I opened the sore, squeezed the pus out, and poured in some Hydrogen Peroxide, then treated it with Triple Anti-Bacterial Ointment, and put a band-aid on it. I thought it was a spider bite, though I had not felt any bites on our hike.

For the next couple of days, instead of starting to heal up, the lesion expanded, until it reached quarter-sized,

still mostly round. It was red-rimmed and oozed clear fluid pretty much continually. See picture left.

At this point, I started on a **10-day course of Bactrim Septra antibiotic** (because that's what we had aboard, and what I could get a refill for in Chuuk at the pharmacy). From about March 20-March 30, I was taking this antibiotic.

From **March 20-30** I was also **scuba-diving daily** in salt water. I would treat the wound (wash+Triple Antibiotic Ointment), put on a bandaid, cover it with a piece of paper towel, and wrap it in duct tape, then put on a sock, and put on my dive booties. It got wet daily in salt water. (We had come 20,000 miles by boat to dive the wrecks at Truk Lagoon, and I wasn't about to skip the diving because of a small sore).

By **1 April**, we were **finished diving** and moved on from Chuuk. At this point, the sore on my right ankle was large, about 1-1¼" in diameter, but looked clean, and I expected it to scab over and heal up quickly once I stopped daily diving.

But a few days afterward, my sore seemed **redder than normal, still not healed**, and the **ankle and foot were swollen** again. *It still looked pretty much the same as it did on Mar 21 (picture above).* I started getting concerned about a serious infection.

On advice from another cruiser (fighting a similar sore acquired in Lamotrek, and who was getting advice from a doctor in Germany), I spent a day with my foot elevated. I also, just for grins, soaked it for about a half an hour in hot water (as hot as I could stand). I also got more serious about washing, and treating, and trying to keep it out of the salt water. This seemed to help the swelling, and the redness to some extent, but the sore was still open and oozing. I was still treating it mainly with Triple-Antibiotic Ointment.

On about **April 14**, **another sore** of a similar nature just popped up out of the blue on my **right knee**. I had had a scrape on that knee (I think which occurred after my last jungle hike), which was nearby and healing. This new sore was about ½" in diameter, looked boil-like (big red round with a white "pimple" on top). It came up spontaneously in an area of unbroken skin on my knee. I opened it up and drained the pus out and started treating it the same way I was treating the sore on my ankle.

At this point I was given some **different stuff to treat the wound with** by the other cruiser who was having a similar problem... **Rivanol** tablets 1.0g (dissolve 1 tablet in 1 liter of water and use to wash the wound when changing dressing). And **Bepanthene** crème.

I had never heard of either of these, but this was recommended by his doctor in Germany, and my Triple Antibiotic Ointment wasn't doing much. So from then on, my treatment was mostly washing with anti-bacterial soap, a rinse and brief soak with the Rivanol solution, and Bepanthene crème. I tried to keep it covered with bandaids, but was running out of appropriate-size bandaids that would stick. So periodically, I would leave it open to the air.

At one point in **late April**, after bandaiding and treating religiously for about 5 days, and trying to keep my foot dry during our "shore landings" the area on my **ankle looked healed, and stopped oozing**. So I quit treating it and quit putting bandaids on it, figuring it was on the mend.

After a day or so, it got crusty (fairly normal scab), then cracked (from movement when I walked, I think), and started oozing again. It got wet and then the scab was gone, and it's open again.

The new smaller sore one on my knee expanded a little bit, but never got much bigger than the original size. I started aggressively treating it with Rivanol and Bepanthene right away, and it had healed up completely by the end of April.

On about **May 1**, within about 24 hours of each other, I developed **two similar small bumps**, one on the top of the same (right) **foot**, and one on the right side of my right **knee**. I opened both of them up and started treating them. At this point, I was beginning to suspect that something more than a simple infection was at play. That's when I started doing internet research.

The sores on my knee have stayed small, and healed up fairly quickly. The new sore on my foot (as of May 4), seems to be following the same path as the one on my ankle... slowly expanding (a widening circle), and oozing clear fluid. What started as pencil-eraser size is now almost quarter-sized. The original sore on the ankle is still not healed (after 6 week), and still oozing a little. My foot is a little bit swollen and a little bit tender.

Also about May 1, we arrived at a place with a dock, so I wasn't having to wade through salt water on a daily basis to go ashore, but this hasn't helped a lot.

On May 2, I started taking Bactrim Septra again. And am treating all 3 sores with Antibacterial Soap, Rivenol rinse, and Bepanthene, and keep it covered.

Finally able to get some medical care in mid-April, I first took a one week course of Cefalexin, and then (going to a different doctor on a different island), I started on a long course of Doxycycline, as recommended by the original 2008 report by Lillis et al. As soon as I started on the Doxycycline, the sores cleared up (and so far have not re-appeared).

### **3 2009 Explorer Club Report on Satawan**

This was a report issued by the Explorer's Club out of California, who visited Satawan in 2009. Their visit included a couple of doctors and attempted to follow up on a previous visit to Satawan to attend to what they called "Spam Disease". This is what led me first to suspect *Mycobacterium marinum*.

Here is the summary of what they hoped to accomplish:

*Conduct research on a unique disease manifestation, "Spam Disease", known to date only on Satowan Island. The research will include taking biopsies and case histories to aid in determining the disease's causative agent, documentation of patient's responses to recently employed empirical therapy and to further investigate possible sources and distribution of the disease. Similar studies will be conducted on Lukunor Island, Lukunor Atoll, Mortlock Islands.*

And their report on "Spam Disease"

Japan's surrender in August, 1945 did not bring an end to the Mortlock's WWII experience.

A unique disease recently described by Lillis, et al., *Clinical Infectious Diseases* 2009; 48:1541–6, is attributed to the mosquito-larvae eating fish (Medaka) introduced to Satowan by the Japanese during the war. Bomb and shell craters formed during U.S. attacks filled with fresh water and became mosquito breeding sites. In an attempt to control the infestation, the Medaka fish were brought from Japan and planted in these pools. Most likely, the fish carried *Mycobacterium marinum*, a bacterium related to the organisms causing tuberculosis and leprosy, inoculating the organism into what became the kid's local swimming holes.

(Note, a link to one of Lillis' articles on his findings is here:  
<http://cid.oxfordjournals.org/content/48/11/1541.full> )

The very recent recognition of this seemingly unique disease on the Mortlock's appears to be directly attributable to the Japanese occupation and U.S. shelling of Satowan, and an interesting way of bringing our story full circle.

In the U.S., the bacterium causes "Fish Tank Granuloma". In the Mortlock's, the often untreated syndrome causes severe abscesses and granulomas that have been named "Spam Disease" because the manifestation resembles the canned meat by that name. Interestingly, the causative organism remains speculative as it has not been cultured from the patients.

Working with Kevin Winthrop, MD, and Joe Lillis, MD, Oregon State University Medical School, and Dr. Kino Ruben, Chuuk Department of Health, we offered to evaluate the treatment regimens that the original investigators had started two years ago but had not been able to follow up. We also proposed to examine new patients with the disease and obtain samples for culture or RNA sequencing in an attempt to identify the causative organism. Since we were going to other atolls, we offered to examine these additional populations in an attempt to understand the disease's epidemiology.

Our medical team consisted of Dr. Mike Day, primary care physician with the University of Florida Medical School, former Marine Corps medic and critical care nurse, Eric Musick, Pharmacist Bruce Latimer and Pam Lambert and myself, clinical microbiologists. In addition, photographers, Luke Twyman and Diana Lackey photo-documented the patients and Peggy Day, Frank Tidikis and Rich Lackey took case histories.

As soon as we had explained our intentions to the chiefs and mayor on Satowan, they gathered "spam disease" patients for evaluation. The local population is acutely aware of the disfiguring syndrome and anxious to find an effective treatment. We looked at patient responses to the "spam disease" therapy regimens established by the Lillis group and observed new patients from which we obtained tissue biopsies. Two clinics were conducted on Satowan. Of the 17 patients evaluated, eight were participants in the Lillis study and nine had not been previously evaluated or treated. From this group, eight tissue biopsies were collected.

Unfortunately, the preservative had been omitted from the collection supplies, necessitating rapid sample shipment to the Center for Disease Control in Atlanta, GA. Serendipitously, Federated States of Micronesia President Mori was conducting "town meetings" at Satowan coincidentally with our visit and offered to carry the samples back to Chuuk for immediate shipment to the CDC in Atlanta. The last we saw of the President and the samples, they were being carried north at full speed on the FSM patrol boat for delivery to Dr. Ruben on Chuuk.

Previous observations had led us to believe the disease had not been found on Lukunor, the neighboring atoll/island to Satowan in the Lower Mortlock group. When we arrived on Lukunor, the local medical liaison, William Sana, was waiting for us with a list of patients to be examined.

Not only were there a large number of untreated patients, some who had never travelled to Satowan, but a group who Mr. Sana had tried to treat. Unfortunately, Lukunor's doxycycline supply (the drug prescribed by the Lillis group) was/is inadequate to support the recommended three month treatment course. Most patients only received a month's worth of the drug, which was generally ineffective. The concern is not only producing a population of uncured, chronically infected patients harboring an unknown organism with an unknown sequela but selecting a microbe that might be developing resistance to the only available effective, cheap antibiotic. We documented twenty one patients but did not obtain any tissue samples as our remaining two biopsy kits were left on Satowan.

It appears that what may be a rather obscure, unique syndrome possibly has a wider distribution than previously expected. On Satowan, it is now evident that the bomb craters pools, the water reservoirs for the taro irrigation canals, are most likely the primary source of the bacterium. However, on Lukunor, according to our guide, Simon Bunluay, only three bombs were dropped on Lukunor (one of which actually hit the elusive island) and it was never shelled. Consequently, there are no craters. We did observe fish (unidentified) in Lukunor's shallow, fresh water wells and taro irrigation ditches.

Because the island did have a large, wartime Japanese garrison, they also may have seeded these standing, fresh water sources with the *Mycobacterium marinum*- infected Medaka fish to help control the mosquito population. The legacy of WWII continues.

The importance of these studies should not be underestimated and minimized simply because they occur in a remote corner of the world. Recently, to reduce the potential for mosquito-borne diseases, local health departments in the U.S. have started offering mosquito-larvae eating fish to homeowners who can no longer financially maintain their swimming pools and spas. The fish are self-sustaining in these pools. There is no evidence the fish are not carriers of *M. marinum*. The research and quest to identify and optimally treat the causative agent is truly in the best interest of all.





Untreated "Spam Disease"



Treated "Spam Disease"



Taro Patch Irrigation Ditch



Satawan Bomb Crater

## 4 Article: Medicine.net

[http://www.medicinenet.com/mycobacterium\\_marinum/article.htm](http://www.medicinenet.com/mycobacterium_marinum/article.htm)

### 4.1 What is *Mycobacterium marinum*?

*Mycobacterium marinum* (*M. marinum*) is a slow-growing atypical mycobacterium that is commonly found in bodies of fresh or saltwater in many parts of the world. Skin infections with *Mycobacterium marinum* in humans are relatively uncommon and are usually acquired from contact with aquariums or fish.

Most infections occur following skin exposure to the bacteria through a small cut or skin scrape.

The first signs of infection with *M. marinum* include a reddish or tan skin bump called a granuloma. Less commonly, a string or batch of the small reddish bumps crop up on the exposed body area in a classic pattern called sporotrichotic lymphangitis.

It is somewhat rare to acquire this infection from well-maintained [swimming](#) pools because of protection afforded by proper chlorination. *Mycobacterium marinum* does not typically grow at normal body temperature, which is why it remains localized to the cooler skin surface. Overall, diagnosis and treatment of this unusual skin infection is often delayed because of a lack of suspicion for this atypical mycobacterium versus more common bacteria like *Staphylococcus*.

### 4.2 What are other names for *Mycobacterium marinum* infections?

Some synonyms for *Mycobacterium marinum* skin infections include tropical fish granuloma, fish tank granuloma and fish tank granuloma.

### 4.3 How common is *Mycobacterium marinum*?

Although rare, infections can occur worldwide, most commonly in individuals with occupational and recreational exposure to fresh or saltwater. In the United States, infections caused by *M. marinum* are rare.

The infection is very rare in children and is typically a disease of adults.

### 4.4 How does a person get infected with *Mycobacterium marinum*?

Human infections with *M. marinum* under normal circumstances are rare. People are prone to this infection when there is minor trauma to an extremity like the forearm before or during contact with marine animals like fish or turtles, or just an aquarium, saltwater or freshwater.

However, people who have minor breaks in the skin such as small cuts or scrapes are at increased risk when in contact with water from an aquarium or fish tank, when handling, cleaning, or processing fish, while swimming or working in fresh or salt water, or while standing in contaminated water.

One form of the infection, known as "swimming pool granuloma," can occur when there is inadequate chlorination of swimming pools. However, in the U.S., most human infections with this bacteria have been associated with contact with fish tanks.

*M. marinum* infection is not spread from person to person. It is also not transmitted in hospitals like other common bacteria.

#### **4.5 Who is at risk for *Mycobacterium marinum* infection?**

People at highest risk include home aquarium hobbyists, swimmers, aquarium workers, marine-life handlers, anglers, and oyster workers. Overall, anyone with frequent or persistent saltwater or freshwater exposure is at potential risk. Here is a list of at risk people:

personal home-aquarium owners

professionals who clean aquariums

marine biologists

fishermen and workers exposed to saltwater fish

immunocompromised patients (HIV/AIDS)

#### **4.6 What are the symptoms of *Mycobacterium marinum* infection?**

Typically, patients may initially notice a small red bump or non-healing red sore on their skin a few weeks after a history of exposure to non-chlorinated water. Ninety percent of the cases involve the arms (upper extremities). They may remember getting a scratch, scrape, or puncture wound several weeks before while in the water. Many people may easily overlook the early signs and try over-the-counter antibiotic creams and disinfectants on their own in an attempt to make the bump or sore go away.

Often, patients may not decide to go to their physician until they can't get rid of the bump for weeks or months, they see more bumps, or see spreading bumps in a "line" pattern up their arm or leg.

Some patients may feel no pain or itch while others commonly have some localized pain and firmness at the site of the infection. Most otherwise healthy people overall feel well during the infection and do not have [fever](#) or [chills](#).

Patients in poor health or those with other health issues like an impaired immune system or other serious illnesses may experience fever, enlarged localized lymph nodes, and systemic infection.

When *M. marinum* infects the skin, it causes localized microscopic nodules to form. These nodules are called granulomas. They occur at sites of skin trauma where there are scratches, cuts, and the like.

The granulomas usually appear within two to three weeks of exposure. Some reported cases have developed two to four months or more after exposure to *M. marinum* because of the very slow-growing nature of this bacterium.

The most frequent sign is a slowly developing nodule (raised bump) at the site the bacteria entered the body. Frequently, the nodule is on the hand or upper arm. Later the nodule can become an enlarging sore (an ulcer).

Swelling of nearby lymph nodes occurs. Multiple granulomas may form in a line along the lymphatic vessel that drains the site. These lesions will usually spontaneously heal in several months. This infection can also involve the joints ([septic arthritis](#)) and bones ([osteomyelitis](#)).

A health-care provider should be consulted if a skin nodule or reddened sore (ulcer) develops following direct skin contact with fresh or saltwater or after handling or processing fish.

For people with compromise of the immune system, *M. marinum* infection can be especially serious and involve disseminated (widespread) disease. If an infection is suspected under such circumstances, a health-care provider should be promptly consulted.

#### **4.7 What tests are available to diagnose the infection?**

Lab tests include cultures where a swab or sample is taken and grown in the laboratory.

Cultures of *M. marinum* are fairly difficult to grow and usually may take several weeks in the lab. The culture may be negative, even if there is an active infection. Treatment may still be considered even if the test results are negative, especially if the patient's history supports past fish or fish-tank exposure.

In the absence of positive culture results, a skin or tissue biopsy may be a helpful test for diagnosis. This may help find the microscopic bacteria.

A special test called polymerase chain reaction (PCR) of tissue may be used in difficult cases to assist in naming the exact type of bacteria or Mycobacterium species involved.

#### **4.8 How is Mycobacterium marinum infection treated?**

Most infections are treated medically with a fairly long course of oral antibiotics.

Medications may be required anywhere from three to six months or more depending on the severity of the disease and spread of the infection. Typically, physicians may recommend continuing the medications for an additional four to six weeks even after all of the symptoms have fully cleared.

The gold standard for treatment of infection by *M. marinum* is oral antibiotics. Clarithromycin is currently the preferred antibiotic selection. Other antibiotic options include rifampin plus ethambutol, tetracyclines, trimethoprim-sulfamethoxazole, and fluoroquinolones.

Some milder infections (mainly in healthy people) have cleared on their own without any treatment. Rarely, surgical treatment and drainage of deeper tissue or skin infections may become necessary in more complicated cases.

However, medical treatment remains the primary and preferred treatment for nearly all cases.

#### **4.9 What is the prognosis for those infected with Mycobacterium marinum?**

The prognosis is excellent for a complete cure with a proper, full course of oral antibiotics and good medical follow-up with your physician. There are no long-term problems after treatment.

#### **4.10 How do I find a specialist?**

Specialists including dermatologists, infectious disease physicians, and rheumatologists may have additional expertise in the diagnosis and treatment of this rare disorder.

#### **4.11 What are possible complications from Mycobacterium marinum?**

*M. marinum* infections are usually localized and typically do not spread past the skin in healthy people. Most patients with a normal immune system don't experience other complications. However, undetected or untreated, the infection may progress and cause deeper and more longstanding infections.



Patients with an impaired immune system (immunocompromised) may be much more prone to serious complications such as spread of infections to involve the bone marrow and internal organs.

Some rare potential problems include infection of the underlying bone called osteomyelitis, infection of the deep muscle tendons called tenosynovitis, inflammation of the joints called arthritis, and widespread bodily infections called disseminated disease.

#### **4.12 Do fish get infected with *Mycobacterium marinum*?**

Yes. There are probably two different types of *M. marinum*. One type only causes a longstanding (chronic) progressive disease in fish without affecting humans. The second type, which can infect humans, seems to cause a deadly sudden illness in fish.

#### **4.13 What else could it be?**

Other conditions may mimic or be confused with *M. marinum* infections. Possible other diagnoses include common things like bug bites, [spider bites](#), foreign body granuloma, bacterial infections like staph or [E. coli](#), fungal infections, tumors, and others.

Additional diagnoses include cowpox infection, [leishmaniasis](#), leprosy, [sarcoidosis](#), and [sporotrichosis](#). More advanced cases may be mistaken for [rheumatoid arthritis](#), gout, traumatic tendon injury, deep fungal infections, or [cancer](#).

#### **4.14 How can I prevent this infection?**

The following steps may help to protect you from contracting an infection with *M. marinum*:

Avoid fresh or saltwater activities if there are open cuts, scrapes, or sores on your skin, especially in bodies of water where this bacterium is known to exist.

If you have a weakened immune system, you can reduce the risk of infection by carefully covering cuts, scrapes, or sores during fresh or saltwater activities and while cleaning fish tanks or handling, cleaning, or processing fish.

Wear heavy gloves (leather or heavy cotton) while cleaning or processing fish, especially fish with sharp spines that may cause cuts, scratches, or sores to the hands and skin. Wash hands thoroughly with soap and water after fish processing or use a waterless cleanser.

Wear waterproof gloves while cleaning home aquariums or fish tanks.

Wash hands and forearms thoroughly with soap and [running](#) water after cleaning the tank, even if gloves were worn.

Ensure regular and adequate chlorination of swimming pools to kill any bacteria that may be present.

#### **4.15 Patient Comments from the internet Comments Section**

**65-74 Year Old Male:** *"I caught this mycobacterium marinum infection 4 years ago from fishing. I had 6 weeks of IV therapy, every day and a year of oral antibiotics. It was misdiagnosed by many doctors. I never went fishing again".*

**55-64 Year Old Female:** *"I had been gardening and got a small puncture wound from brambles. A short while later developed 4 small puncture wounds in a line on back of my hand,*

*they all became infected with pus, saw nurse, gp, ointments, wounds grew bigger reddened, had scan, no debris found, then skin specialist creams biopsy, thought to be psoriasis.*

*Six months later gone from pin prick size to 2-3inch sq spreading. A new doctor asked if I had tropical fish, I have had for years. Brought diagnosis of mycobacteria. On antibiotics for weeks, cleared it slowly, but whilst still active jagged it on something, nail maybe? Small spot within, a year later only slight sign and marking of original problem, but the jagged spot has grown and got infected so back on antibiotics for weeks, cyclosporine type, back for referral in a few weeks, so far subduing infection, but some side effects."*

**25-34 Year Old Male:** *"I got pricked by a frozen prawn that I was feeding to my marine fish. I remember washing my hands with disinfectant. Two to 3 weeks later, a red sore came up on the knuckle of my thumb, about 5mm. It started to form an ulcer after a few more weeks (open wound).*

*I didn't know what this was and tried a few antibiotic creams, fungal creams, etc., that were lying around at home. Nothing worked so I went to the doctor. He prescribed antibiotics, but no result. I went back to the doctor and got referred to skin specialist. I hadn't realized at the time it was connected to the prawn prick, as it was weeks after, that the red nodule came up. Anyway, the skin specialist told me it was rare, and started me on a 4 month treatment on Bactrim DS. It cleared up after around 3 months of antibiotics. I also got 2 nodules popping up on my fore arm, around 5mm. They felt like hard little balls under my skin.*

*The doctor also told me to use heat packs for 5 to 10 minutes on top of the nodules as hot as I could bear. This Mycobacterium marinum bacteria does not like the heat."*

*I'm all fixed now, but not many doctors know about it."*

## **5 Article: Atypical Mycobacterial Infection (NZ)**

<http://www.dermnetnz.org/bacterial/atypical-mycobacteria.html>

Atypical mycobacterial infections are infections caused by a species of mycobacterium other than Mycobacterium tuberculosis, the causative bacteria of pulmonary TB and extrapulmonary TB including cutaneous TB.

Atypical mycobacteria may cause many different types of infections such as septic arthritis, abscesses and skin and bone infection. They may also affect the lungs, gastrointestinal tract, lymphatic system and other parts of the body.

Skin infection tends to result in crusted nodules.

### **5.1 What causes atypical mycobacterial infection?**

There are many different species of mycobacterium. Those that cause atypical mycobacterial infections include:

- Mycobacterium avium-intracellulare
- Mycobacterium kansasii

- *Mycobacterium marinum*
- *Mycobacterium ulcerans*
- *Mycobacterium chelonae*

*Mycobacterium avium-intracellulare* and *Mycobacterium kansasii* primarily cause lung disease similar to pulmonary TB, whilst *Mycobacterium marinum*, *Mycobacterium ulcerans* and *Mycobacterium chelonae* cause skin infections.

## **5.2 What are the clinical features of atypical mycobacterial infection?**

The clinical features of atypical mycobacterial infection depend on the infecting mycobacteria.

*Mycobacterium avium-intracellulare*

- Also known as MAC (*Mycobacterium avium* complex)
- Most common non-tuberculous mycobacterial infection associated with AIDS
- Symptoms include fever, swollen lymph nodes, diarrhoea, fatigue, weight loss
- and shortness of breath
- May develop into pulmonary MAC
- Skin lesions are uncommon

*Mycobacterium kansasii*

- May cause a chronic infection of the lungs similar to pulmonary TB
- Second most common non-tuberculous mycobacterial infection associated with AIDS
- Symptoms include fever, swollen lymph nodes and lung crackles and wheezing
- Skin lesions may occur either alone or as part of a more widespread disease

*Mycobacterium marinum*

- Also known as fish tank granulomas
- Uncommon infection that occurs most often in people with recreational or occupational exposure to contaminated freshwater or saltwater
- Usually a single lump or pustule that breaks down to form a crusty sore or abscess
- Other lumps may occur around the initial lesion, particularly along the lines of lymphatic drainage ("sporotrichoid" forms)
- Most often affects elbows, knees, feet, knuckles or fingers
- Multiple lesions and widespread disease may occur in immunocompromised patients
- Rarely causes red, swollen and tender joints

## Mycobacterium ulcerans

- Also known as Buruli ulcer
- Infection most common in Central and West Africa around areas of lush vegetation and swamps but may also occur in Australia
- Solitary, painless and sometimes itchy nodule of 1-2 cm develops about 7-14 days after infection through broken skin
- Over one to two months the nodule may break down to form a shallow ulcer that spreads rapidly and may involve up to 15% of the patient's skin surface
- Severe infections may destroy blood vessels, nerves, and invade bone

## Mycobacterium chelonae

Worldwide distribution: found in tap water and other water sources

May cause lung disease, joint infection, eye disease and other organ infections

May result in non-healing wound, subcutaneous nodule or abscess

Immunosuppression may cause disseminated lesions throughout the body

### 5.3 *Mycobacterium marinum* infection Photos

*Mycobacterium marinum* infection



<http://www.dermnetnz.org/bacterial/atypical-mycobacteria.html> (page 3 of 4)

### 5.4 *How are atypical mycobacteria diagnosed?*

Atypical mycobacteria are diagnosed on culture of tissue. Specific conditions are required, so the laboratory must be informed of the clinician's suspicion of this diagnosis. The infections have specific pathological features on skin biopsy.



### 5.5 *What is the treatment of atypical mycobacterial infection?*

Treatment of atypical mycobacterial infections depends upon the infecting organism and the severity of the infection. In most cases a course of antibiotics is necessary. These include rifampicin, ethambutol, isoniazid, minocycline, ciprofloxacin, clarithromycin, azithromycin and cotrimoxazole.

Usually treatment consists of a combination of drugs. Some points to consider when treating atypical mycobacterial infections:

**Mycobacterium marinum** species are often resistant to isoniazid. Treatment with other antibiotics should be for at least two months.

**Mycobacterium kansasii** should be treated for at least 18 months.

**Mycobacterium chelonae** is best treated by clarithromycin in combination with another agent. Sometimes surgical excision is the best approach.

AIDS patients on HIV protease inhibitor drugs cannot be treated with rifampicin because rifampicin significantly increases the breakdown of these drugs. Rifabutin is a suitable alternative. Antibiotics are usually ineffective in treating large skin lesions caused by **Mycobacterium ulcerans**. Rifampicin may promote healing of pre-ulcerative lesions. Most lesions eventually spontaneously heal after 6-9 months but may leave behind extensive scarring and disfigurement.

Surgical removal of infected lymph nodes and skin lesions is sometimes necessary. In severe cases, skin grafts may be necessary to repair the surgical wound.

## 6 Article: Rifampicin (NZ)

In my readings, Rifampicin was often combined with something else for a long course of treatment.

<http://www.dermnetnz.org/treatments/rifampicin.html>

Rifampicin is an antibiotic used to treat serious bacterial infections. It may be prescribed by dermatologists for the treatment of:

- Recurrent boils
- Folliculitis decalvans
- Hidradenitis suppurativa
- Tuberculosis
- Leprosy

It is also used to treat brucellosis, serious staphylococcal infections and to clear asymptomatic carriers of *Neisseria meningitidis* (which can cause meningococcal disease).

Rifampicin is active against a variety of organisms including *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Mycobacterium tuberculosis*, *Mycobacterium leprae*, *Neisseria meningitidis* and *Brucella* species.

It should always be prescribed with another antibiotic, in order to prevent bacterial resistance, which can develop rapidly if it is used alone.

It should be taken on an empty stomach at least 30 minutes before a meal or 2 hours after a meal.

Antacids should be given at least 1 hour after rifampicin.

In New Zealand rifampicin is available as:

Rifadin™ tablets, suspension and intravenous infusion

Rifinah™ tablets, where it is combined with isoniazid for the treatment of tuberculosis and leprosy

### **6.1 Side effects**

Rifampicin is usually well tolerated and rarely causes serious toxicity. The commonest side effects involve skin and the gastrointestinal system.

Discoloured urine while on rifampicin

**Bodily fluids:** Tears, sweat and urine may become orange coloured and contact lenses may be permanently stained.

**Skin:** Usually mild and self-limiting flushing and itching with or without rash. Hives are uncommon. Erythema multiforme and toxic epidermal necrolysis have rarely been reported.

**Gastrointestinal:** Loss of appetite, vomiting, abdominal pain and diarrhoea.

**Liver:** Hepatitis, particularly if rifampicin is given with isoniazid

**Blood:** Thrombocytopaenia (low platelets potentially resulting in bruising and bleeding), rarely low white blood cell count and disseminated intravascular coagulation, and very rarely agranulocytosis (severely decreased white blood cell counts).

**Musculoskeletal:** Muscle weakness and myopathy are uncommon

Rifampicin is sometimes used intermittently (less than 2 to 3 doses per week) for the treatment of tuberculosis and leprosy. When rifampicin is used in this way, it may be associated with a 'flu-like syndrome, shortness of breath, low blood pressure, acute renal failure and shock.

### **6.2 Drug interactions**

Rifampicin is a P-glycoprotein inducer and may increase the breakdown of other medications, making them less effective. The dosage of these drugs may need adjustment if taken together with rifampicin:

Anticoagulants (warfarin, dabigatran)

Anticonvulsants (phenytoin)  
Antiarrhythmics (disopyramide, mexilitine)  
Antipsychotics (eg haloperidol)  
Antifungals (eg itraconazole)  
Antiretroviral drugs (eg zidovudine)  
Beta blockers  
Calcium channel blockers (eg diltiazem, verapamil)  
Chloramphenicol  
Clarithromycin  
Corticosteroids  
Ciclosporin  
Dapsone  
Digoxin  
Oral contraceptives  
Benzodiazepines (eg diazepam)

### **6.3 Precautions**

Non-hormonal contraceptive methods may be necessary in women of child bearing age when taking rifampicin, due to its effects on oral contraceptives.

If used in pregnancy, it may be harmful to the fetus without causing malformations. Appears in breast milk.

Baseline blood tests should be done in adults, including a blood count, renal and liver function tests. If there are significant abnormalities, these should be repeated during treatment. Caution should be taken when there is pre-existing liver disease or liver function abnormalities.

## **7 Article: Tropical Fish Disease You Can Catch**

Again, excerpts:

About a year after my friend went through his bout with *Mycobacterium marinum*, in which he almost lost his finger, I noticed a small bump on my right pinky finger knuckle. It looked somewhat like an ingrown hair at first. I covered it with a bandage and used an antibiotic ointment for one week and it seemed to disappear.

However, it reappeared later, this time slightly larger and more dry and scaly. Because I remembered what my friend went through, when it came back, I went straight to my Primary

Care Physician and told him I was concerned that I had a *Mycobacterium marinum* infection. He referred me to a Dermatologist for treatment.

Some time had passed between getting an appointment with my own doctor, having the referral clear the HMO, and then finally getting to see the specialist. By the time I saw the Dermatologist, my single bump had become an ascending lymphangitic granuloma, with a second bump at mid-forearm and a third just short of my elbow. The doctor confirmed my suspicions and prescribed an antibiotic.

I ended up going through three different prescriptions before we found one my body could tolerate. I was prescribed a large dose of a strong antibiotic twice a day for three months. This cured me of the *Mycobacterium marinum* infection, but caused a whole new problem. While my actual encounter with *Mycobacterium marinum* was relatively harmless, I am still getting over the long-term effects of the cure. The long course of strong antibiotics absolutely wrecked my digestive system, and I am still suffering with minor difficulties to this day.

## **8 Dissertation: *Mycobacterium marinum* (UK Essays)**

This is a medical article reproduced on UK Essays.com without attribution to its original author. It appears to be a dissertation from someone who is a non-native-English speaker. It is long and technical. I don't think the person did any original study, just a summary of what others have done. I have just summarized a few things from it, below. The original article I found is here:

<http://www.ukessays.com/dissertations/health/mycobacterium-marinum-water.php>

### **8.1 Introduction of Dissertation**

Below is from the Introduction

*M. marinum* is an environmental atypical mycobacterium ubiquitous in fresh, salt, and brackish water. It is known that infects temperate and tropical species of fish of at least 150 species, including ornamental fish. But also affects frogs, eels, oysters, aquatic mammals, toads and snakes [2, 11].

It cause tuberculosis-like disease in fish, its natural host [129]. The infection in fish has an average incubation period of 3 months. It affects viscera and produce anorexia with emaciation, skin defects, distension of the abdomen, being cause of sudden death [11, 12, 66].

In 1904, Alexander described for first time lesions in a cod fish that were associated with acid fast bacilli. But it was Aronson in 1926 that isolated the bacteria from tubercles of fish that died in an aquarium of Philadelphia. He described the bacilli as acid fast, chromogenic, pleomorphic and growing best at 18°-20°C. Aronson suggested the name of *Mycobacterium marinum* [130].

*M. marinum* is as well the causative agent of the human disease called fish tank granuloma, also known as swimming pool granuloma or fish fancier's finger syndrome. In 1951 Norden and Linell reported for the first time the human disease in a swimming pool outbreak in Örebro, Sweden. They described the lesions as chronic papulous ulcerations, usually located in the elbows. The pathogen was isolated from the walls of that swimming pool as well as from the lesions of the patients. They called the pathogen isolated *Mycobacterium balnei* [131]. It was not

until 1959 when Bojalil demonstrated that *M. marinum* and *M. balnei* were in fact, the same mycobacterium [132].

*M. marinum* is the most frequent cause of skin infection among the environmental mycobacterium that affects humans [86, 133]. Nowadays the frequency of human infections is mainly sporadic. But in the past, outbreaks related with swimming pools were not uncommon [134]. As an example, one of the biggest epidemics was in Glenwood springs pool, Colorado in 1956 with 262 cases reported [135].

That was before chlorination became a common practice. Chlorination makes water safer. As was seen recently in Bologna, where water from swimming pools were free of *M. marinum*. But still could be isolated in 4.5% of the samples from the shower floor of the same [136].

The real global incidence of the disease is not known because the number of cases are underreported, due probably to the difficulties in the diagnosis [94]. It is world wide distributed but with a tendency to aggregate geographically [137]. Like in Chesapeake bay, Maryland, where there is an incidence of 4 cases per 100000 population per year [138]. Meanwhile in California was estimated in 0.27 cases per 100000 adults [139]. Or in Satowan, Micronesia, with an estimated prevalence of 10% of the population [115].

Apparently the global annual incidence remains small and stable [140]. Even though an increase number of reported cases has been noticed in The United States, going from an average of 40 cases per year in the 80s to an average of 198 cases per year in the 90s [141]. What seems clearer is that differing from other atypical mycobacteria the prevalence of *M. marinum* has not increased with the HIV epidemic [140].

Opposite to humans, the incidence in fish is increasing in hatchery fish, probably due to the high population density of fish. Transmission is possible fish to fish and between fish and amphibians. In addition it has been proposed transmission through eggs and through practice of feeding fish with fish carcasses [140].

There are 2 groups or clusters of *M. marinum* with different pathogenicity. Cluster I is characterized by producing acute disease and death in fish and also for affecting humans. On the contrary, cluster II only affects fish producing the classical chronic disease with granuloma formation [9]. This is also supported by a study done in Israel where it was seen that only certain strains of *M. marinum* affected humans. They also demonstrate that in Israel strains affecting humans came from ornamental fish and not from local fish for consumption [5].

The mode of transmission to humans is mainly waterborne and fish borne. Person to person transmission has not been documented [137]. However, It has been described indirect transmission via fomites in at least 3 cases. Two very small children and one infant who acquired the infection after bathing in containers that were previously used to clean the family fish tanks of tropical fish [27, 40, 142].

As other environmental mycobacterium, *M. marinum* has commonly low pathogenicity. For this reason in normal conditions only affects disrupted skin [8]. The main risk factor to contract the infection consists in having lesions or abrasions in the skin with exposure to non chlorinated water or marine animals infected [140]. The most frequently nowadays is the exposure to private aquaria. But some times the source of exposure is unknown. As a consequence, after the description of cases following injuries with plants, it has been suggested the possibility that

could be other reservoirs different from water and fish. Although at the present moment this possibility has not been demonstrated [30, 43].

The incubation period is usually 3 to 4 weeks [135]. Following, the most common manifestation is a cutaneous lesion at the site of inoculation. It initiates as a solitary nodule or pustule that eventually evolves to an ulcer, abscess or verrucous plaque [143]. It affects more frequently the extremities, probably because the pathogen grows better at low temperatures [144].

The severity of the disease depends, among other factors, on the number of microorganisms inoculated [134]. In 20% of the cases the cutaneous lesions spread along ascending lymphatic vessels. This is called sporotrichoid spread or nodular lymphangitis [143]. As a result of direct extension invasion of deep structures as tendons, articulations and bones occurs in 29% of the cases [144]. Systemic dissemination is unusual but has been described in immunocompromised patients [140]. Spontaneous resolution, usually with scarring, has been documented from months after the infection up to 2 years [133, 135].

The diagnosis is based on the history of exposure and risk factors along with the characteristic clinical features. It is supported with histopathology, culture and bacteriological identification that in some cases require molecular biology techniques [94, 120]. The fact is that the diagnosis is not easy and in most of the cases is either delayed or remain being presumptive based in the history and response to treatment.

The objective of the treatment is to increase the speed of resolution and prevent progression of the disease [1]. With this purpose different combinations of antibiotics plus the support, in selected cases, of surgery are the common practices in the treatment of this infection. Although the election of the drugs still depends of the preference of individual authors and is not based on controlled evidence [143]

## **8.2 Excerpts from Review of Case Studies**

Note this is a very long and article. I have picked a few paragraphs out to highlight specific points. The numbers in brackets are references to other articles.

### **8.2.1 Variations on Appearance and Gestation**

The incubation period was documented only in 30 patients. It went from 1 day up to 4 months with an average of 48.9 days. The cases that presented an incubation period less than two weeks had in common to have suffered penetrating injuries with fish (figure 5).

Cutaneous lesions have been described as nodules in most of the cases but also can appear as papules or pustules and eventually abscess or ulcers or even as non-healing wounds[56]. Lesions that do not resolve spontaneously and have a very long time evolution have been described in Satowan island as large warty plaques[115].

The location is predominant in upper limbs but this is related to the site of inoculation and depends on the way of transmission. In other words, meanwhile exposure to aquaria are manifested as lesions in hands, in **Satowan** where the main affected population are taro farmers swimming in bomb crater lakes, knees are the most frequent location[115].

Sporotrichoid spread usually appears 4-8 weeks after the initial lesion but can occur from days up to 18 months of the same [139]. Nodular lymphangitis was one of the main characteristics of the HTD patients. It was also frequently described, almost 40%, in the papers of this review. It is an important sign that shortens the list of differential diagnosis.

### **8.2.2 Variations on Treatment**

About the treatment, 126 patients were treated with antibiotics; in the rest of the cases the management is not mentioned. Surgery was needed in 38 patients (84.4%) with affection of deep structures and in 21 patients (25%) with cutaneous lesions.

Monotherapy was used in 54 cases (42.8%), bitherapy in 38 cases (29.4%), triple therapy in 20 cases (15%) and combination of 4 or more drugs in 5 cases (4%). Finally combinations of drugs that included classical tuberculosis treatment were used in 10 cases (8.7%). In 41 patients the regimen of drugs needed to be changed, either for non effectiveness or non tolerance. The regimen was changed one time in 29 cases (21.8%), two times in 10 cases (7.5%), and up to 3 times in 2 patients (1.5%).

The drug more frequently used as monotherapy was clarithromycin, followed by minocycline, doxycycline, ciprofloxacin and trimethoprim-sulfamethoxazole. The combinations of drugs more frequently used were rifampicin + ethambutol followed by clarithromycin + rifampicin and clarithromycin + ethambutol. (Effectiveness of the different regimens depending on the extension of the disease can be seen in table 4)

Susceptibility tests were reported in 34 patients. Rifampicin was susceptible in 86.4% of the tests, ethambutol in 91.3%, clarithromycin in 95% and minocycline in 62.5%. Isoniazid was resistant in 100% of the tests done and streptomycin in 66.6% (see table 5).

The average time of duration of antibiotic treatment was 5.4 months, with a range of 12 days to 15 months. After completion of the treatment the final evolution of 109 patients (81.9%) was reported as good outcome or cured. Only in 8 patients (6%) the evolution was reported as bad outcome. No mention about the evolution was done in the rest of cases. Among 12 patients in which long follow up was reported, only one patient presented recurrence of the infection after 3 months course of doxycycline.

### **8.3 Recommendations**

Microbiological diagnosis is not always possible. For this reason, history of exposure will be essential in the diagnosis. Granuloma formation is not pathognomonic; however histology is important to support the diagnosis and to facilitate the differential diagnosis. Imaging techniques are useful to assess the extension of the infection. Currently, identification is done mainly after culture with classical biochemical tests. Implementation of molecular biology techniques in more centers will be an advantage for the diagnosis, in terms of accuracy and rapidity.

The literature shows mainly case reports and small series of cases. No clinical trials have been done. Consequently there is not enough evidence to propose any specific treatment. At present, recommendations are based on experts' opinions. However, monotherapy with clarithromycin, minocycline or trimethoprim-sulfamethoxazole has been used with acceptable success for non severe cutaneous lesions. In severe cases seems preferable to use combination therapy with

rifampicin + ethambutol. *M. marinum* is intrinsically a multidrug resistance mycobacterium. Although secondary resistances are rarely documented, susceptibility pattern will be an asset in cases that do not respond to treatment. Surgery indications must be carefully individualized.

A prospective, randomized controlled clinical trial, that probably would need to be interhospitalary, would be valuable to propose a base evident treatment.

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This list came from the dissertation above. It seems fairly complete as of the (unknown, but probably late 2009, early 2010) date of the dissertation

Note that there are two references to articles by Lillis, the guy who did the initial work on Satawan. See [32] and [115]

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