Lesson 2: Mix up with Osps

Discovery Files

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Do you enjoy hiking or camping in the woods?

Then chances are you have seen ticks. Maybe you’ve even found an engorged, or blood-filled, tick attached to your skin. That’s not a good thing.

Ticks are disease vectors. That means they can carry and pass on a disease. Lyme disease is a well-known tick-borne illness. It’s called that because several cases were identified in three towns in southeastern Connecticut in 1975, including the towns of Lyme and Old Lyme.

Lyme disease is caused by a spirochete that is passed to a victim through the saliva of an infected tick. Only the blacklegged tick—or deer tick—can carry Lyme. The scientific name for this tick is Ixodes scapularis.

Ticks are fascinating bloodsuckers.

Arthropods are the largest phylum of animals. Arthropods have segmented bodies, jointed legs and an exoskeleton. The Phylum Arthropoda includes insects, arachnids, crustaceans, and similar species.

Ticks are arthropods, but they are not insects. Insects have six legs. Ticks have eight legs just like spiders, mites, and scorpions.

These creepy crawlies can be either soft- or hard-bodied. Ixodes ticks are hard-bodied.

Ticks are also parasites. Ticks attach themselves to the body of another animal, known as the host. Ticks pierce the skin of the host with special mouthparts and feed on the host’s blood and fluids.

Ticks don’t only bite humans. They can feed on other hosts, including mammals, birds, reptiles, and amphibians. Ixodes ticks become infected with Lyme disease bacteria when they feed on small animals infected with the Borrelia burgdorferi germ —particularly the white-footed mouse.

Ixodes ticks have a two-year lifespan. As they grow, they pass through three growth stages: a six-legged larva, an eight-legged nymph, and an adult that also has eight legs.

To grow from one stage to the next ticks need a blood meal. A tick spends only a few days of each life stage attached to a host, feeding on its blood. Most of the time is spent waiting. Ticks wait for a host. They wait to molt. And they wait for warmer weather.

Larva

In the summer, an Ixodes tick hatches from its egg as a six-legged larva. The larva is about the size of the period at the end of this sentence and nearly transparent. To survive, the larva must hike up a blade of grass, a fallen leaf or twig and wait for a mouse or other small animal to pass by.

Ixodes ticks can also carry and transmit two other diseases, anaplasmosis and babesiosis.

Ticks cannot fly or jump. They must wait for a host to pass close enough to touch.
While waiting, the tick larva holds onto things with its back legs, while it stretches out its two other pairs of legs. A tick can find an animal by sensing carbon dioxide or body odors. It can also sense heat from a nearby animal. When it senses a host animal, the tick becomes excited. It begins waving its outstretched legs in order to grab onto the animal host. Sometimes the wait may take up to a month. If the waiting goes on too long, the larval tick will die.

If a tick finds a host, it may attach quickly or wander around looking for a good spot to bite. When the tick is ready for its meal, it slits the host’s skin with its razor-like mouthparts called chelicerae (say shel-ees-er-ray). It then inserts another mouthpart called the hypostome (say hi-po-stohm), which is a barbed and hollow tube. The successful tick then anchors itself in place with a glue-like substance in its saliva. The bite from most ticks is completely painless. The host usually doesn’t even know it is being fed upon.

A tick larva stays attached for 2 to 3 days as it slowly fills itself with blood. But, don’t worry. Larval ticks do not infect humans because they are not born with Lyme disease germs in their bodies. After its blood meal—often filled with germs—the larval tick falls to the ground to rest and to wait.

**Nymph**

When fall brings cooler weather, the larva tick molts into its next stage. It sheds its hard outer layer and emerges as an eight-legged nymph.

The nymph remains inactive during the cold winter months. It keeps warm and still under fallen leaves or other protection.

With the warming of spring the nymph begins to stir. When it emerges from its hiding place it again climbs a blade of grass or leaf and waits. The nymph is now ready for the second meal of its life.

At this stage they prefer human skin. Studies show that nymphs bite humans more than they bite other animals. Because of their small size (less than 2 mm), nymphs often stay on the body for 2-3 days without being noticed. During this time they slowly take blood. If they are infected, this is also the time when bacteria in their saliva pass into their human host. A tick nymph feeds until it is fully engorged with blood. After this blood meal, the nymph rests. Its body grows. And after 4-6 weeks, it molts again. It emerges from this molt into an adult.

**Adult**

In the fall—usually mid-October—an adult tick becomes active. Only adult ticks can be identified as female or male. And unlike mosquitoes, both sexes are bloodsuckers.

The adult tick behaves just like the nymph or larva. It climbs up grass or leaves and waits for a host. Because the adult is bigger, it can climb a bit higher. This allows it to find a larger animal. Adult *Ixodes* prefer white-tailed deer, but they will also suck blood from humans and dogs.
Females spend 7 days feeding until fully engorged. And they must feed before they can mate. Males do not need to feed before mating. Males also feed for a shorter time.

Mating may or may not occur on a host animal. Either way, 30 days after mating, a female *Ixodes* lays 2,000 to 3,000 eggs on the ground. She then dies within a few days. A male can mate a few times before he dies. Larval ticks emerge from these eggs the following summer, and the cycle begins again.
Discovery File
Bad Bull’s Eye!

Have you ever played a game that uses a target? Did you try to hit the target with an arrow or dart or beanbag? If you had good aim, you may have hit the smallest circle in the center. If so, someone probably yelled, “You hit the bull’s eye!” You felt great about doing your best.

But not all bull’s eyes are good.

Look at the picture to the right. It is a photo of a rash on the skin of a person with Lyme disease. Notice that it looks like a target, too. That’s why the Lyme disease rash is called a bull’s eye rash. The rash is an early symptom of the disease. A deer tick that was carrying Lyme disease bacteria bit this person. The rash appeared within 30 days after the bite. It started out as a small circle but kept getting bigger and bigger. Some bull’s eye rashes can grow to be 12 inches across!

But not every bull’s eye rash is easy to see or find. In fact, not every rash will look like a bull’s eye! The rash can be under your arm, or on your back, or any place else a tick can crawl on your body.

And, what if you have dark skin like the person below? This bull’s eye rash looks more like a bruise. If you look very carefully, you can see that the center is a bit lighter.

To make things even trickier, not everyone who gets Lyme disease develops a rash at all! In fact, 10% to 20% of people infected with Lyme disease won’t have a rash. There is even another reason Lyme disease can be hard to diagnose. Many of the symptoms listed on Lyme disease websites are “non-specific.” This means that the symptoms are not specific for Lyme disease. Fever, chills, fatigue (being tired all the time), and feeling achy are symptoms for lots of illnesses. Can you think of any?

Lyme disease does have some specific symptoms other than the rash. One specific sign of Lyme disease is a swollen knee joint. Another is not being able to move the muscles on one side of your face. When people with this symptom try to smile it only works on one side! You can see the photo of the girl on the right.

If you don’t have a bull’s eye rash, but you do have other specific symptoms, your doctor can do a blood test to tell whether or not you have Lyme disease.

Once you know you have it, Lyme disease can be easily treated with antibiotic medicine. There is no such thing as “long-term” Lyme disease. Once it is treated, the bacteria are gone from a person’s body.

But sadly, some people don’t find out they have Lyme disease until more serious symptoms show up. After several months without treatment, around 60% of people with Lyme disease develop arthritis (painful swollen joints). Another 5% of untreated cases have nervous system problems or changes in their heartbeat. Late Lyme disease can still be treated with medicine, but some of these patients will suffer from post-Lyme disease problems. You can read more about Lyme disease from this trusted website: http://www.cdc.gov/ncidod/dvbid/lyme/ld_resources.htm
Discovery File
Vaccines

The best protection against an infectious disease is one that lasts forever. This permanent protection is called immunity.

There are two kinds of immunity: innate and adaptive.

Innate immunity is a general ability of our immune system to fight invading microbes. Our innate immunity recognizes that an invader doesn’t belong and fights against it.

The problem is, our innate immunity doesn’t give us permanent protection.

It’s good that we also have adaptive immunity. Our immune systems not only spot and kill invaders, they also remember. Special immune cells remember the past invaders. They are ready to attack again the moment the same microbe shows it ugly face again.

How do scientists train our adaptive immunity to help us?

In 1796, an English country doctor named Edward Jenner noticed that people who got a mild disease called cowpox didn’t get the much more serious disease called smallpox.

So, Jenner did an experiment. He tested his theory on the son of his gardener, an 8-year old boy named James Phipps. Jenner injected James with fluid from the cowpox blisters of Sarah Nelmes—a milkmaid who had caught cowpox from a cow named Blossom.

Jenner injected the boy in both arms. This gave him a mild case of cowpox, but he soon recovered.

Next, Doctor Jenner found a person with smallpox and carefully took fluid from a smallpox blister. He injected the smallpox fluid into James Phipps.

What do you think happened?

You’re right! James never got smallpox, and the procedure we now call vaccination had been discovered. (A vaccine can be either a killed or weakened form of the disease germ.) Nowadays you could not do that; scientists must follow strict rules to do experiments with people.

Although it took almost 50 years before Jenner’s discovery became widely accepted, vaccination has saved millions of lives.

Today, there are vaccines for both bacterial and viral diseases. Some common vaccines give immunity against chicken pox, measles, whooping cough, polio, mumps, typhoid, and tuberculosis. Making a new vaccine involves a lot of research and many dollars.

Sadly, not every disease can be prevented by a vaccine.
When a germ gets past your skin, or the lining of your nose or mouth, special immune cells go to work. Together they are called white blood cells. But there are actually several different kinds of white cells each with a different name. Some kill bacteria by eating them or poisoning them. Other white cells fight parasitic worms. Others make antibodies.

Antigens are also proteins. They are found on the outer surfaces of all cells.

An antibody fits its target antigen like a key fits a lock.
are proteins that target **antigens** on the surface of the invaders.) These antibody-producing immune cells also remember the antigen and fight against it if you are exposed to the same germ again.

When you get a vaccination, antibodies are produced. Our antibody-producing cells remember the antigen on the vaccine and fight against it if you are ever exposed to the real germ.

But before either the innate or adaptive immune system attacks it must first identify which cells are part of you and which cells are strangers. If your body wasn’t able to tell the difference, the same immune system that is supposed to protect you might attack you instead.

But you are unique. You have special “self” proteins on the surface of every cell in your body. They make you different from almost everyone else in the world. (It is very rare for two people to have the same exact set of “self” proteins.)

Your immune system not only looks for invaders, it also keeps track of the cells that do belong to you. It makes sure that your cells are growing and behaving normally. If they are not, even your own cells can be attacked and destroyed.

Your **spleen** and your **lymph nodes** are also parts of your immune system. They help to filter your blood. When you have strep throat the “swollen glands” in your neck are really lymph nodes that have collected dead bacteria and dead immune cells to be flushed out of the body.

Not all antigens that don’t belong to you are bad. Foods we eat have antigens on their cell surfaces too. But sometimes, our bodies react to food antigens and otherwise safe antigens by mistake. This is called an **allergy** and the antigens that cause allergic reactions are called **allergens**.

Some people are allergic to strawberries. Their bodies think the antigens on the strawberry cells are dangerous. Other people are allergic to antigens on the skin cells of cats or dogs. Fortunately there are medicines that can help some people with their allergies. Others just have to stay away from the antigens that cause the problem.

**Are you allergic to anything? Can you think of other allergens?**

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**Vaccines** are prepared from disease-causing germs to provide immunity against the disease. They do this by stimulating the body’s antibody-making system with the same antigens found on the real germ.

**Even your “self” proteins are antigens.** Since they are **your** antigens they do not cause antibodies to be produced. Germ antigens do cause an immune response.

Cancer is one kind of abnormal cell growth that can usually be stopped by your self-monitoring immune system. If your immune system can’t stop cancer cells there are medicines and other treatments that help your immune system fight cancer. Sadly, even the most powerful cancer treatments don’t always work.
Phils to the Rescue!

When a germ gets past your skin, special immune cells go to work.

<table>
<thead>
<tr>
<th>Neutrophil</th>
<th>Monocyte</th>
<th>Lymphocyte</th>
<th>Basophil</th>
<th>Eosinophil</th>
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<tr>
<td>59%</td>
<td>4%</td>
<td>34%</td>
<td>0.5%</td>
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The first immune cells that fight the bacteria that get past your skin are the **neutrophils and macrophages**. They kill bacteria by eating them or poisoning them.

If the invader is a parasite, it’s **eosinophils** to the rescue. Eosinophils are best at fighting parasitic worms too.

**Mast** cells and **basophils** play an important role in allergies.

**Adapting from B to T**

Your body also has **adaptive immunity**. Your innate defenses teach cells called **lymphocytes** to fight invaders and remember them so you won’t get the same illness again. Just like the neutrophils, macrophages, eosinophils, mast cells, and basophils, the lymphocytes called **B** and **T** cells are also made in your bone marrow.

When **T cells** leave your bone marrow they travel to your **thymus gland** to mature. That’s why these cells are called **T cells** – **T** is for thymus!

**B cells** have one job – to make **antibodies**. Antibodies are proteins that target **antigens** on the surface of invaders and attack them if they return.

When you get a vaccination, B cells remember the antigen on the vaccine germ and fight against it if you are exposed to that germ again.