JOURNAL OF
HYGIENE SCIENCES
Committed to the advancement of Clinical & Industrial Disinfection & Microbiology
VOLUME - XI ISSUE - III AUG - SEP 2018

Editorial

Contents

Mini Review Section: The correct identification of microorganisms is of fundamental importance to microbial systematists as well as to scientists involved in many other areas of applied research and industry (e.g. agriculture, clinical microbiology and food production). Most bacteria are quite colourless and transparent and have a refractive index similar to that of the aqueous fluids in which they are suspended. Owing to the small size of bacteria little structural details can be seen with the ordinary light microscope unless the organisms are stained. The majority stain readily with aniline dyes.

Current Trends: Perioral dermatitis is a common skin rash. Perioral means 'around the mouth' and dermatitis refers to inflammation of the skin. Perioral dermatitis mainly affects women aged 15-45 years. Perioral dermatitis is uncommon in men and children.

In Profile: Charles-Jules-Henri Nicolle a physician, microbiologist, novelist, philosopher and historian. Nicolle's many accomplishments include the discovery that epidemic typhus is transmitted by body lice (*Pediculus humanis corporis*), discovery of the phenomenon of in apparent infection, and possibly the first isolation of human influenza virus after experimental transmission. Nicolle made many other fundamental contributions to knowledge of infectious diseases.

Bug of the Month: Mosquito season is around the corner, bringing with it a higher risk of catching potentially serious diseases transmitted by their bite. Mosquitoes also may increase the severity of the diseases they transmit, and researchers think that mosquito saliva plays an active role in this process. A team of researchers at Baylor College of Medicine has taken a closer look at the effect of mosquito saliva alone and found that it can trigger an unexpected variety of immune responses in an animal model of the human immune system.

Best Practices: Occupational asthma (OA) is one of the most common chronic occupational lung diseases and workplace factors have been estimated to contribute to ~10% of all adult-onset asthma]. A subset (~10% or less of all OA) can be caused by an acute irritant exposure. This has been termed irritant-induced asthma and includes reactive airways dysfunction syndrome. The majority of OA (80–90%) is caused by specific sensitization to a workplace agent.

Inspire yourself with the motivational quotes in our Relaxed Mood section. We would like to take this opportunity to thank all our esteem readers for their continuous support & encouragement in making this Journal a successful effort.

Looking forward for your feedback & suggestions.

Biological Stains and its Applications (Issue-2)

The correct identification of microorganisms is of fundamental importance to microbial systematists as well as to scientists involved in many other areas of applied research and industry (e.g. agriculture, clinical microbiology and food production). Most bacteria are quite colourless and transparent and have a refractive index similar to that of the aqueous fluids in which they are suspended. Unless the diaphragm is carefully adjusted usually there is considerable difficulty in bringing the organisms into focus. Owing to the small size of bacteria little structural details can be seen with the ordinary light microscope unless the organisms are stained. The majority stain readily with aniline dyes. Some staining techniques, such as the Gram and Ziel Neelsen stains, although of great diagnostic value because of their differential staining properties for specific bacteria, reveal little internal structure. Other such as Feulgen stain for nuclear bodies, demonstrates specific structure. Because of its importance, different important stains are described in some detail.

HANGING DROP TECHNIQUE

The techniques employed are meant for microscopic observation of living bacteria. The motility study of bacterial morphology is performed in two ways:

1. Observing unstained cells live by hanging drop preparation.
2. Observing dead cells by making use of chemical nature of their unicellular, body. This is achieved by staining.

Hanging drop technique enables viewing of size shape, arrangement and motility of live microorganisms in fluid media. It requires the use of special ground slides. In this technique a loopful of bacterial suspension is placed in the centre of a cover slip. In the four corners tiny droplets of mineral oil are placed. The hollow ground slide is placed over the cover slip with the depression side down and the slide is inverted quickly so that the water cannot run off to one side. However, the lack of contrast yields limited though valuable information. For pathogens one tube one plate method can be used. Each method has its advantage and limitations. The method you use will depend on which one is most suitable for the situation at hand.

Hanging Drop Preparation or Motility Test

1. Apply Vaseline around the depression of the hanging-drop slide.
2. Using the inoculation loop, aseptically transfer one drop of the culture to the centre of a clean cover slip.
3. Invert the hanging-drop slide and centre its well over the drop of the culture, Press down on the edge of the cover slip so that the Vaseline makes a firm seal.
4. Quickly and carefully turn the slide right side up so as to suspend the hanging drop in the well. Don't let the drop fall or touch the bottom of the well.
5. To examine, first locate its edge in centre of the microscopic field with low power objective and markedly lower the light. The edge will be visible, as a bright wavy like against a dark background. Now the slide can be focused under oil immersion (Figure 1).

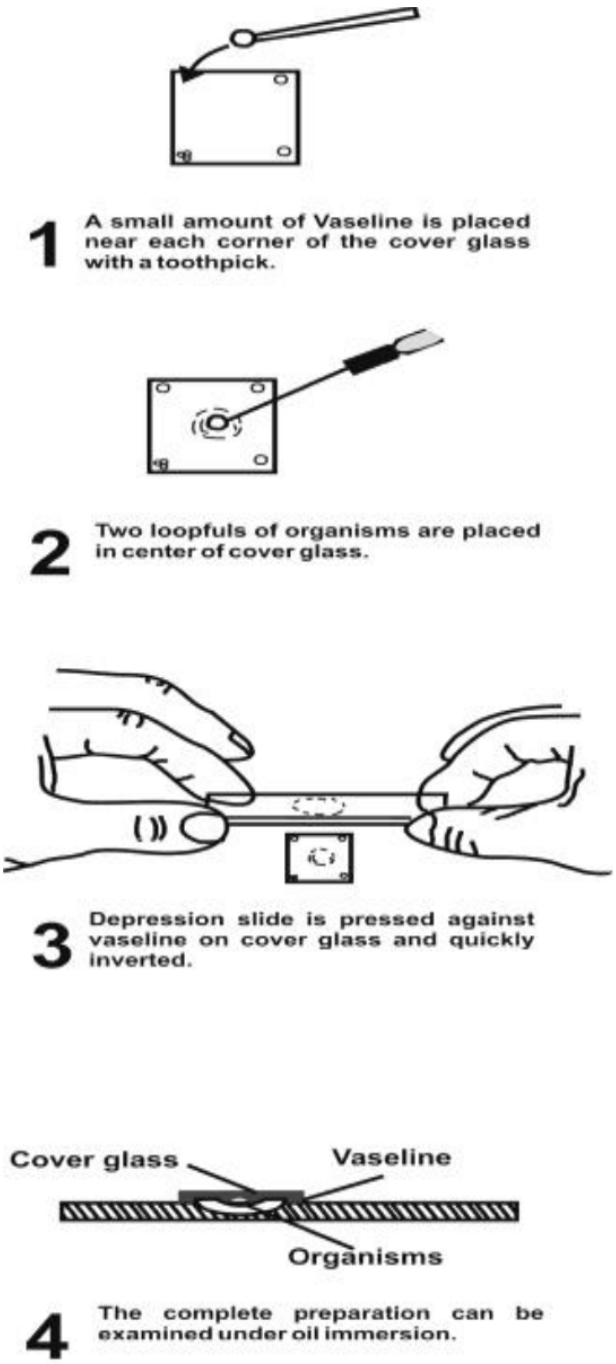


Figure 1. Hanging Drop Preparation

When working with pathogenic microorganisms such as the typhoid bacillus, it is too dangerous to attempt to determine motility with slide techniques. A much safer method is to culture the organisms in a special medium that can demonstrate the presence of motility. The procedure is to inoculate a tube of semisolid or SIM medium that can demonstrate the presence of motility. Both media have a very soft consistency that allows motile bacteria to migrate readily through them causing cloudiness. Following Figure 2 illustrates the inoculation procedure.

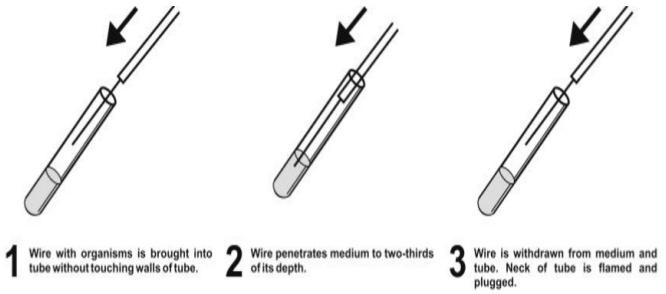


Figure 2. Stab technique for motility test

MICROBIAL STAINING

It is a chemical or a physical union between the dye and like component of a cell. If it is a chemical reaction a new compound is formed and a simple washing with water does not liberate the bound dye but if purely physical it is easy to decolorize such stained organism. Usually it is a combination of chemical and physical reactions.

The main advantages of staining are that it:

(i) Provides contrast between microorganisms and their backgrounds, permitting differentiation among various morphological types;

(ii) Permits study of internal structures of the bacterial cell, such as the cell wall, vacuoles or nuclear bodies and other cellular structures;and

(iii) Enables the bacteriologist to use higher magnifications.

Fixing

Before staining it is essential to fix the bacterial sample on to the slide. Smear is prepared in the following way:

(i) With a loop place a small drop of the broth culture or a loop full of bacteria on a clean slide.

(ii) Place a drop of water over it.

(iii) Spread the culture so as to form a thin film.

(iv) Allow slide to dry in the air or by holding it above a Bunsen flame.

(v) Avoid excess heating.

The purpose of fixation is to kill the microorganisms, coagulate the protoplasm of the cell and cause it to adhere to the slide.

Type of Stains

1. SIMPLE STAINING

The use of a single stain to colour a bacterial organism is commonly referred as simple staining. All these dyes work well on bacteria as they have colour bearing ions (chromatophores) and are positively charged. The fact that bacteria are slightly negatively charged when the pH of the surrounding is near neutrality and produces a pronounced attraction between these cationic chromatophores and the organism so that the cell is stained. Such dyes are classified as basic dyes (Figure 3). Crystal violet and carbolfuschin are some other examples.

Those dyes that have anionic chromatophores are called acidic dyes. Eosin (sodium^{+}eosine^{-}) is such a dye. The anionic chromatophores, eosine^{-}, will not stain bacteria because of the electrostatic repelling forces that are involved.

The staining times for most simple stains are relatively short, usually from 30 seconds to 2 minutes, depending on the affinity of the dye. After a smear has been stained for the required time, it is washed off gently, blotted dry, and examined directly under oil immersion. Such aslide is useful in determining basic morphology and the presence or absence of certain kinds of granules.

Figure 3. Demonstration of staining procedure

NEGATIVE STAINING

A better way to observe bacteria for the first time is to prepare a slide by a process called negative or background staining. This method consists of mixing the microorganisms in a small amount of nigrosine or India ink and spreading the mixture over the surface of the slide (nigrosine is far superior to India ink). Since these two pigments are not really bacterial stains, they do not penetrate the microorganisms; instead they obliterate the background, leaving the organisms transparent and visible in a darkened field. Although this technique has a limitation, it can be useful for determining cell morphology and size. Since no heat is applied to the slide, there is no shrinkage of the cell and consequently more accurate cell size determination result than with some other methods. This method is also useful for studying spirochetes that does not stain readily with ordinary dyes.

Negative staining can be performed by one of the following methods. Figure 4 illustrates the more commonly used method in which the organisms are mixed in a drop of nigrosine and spread over the slide with another slide in order to prepare a smear that is thick at one end and feather thin at the other end. Somewhere between the too thick and too thin areas will be an ideal spot to study the organisms.

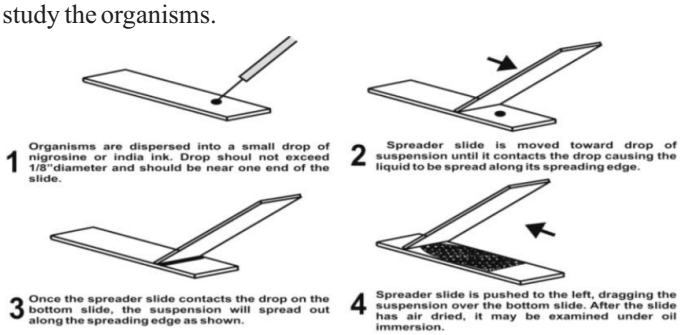


Figure 4. Demonstration of method of negative staining

In second method, the organisms are mixed in only a loopful of nigrosine instead of a full drop. The organisms are spread over a smaller area in the centre of the slide with an inoculating needle. No spreader slide is used in this method. It gives more accurate view of the bacterial cell (Figure 5).

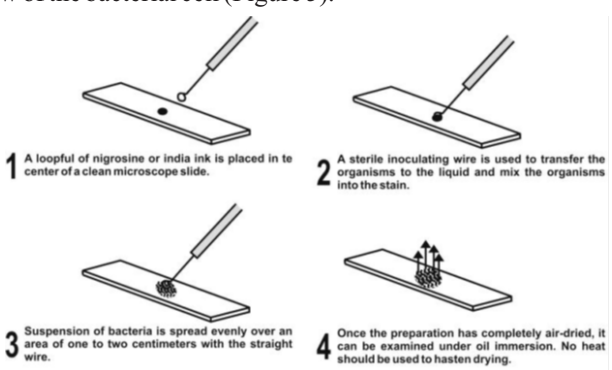


Figure 5. Method of negative staining by Nigrosine

While negative staining is a simple enough process to make bacteria more visible with a bright-field microscope, it is of little help when one attempts to observe anatomical microstructures such as flagella, granule, and endospore. Only by applying specific bacteriological stains to organisms one can see such organelles. However, success at bacterial staining depends on the preparation of a suitable smear of the organisms. A properly prepared bacterial smear is one that withstands one or more washings during staining without loss of organisms; should not be too thick; and does not result in excessive distortion due to cell shrinkage. The procedure is illustrated in Figure 6.

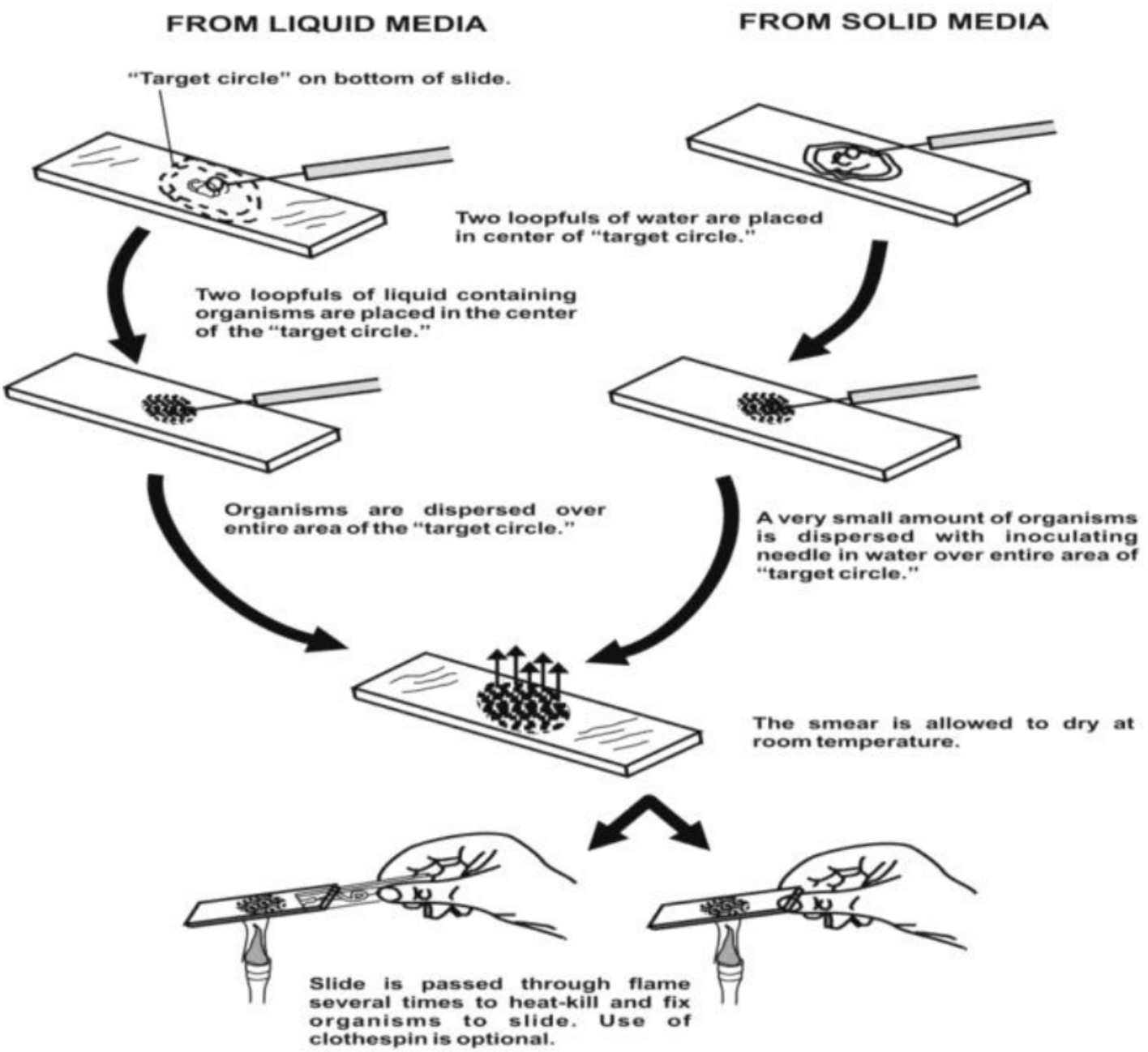


Figure 6. Complete ideal staining procedure from two different sources

…To be continued.

Personal Dermatitis

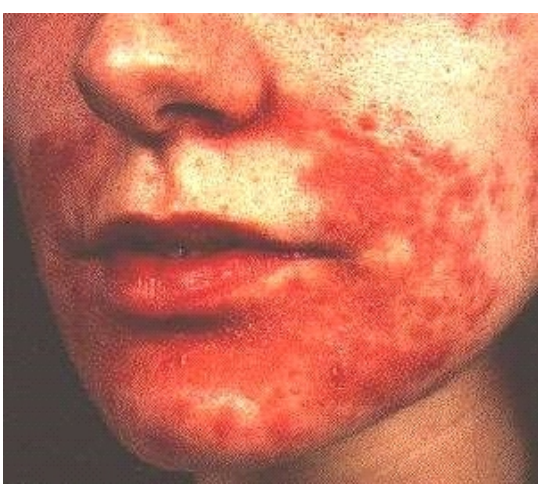
Perioral dermatitis is a common skin rash. Perioral means 'around the mouth' and dermatitis refers to inflammation of the skin.

Who gets it?

Perioral dermatitis mainly affects women aged 15-45 years. It's fairly common and is thought to affect 1 in 100 women. Men don't get away scot-free. The increasing trend for 'cool dudes' to use skincare products has meant that we're starting to see perioral dermatitis in men too.

Typically, small red or pink lumpy spots develop on the skin anywhere around the outside of the mouth. That is, they may appear on the chin, the cheeks and the skin next to and below the nose.

They look a little like acne spots but perioral dermatitis is not acne. The skin under and next to each spot is often red or pink. If there are a lot of spots next to each other then the area of affected skin can just look red and lumpy. Sometimes the skin surface can become dry and flaky.



Typically, the skin just next to the lips is not affected, or is affected much less than the skin just a little further away from the lips. So, in some cases, it looks like the rash forms almost a ring around the mouth but sparing a small border of skin next to the lips. Occasionally, the skin around the eyes is also affected.

The severity of the rash can vary from a few minor spots that are barely noticeable, to a definite and obvious lumpy rash that is around the mouth. The rash is not usually painful or itchy. However, some people report a mild burning or itchy feeling. Others report that the affected skin feels tense. The rash is not serious and is not associated with any underlying disease. However, it can be unsightly.

Who develops perioral dermatitis?

Almost all cases occur in young women, most commonly between the ages of 15 and 45 years. It is thought to affect up to 1 in 100 women at some point in their lives. Perioral dermatitis is uncommon in men and children. However, as the number of men using facial skin products increases, the number of men with perioral dermatitis is increasing.

- Children 6 months to 16 years old

What causes perioral dermatitis?

The exact cause is not clear. However, in many cases the rash seems to be triggered by one or more of the following:

- Steroid creams and ointments are a main trigger. See below for details.
- Make-up, cleansers and cosmetics applied to the area affected on the face. It may be that certain ingredients of cosmetics may act as the trigger. For example, one study found that make-up foundation seemed to be a particular provoking factor.
- Physical factors such as strong winds and UV light.
- Fluoridated toothpaste has been suggested as a possible trigger.

Fluorinated Toothpastes/Creams

Another factor seen to cause perioral dermatitis is the use of fluorinated toothpastes, or other fluorine containing compounds. In a study conducted to 65 patients with perioral dermatitis, it was found that almost all of them used fluoride toothpastes. Replacing the toothpastes with non-fluoride relieved the patients of their symptoms.

- Yeasts and germs (bacteria) that live on the skin and in hair follicles have been suggested as a possible trigger. (However, perioral dermatitis is not just a simple skin infection.)

Microorganisms

In certain cases, particular microbial specie is attributed to perioral dermatitis. Although studies are not yet conclusive, certain cases were seen to be related to the emergence of this condition. Microorganisms seen to cause perioral dermatitis include:

1. Fusiform Bacteria
2. Dermodexfolliculorum
3. Microscopic mites

Certain expert say that the "immune suppressing" effect of corticosteroids may have contributed to the proliferation of such microorganisms. This then leads to perioral dermatitis.

- Hormone factors may play a part, as some women find that the rash becomes worse just before a period.
- The oral contraceptive pill may be a factor in some cases.

Recently, a study has found that some sun creams used on the face

may be a trigger for perioral dermatitis in some children and adults. A liquid, gel or light milk sunscreen may be the best to use.

What about steroid creams and ointments?

There is a well-known link between using a topical steroid (steroid creams, gels, ointments, etc) and developing perioral dermatitis. Many cases develop soon after using a topical steroid on the face for another condition, such as mild eczema. Without realising you are doing so, you may even rub some steroid on your face if you are treating another part of your body with a topical steroid. For example, you may scratch the treated area of your skin (say, your elbow) and then, without realising you are doing so, rub the finger used for scratching on to your face.

Topical steroids can also clear a mild patch of perioral dermatitis temporarily. Some people will have tried a steroid cream, which can be bought at pharmacies, to treat what they think is mild eczema. However, as soon as the rash clears and the steroid is stopped, the rash reappears, only even worse. This can become a vicious circle as they may then put more steroid cream on to clear the new rash, which may clear again. They may stop the steroid again, only for the rash to come back yet again and even worse, etc.

How is perioral dermatitis diagnosed?

Perioral dermatitis is usually diagnosed from its appearance. There is not much else that looks like it, but there are a few other conditions it can be mistaken for:

- Rosacea
- Acne vulgaris
- Contact dermatitis

Tests are usually not needed unless perioral dermatitis does not improve with treatment and one of these other conditions needs to be ruled out.

What is the treatment for perioral dermatitis?

Without treatment, the condition may last for months or years. The following treatments can usually help to clear the rash. However, it may take some time for the treatment to work.

Stop using anything on your face

Firstly, your doctor is likely to advise you to stop using any cream, ointment, cosmetic, etc, on your face. In particular, your doctor may advise you to stop using any topical steroid. If you have been using a topical steroid, the rash will worsen for several days before it gets any better. You need to anticipate and accept this. Whilst the rash is present, just wash your face with water only. Some doctors advise not using toothpaste that contains fluoride.

Even when the rash has gone, it is best not to use any cosmetics or creams on the affected area, as the rash may reappear. And use only a bland liquid face cleaner to wash your face, rather than bar soap.

Antibiotic medicines

Your doctor may prescribe an antibiotic tablet in the tetracycline group. Doxycycline or tetracycline topical antibiotics are sometimes used in milder cases. The course of treatment is usually for six to twelve weeks. You may not notice any improvement for the first few weeks of treatment. However, there is an improvement in most cases within two months after starting antibiotic treatment. So, do persevere if an antibiotic is prescribed. The way antibiotics work in this condition is not clear. It is not a simple skin infection. However, tetracyclines and some other antibiotics have an action to reduce inflammation in addition to killing germs (bacteria) and this may be why they work.

Other treatments

Other treatments are sometimes used for perioral dermatitis. These include pimecrolimus cream. This cream works to reduce skin inflammation. It seems to be particularly effective in perioral dermatitis that has been caused by using topical steroids.

Perioral dermatitis is notcontagious (cannot be spread from person to person).

Diet for Perioral Dermatitis

Diet Rich In Vitamins A, E and B12

These vitamins promote a healthy skin. Vitamin A helps in regeneration new skin cells, while Vitamin E helps reduce inflammation and protects your skin cells from further damage. Vitamin B12, on the other hand strengthens the skin's protective barrier preventing further damage and irritation.

Failsafe Diet

The failsafe regimen stands for a diet free of additives, low in salicylates, amines and artificial ingredients. Such compounds can worsen or start an eruption, following this diet for at least 2 to four weeks can help lessen the lesions.

Probiotics and Vitamin C

Probiotics and Vitamin C share one action, and that is to boost the immune system in fighting infections and controlling inflammation. Since perioral dermatitis can be worsened by a secondary infection, preventing such would hasten your recovery.

Reference:

h00ttps://patient.info/health/skin-rashes/perioral-dermatitis
https://ehealthwall.com/perioral-dermatitis/
https://www.google.co.in/search?rlz=1C1NHXL_enIN804IN804&biw=1280&bih=918&tbm=isch&sa=1&ei=471RW6iJEYH6vgTJ17mADQ&q=healthy+skin+perioral+area&oq=healthy+skin+perioral+area&gs_l=img.3...10914.20235.0.20958.14.14.0.0.0.0.185.2089.0j14.14.0....0...1c.1.64.img..0.4.655...0j0i30k1j0i5i30k1j0i8i30k1.0.bde08BB-_uw#imgrc=6U9RexCD5a1MGM:

Charles Nicolle

Charles-Jules-Henri Nicolle (1866–1936), a physician, microbiologist, novelist, philosopher, and historian. From 1903 until his death in 1936, he was Director of the Institute Pasteur in Tunis, Tunisia. Nicolle's many accomplishments include the discovery that epidemic typhus is transmitted by body lice (*Pediculus humanis corporis*), discovery of the phenomenon of in apparent infection, and possibly the first isolation of human influenza virus after experimental transmission. Nicolle made many other fundamental contributions to knowledge of infectious diseases. This year is the centenary of his discovery about typhus transmission, made in the summer of 1909, for which he was awarded the 1928 Nobel Prize in Physiology or Medicine.

Nicolle was born on September 21, 1866, in Rouen, the ancient capital of Normandy, France. He obtained a classical education and was greatly attracted to literature, history, and the arts, interests he nurtured throughout his life. Bowing to the wish of his physician father, however, Nicolle studied medicine. After 3 years at the medical school in Rouen, he proceeded to Paris for further training and received a medical degree from the Institute Pasteur in 1893. At 27 years of age, Nicolle returned to his hometown, where he served as a member of the medical faculty and as Director of the Bacteriological Laboratory His 8 years in Rouen were difficult: his position was untenured, his colleagues were reluctant to accept his modern ideas about bacteriology, and he experienced a hearing loss that prevented him from effectively using a stethoscope. These challenges may have motivated him to take a leap that he might otherwise not have taken when the post of directorship of the Institute Pasteur in Tunis became open. It was offered to his elder brother, Maurice (1862–1932), an established experimental scientist, who refused it. Charles then applied and obtained the position.

Nicolle arrived in Tunis in 1902, when he was 36 years old. North Africa was a good place to study infectious diseases, including brucellosis, diphtheria, leishmaniasis, leprosy, malaria, measles, Mediterranean spotted fever, relapsing fever, scarlet fever, tuberculosis, and typhus. Of all the problems Nicolle faced in Tunis, however, epidemic typhus was, in his words, “the most important and the least explored.” He studied it for the next 7 years. He was well aware of the clinical presentation of typhus—its triad of fever, rash, and stupor—and of its link to poverty. Throughout history, typhus had been a highly communicable and frequently fatal disease. Before it began to be understood as a single infectious disease distinguished epidemiologically from typhoid (in the mid to late 19th century), typhus had been considered a collection of distinctive diseases that affected specific populations. It devastated armies during wars (“war typhus”) and prisoners living under unsanitary conditions (“jail typhus” or “jail fever”); it affected displaced populations suffering from famine, floods, and other natural disasters; and in general, it was a disease of poverty.

In Tunis, typhus struck in seasonal waves during the cooler months and disappeared during the summer. It spread through overcrowded prisons, asylums, and tent villages, taking a heavy toll in hospitals among admissions personnel and sometimes even among examining physicians. Most of the doctors in the Tunisian health system, especially those in rural districts, had contracted typhus; approximately one third of them died from it. Nicolle's first encounter with typhus could have potentially been his last. In 1903, he escaped death when at the last moment he cancelled a trip to investigate a prison outbreak. His 2 colleagues went on to the prison without him and spent the night there; both became ill with typhus and died.

Nicolle's discovery of how typhus is transmitted came from observations at the entrance and waiting room of the Sadiki Hospital, which primarily served indigent patients. He often had to step over the bodies of typhus-infected patients who had fallen and died at the doorway. Nicolle observed that typhus patients who were admitted spread their infections to others up to the point at which they entered the hospital waiting room. Included among these secondary cases were persons who took charge of their clothing. However, patients became completely noninfectious as soon as they were bathed and dressed in a hospital uniform. They could then enter the general wards without posing a risk to others. Once Nicolle realized this, he reasoned that lice on patients' clothes were most likely the vectors.

To test his hypothesis about lice, Nicolle requested and promptly received a chimpanzee (*Pan troglodytes*) from his mentor, Pierre-Paul-Émile Roux (1853–1933), at the Paris Institut Pasteur. Nicolle injected the chimpanzee with blood from a typhus patient. Twenty-four hours later, the chimpanzee was febrile, had new skin eruptions, and was prostrate. Because chimpanzees were costly, Nicolle then injected a toque macaque (*Macaca sinica*) with blood from the ill chimpanzee. Thirteen days later the macaque became febrile. Nicolle fed 29 lice on the ill macaque, and over the next few days transferred the lice to feed on other macaques. Eventually, macaques in this latter group became ill as well.

Thus, in June 1909, Nicolle reproduced typhus in a chimpanzee; in August 1909, he demonstrated that lice are the carriers of typhus; and in September 1909, he communicated his discovery to the French Académie des sciences. In these simple experiments, Charles Nicolle had solved the mystery surrounding the transmission of one of humankind's most dreaded scourges, a disease that had been a major force in shaping world history. Later research showed that the principal transmission method was not the bites of lice but the excrement of lice rubbed into the skin or eyes.

Indeed, in the year after Nicolle's typhus discovery, Howard Taylor Ricketts (1871–1910) and Russell Morse Wilder (1885–1959), working in Mexico, confirmed louse transmission of typhus. In 1916, Henrique da Rocha-Lima (1879–1956)

identified the causative organism and named it *Rickettsia prowazeki*in memory of Ricketts and Stanislaus Joseph Matthias von Prowazek (1875–1915), both of whom had died of typhus contracted during their scientific investigations.

Although Nicolle is not credited with discovering the cause of human influenza, his contributions were seminal. In 1903, when he had just joined the Institut Pasteur in Tunisia, his mentor Émile Roux reviewed the literature on "filter-passing" agents (hypothetical subbacterial agents that passed through Berkfeld and Chamberland filters). Roux identified 10 of them that he believed to be scientifically proven as causative agents of disease, among them what we now know to be viruses and mycoplasmas. Working at Turkey's Imperial Institute of Bacteriology, Nicolle's brother Maurice and colleagues had isolated the filter-passing agent of rinderpest (later characterized as a paramyxovirus). Charles Nicolle, who had also worked with rinderpest, was familiar with these new techniques.

When the deadly influenza pandemic struck in 1918, Nicolle was among the few scientists in the world prepared to study its etiology. At the time, the cause of influenza was unknown, but many doubted the conventional explanation that it was a bacterial disease. Beginning on September 1, 1918, Nicolle injected Chamberland-filtered and unfiltered sputum samples from ill patients into human volunteers and into monkeys, reproducing in some experiments a febrile influenza-like illness. However, the scarcity of clinical material and the rapidity with which the epidemic advanced precluded large-scale controlled studies. Within a few months, a Japanese group appeared to reproduce and extend the results of the 2 French scientists, but other investigators had trouble doing so. As the pandemic faded into endemicity, further experimentation became difficult for all researchers. When influenza viruses were eventually isolated and characterized in mice and in ferrets more than a decade later, Nicolle was finally acknowledged as having made the first isolation and as having taken the first important steps toward finding influenza's cause.

In addition to increasing knowledge about typhus and influenza, Nicolle made important contributions to the understanding of brucellosis, leishmaniasis, measles, rinderpest, scarlet fever, Mediterranean spotted fever, toxoplasmosis, trachoma, and tuberculosis. Perhaps his greatest discovery, a critical key to understanding the epidemiology of many infectious diseases, was characterization of the phenomenon of in apparent infection, the acquisition and transmission of infection without signs of illness. This line of work began with Nicolle's observations on experimental typhus. He learned that guinea pigs were good hosts for the typhus organism and showed that certain guinea pigs could have apyretic typhus after a primary infection of pyretic typhus. Nicolle extended his observation to other infections—viral, bacterial, and parasitic—finding similar phenomena in each. As Charles-Edward Amory Winslow (1877–1957) emphasized in his classical work, The Conquest of Epidemic Diseases: A Chapter in the History of Ideas (1943), in apparent infection is one of the most important concepts in infectious disease epidemiology, and it had for centuries been one of the key missing links, which prevented full understanding of the principles of disease transmission. In apparent infection of symptomless carriers is now generally accepted as the source for dissemination of many communicable diseases. Nicolle considered it his most important discovery.

Motivational Quotes

1. "Success is most often achieved by those who don't know that failure is inevitable." -- *Coco Chanel*
2. "Things work out best for those who make the best of how things work out. - *John Wooden*
3. "Courage is grace under pressure." -- *Ernest Hemingway*
4. "If you are not willing to risk the usual, you will have to settle for the ordinary."- *Jim Rohn*
5. "Learn from yesterday, live for today, hope for tomorrow. The important thing is not to stop questioning." -- *Albert Einstein*
6. "Take up one idea. Make that one idea your life -- think of it, dream of it, live on that idea. Let the brain, muscles, nerves, every part of your body be full of that idea, and just leave every other idea alone. This is the way to success." -- *Swami Vivekananda*
7. "Sometimes you can't see yourself clearly until you see yourself through the eyes of others." -- *Ellen DeGeneres*
8. "All our dreams can come true if we have the courage to pursue them."- *Walt Disney*
9. "It does not matter how slowly you go, so long as you do not stop."- *Confucius*
10. "Success is walking from failure to failure with no loss of enthusiasm."- *Winston Churchill*
11. "Someone is sitting in the shade today because someone planted a tree a long time ago." -- *Warren Buffett*
12. "Whenever you see a successful person, you only see the public glories, never the private sacrifices to reach them." -- *Vaibhav Shah*
13. "Don't cry because it's over, smile because it happened." -- *Dr. Seuss*
14. "Success? I don't know what that word means. I'm happy. But success, that goes back to what in somebody's eyes success means. For me, success is inner peace. That's a good day for me." -- *Denzel Washington*
15. "You only live once, but if you do it right, once is enough." -- *Mae West*
16. "Opportunities don't happen. You create them." -- *Chris Grosser*
17. "Once you choose hope, anything's possible." -- *Christopher Reeve*
18. "Try not to become a person of success, but rather try to become a person of value." -- *Albert Einstein*
19. "There is no easy walk to freedom anywhere, and many of us will have to pass through the valley of the shadow of death again and again before we reach the mountaintop of our desires." -- *Nelson Mandela*
20. "It is not the strongest of the species that survive, nor the most intelligent, but the one most responsive to change." -- *Charles Darwin*
21. "The best and most beautiful things in the world cannot be seen or even touched -- they must be felt with the heart." -- *Helen Keller*
22. "Great minds discuss ideas; average minds discuss events; small minds discuss people." -- *Eleanor Roosevelt*
23. "Live as if you were to die tomorrow. Learn as if you were to live forever." -- *Mahatma Gandhi*
24. "The best revenge is massive success." -- *Frank Sinatra*
25. "The difference between winning and losing is most often not quitting."- *Walt Disney*

Moraxella catarrhalis

Moraxella catarrhalis (M. catarrhalis) is a type of bacteria that's also known as *Neisseria catarrhalis* and *Branhamella catarrhalis*.

It used to be considered a normal part of the human respiratory system, but more recent research shows that can it sometimes causes infections.

Many young children have *M. catarrhalis* in their respiratory tract in the first few years of life, but it doesn't always cause infections. When it does, it often results in a simple ear or sinus infection. In children with weakened immune systems, it can cause more serious infections, such as pneumonia or bronchitis.

Adults, on the other hand, usually don't have *M. catarrhalis* in their respiratory tract. When they do, they typically have a weakened immune system due to an underlying condition, such as an autoimmune disorder, or from treatment such as chemotherapy.

Adults with lung conditions, especially cystic fibrosis and chronic obstructive pulmonary disease (COPD), are also more likely to develop an *M. catarrhalis* infection. This is because chronic lung conditions make it harder for your lungs to clear out bacteria.

What does it cause?

Middle ear infection

M. catarrhalis is increasingly recognized as a common cause of acute otitis media, also known as a middle ear infection, in children. Many young children have this bacterium in their noses, and it can sometimes move into the middle ear, causing infection.

Pneumonia

Pneumonia is an infection in the lungs that's often caused by bacteria. While *M. catarrhalis* typically doesn't cause pneumonia, it can in adults with weakened immune systems or chronic lung diseases. People with a lung disease who spend a lot of time in hospitals have the highest risk of developing pneumonia due to *M. catarrhalis*.

Bronchitis

Bronchitis is an inflammation of the lungs that's usually caused by a virus, not bacteria. However, in adults with weakened immune systems or chronic lung conditions, *M. catarrhalis* can cause bronchitis. Like pneumonia, bronchitis due to *M. catarrhalis* is most common in adults with lung conditions in hospitals.

Both pneumonia and bronchitis produce similar symptoms, the main one being a cough that produces mucus and often lasts for weeks. However, the symptoms of pneumonia are usually more severe.

Sinus infection

M. catarrhalis can also cause sinus infections in children as well as adults with weakened immune systems. Symptoms of a sinus infection are similar to those of a cold, but tend to get worse over the course of a week rather than better. They can also cause greenish-yellow discharge in your nose, pressure or pain in your face, and a fever.

COPD

COPD refers to a group of lung diseases that worsen over time. These include chronic bronchitis, emphysema, and refractory asthma, which is asthma that doesn't get better with regular treatment.

The main symptoms of COPD are coughing, wheezing, coughing up mucus, chest tightness, shortness of breath, and difficulty breathing.

While COPD slowly gets worse over time, infections can speed up the process and cause serious complications, including death.

M. catarrhalis is the second most common bacterial cause of worsening COPD. It can increase mucus production, make mucus thicker, and make it even harder to breath.

Pink eye

Conjunctivitis, commonly known as pink eye, is an infection of the outer layer of your eye. *M. catarrhalis* can cause pink eye in both children and newborns.

Meningitis

In very rare cases, *M. catarrhalis* can cause meningitis, especially in newborns. Meningitis refers to inflammation of the meninges, which are layers of tissue that surround the brain. While most cases of meningitis are preventable with a vaccine, there's no vaccine for *M. catarrhalis* yet.

Can you treat it?

Infections caused by *M. catarrhalis* usually respond well to antibiotics. However, almost all strains of *M. catarrhalis* produce an enzyme called beta-lactamase, which makes them resistant to some common antibiotics, such as penicillin and ampicillin.

Common antibiotics used to treat *M. catarrhalis* infections include:

- amoxicillin-clavulanate (Augmentin)
- trimethoprim-sulfamethoxazole (Bactrim)
- extended-spectrum cephalosporins, such as cefixime (Suprax)
- macrolides, such as azithromycin (Zithromax)

Adults can also take tetracycline and fluoroquinolone antibiotics.

Regardless of which antibiotic you use, it's very important to take them exactly as prescribed. Even if your symptoms start to improve and you don't feel sick, make sure you complete the full course of antibiotics. Otherwise, your infection may return and be resistant to the original antibiotic used.

Can you prevent it?

Scientists are currently working to develop a vaccine that protects against *M. catarrhalis* infections. This would be a major breakthrough in helping to prevent ear infections and pink eye in children. It will also be valuable for adults with COPD who are vulnerable to *M. catarrhalis* infections.

Until then, the best way to avoid *M. catarrhalis* infections is to keep your immune system healthy through following a balanced diet and getting regular exercise. If you have a compromised immune system or lung condition, make sure you regularly wash your hands and carry hand sanitizer. If you need to go to a hospital or doctor's office, consider wearing an N95 respirator mask while you're there.

The bottom line

Most people have *M. catarrhalis* in their respiratory tract at some point their lives, usually during childhood. While it was initially thought to be relatively harmless, more recent research has found that it can do more damage than previously thought, especially for people with weakened immune systems or lung conditions.

While *M. catarrhalis* infections are resistant to some common antibiotics, there are plenty of other antibiotics that do work. Just make sure to follow your doctor's instructions for taking them

Mosquito saliva alone triggers unexpected immune response

Mosquito season is around the corner, bringing with it a higher risk of catching potentially serious diseases transmitted by their bite. Mosquitoes also may increase the severity of the diseases they transmit, and researchers think that mosquito saliva plays an active role in this process. A team of researchers at Baylor College of Medicine has taken a closer look at the effect of mosquito saliva alone and found that it can trigger an unexpected variety of immune responses in an animal model of the human immune system. These results offer an opportunity to develop effective strategies to prevent mosquito-based transmission of disease. The study appears in the journal *PLOS Neglected Tropical Diseases*.

"Billions of people worldwide are exposed to diseases transmitted by mosquitoes, and many of these conditions do not have effective treatments," said corresponding author Dr. Rebecca Rico-Hesse, professor of molecular virology and microbiology at Baylor College of Medicine. "One of the interests of my lab is to study the development of dengue fever, which is caused by the dengue virus transmitted by mosquito *Aedes aegypti*."

The World Health Organization has estimated that 100 million dengue virus infections and 22,000 deaths occur yearly worldwide, mostly among children. According to the Centers for Disease Control and Prevention, more than one-third of the world's population lives in areas at risk of infection, making the dengue virus a leading cause of illness and death in the tropics and subtropics.

"One of the main limitations for studying dengue fever is that the dengue virus only causes the disease in humans; no other animals can be used as models of the condition to develop preventive and therapeutic measures," Rico-Hesse said. "To overcome this challenge, we have been working with a mouse model of the human immune system."

These 'humanized mice' were developed by other research groups from mice naturally born without their own immune system. These severely immunodeficient mice received human stem cells that gave rise to many of the components of the human immune system, creating a living humanized animal model in which Rico-Hesse and her colleagues can study factors that may affect the development of dengue fever.

"In 2012, we demonstrated in these humanized mice that mosquito-bite delivery and needle-injection delivery of dengue virus led to significantly different disease developments," Rico-Hesse said. "Importantly, mosquito-bite delivery of the virus resulted in a more human-like disease than the one we observed after needle-injection delivery of the virus. When the mosquitoes delivered the virus, the mice had more of a rash, more fever and other characteristics that mimic the disease presentation in humans."

These observations support the idea that mosquitoes are not just acting like 'syringes,' merely injecting viruses into the animals they feed on. Their saliva seems to contribute significantly to the development of the disease, which has prompted Rico-Hesse and her colleagues to investigate what this role might be. They began by determining the effect of bites from virus-free mosquitoes on the human immune response of humanized mice.

An unexpected complex response

To test the effect of virus-free mosquito saliva on humanized mice, the researchers held a vial containing mosquitoes against a footpad of anesthetized humanized mice, allowing a total of four mosquitoes to feed on both footpads.

The researchers then took blood and a number of other tissue samples six hours, 24 hours and seven days after the mosquitoes bit the mice, and determined the levels of cytokines, molecules that modulate the immune response, as well as the number and activity of different types of immune cells. They compared these results with those obtained from humanized mice that had not been bitten by mosquitoes.

To make the above determinations, the researchers used highly-sensitive techniques -- flow cytometry for immune cell analysis and multiplex cytokine bead array analysis for cytokines -- that allowed them to dissect the immune responses in great detail. This approach produced surprising results.

"We found that mosquito-delivered saliva induced a varied and complex immune response we were not anticipating," said co-author Dr. Silke Paust, assistant professor of pediatrics at Baylor and Texas Children's Hospital. "For instance, both the immune cell responses and the cytokine levels were affected. We saw activation of T helper cells 1, which generally contribute to antiviral immunity, as well as activation of T helper cells 2, which have been linked to allergic responses."

At various time points, the levels and activities of other types of immune cells also increased as others decreased. Overall, the researchers found evidence that mosquito saliva alone can trigger long-lasting immune responses -- up to seven days post-bite -- in multiple tissue types, including blood, skin and bone marrow.

"The diversity of the immune response was most striking to me. This is surprising given that no actual infection with any type of infectious agent occurred," said Paust, who also is a member of the Dan L Duncan Comprehensive Cancer Center at Baylor College of Medicine. "These results are evidence that components in the mosquito saliva can modulate the immune response in humanized mice."

The researchers will continue this study by investigating which of the more than 100 proteins in mosquito saliva are mediating the effects on the immune system, or may help the virus become more infectious. Identifying these proteins could help design strategies to fight transmission of dengue fever, as well as other diseases caused by viruses also transmitted by *Aedes aegypti*, such as Zika virus, chikungunya virus and yellow fever virus.

"We hope that our work will inspire more research in this area with the long-term goal of using our understanding of how saliva manipulates the immune system for therapeutic purposes," said Paust.

Best practices to avoid occupational asthma (part 1)

Prevention of occupational asthma—practical implications for occupational physicians

Chemical Routes of Entry

Occupational factors have been estimated to contribute to ~10% of adult-onset asthma and occupational asthma (OA) is one of the most common occupational lung diseases

Primary prevention has been effective for OA

Medical health surveillance has been effective in settings such as the detergent enzyme industry, workers exposed

Tertiary prevention is still required for workers with OA and can improve prognosis.

Conclusions OA is potentially preventable.

Medical health surveillance programs combined with occupational hygiene measures and worker education have been associated with improved outcomes but further studies are needed to understand the optimum frequency and measures for such programs and to identify the separate contribution of the components. Until primary and secondary prevention is better understood and implemented, there will also remain a need for tertiary preventive measures.

Occupational asthma (OA) is one of the most common chronic occupational lung diseases and workplace factors have been estimated to contribute to ~10% of all adult-onset asthma]. A subset (~10% or less of all OA) can be caused by an acute irritant exposure. This has been termed irritant-induced asthma and includes reactive airways dysfunction syndrome. The majority of OA (80–90%) is caused by specific sensitization to a workplace agent. For many specific sensitizers, especially high-molecular-weight agents, such as animal, plant, insect or fungal agents, and also some low-molecular-weight chemical agents, such as complex platinum salts or acid anhydrides, this is often associated with specific IgE antibodies to that agent. For other low-molecular-weight chemical sensitizers, sensitization and OA are less clearly IgE antibody mediated and may be produced by other specific, presumed immunologic mechanisms.

Once OA has developed in a worker, outcome is best with early diagnosis, early removal from further exposure to the causative agent and milder asthma at the time of removal from further exposure. Nevertheless, asthma may persist even after removal from exposure to the causative workplace agent and the socio-economic consequences of OA have been poor in reports from several different countries.

Given the burden of illness and potential for adverse outcomes, prevention of this relatively common occupational disease is therefore of importance.

Primary preventive measures

These measures can potentially include:

- (i) Identification of highly susceptible workers and locating them to areas without exposure to known sensitizers.
- (ii) Limitation of exposure to potential respiratory irritants among those with pre-existing asthma to reduce work-related aggravation of asthma.
- (iii) Use of engineering controls, such as elimination of a responsible agent, substitution with a safer substance/chemical, ventilation, process or equipment modification, process enclosure, dust reduction techniques, housekeeping and work practices.
- (iv) Administrative controls to reduce number of workers exposed or duration of exposure, e.g. job rotation, rest periods, shift or location changes where fewer people are working with sensitizers or irritant exposures.
- (v) Personal protective equipment (at the worker), which includes respirators, gloves, goggles and coveralls.

Irritant-induced OA

Irritant-induced asthma as currently understood results from acute exposure to an expected respiratory irritant. This is usually an accidental workplace occurrence, as in a spill or fire. Appropriate measures of occupational hygiene such as containment measures 'at the source' (isolation/enclosure), ventilation measures 'along the path to the worker' and appropriate respiratory protective devices 'at the worker' may prevent some cases of irritant-induced asthma. As an example, the lack of usage of appropriate respiratory protection among firefighters working at the site of the World Trade Center collapse has been suggested to be a significant factor contributing to the relatively high prevalence of irritant-induced asthma and airway hyper-responsiveness in these workers following inhalation of high concentrations of alkaline respirable dust.

Lowering exposure to concentrations of respiratory irritant agents, benefits workers with coincidental asthma by reducing

the likelihood of work-related aggravation of asthma. The induction of asthma by chronic moderate or low exposures to respiratory irritants is suggested by epidemiologic studies but to date is unproven. If confirmed, there would be an additional potential primary preventive role for limiting such exposures.

Sensitizer-induced OA

Host factors.

Since sensitization and OA from occupational agents occurs in a minority of exposed workers (5% or less in many studies), there clearly are host susceptibility factors. These include specific genotypes for which to date there is limited information, atopy and smoking history, as previously reviewed . The importance of underlying atopy appears to be greatest in those who become sensitized by an IgE antibody-mediated response, particularly to high-molecular-weight allergens such as animal proteins and plant products. However, the high prevalence of atopy in the general population (~20% or higher in some studies), compared with the relatively low risk of occupational sensitization, precludes this from being a useful determinant of employment, i.e. there is a low predictive value and it would exclude many who would not develop OA. Similarly, although smoking has been a significant risk factor for laboratory animal asthma, and the most significant associated host factor in sensitization to some occupational agents such as complex platinum salts and acid anhydrides, the high proportion of the working-age population who still smoke precludes this from usefulness in pre-employment screening to determine employment. Thus, these factors cannot be justifiably used to prevent individuals from working in jobs that may lead to OA. Nevertheless, physicians caring for older children with asthma and allergic diseases may offer useful advice to their patients regarding careers in which underlying allergy increases the risks for work-related sensitization, e.g. to natural rubber latex (NRL) or to animal proteins.

Exposure factors.

In order for immunologic sensitization to a specific workplace agent to occur, there clearly has to be exposure to that agent. In addition, it has been shown for some agents, as recently reviewed by Baur *et al.* and by Bush and Stave, that the higher the exposure levels to a sensitizer, the greater the proportion of exposed workers who will become sensitized (i.e. there are dose–response relationships). As an example, we reported that among diisocyanate-using companies, those companies with workers who had claims accepted for OA due to diisocyanates, were more likely to have measured concentrations of diisocyanates>0.005 ppm than companies who did not have workers with claims over a 4-year period.

An effective primary prevention measure would therefore be to avoid the use of known sensitizers in a workplace (i.e. elimination), or to reduce the exposure levels to a minimum (e.g. by isolation/control at source), aiming for levels which are not likely to induce sensitization except in those with the strongest genetic susceptibility. Unfortunately, this may not be possible in many workplaces.

An example where this strategy has been very effective is in the case of sensitization and occupational allergy including OA, from NRL. This was recognized to be common in health care workers and other workers with exposure to powdered NRL gloves in the early 1990s. Factors thought to have increased the risk for sensitization at that time include increased glove usage with universal precautions to prevent infection with blood-borne pathogens in health care workers, resulting in an increased production of NRL gloves with increased tapping of rubber trees which may have altered proteins in the rubber latex, reduced leaching out of proteins from gloves during manufacture, and possibly earlier usage of gloves after manufacture. The NRL proteins became airborne in association with glove-donning powder in particles of a size which could be inhaled and could lead to respiratory allergic manifestations in addition to the mucocutaneous contact allergic manifestations. These factors may have contributed to increased exposure to NRL proteins by those wearing NRL gloves, and recognition of NRL allergy and asthma increased markedly during this time.

Following understanding of the problem, recommendations were made to change to non-NRL gloves where possible and to reduce the powder and the NRL protein content of NRL gloves if these needed to be used. Such changes have been associated with significant reductions in airborne glove powder and protein concentrations and declines in the incidence of NRL allergy and asthma as reflected in hospital series, compensation data and national figures, reported from Ontario (Canada) and Germany.

Removal of a sensitizer from the workplace and substitution with a non-sensitizing and non-toxic agent is an ideal approach which may not often be practical. The experience with NRL has shown, however, that if complete removal is not feasible, then changes to reduce exposure to a minimum, such as that currently occurring in many areas with NRL glove use (by use of minimal powder and low-protein gloves), are likely to reduce, if not completely eliminate, sensitization.

A further example of primary prevention is the encapsulation of detergent enzymes (i.e. process modification; isolation) to reduce exposure. This was very successful when first introduced and as recently reviewed in a large company with associated medical surveillance measures. In contrast, introduction of new enzymes into a plant and failure of preventive measures led to further 'outbreaks' of sensitization and OA. The use of robots (automation) in addition to separated and ventilated areas, as well as appropriate respiratory protective devices for workers with unavoidable intermittent potential exposures, in plants manufacturing polyurethane foam has coincided with declining rates of sensitization to diisocyanates as suggested by compensation rates in Ontario. However, there is no direct evidence to determine whether these changes or other temporally associated interventions have been responsible.

Substitution of occupational sensitizers with newer chemicals, which may not cause sensitization, might be effective but there is currently no accurate method of determining the potential of new agents to cause human sensitization, despite the ability to obtain suggestive information from animal studies. The introduction of less volatile or more complex forms of some sensitizers such as diisocyanates requires further investigation to determine relative rates of human sensitization.

Although primary prevention by complete avoidance of respiratory sensitizers is an ideal intervention, it is clearly not feasible in many settings, such as bakeries and animal care facilities. However, even in these settings, reduction of exposure

sufficient to significantly reduce risks of sensitization can be feasible as described in laboratory care facilities and suggested for bakeries . The aim in these settings is to reduce the exposure to the lowest feasible level, but currently there is no known exposure concentration (other than zero) which will prevent sensitization in all susceptible workers. The introduction of a surveillance program for diisocyanates in Ontario in 1983 included monitoring of diisocyanate concentrations in the workplace with a maximum allowable 8-h time-weighted average (TWA) concentration of 5 ppb. The combination of this monitoring of workplace exposures in addition to a medical surveillance program (see Secondary Preventive Measures) was associated with a decline in new diisocyanate-related OA compensation claim. However, it could not be determined from the information available whether the decline was due to reduced exposure (primary prevention—from compliance with exposure monitoring or from increased use of robots and better worker education as to appropriate protective respirator use) or to detection by the surveillance program of early reversible asthma in workers who were then moved away from diisocyanate exposure.

Secondary preventive measures

Secondary preventive measures are aimed at detecting indicators of early sensitization or early changes of sensitizer-induced OA before there is permanent disease. This identification and early intervention with removal from further exposure can prevent permanent asthma. There is no equivalent process for irritant-induced asthma since disease starts with one or more very high irritant exposures.

Medical surveillance programs for OA typically include a symptom questionnaire, skin prick testing (if the sensitizer is a high-molecular-weight allergen for which skin testing can detect specific IgE antibodies) and spirometry. Although there is some support for the effectiveness of such programs in some settings, it is often difficult to determine which component of the program is effective and what is the optimum frequency of delivering such programs. In addition to serving as secondary prevention, such medical surveillance programs may lead to better control measures in the workplace, resulting in primary prevention for co-workers who are not yet sensitized and for future workers.

An example of such a program for which skin testing has been feasible and which appears to have been very successful is in the detergent enzyme industry. The medical surveillance program that has been recommended for people who work with enzymes includes periodic questionnaires, skin prick tests with a dilute solution of the enzyme and spirometry every 6 months for 2 years and then yearly. As with other high-molecular-weight occupational allergens, upper respiratory allergic symptoms often precede the onset of allergic asthma from the sensitizer. Workers who developed symptoms suggestive of an allergic upper or lower respiratory response at work and who had a positive skin prick test to the enzyme solution were moved away from further exposure in one company and rates of OA in this setting significantly declined in temporal association with this program .

Similarly, a medical surveillance program for workers exposed to complex platinum salts has been reported to be very effective. A positive skin prick test to complex platinum salts has been found to be highly predictive of the development of later OA if exposure is continued (100% developed work-related symptoms in some studies). Therefore, those found to have a positive skin prick test on surveillance have been removed early from further exposure. Limitations of these programs are that the outcomes are usually compared to historical experience, rather than a concurrent setting not undergoing medical surveillance, so one cannot separate the role of parallel hygiene and engineering control measures.

In the case of low-molecular-weight sensitizers such as diisocyanates, the immunologic mechanism is less clear. Only a minority of those with OA from sensitization to diisocyanates have been demonstrated to have serum IgE antibodies to diisocyanates with currently available methods of detection. In addition, the development of work-related nasal symptoms is not known to be a sensitive or specific marker of development of OA in this setting. Therefore, medical surveillance programs for diisocyanates to date have relied on symptom questionnaires and spirometry, with referral for more specific testing based on these results. The program which was mandated by the Ontario Ministry of Labour in 1983 in the province of Ontario, Canada, required exposure monitoring and also a medical questionnaire administered every 6 months and performance of spirometry if indicated by the questionnaire or at least every 24 months. Referral for further medical assessment was to be made if asthma-like symptoms were reported or if the spirometry showed that the FEV_1 or FVC had declined at least 15% from previous results. No medical surveillance programs are mandated in Ontario for other asthmagens.

Unfortunately, there was no prospective evaluation of this program when it was introduced. Retrospective evaluation has suggested benefit from some component of the program. Review of workers' compensation data has shown that in the period after introduction of the program, annual claims accepted for diisocyanate-induced asthma initially rose, consistent with increased case finding, then fell below baseline, suggesting a true reduced incidence, while claims due to other causes also rose and then remained stable. In addition, the compensation claimants with accepted diisocyanate-induced OA during the period before the program was likely to have been fully in effect had a longer duration of work-related symptoms before diagnosis was made, and had markers of more severe asthma, showing a temporal relationship between earlier diagnosis of asthma at a milder stage with introduction of the medical surveillance program. Also, as compared with OA due to other causes, OA due to diisocyanates had a shorter duration with symptoms before diagnosis, and these workers had milder asthma and were less likely to be hospitalized.

However, it remains possible that the earlier diagnosis may have resulted from other factors such as better knowledge of OA by family physicians and pulmonary physicians during the later time period or better education of workers with potential diisocyanate exposure so that they may have sought medical attention for work-related symptoms at an earlier stage. An analysis of companies known to be in compliance with the program showed an earlier diagnosis of OA (mean 1.7 years) compared with those not known to be in compliance (mean 2.7 years), and a trend to better outcome.

Although these studies have suggested a benefit from the

program, it was difficult to determine which component was responsible. A small analysis of the relative role of spirometry as part of this surveillance program in one polyurethane foam-making company showed a high proportion of false-positive responses among those who had apparent spirometric changes in the absence of asthma symptoms on questionnaire (and surveillance spirometry did not add benefit to the questionnaire). However, conversely, in a medical surveillance program of a bakery, screening questionnaires were found to have a significant number of false-negative reports and the addition of an objective test has been advised where possible. The difference in these two reports may in part reflect the lack of job security among those reporting symptoms in the bakery in contrast to the foam-making company where transfer to areas away from diisocyanate exposure was feasible.

Tertiary preventive measures

Tertiary prevention is aimed at limiting medical impairment among those with established OA.In general, the sensitized worker is advised to completely avoid further areas of exposure to the sensitizer. For those who cannot leave exposure completely, a few reports have indicated that use of an air supply helmet respirator for occasional work in areas of potential exposure may prevent asthma exacerbation

- (i) For those with irritant-induced asthma, it has been suggested that early treatment with oral corticosteroids may improve long-term prognosis . However, this is based only on a few case reports. For patients with persistent asthma induced by irritants, standard asthma management modalities, such as patient education, limitation of non-occupational irritant exposure and relevant allergen exposure as well as pharmacologic management as for non-occupational asthmatics, should be utilized. Depending on the severity of asthma, subsequent job modification and/or occupational hygiene measures may be needed to reduce exposure to potential respiratory irritants in order to avoid resulting aggravation of asthma symptoms.
- (ii) For workers with sensitizer-induced OA, the best prognosis generally requires complete avoidance of re-exposure to the sensitizing occupational agent and any immunologically cross-reacting agents, in addition to standard asthma management. Very low levels of NRL allergens may be tolerated by health care workers with NRL-induced OA , such as those achieved by avoidance of personal use of NRL products and use by co-workers when needed of only low-protein, powder-free NRL gloves. Residual powder should be removed from floors, furniture surfaces and ceiling plenums. However, the continuing presence of potential low exposures to airborne NRL allergen requires ongoing individual monitoring of the sensitized worker to ensure that there is no further work relationship of asthma.

At this time, specific allergen immunotherapy is not a standard treatment for occupational asthma. Early reports have suggested that it might be of some effect for allergy to NR, but more studies with larger groups are needed. Medical outcome is best when workers have an early, objective diagnosis of OA, soon after the onset of work-related symptoms; have mild asthma at the time of diagnosis and are removed early from further exposure. Overall, a significant proportion of patients have ongoing asthma and suffer significant socio-economic consequences from the diagnosis, emphasizing on the preference for primary preventive measures.

References:

https://academic.oup.com/occmed/article/55/8/588/1457179

https://www.levitt-safety.com/blog/chemical-routes-of-entry/?utm_expid=88473856-0.4Y33gbhdTbuBJL3q7w0UXQ.0&utm_referrer=https%3A%2F%2Fwww.levitt-safety.com%2Fblog%2Fchemical-routes-of-entry%2F

https://proshieldsafetysigns.co.uk/product/dont-turn-your-back-on-safety-poster-58184/

Highlights of the coming issue


JOURNAL OF HYGIENE SCIENCES
Committed to the advancement of Clinical & Industrial Disinfection & Microbiology
Contents

Mini Review
Biological Stains and its Applications (Issue-3).

Current Trends
Detergent & water miscibility.

In Profile
Salvador Luria.

Bug of the Month
Propionibacterium freudenreichii.

Did You Know
Formaldehyde and Cancer Risk

Best Practices
Best practices to have healthy lungs for inhalation.

Printed and published by D.G. Tripathi, Edited by Akshaya Tendulkar for and on behalf of Tulip Diagnostics (P) Ltd., Tulip House, Dr. Antonio Do Rego Bagh, Alto Santacruz, Bambolim Complex, Post Office Goa - 403 202, India. Fax: 0832 2458544, **Website: www.tulipgroup.com.**