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Editorial

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Mini review section – Dengue virus (DENV) belongs to the family Flaviviridae, genus Flavivirus, and is transmitted to humans by Aedes mosquitoes, mainly *Aedes aegypti*. The dengue viruses are positive stranded RNA viruses. There are four distinct dengue virus (DENV) serotypes that share antigenic relationships (DENV-1, DENV-2, DENV-3, and DENV-4), and although infection with one serotype confers lifelong protection against that serotype, it does not necessarily protect against a secondary infection with a heterologous serotype.

Current Trends section – disinfection of environmental surfaces could be best characterized as an interim state. For example, a surface becomes contaminated, a disinfectant is then applied for the prescribed contact/wet/kill time – effectively reducing the number of contaminating organisms by several log reductions, and over time, that same surface in the healthcare setting becomes re-contaminated – warranting another application of disinfectant at regular intervals. These regular intervals make a difference. Arguably the most significant difference in contaminating healthcare worker hands are the vectors between the infected/colonized patient and the susceptible patient.

In Profile Scientist – In 1878 Koch summarized his experiments on the etiology of wound infection. By inoculating animals with material from various sources, he produced six types of infection, each caused by a specific microorganism. He then transferred these infections by inoculation through several kinds of animals, reproducing the original six types. In that study, he observed differences in pathogenicity for different species of hosts and demonstrated that the animal body is an excellent apparatus for the cultivation of bacteria.

Bug of the month – *Y. enterocolitica* is widespread in nature, occurring in reservoirs ranging from the intestinal tracts of numerous mammals, avian species, cold-blooded species, and even from terrestrial and aquatic niches. Most environmental isolates are avirulent; however, isolates recovered from porcine sources contain human pathogenic serogroups. In addition, dogs, sheep, wild rodents, and environmental water may also be a reservoir of pathogenic *Y. enterocolitica* strains. Human pathogenic strains are usually confined to the intestinal tract and lead to enteritis/diarrhea.

Did You Know? – With the help of artificial intelligence, researchers at Chalmers University of Technology, Sweden, have succeeded in designing synthetic DNA that controls the cells' protein production. The technology can contribute to the development and production of vaccines, drugs for severe diseases, as well as alternative food proteins much faster and at significantly lower costs than today.

Best Practices – As you touch people, surfaces, and objects throughout the day, you accumulate germs on your hands. You can infect yourself with these germs by touching your eyes, nose, or mouth, or spread them to others. Although it's impossible to keep your hands germfree, washing your hands with soap and water frequently can help limit the transfer of bacteria, viruses, and other microbes.

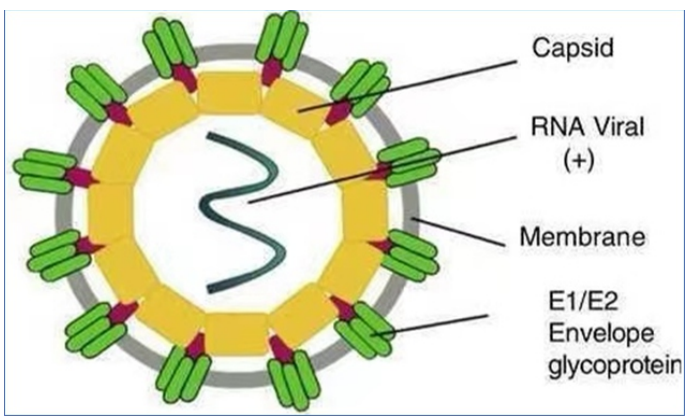
Tickle yourself enjoying the jokes in our **Relax Mood section**.

Our JHS team is thankful to all our readers for their ever-increasing appreciation that has served as a reward & motivation for us. Looking forward for your continuous support.

Understanding Dengue (I)

Dengue fever is a mosquito-borne virus disease of humans. In terms of numbers of individuals infected, it is by far the most devastating of all the recognised arthropod-transmitted virus diseases, also known as breakbone fever due to the severity of muscle spasms and joint pain, dandy fever, or seven-day fever because of the usual duration of symptoms. Although most cases are asymptomatic, severe illness and death may occur. *Aedes* mosquitoes transmit the virus and are common in tropical and subtropical parts of the world. The incidence of dengue has increased dramatically over the past few decades. The infection is now endemic in some parts of the world. It is estimated that more than 3 billion humans live in dengue endemic regions of the world, and currently, more than 50 million infections occur annually with at least 500,000 individuals requiring hospitalisation. Of these, tens of thousands have a high risk of developing haemorrhagic disease, potentially with fatal consequences depending to a large extent on the quality of the available medical services.

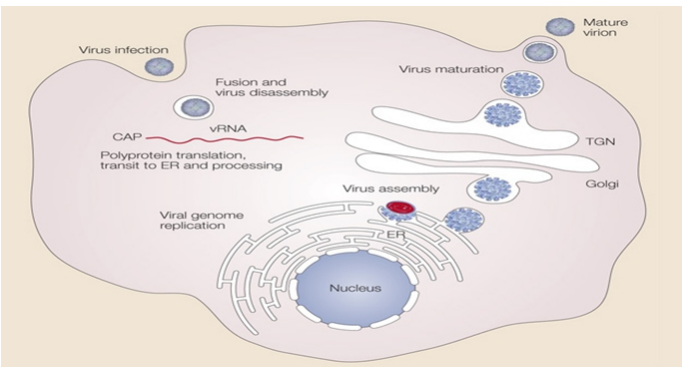
Dengue Virus



Dengue virus (DENV) belongs to the family *Flaviviridae*, genus *Flavivirus*, and is transmitted to humans by *Aedes* mosquitoes, mainly *Aedes aegypti* The dengue viruses are positive stranded RNA viruses .There are four distinct dengue virus (DENV) serotypes that share antigenic relationships (DENV-1, DENV-2, DENV-3, and DENV-4), and although infection with one serotype confers lifelong protection against that serotype, it does not necessarily protect against a secondary infection with a heterologous serotype. Serotype is a group of viruses classified together based on their antigens on the surface of the virus. These four subtypes are different strains of dengue virus that have 60-80% homology between each other. The major difference for humans lies in subtle differences in the surface proteins of the different dengue subtypes. Infection induces long-life protection against the infecting serotype, but it gives only a short time cross protective immunity against the other types. The first infection cause mostly minor disease, but secondary infections has been reported to cause severe diseases (DHF or DSS) in both children and adults. This phenomenon is called Antibody-Dependent Enhancement.

The structure of the dengue virus is roughly spherical, with a diameter of approximately 50 nm (1 nm is one millionth of 1 mm). The core of the virus is the nucleocapsid, a structure that is made of the viral genome along with C proteins. The nucleocapsid is surrounded by a membrane called the viral envelope, a lipid bilayer that is taken from the host. Embedded in the viral envelope are 180 copies of the E and M proteins that span through the lipid bilayer. These proteins form a protective outer layer that controls the entry of the virus into human cells.

The dengue viral replication process begins when the virus attaches to a human skin cell After this attachment, the skin cell's membrane folds around the virus and forms a pouch that seals around the virus particle. This pouch is called an endosome. A cell normally uses endosomes to take in large molecules and particles from outside the cell for nourishment. By hijacking this normal cell process, the dengue virus is able to enter a host cell.



Once the virus has entered a host cell, the virus penetrates deeper into the cell while still inside the endosome. Two conditions are needed for the dengue virus to exit the endosome:

- The endosome must be deep inside the cell where the environment is acidic.
- The endosomal membrane must gain a negative charge.

These two conditions allow the virus envelope to fuse with the endosomal membrane, and that process releases the dengue nucleocapsid into the cytoplasm of the cell.

Once it is released into the cell cytoplasm the nucleocapsid opens to uncoat the viral genome. This process releases the viral RNA into the cytoplasm. The viral RNA then hijacks the host cell's machinery to replicate itself. The virus uses ribosomes on the host's rough endoplasmic reticulum (ER) to translate the viral RNA and produce the viral polypeptide. This polypeptide is then cut to form the ten dengue proteins.

The newly synthesized viral RNA is enclosed in the C proteins, forming a nucleocapid. The nucleocapsid enters the rough ER and is enveloped in the ER membrane and surrounded by the M and E proteins. This step adds the viral envelope and protective outer layer. The immature viruses travel through the Golgi apparatus complex, where the viruses mature and convert into their infectious form. The mature dengue viruses are then released from the cell and can go on to infect other cells.

Dengue vector

A vector is a vehicle that carries and transmits a disease to its host organism. Vectors include animals and microorganisms that transmit different diseases. The most common vectors are

arthropods, which are invertebrate animals with an external skeleton called an exoskeleton. Arthropods include mosquitoes, ticks, lice, flies, and fleas. For instance, ticks can carry Lyme disease, and some mosquitoes can carry yellow fever, malaria, and dengue fever. The dengue virus is transmitted to humans via the bite of an infected mosquito. Only a few mosquito species are vectors for the dengue virus.

Aedes aegypti is a small, dark mosquito that can be identified by the white bands on its legs and a silver-white pattern of scales on its body. *Aedes aegypti* dwell in tropical and subtropical regions all over the world, mainly between the latitudes of 35°N and 35°S where the winter temperature is no colder than 10°C. Although some mosquitoes may travel farther north or south of these latitudes, they are unable to survive cold winters. Because *Aedes aegypti* require a warm climate, they typically do not live at altitudes above 1000 m, where the temperature is colder. These mosquitoes are associated with the living spaces of humans. They generally spend their entire lives in and around the houses where their eggs hatched.

Identifying Aedes mosquito

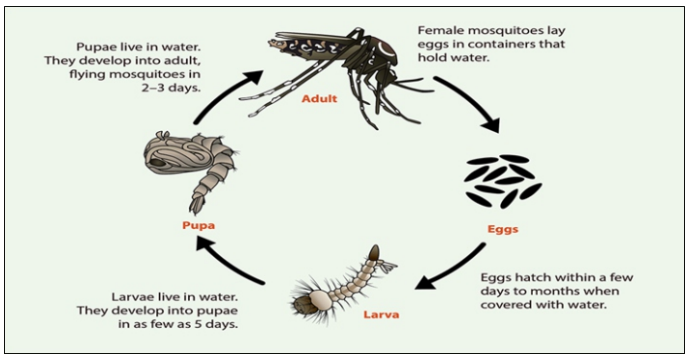
The Aedes Aegypti mosquito, or dengue mosquito, is dark coloured and has typical white markings on the legs and lyre like markings on the thorax. It is significantly smaller in size, being only 4 to 7 millimetres long. In this species, the female mosquitoes are longer than males. The Aedes Aegypti mosquito cannot stay alive during the winter season and thus only lays eggs in the summers or monsoons. They usually lay their eggs near households due to the proximity to potted plants and still water.

The dengue mosquito mostly bites its victims during the day. The mosquito is most active during the daytime, approximately two hours after sunrise and several hours before sunset. Only the females of this species feed on blood.

These mosquitoes usually rest in cool and shaded areas, like closets and under the beds. Though they usually bite during the day, there are chances they may bite humans during the night as well. They usually target areas such as ankles and elbows.

Life Cycle of Aedes aegypti

Aedes aegypti is a so-called holometabolous insect. This means that the insects goes through a complete metamorphosis with an egg, larvae, pupae, and adult stage. The adult life span can range from two weeks to a month depending on environmental conditions. The life cycle of Aedes aegypti can be completed within one-and-a-half to three week



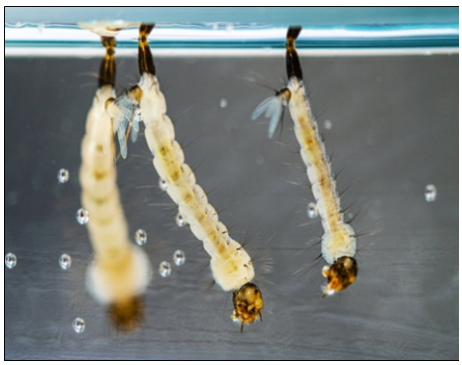
Egg

After taking a blood meal, female Aedes aegypti mosquitos produce on average 100 to 200 eggs per batch. The females can produce up to five batches of eggs during a lifetime. The number of eggs is dependent on the size of the bloodmeal. Eggs are laid on damp surfaces in areas likely to temporarily flood, such as tree holes and man-made containers like barrels, drums, jars, pots, buckets, flower vases, plant saucers, tanks, discarded bottles, tins, tyres, water cooler, etc. and a lot more places where rain-water collects or is stored. The female Aedes aegypti lays her eggs separately unlike most species. Not all eggs are laid at once, but they can be spread out over hours or days, depending on the availability of suitable substrates. Eggs will most often be placed at varying distances above the water line. The female mosquito will not lay the entire clutch at a single site, but rather spread out the eggs over several sites.

The eggs of Aedes aegypti are smooth, long, ovoid shaped, and roughly one millimeter long. When first laid, eggs appear white but within minutes turn a shiny black. In warm climates eggs may develop in as little as two days, whereas in cooler temperate climates, development can take up to a week. Laid eggs can survive for very long periods in a dry state, often for more than a year. However, they hatch immediately once submerged in water. This makes the control of the dengue virus mosquito very difficult.

Larvae

After hatching of the eggs, the larvae feed on organic particulate matter in the water, such as algae and other microscopic organisms. Most of the larval stage is spent at the water's surface, although they will swim to the bottom of the container if disturbed or when feeding. Larvae are often found around the home in puddles, tires, or within any object holding water. Larval



development is temperature dependent. The larvae pass through four instars, spending a short amount of time in the first three, and up to three days in the fourth instar. Fourth instar larvae are approximately eight millimeters long. Males develop faster than females, so males generally pupate earlier. If temperatures are cool, Aedes aegypti can remain in the larval stage for months so long as the water supply is sufficient.

Pupae

After the fourth instar, the larvae enters the pupal stage. Mosquito pupae are mobile and respond to stimuli. Pupae do not feed and take approximately two days to develop. Adults emerge by ingesting air to expand the abdomen thus splitting open the pupal case and emerge head first.

Adult

The adult mosquito is able to fly and is no longer aquatic. It has a terrestrial habitat.

After adult mosquitoes emerge: male mosquitoes feed on nectar from flowers and female mosquitoes feed on humans and animals for blood to produce eggs. After feeding, female mosquitoes will look for water sources to lay more eggs.

Impact of continuous active disinfectants (CAD)

Environmental surfaces are frequently contaminated with microbes and contribute to the spread of infectious agents. Despite effective surface disinfectants, maintaining hygienic surfaces is difficult as commonly touched surfaces are easily recontaminated. One of the limitations of current cleaning and disinfection strategies is that cleaned surfaces rapidly become recontaminated.

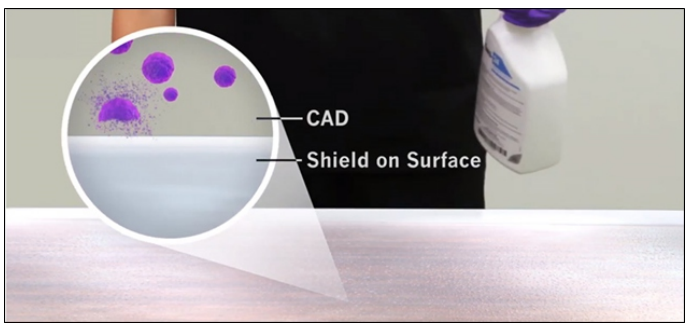
For about a decade, prior to the pandemic, there was a shift towards the control of healthcare-associated infections (HAIs) through a focus on environmental cleaning, especially in relation to reusable medical equipment. The novel idea of continuous room disinfection was an ongoing topic of discussion among environmental experts Even with optimal cleaning and disinfecting practices, recontamination of the environment and equipment occurs quickly, especially in healthcare settings.

Until recently, disinfection of environmental surfaces could be best characterized as an interim state. For example, a surface becomes contaminated, a disinfectant is then applied for the prescribed contact/wet/kill time – effectively reducing the number of contaminating organisms by several log reductions, and over time, that same surface in the healthcare setting becomes re-contaminated – warranting another application of disinfectant at regular intervals.

These regular intervals make a difference. Arguably the most significant difference in contaminating healthcare worker hands are the vectors between the infected/colonized patient and the susceptible patient.

Continuous Active Disinfection

Continuous Active Disinfection is the application of a surface chemical that has intermediate level disinfection on contact and then continuously disinfects potential pathogens that land on the surface thereafter. Think of it as a shield that can withstand organisms for some prescribed time. Inherently it leaves behind a residue or film – letting you know it is there.



The EPA has approved these products based on scientific data submitted. They are based on the premise that you can spray or wipe once and expect residual activity thereafter for a prescribed period of time or a defined number of touches Essentially, you disinfect a surface with a CAD, walk away, and it continues killing specific organisms for the prescribed time or touches to the surface – like a temporary shield.

It may seem tempting to use this CAD in immediate care areas–patient rooms, bedside tables, and bedrails–where it has been shown to be effective but it makes sense to use it in areas or processes where disinfection is inherently impaired.

Common areas, or more commonly, waiting areas in ambulatory care, are often underserved. Waiting areas, ambulatory care lounges, the places that are often disinfected no more than once a day.

These areas are still disinfected once per day ,but you use a chemical that continues disinfecting throughout the following day. Patient 1 sits down, sets their coffee on the nearby table, gets up, goes to their appointment and Patient 2 sits down – but in the interim, the organisms from Patient 1 have been killed. That is the potential of a CAD.

This persistent decontamination technology may reduce or eliminate the problem of recontamination and minimize the role of contaminated environmental surfaces and equipment in transmitting healthcare pathogens. CAD is an adjunct to traditional modalities-not a replacement.

Robert Koch

Robert Koch, in full **Robert Heinrich Hermann Koch**, (born Dec. 11, 1843, Clausthal, Hannover [now Clausthal-Zellerfeld, Ger.]—died May 27, 1910, Baden-Baden, Ger.), German physician and one of the founders of bacteriology. He discovered the anthrax disease cycle (1876) and the bacteria responsible for tuberculosis (1882) and cholera (1883). For his discoveries in regard to tuberculosis, he received the Nobel Prize for Physiology or Medicine in 1905.

Koch attended the University of Göttingen, where he studied medicine, graduating in 1866. He then became a physician in various provincial towns. After serving briefly as a field surgeon during the Franco-Prussian War of 1870–71, he became district surgeon in Wollstein, where he built a small laboratory. Equipped with a microscope, a microtome (an instrument for cutting thin slices of tissue), and a homemade incubator, he began his study of algae, switching later to pathogenic (disease-causing) organisms. One of Koch's teachers at Göttingen had been the anatomist and histologist Friedrich Gustav Jacob Henle, who in 1840 had published the theory that infectious diseases are caused by living microscopic organisms. In 1850 the French parasitologist Casimir Joseph Davaine was among the first to observe organisms in the blood of diseased animals. In 1863 he reported the transmission of anthrax by the inoculation of healthy sheep with the blood of animals dying of the disease and the finding of microscopic rod-shaped bodies in the blood of both groups of sheep. Inspired by the work of the French microbiologist Louis Pasteur, Davaine showed that it was highly probable that, because the sheep did not become diseased in the absence of these rodlike bodies, anthrax was due to the presence of such organisms in the blood. The natural history of the disease was, nevertheless, far from complete.

It was at that point that Koch began. He cultivated the anthrax organisms in suitable media on microscope slides, demonstrated their growth into long filaments, and discovered the formation within them of oval, translucent bodies—dormant spores. Koch found that the dried spores could remain viable for years, even under exposed conditions. The finding explained the recurrence of the disease in pastures long unused for grazing, for the dormant spores could, under the right conditions, develop into the rod-shaped bacteria (bacilli) that cause anthrax. The anthrax life cycle, which Koch had discovered, was announced and illustrated at Breslau in 1876, on the invitation of Ferdinand Cohn, an eminent botanist. Julius Cohnheim, a famous pathologist, was deeply impressed by Koch's presentation. “It leaves nothing more to be proved,” he said.

Cohn, whose discovery of spores had been published in 1875, was also very much impressed and generously helped to prepare the engraving for Koch's epochal paper, which he also published. One of Cohn's pupils, Joseph Schroeter, found that chromogenic (colour-forming) bacteria would grow on such solid substrates as potato, coagulated egg white, meat, and bread and that those colonies were capable of forming new colonies of the same colour, consisting of organisms of the same type. That was the starting point of Koch's pure-culture techniques, which he worked out a few years later. That a disease organism might be cultured outside the body was a concept introduced by Louis Pasteur, but the pure-culture techniques for doing so were perfected by Koch, whose precise and ingenious experiments demonstrated the complete life cycle of an important organism. The anthrax work afforded for the first time convincing proof of the definite causal relation of a particular microorganism to a particular disease.

In 1877 Koch published an important paper on the investigation, preservation, and photographing of bacteria. His work was illustrated by superb photomicrographs. In his paper he described his method of preparing thin layers of bacteria on glass slides and fixing them by gentle heat. Koch also invented the apparatus and the procedure for the very useful hanging-drop technique, whereby microorganisms could be cultured in a drop of nutrient solution on the underside of a glass slide.

In 1878 Koch summarized his experiments on the etiology of wound infection. By inoculating animals with material from various sources, he produced six types of infection, each caused by a specific microorganism. He then transferred these infections by inoculation through several kinds of animals, reproducing the original six types. In that study, he observed differences in pathogenicity for different species of hosts and demonstrated that the animal body is an excellent apparatus for the cultivation of bacteria.

Koch, now recognized as a scientific investigator of the first rank, obtained a position in Berlin in the Imperial Health Office, where he set up a laboratory in bacteriology. With his collaborators, he devised new research methods to isolate pathogenic bacteria. Koch determined guidelines to prove that a disease is caused by a specific organism. These four basic criteria, called Koch's postulates, are:

1. A specific microorganism is always associated with a given disease.
2. The microorganism can be isolated from the diseased animal and grown in pure culture in the laboratory.

3. The cultured microbe will cause disease when transferred to a healthy animal.
4. The same type of microorganism can be isolated from the newly infected animal.
5. Koch concentrated his efforts on the study of tuberculosis, with the aim of isolating its cause. Although it was suspected that tuberculosis was caused by an infectious agent, the organism had not yet been isolated and identified. By modifying the method of staining, Koch discovered the tubercle bacillus and established its presence in the tissues of animals and humans suffering from the disease. A fresh difficulty arose when for some time it proved impossible to grow the organism in pure culture. But eventually Koch succeeded in isolating the organism in a succession of media and induced tuberculosis in animals by inoculating them with it. Its etiologic role was thereby established. On March 24, 1882, Koch announced before the Physiological Society of Berlin that he had isolated and grown the tubercle bacillus, which he believed to be the cause of all forms of tuberculosis.
6. Meanwhile, Koch's work was interrupted by an outbreak of cholera in Egypt and the danger of its transmission to Europe. As a member of a German government commission, Koch went to Egypt to investigate the disease. Although he soon had reason to suspect a particular comma-shaped bacterium (vibrio) as the cause of cholera, the epidemic ended before he was able to confirm his hypothesis. Nevertheless, he raised awareness of amebic dysentery and differentiated two varieties of Egyptian conjunctivitis. Proceeding to India, where cholera is endemic, he completed his task, identifying both the organism responsible for the disease and its transmission via drinking water, food, and clothing.
7. Resuming his studies of tuberculosis, Koch investigated the effect an injection of dead bacilli had on a person who subsequently received a dose of living bacteria and concluded that he may have discovered a cure for the disease. In his studies he used as the active agent a sterile liquid produced from cultures of the bacillus. However, the liquid, which he named tuberculin (1890), proved disappointing, and sometimes dangerous, as a curative agent. Consequently, its importance as a means of detecting a present or past tubercular state was not immediately recognized (see tuberculin test). Additional work on tuberculosis came later, but, after the seeming debacle of tuberculin, Koch was also occupied with a great variety of investigations into diseases of humans and animals—studies of leprosy, bubonic plague, livestock diseases, and malaria.

In 1901 Koch reported work done on the pathogenicity of the human tubercle bacillus in domestic animals. He believed that infection of human beings by bovine tuberculosis is so rare that it is not necessary to take any measures against it. That conclusion was rejected by commissions of inquiry in Europe and America but extensive and important work was stimulated by Koch. As a result, successful measures of prophylaxis were devised.

Not an eloquent speaker, Koch was nevertheless by example, demonstration, and precept one of the most effective of teachers, and his numerous pupils—from the entire Western world and Asia—were the creators of the new era of bacteriology. His work on trypanosomes was of direct use to the eminent German bacteriologist Paul Ehrlich; that is only one example of Koch's instigation of epochal work both within and beyond his own immediate sphere. His discoveries and his technical innovations were matched by his fundamental concepts of the etiology of disease. Long before his death, his place in the history of science was universally recognized.

Jokes

Saw It With My Eyes But Couldn't Understand It

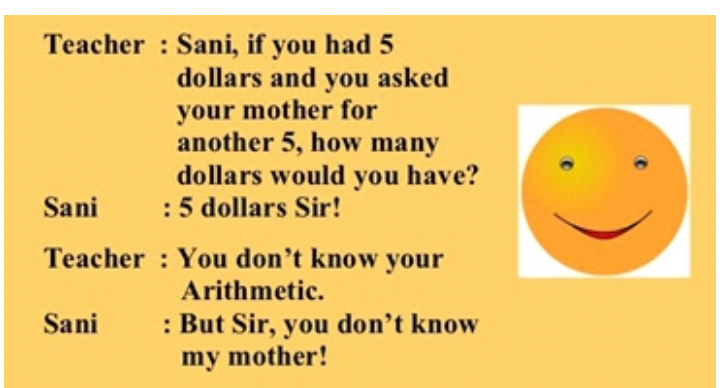
Took It In My Hands, But Couldn't Understand It

Keep Thinking For A Long Time, But Again Couldn't Understand It

It was Not A Dream,

It was Is Not A Love,

It was Not Even Friendship.



Question by a student !!

If a single teacher can't teach us all the subjects,

Then…

How could you expect a single student to learn all subjects?

****

New Teacher: anybody who thinks he is stupid, stand up

pappu stoodup

Teacher: R U stupid?

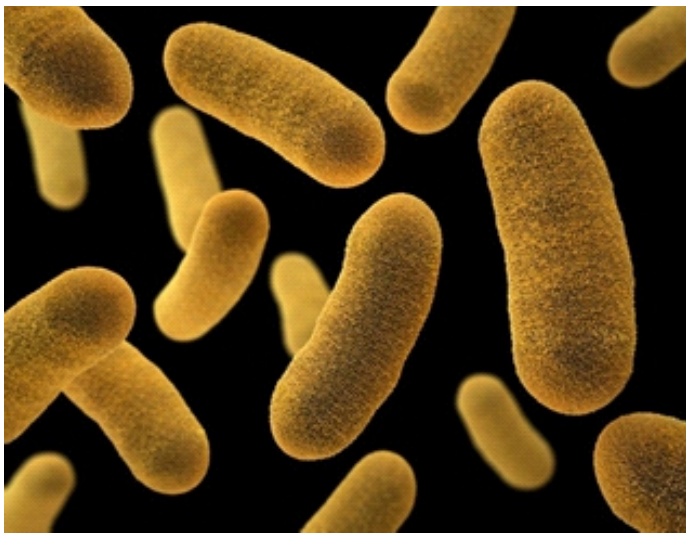
Pappu: "nhi, Aap akeli khari theen mujhe acha nhi lag raha tha"

****

" Women wont play football not coz they aren't gud at it..

But coz its against their ego to b dressed up exactly like 10 other women in front of 10,000 people.

Yersinia enterocolitica



Yersinia enterocolitica is a Gram-negative, bacillus-shaped bacterium, belonging to the family Yersiniaceae. It is motile at temperatures of 22–29°C (72–84°F), but becomes nonmotile at normal human body temperature. *Y. enterocolitica* infection causes the disease yersiniosis, which is an animal-borne disease occurring in humans, as well as in a wide array of animals such as cattle, deer, pigs, and birds. Many of these animals recover from the disease and become carriers; these are potential sources of contagion despite showing no signs of disease. The bacterium infects the host by sticking to its cells using trimeric autotransporter adhesins.

The genus *Yersinia* includes 20 species: *Y. aldovae*, *Y. aleksiciae*, *Y. bercovieri*, *Y. canariae*, *Y. enterocolitica*, *Y. entomophaga*, *Y. frederiksenii*, *Y. hibernica*, *Y. intermedia*, *Y. kristensenii*, *Y. massiliensis*, *Y. mollaretii*, *Y. nurmii*, *Y. pekkanenii*, *Y. pestis*, *Y. pseudotuberculosis*, *Y. rohdei*, *Y. ruckeri*, *Y. similis*, and *Y. wautersii*. Among them, only *Y. pestis*, *Y. pseudotuberculosis*, and certain strains of *Y. enterocolitica* are of pathogenic importance for humans and certain warm-blooded animals, whereas the other species are of environmental origin and may, at best, act as opportunists. However, *Yersinia* strains can be isolated from clinical materials, so they must be identified at the species level.

Y. enterocolitica is a heterogeneous group of strains, which are traditionally classified by biotyping into six biogroups based on phenotypic characteristics, and by serotyping into more than 57 O serogroups, on the basis of their O (lipopolysaccharide or LPS) surface antigen. Five of the six biogroups (1B and 2–5) are regarded as pathogens. However, only a few of these serogroups have been associated with disease in either humans or animals. Strains that belong to serogroups O:3 (biogroup 4), O:5,27 (biogroups 2 and 3), O:8 (biogroup 1B), and O:9 (biogroup 2) are most frequently isolated worldwide from human samples. However, the most important *Y. enterocolitica* serogroup in many European countries is serogroup O:3 followed by O:9, whereas the serogroup O:8 is mainly detected in the United States.

Y. enterocolitica is widespread in nature, occurring in reservoirs ranging from the intestinal tracts of numerous mammals, avian species, cold-blooded species, and even from terrestrial and aquatic niches. Most environmental isolates are avirulent; however, isolates recovered from porcine sources contain human pathogenic serogroups. In addition, dogs, sheep, wild rodents, and environmental water may also be a reservoir of pathogenic *Y. enterocolitica* strains. Human pathogenic strains are usually confined to the intestinal tract and lead to enteritis/diarrhea.

The portal of entry is the gastrointestinal tract. The organism is acquired usually by insufficiently cooked pork or contaminated water, meat, or milk. In recent years *Y. enterocolitica* has increasingly been causing smaller outbreaks via Ready-To-Eat (RTE) vegetables. Acute *Y. enterocolitica* infections usually lead to mild self-limiting enterocolitis or terminal ileitis and adenitis in humans. Symptoms may include watery or bloody diarrhea and fever, resembling appendicitis or salmonellosis or shigellosis. After oral uptake, *Yersinia* species replicate in the terminal ileum and invade Peyer's patches. From here they can disseminate further to mesenteric lymph nodes causing lymphadenopathy. This condition can be confused with appendicitis, so is called pseudoappendicitis. In immunosuppressed individuals, they can disseminate from the gut to the liver and spleen and form abscesses. Because *Yersinia* species are siderophilic (iron-loving) bacteria, people with hereditary hemochromatosis (a disease resulting in high body iron levels) are more susceptible to infection with *Yersinia* (and other siderophilic bacteria). In fact, the most common contaminant of stored blood is *Y. enterocolitica*.

Yersiniosis is usually self-limiting and does not require treatment. For sepsis or severe focal infections, especially if associated with immunosuppression, the recommended regimen includes doxycycline in combination with an aminoglycoside. Other antibiotics active against *Y. enterocolitica* include trimethoprim-sulfamethoxasole, fluoroquinolones, ceftriaxone, and chloramphenicol. *Y. enterocolitica* is usually resistant to penicillin G, ampicillin, and cefalotin due to beta-lactamase production, but multi-drug resistant strains have been reported in Europe.

Y. enterocolitica infections are sometimes followed by chronic inflammatory diseases such as arthritis, erythema nodosum, and reactive arthritis. This is most likely because of some immune-mediated mechanism.

Y. enterocolitica seems to be associated with autoimmune Graves-Basedow thyroiditis. Whilst indirect evidence exists, direct causative evidence is limited. *Y. enterocolitica* is probably not a major cause of this disease, but may contribute to the development of thyroid autoimmunity arising for other reasons in genetically susceptible individuals. *Y. enterocolitica* infection has also been suggested to be not the cause of autoimmune thyroid disease, but rather an associated condition, with both sharing a common inherited susceptibility. More recently, the role for *Y. enterocolitica* has been disputed.

AI tailors artificial DNA for future drug development

With the help of artificial intelligence, researchers at Chalmers University of Technology, Sweden, have succeeded in designing synthetic DNA that controls the cells' protein production. The technology can contribute to the development and production of vaccines, drugs for severe diseases, as well as alternative food proteins much faster and at significantly lower costs than today.

How our genes are expressed is a process that is fundamental to the functionality of cells in all living organisms. Simply put, the genetic code in DNA is transcribed to the molecule messenger RNA (mRNA), which tells the cell's factory which protein to produce and in which quantities.

Researchers have put a lot of effort into trying to control gene expression because it can, among other things, contribute to the development of protein-based drugs. A recent example is the mRNA vaccine against Covid-19, which instructed the body's cells to produce the same protein found on the surface of the coronavirus. The body's immune system could then learn to form antibodies against the virus. Likewise, it is possible to teach the body's immune system to defeat cancer cells or other complex diseases if one understands the genetic code behind the production of specific proteins.

Most of today's new drugs are protein-based, but the techniques for producing them are both expensive and slow, because it is difficult to control how the DNA is expressed. Last year, a research group at Chalmers, led by Aleksej Zelezniak, Associate Professor of Systems Biology, took an important step in understanding and controlling how much of a protein is made from a certain DNA sequence.

"First it was about being able to fully 'read' the DNA molecule's instructions. Now we have succeeded in designing our own DNA that contains the exact instructions to control the quantity of a specific protein", says Aleksej Zelezniak about the research group's latest important breakthrough.

DNA molecules made-to-order

The principle behind the new method is similar to when an AI generates faces that look like real people. By learning what a large selection of faces looks like, the AI can then create completely new but natural-looking faces. It is then easy to modify a face by, for example, saying that it should look older, or have a different hairstyle. On the other hand, programming a believable face from scratch, without the use of AI, would have been much more difficult and time-consuming. Similarly, the researchers' AI has been taught the structure and regulatory code of DNA. The AI then designs synthetic DNA, where it is easy to modify its regulatory information in the desired direction of gene expression. Simply put, the AI is told how much of a gene is desired and then 'prints' the appropriate DNA sequence.

“DNA is an incredibly long and complex molecule. It is thus experimentally extremely challenging to make changes to it by iteratively reading and changing it, then reading and changing it again. This way it takes years of research to find something that works. Instead, it is much more effective to let an AI learn the principles of navigating DNA. What otherwise takes years is now shortened to weeks or days”, says first author Jan Zrimec, a research associate at the National Institute of Biology in Slovenia and past postdoc in Aleksej Zelezniak's group.

The researchers have developed their method in the yeast Sacc*haromyces cerevisiae*, whose cells resemble mammalian cells. The next step is to use human cells. The researchers have hopes that their progress will have an impact on the development of new as well as existing drugs.

"Protein-based drugs for complex diseases or alternative sustainable food proteins can take many years and can be extremely expensive to develop. Some are so expensive that it is impossible to obtain a return on investment, making them economically nonviable. With our technology, it is possible to develop and manufacture proteins much more efficiently so that they can be marketed", says Aleksej Zelezniak.

Hand Hygiene

As you touch people, surfaces and objects throughout the day, you accumulate germs on your hands. You can infect yourself with these germs by touching your eyes, nose or mouth, or spread them to others. Although it's impossible to keep your hands germ-free, washing your hands with soap and water frequently can help limit the transfer of bacteria, viruses and other microbes.

Washing hands can keep you healthy and prevent the spread of respiratory and diarrheal infections. Germs can spread from person to person or from surfaces to people when you:

- Touch your eyes, nose, and mouth with unwashed hands
- Prepare or eat food and drinks with unwashed hands
- Touch surfaces or objects that have germs on them
- Blow your nose, cough, or sneeze into hands and then touch other people's hands or common objects

Times to Wash Hands

Always wash your hands before and after:

- Preparing and eating food
- Treating wounds or caring for a sick person
- Touching an item or surface that is frequently touched by other people, such as door handles, gas pumps or shopping carts
- Entering or leaving a public place
- Inserting or removing contact lenses

Always wash your hands after:

- Using the toilet, changing a diaper or cleaning up a child who has used the toilet
- Touching an animal, animal feed or animal waste
- Blowing your nose, coughing or sneezing
- Handling garbage
- Handling pet food or pet treats

Also, wash your hands when they are visibly dirty.

If soap and water are not readily available, use a hand sanitizer with at least 60% alcohol to clean your hands.

5 Steps to proper hand-washing

1. Wet your hands
   It is important to use clean water to wet your hands. The temperature of the water doesn't really make a difference for effectiveness but most people like warm water when available. Make sure to get all of your skin wet and then turn off the faucet and apply soap.
2. Lather your hands
   Many people quickly rub their hands together while the water is running. This causes the soap to quickly run off before you have had a chance to thoroughly clean your skin. With the water off apply the soap and rub your hands together until the bubbles begin to build up. This "lathering" is a sign you are doing a good job. Be sure to scrub every part of your hands including both front and back, under your nails and between your fingers.
3. Scrub your hands for 20 seconds
   Simply applying water and soap isn't enough. It is important you scrub your hands for at least 20 seconds to remove dirt, bacteria or any germs that could be transmitted. Count to 20 or hum the happy birthday song twice before you begin to rinse.
4. Rinse your hands
   Turn the tap back on and rinse your hands well under clean running water. Be sure to rinse off all of the soap residue under your nails and from every part of your hands. Rinse your hands well under clean, running water.
5. Dry your hands using a clean towel
   If in a public place, be sure to get your paper towel before turning off the faucet. Germs are all over bathroom fixtures. Reach for the towel, dry your hands thoroughly and then use the towel to turn off the faucet. Use that same towel to open the bathroom door. The door is another germ-filled object. The waste basket should be positioned near the door where you can discard your towel after opening the door with it.

Use Hand Sanitizer When You Can't Use Soap and Water

Washing hands with soap and water is the best way to get rid of germs in most situations. If soap and water are not readily available, you can use an alcohol-based hand sanitizer that contains at least 60% alcohol.

How to Use Hand Sanitizer

1. Apply the gel product to the palm of one hand (read the label to learn the correct amount).
2. Cover all surfaces of hands.
3. Rub your hands and fingers together until they are dry. This should take around 20 seconds.

Highlights of the coming issue

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