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Editorial

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Mini Review Section – Fungi are major spoilage of foods and feedstuffs. The proliferation of various fungi in agricultural products leads to reduction in yield and quality with significant economic losses. Fungi produce secondary metabolites which are referred to as mycotoxins which have been found to be present in most food substances. The mycotoxins are low weight metabolites which cause harm known as mycotoxicoses, in livestock, domestic animals and humans and therefore of public health significance.

Current Trend Section -Careful cleaning and disinfection of environmental surfaces are essential elements of effective infection prevention programs. Improved hydrogen peroxide-based liquid surface disinfectants and a combination product containing peracetic acid and hydrogen peroxide are effective alternatives to disinfectants currently in widespread use, and electrolyzed water (hypochlorous acid) and cold atmospheric pressure plasma show potential for use in hospitals.

In Profile Scientist - Elion decided to dedicate her life to medicine after the deaths of her grandfather from stomach cancer and fiancé from an inflammation of the heart lining. - They soon discovered, from observing the role of purines in nucleic acid metabolism, that bacterial cells require certain purines to make DNA. Hitchings hypothesized that by preventing those purines from entering the metabolic pathway that leads to DNA synthesis, they could stop the production of DNA and thereby stop cell growth. Elion published 225 papers on her findings.

Bug of The Month - *Giardia duodenalis,* a major cause of parasite-induced diarrheal disease. Transmission occurs via the fecal–oral route, and sources of *G. duodenalis* infection include contaminated water or food, or direct contact with infected people or animals. The microorganism has an outer membrane that makes it possible to survive even when outside of its host, and which can render it tolerant to certain disinfectants. Giardia trophozoites are anaerobic and absorb their nutrients from the intestinal lumen.

Did You Know – Within a minute after falling asleep, notable changes start to affect both the brain and body. Body temperature drops, brain activity ramps down, and heart rate and respiration slow as well. Not surprisingly, the body's energy expenditure is lower during sleep.It is important to recognize, though, that what happens during sleep is dynamic. Over the course of one night, you actually progress through multiple sleep cycles

Best Practices - Most people who get dengue will not have symptoms. But for those who do, the most common symptoms are high fever, headache, body aches, nausea, and rash. Most will get better in 1–2 weeks. Some people develop severe dengue and need care in a hospital. In severe cases, dengue can be fatal.

Fungal mycotoxins in foods: A review

Moulds are microscopic, plant-like organisms, composed of long filaments called hyphae. Mould hyphae grow over the surface and inside nearly all substances of plant or animal origin. Because of their filamentous construction and consistent lack of chlorophyll they are considered by most biologists to be separate from the plant kingdom and members of the kingdom of fungi. Moulds are multicellular fungi. They are widely distributed and found wherever moisture is present. The genera of mycotoxigenic fungi are mainly represented by *Aspergillus*, *Penicillium* and *Fusarium*, but *Trichoderma*, *Trichothecium* and *Alternaria* are also important as food contaminants or pathogens for plants, among others.

Fungi are major spoilage of foods and feedstuffs. The proliferation of various fungi in agricultural products leads to reduction in yield and quality with significant economic losses. Fungi produce secondary metabolites which are referred to as **mycotoxins** which have been found to be present in most food substances. The mycotoxins are low weight metabolites which cause harm known as mycotoxicoses, in livestock, domestic animals and humans and therefore of public health significance. The production of mycotoxins is stimulated by certain environmental factors: Therefore, the extent of contamination will differ with geographic location, agricultural methods and the susceptibility of commodities to the penetration of fungi during storage and processing periods. Fungi that produce toxins in food are therefore classified into field fungi and storage fungi based on their ecological requirements for growth. Mycotoxins have been reported in several food products such as cereals, legumes, processed flour, and smoked-dried fish and in dried meats.

Major groups of mycotoxins in foods

Aflatoxins are a type of mycotoxin produced by *Aspergillus* species of fungi, such as *A. flavus* and *A. parasiticus.* The umbrella term aflatoxin refers to four different types of mycotoxins produced, which are B1, B2, G1, and G2. Aflatoxin B, the most toxic, is a potent carcinogen and has been directly correlated to adverse health effects, such as liver cancer, in many animal species. Aflatoxins are largely associated with commodities produced in the tropics and subtropics, such as cotton, peanuts, spices, pistachios, and maize.

Sources of Aflatoxins

Ochratoxin is a mycotoxin that comes in three secondary metabolite forms, A, B, and C. All are produced by *Penicillium* and *Aspergillus* species. The three forms differ in that Ochratoxin B (OTB) is a nonchlorinated form of Ochratoxin A (OTA) and that Ochratoxin C (OTC) is an ethyl ester form Ochratoxin A. *Aspergillus ochraceus* is found as a contaminant of a wide range of commodities including beverages such as beer and wine. *Aspergillus carbonarius* is the main species found on vine fruit, which releases its toxin during the juice making process. OTA has been labelled as a carcinogen and a nephrotoxin and has been linked to tumours in the human urinary tract, although research in humans is limited by confounding factors.

Citrinin is a toxin that was first isolated from *P. citrinum*, but has been identified in over a dozen species of *Penicillium* and several species of *Aspergillus*. Some of these species are used to produce human foodstuffs such as cheese (*Penicillium camemberti*), sake, miso, and soy sauce (*Aspergillus oryzae*). Citrinin is associated with yellowed rice disease in Japan and acts as a nephrotoxin in all animal species tested. Citrinin can also act synergistically with Ochratoxin A to depress RNA synthesis in murine kidneys.

Ergot is a compound produced as a toxic mixture of alkaloids in the sclerotia of species of *Claviceps*, which are common pathogens of various grass species. The ingestion of ergot sclerotia from infected cereals, commonly in the form of bread produced from contaminated flour, cause ergotism the human disease historically known as St. Anthony's Fire. There are two forms of ergotism: Gangrenous, affecting blood supply to extremities, and convulsive, affecting the central nervous system. Modern methods of grain cleaning have significantly reduced ergotism as a human disease, however it is still an important veterinary problem. Ergot alkaloids have been used pharmaceutically.

Patulin is a toxin produced by the *P. expansum*, *Aspergillus*, *Penicillium*, and *Paecilomyces* fungal species. *P. expansum* is especially associated with a range of mouldy fruits and vegetables, in particular rotting apples and Figs. It is destroyed by the fermentation process and so is not found in apple beverages, such as cider. In 2004, the European Community set limits to the concentrations of patulin in food products. They currently stand at 50 µg/kg in all fruit juice concentrations, at 25 µg/kg in solid apple products used for direct consumption, and at 10 µg/kg for children's apple products, including apple juice.

Fusarium toxins are produced by over 50 species of *Fusarium* and have a history of infecting the grain of developing cereals such as wheat and maize. They include a range of mycotoxins, such as: the fumonisins, which affect the nervous systems of horses and may cause cancer in rodents; the trichothecenes, which are most strongly associated with chronic and fatal toxic effects in animals

and humans; and zearalenone, which is not correlated to any fatal toxic effects in animals or humans. Some of the other major types of Fusarium toxins include: beauvercin and enniatins, butenolide, equisetin, and fusarins.

Occurrence of mycotoxins in foods

The most well-known mycotoxin, the potent human hepatocarcinogen aflatoxin, is produced by *A. flavus* and *A. parasiticus*. These moulds occur in warm climates and produce aflatoxin in drought-stressed maize and groundnuts in the field. They also affect these crops and many other commodities (copra, cottonseed, pepper) which are stored under improper conditions of temperature and humidity.

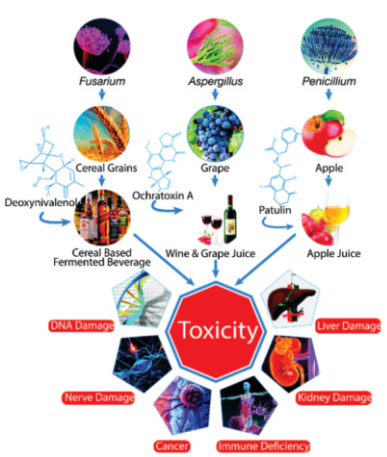
There are 24 toxigenic species of Fusarium which are increasingly viewed as having an important effect on human and animal health. *Fusarium graminearum*, which causes head blight and ear rot, produces a variety of potent mycotoxins including deoxynivalenol, zearalenone and fusarin C. F. *sporotrichioides* produces T-2 toxin and related compounds which were responsible for the large scale human toxicosis in Eastern Europe. The poorest quality grain is used for animal feed. Animal feeds with ingredients such as oilseed cakes, peanut, cottonseed and coconut cake or corn grits often contain mycotoxins.

Mycotoxins in staple grains and seeds

Mycotoxin	Commodity	Fungal source(s)	Effects of ingestion
Deoxynivalenol/nivalenol	Wheat, maize, barley	*Fusarium graminearum* *Fusarium crookwellense* *Fusarium culmorum*	Human toxicoses India, China, Japan, and Korea. Toxic to animals, especially pigs
Zearalenone	Maize, wheat	*F. graminearum* *F. culmorum* *F. crookwellense*	Identified by the International Agency for Research on Cancer (IARC) as a possible human carcinogen. Affects reproductive system in female pigs
Ochratoxin A	Barley, wheat, and many other commodities	*Aspergillus ochraceus* *Penicillium verrucosum*	Suspected by IARC as human carcinogen. Carcinogenic in laboratory animals and pigs
Fumonisin B1	Maize	*Fusarium moniliforme* plus several less common species	Suspected by IARC as human carcinogen. Toxic to pigs and poultry. Cause of equine eucoencephalomalacia (ELEM), a fatal disease of horses
Aflatoxin B1, B2	Maize, peanuts, and many other commodities	*Aspergillus flavus*	Aflatoxin B1, and naturally occurring mixtures of aflatoxins, identified as potent human carcinogens by IARC. Adverse effects in various animals, especially chickens
Aflatoxin B1, B2, G1, G2	Maize, peanuts	*Aspergillus parasiticus*	

Health implications of eaten foods contaminated by mycotoxins

The consumption of mycotoxin-contaminated commodities is related to several acute and chronic diseases in humans as well as in animals. Among the mycotoxins, aflatoxins have been implicated in human diseases including liver cancer, Reye's syndrome, Indian childhood cirrhosis, chronic gastritis, kwashiorkor and certain occupational respiratory diseases in various parts of the world. Fusarium toxins have been suspected to have a role in diseases such as Kashin Beck syndrome in the USSR, China and Viet Nam; Mseleni joint disease in southern Africa; endemic familial arthritis in India; alimentary toxic aleukia in the USSR; and oesophageal cancer in southern Africa. Ochratoxins have been associated with Balkan endemic nephropathy and urinary tract tumours.



The acute diseases for which there is some evidence of an association with mycotoxins include: Aflatoxic hepatitis in Indiaand Kenya; enteric ergotism in India; vascular ergotism in Ethiopia; and deoxynivalenol mycotoxicosis in India and China. A common feature in all these outbreaks has been the involvement of staple foods such as corn, wheat or pearl millet, following unseasonable rains or drought during either the growing season or harvest.

Modern technologies for improving cleaning and disinfection of environmental surfaces in hospitals

Careful cleaning and disinfection of environmental surfaces are essential elements of effective infection prevention programs.

Improved hydrogen peroxide-based liquid surface disinfectants and a combination product containing peracetic acid and hydrogen peroxide are effective alternatives to disinfectants currently in widespread use, and electrolyzed water (hypochlorous acid) and cold atmospheric pressure plasma show potential for use in hospitals.

Creating "self-disinfecting" surfaces by coating medical equipment with metals such as copper or silver or applying liquid compounds that have persistent antimicrobial activity surfaces are additional strategies.

New liquid disinfectants

New disinfectants that are currently available or under development include improved hydrogen peroxide liquid disinfectants, peracetic acid-hydrogen peroxide combination, electrolyzed water, cold atmospheric pressure plasma, and polymeric guanidine.

A new **sporicidal disinfectant** that contains both peracetic acid and hydrogen peroxide has been shown to reduce bacterial levels on surfaces to a greater degree than a quaternary ammonium disinfectant in one study, and reduced contamination by *C.difficile*, MRSA, and VRE as effectively as sodium hypochlorite. The product has a smell like vinegar that may be of concern when it is initially introduced. The combination product gives hospitals a potential alternative to sodium hypochlorite when a sporicidal disinfectant is needed.

Electrolyzed water (hypochlorous acid) disinfectant is produced by passing current through a solution of water and salt. This promising disinfectant was shown to reduce bacterial levels on surfaces near patients to greater degree than a quaternary ammonium disinfectant. In another study, an electrolyzed water disinfectant significantly reduced MRSA, VRE and *C. difficile* spores in in-vitro experiments, and significantly reduced aerobic bacteria and *C. difficile* spores when sprayed onto medical equipment. Electrolyzed water has the advantage of not leaving any toxic residues on surfaces.

Cold-air atmospheric pressure plasma systems are being investigated for possible use as surface disinfectants in healthcare facilities. Reactive oxygen species generated by these systems have bactericidal activity against a variety of pathogens, with variable activity against *C. difficile* spores.

New methods for applying disinfectants

Microfiber cloths or mops and ultramicrofiber cloths are among the relatively newer methods for applying liquid disinfectants to surfaces. Some studies have shown increased cleaning efficacy of microfiber or ultramicrofiber cloths compared to standard cotton cloth or mops. When using microfiber cloths or mops, is important to know that the durability of these products is adversely affected by hypochlorite and high temperatures used during laundering and drying, and that their performance may decrease after multiple washings. One of the advantages of microfiber over cotton cloths is that microfiber is less likely than cotton cloths to bind quaternary ammonium disinfectants.

Self-disinfecting surfaces

Creating "self-disinfecting surfaces" by coating surfaces with heavy metals such as copper or silver that have innate antimicrobial properties or applying to surfaces compounds that retain their antimicrobial activity for weeks or months has received some attention as a new strategy for disinfecting or preventing the growth of bacteria on surfaces in hospitals. Silver binds strongly with disulfide and sulfhydryl groups present in proteins of microbial cell walls, leading to cell death. The antimicrobial activity of copper may be due primarily to its ability to form reactive oxygen radicals that damage nucleic acid and proteins. Impregnating equipment surfaces with copper alloys has been shown to reduce bacterial contamination of surfaces.

Organosilane compounds are comprised of a surfactant plus an antimicrobial substance such as a quaternary ammonium moiety. These compounds are designed to minimize bacterial contamination of surfaces by maintaining their antimicrobial activity on surfaces for weeks or months. Polyhexamethylene biguanide disinfectant was found to reduce bacterial levels on surfaces for at least 24 h after application.

No-touch room decontamination methods

Examples of no-touch room decontamination technologies include aerosolized hydrogen peroxide, hydrogen peroxide vapor systems, gaseous ozone, chlorine dioxide, saturated steam systems, peracetic acid/hydrogen peroxide fogging, mobile continuous ultraviolet devices, pulsed xenon light devices, and high-intensity narrow-spectrum (405 nm) light.

Aerosolized hydrogen peroxide

Aerosolized hydrogen peroxide systems that utilize 3 to 7 % hydrogen peroxide with or without the addition of silver ions. Aerosols (which are not vapor) generally have particle sizes ranging from 2 to 12 µ, are injected into a room, followed by passive aeration. These systems have been shown to significantly reduce bacteria, generally a 4 $\log^{10}$ reduction of spores.

Hydrogen peroxide vapor

A "dry gas" vaporized hydrogen peroxide system that utilizes 30 % hydrogen peroxide has been shown to be effective against a variety of pathogens, including *Mycobacterium tuberculosis*, *Mycoplasma*, *Acinetobacter*, *C. difficile*, *Bacillus anthracis*, viruses, and prions. Dry gas vaporized hydrogen peroxide system, when combined with other infection control measures, appears to have contributed to control of outbreaks of *Acinetobacter* in a long-term care facility and in two intensive care units in a hospital.

A micro-condensation hydrogen peroxide vapor system, which utilizes 35 % hydrogen peroxide, is effective in eradicating important pathogens including MRSA, VRE, *C. difficile*, *Klebsiella*, *Acinetobacter*, *Serratia*, *Mycobacterium*

tuberculosis, fungi, and viruses. It has also been used to decontaminate the packaging of unused medical supplies removed from isolation rooms, instead of discarding such items. This system has also been used to decontaminate rooms previously occupied by patients with the Lassa fever and Ebola virus infection.

Ultraviolet light devices

Automated mobile ultraviolet light devices that continuously emit UV-C in the range of 254 nm can be placed in patient rooms after patient discharge and terminal cleaning has been performed. A number of these devices can be set to kill vegetative bacteria or to kill spores. These systems often reduce the VRE and MRSA by four or more $\log^{10}$, and *C. difficile* by 1–3 $\log^{10}$. Advantages of the mobile, continuous UV-C light devices include their ease of use, minimal need for special training of environmental services personnel, and unlike hydrogen peroxide vapor systems, the ability to utilize the devices without having to seal room vents or doors.

A pulsed-xenon device, which does not use mercury bulbs to produce UV light, emits light in the 200– 320 nm range. It has been shown to significantly reduce pathogens in patient rooms.

High-intensity narrow-spectrum light

High-intensity narrow-spectrum (HINS) light, which is visible violet-blue light in the range of 405 nm has been tested as a means of disinfecting air and surfaces and hospital rooms. This technology targets intracellular porphyrins that absorb the light and produce reactive oxygen species. Its antimicrobial efficacy is lower than UV-C light, but it can be used in areas occupied by patients. In one study, continuous HINS light showed a 27 to 75 % reduction in surface contamination by *Staphylococci* compared to control areas.

Photocatalytic disinfection

An enclosed air purifying system designed for use by NASA utilizes UV-activated titanium dioxide photocatalytic reactions to oxidize volatile organic compounds and airborne microorganisms. Since aerosolization of pathogens such as *S. aureus* and *C. difficile* during patient care activities is known to occur, there may be some interest in using such systems in patient rooms to reduce airborne bacteria may settle onto environmental surfaces.

Gertrude Elion

Leukemia, Herpes Drug Pioneer

Gertrude Elion (1918–1999) and colleague George Hitchings (1905–1998) went off the beaten path of trial-and-error drug development to revolutionize drug making. Using a method known as "rational drug design," Elion and Hitchings were able to successfully interfere with cell growth, giving way to a number of effective drugs for treating leukemia, gout, malaria, herpes, and many other illnesses.

Academic Highs and Lows

Elion decided to dedicate her life to medicine after the deaths of her grandfather from stomach cancer and fiancé from an inflammation of the heart lining. In 1937, she graduated summa cum laude from Hunter College with a degree in chemistry, but her hopes of becoming a research scientist were dashed by 15 rejections for financial assistance from graduate schools throughout the country. Not only did she face discrimination in academia, she also couldn't land a job because of her gender, so she enrolled in secretarial school. Looking back, she said: "I hadn't been aware that any doors were closed to me until I started knocking on them."

While volunteering in a chemistry lab that could only offer her a dishwashing job, an opportunity opened up at Burroughs Wellcome Company. This enabled her to save money for graduate work at New York University, where she was the only woman in her chemistry classes.

Elion completed her master's degree in 1941, but it wasn't until 1944 that she would be hired as a research chemist by Johnson & Johnson. Later that year, Elion was offered another position working with nucleic acids alongside Hitchings at Burroughs Wellcome Company. She also enrolled as a doctoral student at Brooklyn Polytechnic Institute (now Polytechnic Institute of New York University), where she took evening courses. Faced with a decision to enroll full-time to complete a Ph.D. or to continue on as a research chemist, Elion elected to side with the research at Wellcome with Hitchings, rather than pursue her doctoral degree.

"Rational" Research for Leukemia

At the lab, Hitchings had been looking for a more "rational" method of research. The introduction of sulfa drugs inspired him to consider that other substances that interfere with the metabolism of microbes could also be developed as drugs. His approach was to observe the differences in nucleic acid (DNA and RNA) metabolism among protozoa, normal human cells, and abnormal cells (e.g., cancer cells, bacteria and viruses).

Hoping to develop drugs that selectively block the growth of cancer cells and poisonous organisms, Hitchings assigned Elion to investigate organic compounds called purines—specifically, the purine bases adenine and guanine, which are building blocks of DNA. They soon discovered, from observing the role of purines in nucleic acid metabolism, that bacterial cells require certain purines to make DNA. Hitchings hypothesized that by preventing those purines from entering the metabolic pathway that leads to DNA synthesis, they could stop the production of DNA and thereby stop cell growth.

Along the way, Elion published 225 papers on her findings. By 1950, Hitchings and Elion successfully synthesized two compounds—diaminopurine and thioguanine—which attract metabolic enzymes to latch onto them instead of natural purines, thereby blocking DNA production. For the first time, a treatment that could interfere with the formation of leukemia cells was now available to put Leukemia patients in remission.

While the new chemotherapy drugs proved effective in treating the cancer, they were too toxic and caused severe vomiting. Elion began searching for a less poisonous compound, testing over 100 purine compounds. She finally discovered 6-mercaptopurine (6-MP), which she created by replacing one sulphur atom with an oxygen atom.

Gertrude Elion preparing radioactive 6-MP

In testing, mouse tumors failed to grow, and treated mice live twice as long as those left untreated. Children given the treatment went into complete remission, but they were not cured. Elion decided to focus on understanding the metabolism of the drug to come up with a better solution.

Previously that year, she had discovered a close relative of 6-MP called thioguanine. A physician found that by combining either 6-

MP or thioguanine with other drugs, children with leukemia could be treated more effectively. Today, this method—along with maintenance therapy—is responsible for curing 80 percent of children with Leukemia. Thioguanine is also used to treat acute myelocytic leukemia (AML) in adults.

On the Road to the Nobel

In addition to 6-MP, Elion went on to discover a series of drugs that attack the life cycle of nucleic acid, including allopurinol—which inhibits uric acid synthesis, making it a viable treatment for gout—and azathioprine (Imuran), an effective immunosuppressive drug. Her discovery of azathioprine was extremely important to medicine, because it made possible for people with compromised immune systems to receive organ transplants without their body rejecting them.

In the 1960s, Hitchings and Elion also found more success in combating infectious diseases by targeting bacterial and viral DNA: the development of pyramethamine, used to treat malaria; and trimethoprim (Septra), which treats meningitis, septicemia, and bacterial infections of the urinary and respiratory tracts.

On the heels of Hitchings' retirement in 1967, Elion became head of the Department of Experimental Therapy. At that point, she turned her attention to antiviral activity of purines. Testing the compound arabinosyldiaminopurine, Elion and her assistants altered sidechains to produce a more active compound to interfere with the replication of the herpes virus.

The approach proved successful with the synthesis of acycloguanosine, also known as acyclovir (Zovirax). This work proved that drugs can be selective. On this principle, her colleagues later developed the AIDS drug azisothymidine (AZT). That would seal her fate as a recipient of the distinguished Nobel Prize. Elion, Hitchings, and James Whyte Black received the 1988 Nobel Prize in physiology or medicine for discovering important principles of drug treatment.

Elion retired in 1983, eight years after Hitchings. Though she was unable to complete her Ph.D., George Washington University and Brown University awarded Elion honorary doctorates. Elion is also one of the few women recipients of the American Chemical Society's prestigious Garvan Medal, awarded in 1968.

Jokes

Bobby: Pa, does a cup of coffee do any harm?
Pa: No, Bobby.
Bobby: That's lucky! I've just spilled one over your new suit.

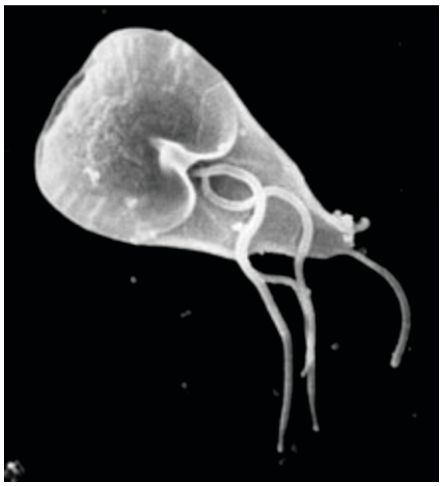
Seema: Why did the firefly get bad grades in school?
Leela: Because he didn't study.
Seema: No, He wasn't very bright.

Lata: Is it right to punish someone for something they have not done?
Teacher: Of course, not!
Lata: I have not done my homework.

Teacher: What makes you see?
Jack: My eyes, My nose and my ears.
Teacher: True for the eyes but why for your ears and nose?
Jack: Its to hold my glasses!

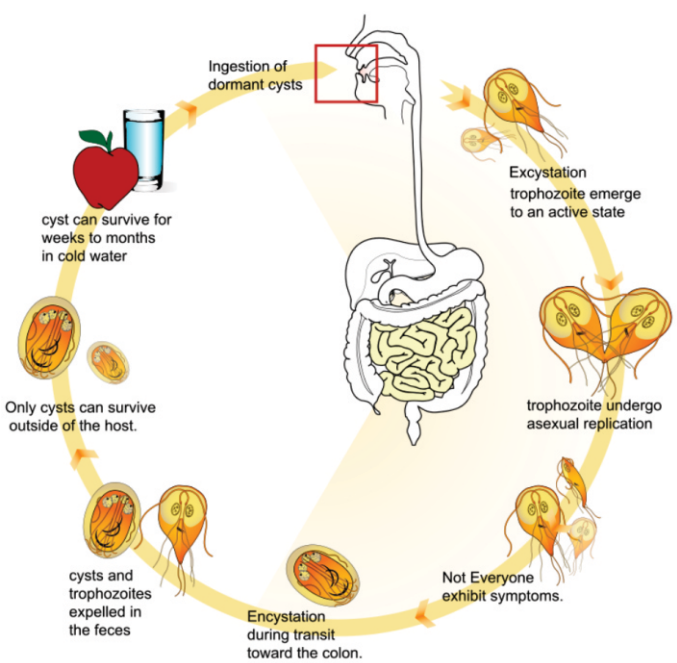
John: Dad, are caterpillars good to eat?
Dad: Have I not told you never to mention such things during meals!
Mom: Why did you say that, John?
John: It's because I saw one on daddy's salad but now its gone.

Giardia duodenalis



Giardia duodenalis (syn. *Giardia lamblia* and *Giardia intestinalis*) is a major cause of parasite-induced diarrheal disease. Transmission occurs via the fecal–oral route, and sources of *G. duodenalis* infection include contaminated water or food, or direct contact with infected people or animals. The parasite attaches to the intestinal epithelium by an adhesive disc or sucker and reproduces via binary fission. Giardiasis does not spread to other parts of the gastrointestinal tract but remains confined to the lumen of the small intestine. The microorganism has an outer membrane that makes it possible to survive even when outside of its host, and which can render it tolerant to certain disinfectants. Giardia trophozoites are anaerobic and absorb their nutrients from the intestinal lumen.

Giardia completes its life cycle by forming cysts (encystation) in the lower intestinal region. Cysts are immediately infectious upon excretion in feces. Replication of the parasite can result in profuse, fatty diarrhoea in infected individuals, but asymptomatic infections are also common.



G. duodenalis takes on two morphologically distinct forms during its lifecycle.

- Trophozoite: This is the active feeding stage of the parasite, residing freely within the human small intestine.
- Cyst: This is the dormant and hardy stage, passed into the environment.

The replicative form is a motile, pear-shaped, flagellated cell that survives only in the small intestine of the host, called a trophozoite. Trophozoites swim through the intestinal mucus until they eventually adhere to the intestinal epithelium. Adhered trophozoites then divide by binary fission, forming either more trophozoites or the nonreplicative cyst stage. Cysts and trophozoites pass through the host's large intestine and are shed in the feces. While the trophozoites cannot survive outside of the host, the cysts can survive for months outside the host especially in cold water because they have a slower metabolic rate than the trophozoites. The cysts remain dormant until ingested by a host animal. When a new potential host ingests water or food contaminated with this feces, the cysts gain entry to the gastrointestinal tract of the new host. In the new host, environmental conditions trigger the cyst to produce two trophozoites, which then attach to epithelial cells, starting the cycle anew.

Giardia duodenalis is common around the world because the parasite resides in bodies of water; typically rivers, lakes, and recreational swimming pools. Additionally, cases of giardiasis tend to be more frequent in developing countries, where the sanitation and overall hygiene is poorer, compared to countries that are more developed and have more advanced sanitary regulations and procedures. In developed nations, giardiasis has a prevalence of 2%-5%, and in developing nations giardiasis has a prevalence of 20%-30%. In addition to waterborne sources, Giardia infections are more commonly found in children than adults, this is believed to be due to fecal-oral transmission of the cysts. Those who work with children are also at risk of being infected, as are family members of infected individuals. Not all Giardia infections are symptomatic, and many people can unknowingly serve as carriers of the parasite. Re- infection and chronic infections of the parasite can occur.

Giardia infects humans but is also one of the most common parasites infecting cats, dogs, and birds. Mammalian hosts also include dozens of species, including cattle, sheep, and goats. Cats can be cured easily, and lambs usually simply lose weight, but in calves, the parasites can be fatal and often are not responsive to antibiotics or electrolytes. Carriers among calves can also be asymptomatic.

G. duodenalis primarily generates its energy by breaking down glucose via glycolysis, as well as the arginine deiminase pathway. It is unable to synthesize nucleotides on its own, instead salvaging them from its host. Synthesis of iron–sulfur clusters is done in a double-membrane-bound compartment called the mitosome, which is likely a remnant of mitochondria. Each cell contains 25 to 100 mitosomes divided into two categories - peripheral mitosomes, which are scattered throughout the cell, and central mitosomes, which gather at the center of the cell for unknown reasons. As in mitochondria, proteins with a certain

peptide signal sequence are trafficked to and imported into the mitosome. Unlike mitochondria, mitosomes have no genome of their own. All mitosomal genes are encoded by the Giardia nuclear genome.

Infections with *Giardia* are self-limited in immunocompetent individuals, while people with immunodeficiency disorders may develop chronic giardiasis. The first physical barrier is the mucus layer where the organism interacts with epithelial, immune cells, and some antimicrobial peptides released by those cells as well as nitric oxide and inflammatory cytokines like interleukin 6. TLR2 and TLR4 also can be activated by *Giardia*.

Frances Gillin of the University of California, San Diego, and her colleagues cultivated the entire lifecycle of this parasite in the laboratory and identified biochemical cues in the host's digestive system that trigger *Giardia*'s lifecycle transformations. They also uncovered several ways in which the parasite evades the defences of the infected organism. One of these is by altering the proteins on its surface, which confounds the ability of the infected animal's immune system to detect and combat the parasite. Gillin's work reveals why *Giardia* infections are extremely persistent and prone to recur. In December 2008, Nature published an article showing the discovery of an RNA interference mechanism that allows Giardia to switch variant-specific surface proteins to avoid host immune response.

Most patients presenting with giardiasis are nontoxic and may necessitate only oral rehydration for initial fluid resuscitation. In severe cases, intravenous (IV) fluids might be necessary. If patients experience persistent symptoms despite therapy, a medication from another class should be used.

The prognosis for patients with giardiasis is generally excellent, as most infections are self-limited. The mortality risk associated with giardiasis is low, with infants or malnourished children facing a slightly higher risk in cases of extreme dehydration. Several antibiotics are available and are reasonably successful in shortening the illness duration, although drug resistance has been reported in clinical cases. If left untreated, giardiasis can persist for weeks, as the parasite stays in the stool, and reinfection is possible.

How Sleep Works: The Science of Sleep

Before the 1950s, most people believed sleep was a passive activity during which the body and brain were dormant. "But it turns out that sleep is a period during which the brain is engaged in a number of activities necessary to life which are closely linked to *quality* of life". Sleep is important to a number of brain functions, including how nerve cells (neurons) communicate with each other. In fact, your brain and body stay remarkably active while you sleep.

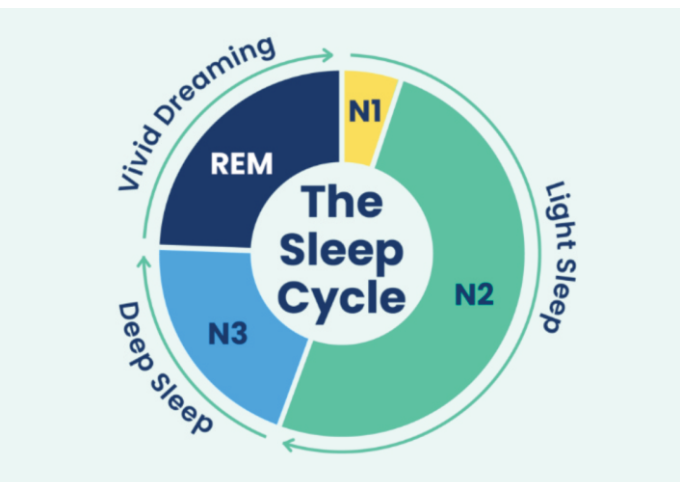
What Happens When You Sleep?

Within a minute after falling asleep, notable changes start to affect both the brain and body. Body temperature drops, brain activity ramps down, and heart rate and respiration slow as well. Not surprisingly, the body's energy expenditure is lower during sleep.

It is important to recognize, though, that what happens during sleep is dynamic. Over the course of one night, you actually progress through multiple sleep cycles, each of which lasts between 70 and 120 minutes and is composed of separate sleep stages. These sleep stages are fundamental to how sleep works.

What Are the Sleep Stages?

Throughout your time asleep, your brain will cycle repeatedly through four stages of sleep divided into two categories. The first three stages fall into the category of non-REM (rapid eye movement) sleep. The fourth stage is REM sleep.



In stage 1, you've just dozed off and started transitioning to stage 2, which involves further slowing of activity in the brain and body. It's much easier to be awoken during these early stages of the sleep cycle.

Stage 3 is the deepest part of NREM sleep. In this stage, your muscles and body relax even more, and brain waves show a clear pattern of slowed activity that is markedly different from waking brain activity. It is believed that deep sleep plays an important role in recuperation of the body as well as effective thinking and memory.

Stage 4 is the only stage of REM sleep. During this time, brain activity picks up significantly, and most of the body (except the eyes and breathing muscles experience) temporary paralysis. Although dreams can happen during any stage, the most intense dreaming takes place during REM sleep.

Though REM sleep was previously believed to be the most important sleep phase for learning and memory, newer data suggests that non-REM sleep is more important for these tasks, as well as being the more restful and restorative phase of sleep.

The body regulates sleep with two key drivers: sleep-wake homeostasis and the circadian alerting system.

Sleep-wake homeostasis. This technical term describes something most of us know implicitly from experience: the longer you're awake, the more you feel a need to sleep. This is because of the homeostatic sleep drive, the body's self-regulating system in which pressure to sleep builds up based on how long you've been awake. This same drive causes you to sleep longer or more deeply after a period of insufficient sleep.

The circadian alerting system. Circadian rhythms are controlled by a biological clock located in the brain. One key function of this clock is responding to light cues, ramping up production of the hormone melatonin at night, then switching it off when it senses light. People with total blindness often have trouble sleeping because they are unable to detect and respond to these light cues.

In addition, a wide range of external factors, can influence sleep-wake homeostasis and the circadian alerting system. For example, stress or hunger may disrupt your normal process for sleep regulation. Caffeine intake or exposure to light from electronic devices are other examples of how behavioural choices can alter the body's underlying systems for managing sleep.

What Chemicals and Hormones Regulate Sleep?

To date, there is much that is still unknown about the intricate processes that control sleep, but researchers have discovered some substances that appear to be important cogs in the machinery of sleep.

A chemical called adenosine is believed to play a central role in sleep-wake homeostasis. Adenosine builds up when we're awake and appears to increase sleep pressure. Caffeine, on the other hand, suppresses adenosine, which may explain part of how it promotes wakefulness.

Neurotransmitters are chemicals that send signals within the nervous system to activate or deactivate certain cells. Examples

of neurotransmitters involved in promoting wakefulness or sleep include GABA, acetylcholine, orexin, and serotonin.

Hormones also play an integral role in signalling and regulating sleep-wake states. Melatonin, which promotes sleep and is naturally produced as light exposure decreases, is one of the best-known hormones related to sleep. Other important sleep-related hormones include adrenaline, cortisol, and norepinephrine. Sleep can also affect the production of vital hormones such as growth hormone as well as leptin and ghrelin that regulate appetite, which may exert influence on sleep-wake homeostasis and circadian rhythms.

Why You Need Sleep

Even after decades of research, the exact reason why we sleep remains one of the most enduring and intriguing mysteries in health science. While even experts haven't reached a consensus explanation for why we sleep, numerous indicators support the view that it serves an essential biological function.

If you have ever felt foggy after a poor night's sleep, it won't surprise you that sleep significantly impacts brain function. From an evolutionary perspective, the fact that sleep exists in almost all animal species despite the fact that it creates vulnerability and takes time away from feeding or procreating is a strong indication that it is fundamental to well-being.

Strategies for Dengue Prevention

Dengue is a viral infection transmitted to humans through the bite of infected mosquitoes. About half of the world's population is now at risk of dengue with an estimated 100–400 million infections occurring each year. Dengue is found in tropical and sub-tropical climates worldwide, mostly in urban and semi-urban areas. While many dengue infections are asymptomatic or produce only mild illness, the virus can occasionally cause more severe cases, and even death. Prevention and control of dengue depend on vector control. There is no specific treatment for dengue/severe dengue, and early detection and access to proper medical care greatly lower fatality rates of severe dengue.

Most people who get dengue will not have symptoms. But for those who do, the most common symptoms are high fever, headache, body aches, nausea, and rash. Most will get better in 1–2 weeks. Some people develop severe dengue and need care in a hospital. In severe cases, dengue can be fatal.

Dengue, an acute febrile illness, is caused by infection with any of 4 related single-stranded RNA viruses of the genus *Flavivirus*, dengue virus 1, 2, 3, or 4 (DENV1–4). Infection with one DENV confers long-term immunity to that virus but conveys only short-lived protection against the other dengue viruses. The risk for severe dengue is greater during a second DENV infection; although severe dengue also can occur during the first, third, or fourth infection.

Symptoms

Most people with dengue have mild or no symptoms and will get better in 1–2 weeks. Rarely, dengue can be severe and lead to death.

Dengue	Dengue with Warning Signs	Severe Dengue
Probable Dengue Live in/travel to endemic area. **Fever** and 2 of the following criteria • Nausea/vomiting • Rash • Aches and pains • Tourniquet test positive • Leukopenia • Any warning sign **Laboratory-confirmed dengue** • Molecular techniques/ IgM or IgG seroconversion	Presence of warning signs • Abdominal pain or tenderness • Persistent vomiting • Clinical fluid accumulation (ascites, pleural effusion) • Mucosal bleeding • Lethargy, restlessness • Postural hypotension • Liver enlargement >2 cm • Progressive increase in hematocrit	One of the following manifestations • Shock or respiratory distress due to severe plasma leakage • Severe bleeding (based on evaluation by attending physician) • Severe organ involvement (such as liver or heart)

If symptoms occur, they usually begin 4–10 days after infection and last for 2–7 days. Symptoms may include:

- high fever (40°C/104°F)
- severe headache
- pain behind the eyes
- muscle and joint pains
- nausea
- vomiting
- swollen glands
- rash

Individuals who are infected for the second time are at greater risk of severe dengue.

Severe dengue symptoms often come after the fever has gone away:

- severe abdominal pain
- persistent vomiting
- rapid breathing
- bleeding gums or nose
- fatigue
- restlessness
- blood in vomit or stool
- being very thirsty
- pale and cold skin
- feeling weak.

People with these severe symptoms should get care right away.

After recovery, people who have had dengue may feel tired for several weeks.

Prevention and control

The mosquitoes that spread dengue are active during the day.

Lower the risk of getting dengue by protecting yourself from mosquito bites by using:

- clothes that cover as much of your body as possible;
- mosquito nets if sleeping during the day, ideally nets sprayed with insect repellent;
- window screens;
- mosquito repellents (containing DEET, Picaridin or IR3535); and
- coils and vaporizers.

Mosquito breeding can be prevented by:

- preventing mosquitoes from accessing egg-laying habitats by environmental management and modification;
- disposing of solid waste properly and removing artificial man-made habitats that can hold water;
- covering, emptying and cleaning domestic water storage containers on a weekly basis;
- applying appropriate insecticides to outdoor water storage containers.

If you get dengue, it's important to:

- rest;
- drink plenty of liquids;
- use acetaminophen (paracetamol) for pain;
- avoid non-steroidal anti-inflammatory drugs, like ibuprofen and aspirin; and
- watch for severe symptoms and contact your doctor as soon as possible if you notice any.

So far one vaccine (QDenga) has been approved and licensed in some countries. However, it is recommended only for the age group of 6 to 16 years in high transmission settings. Several additional vaccines are under evaluation.

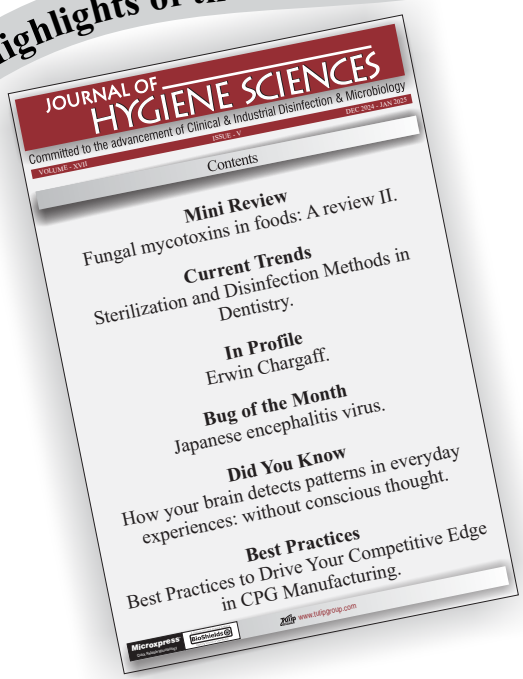
Diagnostics and treatment

There is no specific treatment for dengue. The focus is on treating pain symptoms. Most cases of dengue fever can be treated at home with pain medicine.

Acetaminophen (paracetamol) is often used to control pain. Non-steroidal anti-inflammatory drugs like ibuprofen and aspirin are avoided as they can increase the risk of bleeding.

For people with severe dengue, hospitalization is often needed.

Highlights of the coming issue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