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Editorial

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Mini review section – **wound infection** is defined as the presence of multiplying organisms which overwhelm the body's immune system resulting in spreading cellulitis (inflammation of the tissues). This definition indicates that wound infection results in active disease that is likely to delay the wound healing process and is determined by clinical diagnosis. It involves not only identifying which type of micro-organism(s) are present within a wound through microbiological testing but also assessing the patient for clinical signs and symptoms suggestive of infection, rather than the mere presence of microorganisms within the wound.

Current Trends section – Healthcare-associated infections remain one of the leading causes of increased morbidity and mortality among patients. The patient's endogenous bacterial flora is the main source of nosocomial infections, however 20 to 40% of nosocomial infections are caused by cross-contamination, where the vector of pathogen transmission is the hands of medical personnel.

In Profile Scientist – Ludwig Haberlandt, born in 1885 in Graz, Austria-Hungary, is a name that resonates quietly but significantly in the history of medical science. Though largely unrecognized during his lifetime, he is now considered a visionary and pioneer in the field of hormonal contraception. His early research laid the theoretical groundwork for what would eventually become one of the most transformative medical innovations of the 20th century: the birth control pill.

Bug of the month – The **rhinovirus** is a positive-sense, single-stranded RNA virus belonging to the genus *Enterovirus* in the family *Picornaviridae*. Rhinovirus is the most common viral infectious agent in humans and is the predominant cause of the common cold.

Did You Know? – Influenza viruses are among the most likely triggers of future pandemics. A research team from the Helmholtz Centre for Infection Research (HZI) and the Medical Center - University of Freiburg has developed a method that can be used to study the interaction of viruses with host cells in unprecedented detail. With the help of their new development, they have also analyzed how novel influenza viruses use alternative receptors to enter target cells. The results were recently published in two papers in the journal Nature Communications.

Best Practices – Can vitamin B12 damage kidneys? 5 things to know before regularly taking this supplement.

Tickle yourself to enjoy 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 to your continuous support.

UNDERSTANDING WOUND INFECTION AND COLONISATION

The type of wounds patients present with vary from one setting to another, ranging from acute surgical wounds, traumatic wounds such as those that occur following an accident, burn wounds or chronic wounds such as leg and pressure ulcers.
Healthcare professionals should have a good understanding of wound management issues, including wound colonisation and infection, as failure to distinguish between the two may result in inappropriate patient care and potentially serious complications.

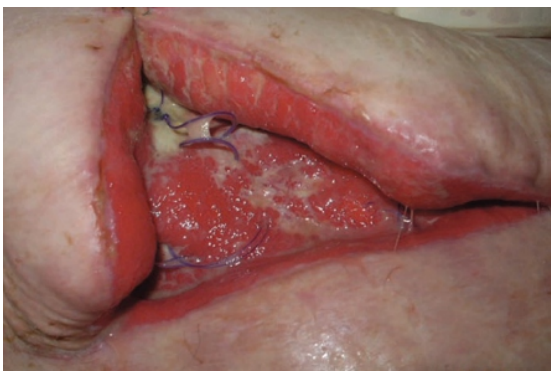
What is wound colonisation and infection?
The first step to understanding wound colonisation and infection is recognising that all wounds are contaminated with microorganisms; **contamination** is the presence of organisms on the surface of a wound. The type and quantity of organisms vary from one wound to another, and contamination can occur in a variety of ways. Often it arises through the transfer of normal body bacteria.
All individuals are covered by a range of bacteria, known as normal body flora, which generally, live quite harmlessly in various body sites without causing any active disease or ill health and often offer protection from more harmful or pathogenic organisms.
Examples of normal body flora include *Staphylococcus aureus* and *Staphylococcus epidermidis*, both of which commonly live on the skin of many individuals without causing any harm.

Wound colonisation is defined as the presence of multiplying micro-organisms on the surface of a wound, but with no immune response from the host and with no associated clinical signs and symptoms. This indicates that wound colonisation, like wound contamination, is a normal state and is not associated with active disease, ill health or delayed wound healing. Within the wound infection continuum, wound colonisation may lead to heavier colonisation, known as critical colonisation. This is the point at which the multiplying micro-organisms can no longer be controlled by the body's immune system and may lead to wound infection.

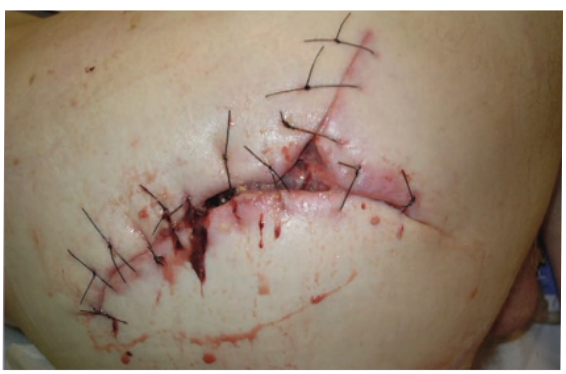
Table 1

Summary of definitions of terminology used

Term	Definition
Contamination	The presence of bacteria on the surface of a wound, before multiplication takes place
Colonisation	The presence of multiplying bacteria in a wound, but with no patient immune response (Ayton, 1985). There is no active disease or ill-health, therefore no signs or symptoms
Critical colonisation	The point when the patient's immune system is no longer able to control the colonising bacteria in a wound
Infection	The presence of multiplying bacteria that overwhelms the patient's immune system and results in spreading cellulitis (Kingsley, 2001). Active signs and symptoms of disease present



Colonised wound



Infected wound

In contrast, **wound infection** is defined as the presence of multiplying organisms which overwhelm the body's immune system resulting in spreading cellulitis (inflammation of the tissues). This definition indicates that wound infection results in active disease that is likely to delay the wound healing process and is determined by clinical diagnosis.
It involves not only identifying which type of micro-organism(s) are present within a wound through microbiological testing but also assessing the patient for clinical signs and symptoms suggestive of infection, rather than the mere presence of microorganisms within the wound.
There are several classic signs and symptoms of infection, such as pus and cellulitis, however, in some types of wounds, for instance chronic wounds with heavy exudate (discharge), identifying signs and symptoms can be difficult.
In addition, in older or immunosuppressed patients, such as those receiving chemotherapy or long-term steroid therapy, the immune response is less efficient and therefore clinical signs and symptoms of infection are less apparent. Two of the most accurate signs and symptoms of wound infection are increasing pain and wound breakdown. Healthcare professionals should therefore regularly assess the patient, including regular visualisation of the patient's wound for signs and symptoms of infection. It has been recommended that wounds should be assessed at all dressing changes.

Pain assessment and management

To accurately assess the patient's level of pain, a pain assessment tool should be used. The Numerical Rating Scale is one such took, where a scale of one to 10 (with one representing no pain and 10 representing severe pain), may be utilised. If the patient does complain of unexpected or increasing pain, the practitioner should provide analgesia as prescribed and report to the relevant medical staff for review.

Factors that determine wound colonisation or infection

Several factors determine whether a wound remains harmlessly colonised or becomes infected. there needs to be a balance between the patient's immune response and type of wound versus the type, quantity and disease-causing ability (known as virulence) of the micro-organisms present within a wound. Wound infection will occur when this balance is lost.

Individual resistance to infection

Individual vulnerability

The patient's individual vulnerability and immune response will significantly influence what effect bacteria have within a wound. Factors include:

- Stress
- Nutritional status
- Circulatory system
- Metabolic disorders
- Increasing age
- Concurrent infections
- Drugs that lower the immune system
- Body size

These factors indicate that the healthier the patient, the more likely it is that a wound will remain harmlessly colonised with microorganisms and there will be a lower risk of wound infection. Patients who have a compromised immune system due to some or all these factors will have a greater risk of wound infection.

Reducing the risk of infection

Nutrition

Healthcare professionals have a key role in assessing whether the patient has a healthy, well-balanced diet with enough calories, protein, vitamins and minerals to aid the wound healing process and reduce the risk of wound infection. All these dietary components are crucial for uncomplicated wound healing.

Wound characteristics

Wound characteristics such as size, position, duration and presence of dead tissue or blood clots are all important factors that also impact on wound colonisation and infection. In terms of wound size, the larger the wound the greater the surface space for bacteria to contaminate, multiply and potentially lead to either wound colonisation or infection.

The position of a wound on the patient's body may also influence the amount of wound contamination that occurs, thus increasing the risk of wound colonisation and infection. The duration of the wound also impacts on wound colonisation and infection.

Microbial characteristics

The type and/or number of bacteria present within a wound may influence whether it remains harmlessly colonised or becomes infected. Both factors are important as all organisms have differing levels of virulence, or ability to cause disease. The bacterial loading or bioburden within the wound is particularly significant for patients with a reduced immune response, for instance those receiving chemotherapy. In contrast, other organisms have high virulence and will produce infection readily even if present in very low numbers. *S. aureus*, including methicillin-resistant *S. aureus* (MRSA), is often found in chronic wounds such as leg ulcers in large quantities but does not cause wound infection, while in contrast other organisms, such as beta-haemolytic *streptococci*, can cause wound infection even in low concentrations as these bacteria have much higher virulence.

Managing wound colonisation and infection

Healthcare professionals play a pivotal role in correctly identifying and managing wound colonisation and infection.

For colonised wounds it is important to monitor the wound regularly for any signs of change that may indicate the development of infection. Once assessed as being colonised, the wound should be dressed with an appropriate dressing product most suited to the wound type, following moist wound healing principles. Patients with particularly large, deep wound infections are potentially at risk of systemic infection, such as septicaemia (infection in the bloodstream) and consequently are at risk of multi-organ failure and possibly death.

It is vital to monitor the patient carefully for signs of deterioration. This includes regular visualisation of the wound and monitoring the patient's body temperature to detect for pyrexia (raised body temperature), which may be indicative of spreading infection throughout the body.

Patients diagnosed with a wound infection will require systemic antibiotics; these will be prescribed according to available antibiotic sensitivities.

Antiseptics are available in a variety of preparations, including solutions and dressings. Care needs to be taken when using antiseptic solutions as they can be toxic to wounds when used at high concentrations and may cause delayed healing.

Examples of antiseptic impregnated dressings which may be useful include iodine and silver dressings, both of which have good action against most bacteria and fungi.

Reducing the risk of cross-infection

Patients with wounds represent a cross-infection risk, regardless of whether they have a wound that is colonised or infected. It is important to remember that one patient's wound colonisation may be another patient's wound infection if they are more susceptible to infection.

For example, MRSA may colonise some patients' wounds but equally can cause wound infection even if present in the same numbers in those patients who are vulnerable.

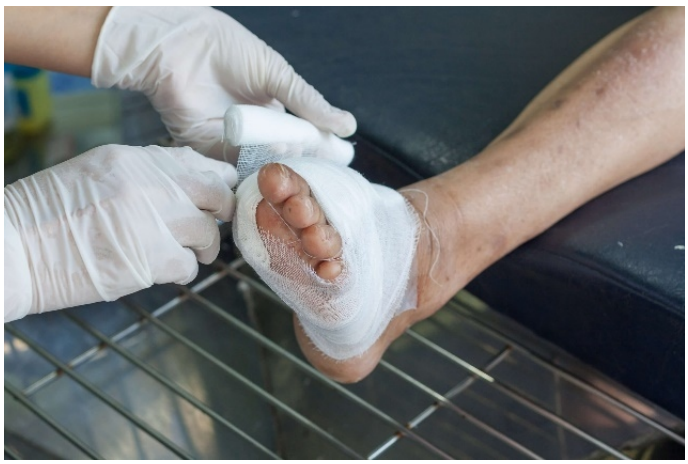
Several factors may increase the risk of spreading microorganisms from one patient to another. A primary means of cross infection is via healthcare workers' hands.

A primary means of cross infection is via healthcare workers' hands which become readily contaminated during wound cleansing and dressing, even if disposable gloves are worn. This is because hands often become contaminated during glove removal. Furthermore, gloves can leak through micro-tears that are not visible to the naked eye but are large enough to allow microorganisms through the gloves. If hands are not thoroughly washed following glove removal these bacteria may be spread to other patients.

To minimise this risk healthcare professionals should undertake meticulous hand hygiene. Hands should be thoroughly cleaned before and after all clinical contact with patients where they are likely to become contaminated with micro-organisms, including following glove removal.

Contaminated equipment and instruments may also spread micro-organisms from one wound to another. For example, a footstool used by a patient with a heavily discharging wound may act as an indirect source of cross-infection if the stool is not thoroughly cleaned before use by another patient.

Poor wound dressing technique may be another important means of spreading infection. If aseptic or clean technique principles are not strictly adhered to while undertaking wound dressings, micro-organisms may be transferred from one wound to another. When adopting aseptic principles, it is important that only sterile items have contact with the vulnerable wound site to reduce the risk of wound infection; surgical wounds healing by primary intention will have few contaminating organisms present and it is important to keep microbial numbers low to facilitate healing.



When using a clean technique for wounds healing by secondary intention, clean rather than sterile items may be used, for example, clean rather than sterile gloves, as these types of wounds are already likely to be heavily contaminated with microorganisms.

Bacteria are present in all wounds; however, their mere presence does not indicate wound infection. The role bacteria play within a wound will depend on a variety of factors, including the type of bacteria present, numbers and their disease-causing ability, all balanced against individual patient vulnerability to infection and the nature of the wound itself. It is crucial for healthcare professionals to understand these contributing factors to comprehend the difference between wound colonisation and infection so that patients with wounds receive the most appropriate care.

Contamination, Disinfection, and Cross-Colonization: Are Hospital Surfaces Reservoirs for Nosocomial Infection?

Healthcare-associated infections remain one of the leading causes of increased morbidity and mortality among patients. The patient's endogenous bacterial flora is the main source of nosocomial infections, however 20 to 40% of nosocomial infections are caused by cross-contamination, where the vector of pathogen transmission is the hands of medical personnel.

Contamination of the hands of medical personnel may result from direct contact with the patient or be caused by touching infected surfaces in the patient's environment.

A patient can become infected by pathogens that cause nosocomial infections by meeting contaminated surfaces in a medical facility. All surfaces in healthcare facilities should be visibly clean, i.e., free of visible residues, e.g., body fluids.

The risk of nosocomial infections is related to microbial contamination of the surface by Gram (−) bacteria, e.g., *Acinetobacter*, Gram (+), e.g., *Staphylococcus aureus*, viruses, such as corona-, Noro- and rotaviruses, and fungi, e.g., *Candida*. Even a single contact of human skin with a contaminated surface can contribute to the transmission of the pathogen. The most easily transmitted diseases from inanimate surfaces to the skin are *Escherichia coli*, *Salmonella* spp., *Staphylococcus aureus* (100% of cases), *Candida albicans* (90%), rhinoviruses (61%), HAV (33%) and rotaviruses (16%).

Different countries have different rules for cleaning and disinfecting of surfaces, but surface disinfection always increases the level of microbiological cleanliness of the patient's surroundings, thus preventing the occurrence of additional infectious complications, breaking the chain of infections and contributing to the prevention of infectious diseases. Therefore, there is a strong need to constantly improve the procedures of cleaning and disinfecting surfaces in healthcare facilities. Very high concentrations of some pathogens in body fluids, such as blood, which can remain on surfaces in the patient's environment in very small amounts on the order of a few µL, pose a serious risk of infection. Therefore, disinfection procedures, not simply cleaning procedures, are important, because even the most effective washing does not completely remove microscopic residues of body fluids, which can be a carrier of infectious agents.

Hygiene measures are an important element in infection prevention procedures, which reduce the risk of transmission of pathogens. Infection control to prevent hospital epidemics is mandatory and essential, especially in hospitals and other healthcare settings.

The quality of the evidence that examines the contamination of the inanimate environment should be judged according to whether the following 4 factors have been measured: (1) the degree of contamination of the nosocomial environment by specific pathogens; (2) whether temporality is addressed (i.e., whether the environment is contaminated before or after patient colonization); (3) the assessment of confounders, such as hand hygiene and the quality of cleaning of fomites; and (4) whether improved cleaning, after controlling for other interventions, reduces the risk of patient infection.

Contamination of the hospital environment by nosocomial pathogens

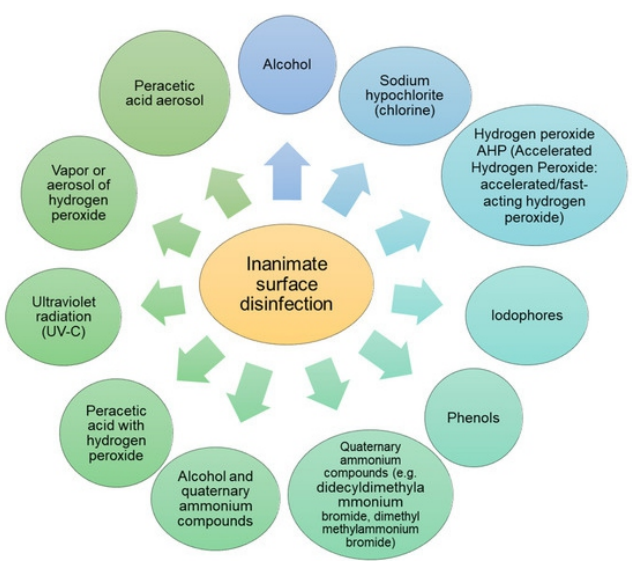
Table 1. Summary of nosocomial pathogens and environmental contamination.

Pathogen	Types of environmental contamination	Length of survival of organism
Influenza virus	Aerosolization after sweeping [3]; survival on fomites	24–48 hours on nonporous surfaces [3]
Parainfluenza virus	Survival on clothing and nonporous surfaces [5]	10 hours on nonporous surfaces; 6 h on clothing [5]
Noroviruses	Persistent outbreaks on ships [6]; extensive environmental contamination [7]; possible aerosolization [8]	≤14 days in stool samples [6]; ≤12 d on carpets [7]
Hepatitis B virus	Environmental contamination with blood	7 days [9]
SARS-associated coronavirus	Positive results of cultures of samples from an ED environment [12]; high–secondary attack rate events (i.e., super spreading events)[a] [15]	24–72 hours on fomites and in stool samples [13]
Candida species	Contamination of fomites [16]	3 days for *Candida albicans* [17] and 14 days for *Candida parapsilosis* [17]
Clostridium difficile	Extensive environmental contamination [18–20]	5 months on hospital floors [19]
Pseudomonas aeruginosa	Contamination of sink drains [21]	7 hours on glass slide [22]
Acinetobacter baumannii	Extensive environmental contamination [24, 25]	33 days on plastic laminate surfaces [26]
MRSA	Burn units extensively contaminated [28]	≤9 weeks after drying [29]; 2 days on plastic laminate surfaces [30
VRE	Extensive environmental contamination [32–34]	≤58 days on countertops [35]

INTERVENTION STRATEGIES

Two major categories for the intensity of cleaning exist: sterilization and disinfection. Sterilization destroys all microbial life on an object or surface and occurs using heat, pressure, or chemical methods.

Disinfection eliminates most microbes, excluding bacterial spores, and typically involves the use of chemical agents. The degree of destruction of organisms depends on their sensitivity to chemical disinfection. High-level disinfection involves the elimination of all but large quantities of spores, intermediate-



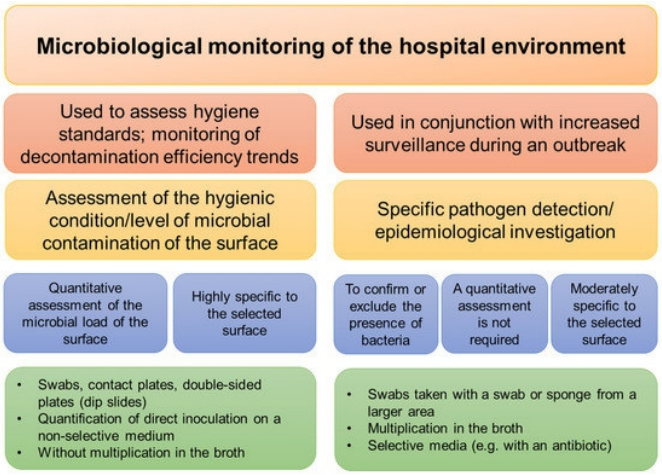
level disinfection leads to destruction of all life except spores, and low-level disinfection will not reliably kill mycobacteria or spores.

"Cleaning" is the process of removal of foreign material from a surface or object and may involve both mechanical processes and the use of detergents with water. Cleaning, alone, can reduce the organism load on a surface and, if used in conjunction with disinfection, may lead to significant reductions in organism load in shorter spans of time. Three types of available solutions can be used during cleaning: detergents, which remove organic material and suspend grease or oil; disinfectants, which rapidly kill or inactivate infectious particles; and detergent-disinfectants, which achieve both aims.

Changes in cleaning products or cleaning practices are generally not required to eliminate specific pathogens. Areas with high rates of *C. difficile* infection may warrant the use of hypochlorite-based products because of the more reliable sporicidal activity of these agents. Most commercial disinfectants used for environmental cleaning have activity against viruses. enveloped viruses are more susceptible to detergents than are nonenveloped viruses.

Decontamination performed after outbreaks of norovirus should involve the use of a germicidal product, such as 10% sodium hypochlorite solution (i.e., household bleach), and closure of an affected institution or facility may be necessary.

Inanimate environmental surfaces can become durably contaminated after exposure to colonized patients; although an organism may be endemic within an institution, specific isolates may predominate in the inanimate environment; and contaminated rooms may be a risk factor for the acquisition of nosocomial pathogens by unaffected patients.



Scientific research has confirmed that contaminated hospital surfaces can be the cause of infection; therefore, cleaning and disinfection procedures should be carried out very carefully, selecting appropriate washing, disinfecting or washing-disinfecting agents with a broad biocidal spectrum and high efficiency, as well as in accordance with applicable standards and recommendations. Molecular epidemiology has helped to enhance understanding of the role of the environment in nosocomial infection by confirming that isolates in the environment are the same as isolates recovered from patients.

The use of the correct disinfectant and following an effective cleaning procedure are key to preventing health and safety risks. A properly selected preparation for a specific surface and the degree of its contamination ensures that the microorganisms will be completely removed, not spread accidentally, and any additional threats will be minimized.

Ludwig Haberlandt

Ludwig Haberlandt, born in 1885 in Graz, Austria-Hungary, is a name that resonates quietly but significantly in the history of medical science. Though largely unrecognized during his lifetime, he is now considered a visionary and pioneer in the field of hormonal contraception. His early research laid the theoretical groundwork for what would eventually become one of the most transformative medical innovations of the 20th century: the birth control pill.

Early Life and Academic Foundation

Ludwig Haberlandt was born into a family of scientific distinction. His father, Gottlieb Haberlandt, was a well-known botanist and professor at the University of Graz. Growing up in an intellectually stimulating environment, Ludwig pursued a career in medicine, focusing on human physiology and the intricacies of reproduction.

Haberlandt studied medicine at the University of Innsbruck and later at the University of Vienna, where he developed an interest in the endocrine system. Influenced by emerging studies on hormones and their roles in bodily functions, he began exploring how internal chemical messengers regulate reproductive processes. It was during these formative years that the seeds for his groundbreaking ideas on hormonal contraception were planted.

Groundbreaking Research in Hormonal Control

In the 1920s, Ludwig Haberlandt conducted a series of experiments on animals, mainly rabbits and guinea pigs, to explore whether ovarian extracts containing hormones could suppress ovulation. At the time, hormone science was still in its infancy. The concept that female fertility could be pharmacologically regulated was radical and largely speculative.

Haberlandt's experiments demonstrated that by administering extracts from pregnant animals' ovaries, ovulation in healthy female animals could be temporarily suppressed. This was a remarkable finding—it implied that the female reproductive cycle could be manipulated by introducing specific hormones externally. He published his research with cautious optimism, proposing the development of a "chemical method" of birth control.

His most notable publication came in 1931, titled **"Hormonale Sterilisation des Weibes"** (Hormonal Sterilization of the Female). In this paper, he clearly articulated the possibility of a contraceptive pill that could temporarily halt fertility by using hormones. He even envisioned a future where women could have access to a reliable, reversible method of birth control—a concept that would not be realized until two decades after his death.

Scientific Vision Ahead of Its Time

What made Haberlandt's work so revolutionary was not just the scientific method, but the foresight. He understood that hormonal contraception, if developed successfully, could have profound effects on public health, family planning, and the autonomy of women.

His vision preceded the discovery and synthesis of key sex hormones like progesterone and estrogen, which would later become essential components in oral contraceptives. At a time when many scientists were still unraveling the basic principles of endocrinology, Haberlandt was already thinking ahead about practical applications for reproductive control.

Opposition and Isolation

Despite the scientific merit of his research, Haberlandt faced substantial resistance. His work challenged not only the scientific norms of his time but also the cultural, religious, and moral frameworks surrounding sexuality and reproduction. In the early 20th century, the idea of birth control was taboo in many societies. Religious groups denounced it, lawmakers censored it, and even many in the scientific community shied away from publicly supporting it.

This lack of support, combined with the absence of advanced biochemical tools needed to develop a reliable contraceptive, left Haberlandt professionally isolated. His pioneering ideas did not receive the recognition they deserved, and funding for further research was limited.

The psychological toll of rejection and stagnation took a heavy toll on Haberlandt. Tragically, in 1932, at the age of 47, he took his own life—just years before hormonal science would begin to accelerate and eventually validate his theories.

Posthumous Recognition and Influence

Although Haberlandt did not live to see the realization of his vision, his work was not in vain. In the 1950s, scientists such as **Gregory Pincus**, **Carl Djerassi**, **John Rock**, and **Margaret Sanger** played key roles in developing and promoting the first oral contraceptive pill. Their work was made possible by advancements in hormone synthesis and biochemistry that were unavailable during Haberlandt's time.

Historians of medicine and reproductive health now acknowledge Haberlandt as a foundational figure in contraceptive research. While he did not directly invent the pill, his research anticipated the basic principle on which it is based: that synthetic hormones can be used to safely and effectively regulate fertility.

Many modern reproductive scientists and advocates now consider Haberlandt a visionary whose ideas helped catalyze one of the most liberating inventions for women in modern history.

Impact on Society and Medicine

The invention of the contraceptive pill in the mid-20th century had profound and far-reaching effects, especially for women. It gave them greater control over their reproductive choices, contributed to the rise of the feminist movement, and transformed social and sexual norms.

Though Haberlandt did not live to witness this transformation, his early work is often cited in medical history as a turning point in our understanding of reproductive endocrinology. The field of family planning and women's health owes a conceptual debt to his early insights.

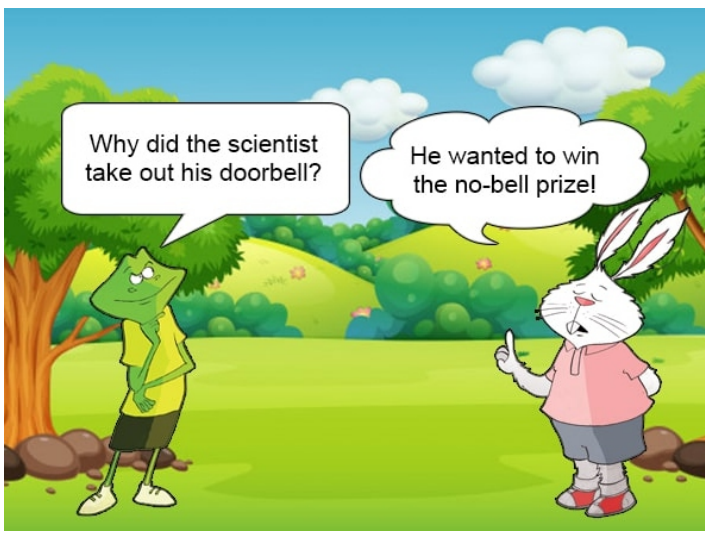
Today, hormonal contraceptives are used by hundreds of millions of people worldwide and continue to evolve with advancements in medicine. From pills and patches to implants and hormonal IUDs, the legacy of Haberlandt's vision lives on in every modern contraceptive method based on hormonal regulation.

Conclusion

Ludwig Haberlandt's life is a poignant story of brilliance, foresight, and tragic obscurity. A man decades ahead of his time, he dared to imagine a world where fertility could be safely and reversibly controlled—a vision that would only be realized long after his death. Though once dismissed and forgotten, his name now holds a place of honor among the pioneers of reproductive science.

In recognizing Ludwig Haberlandt today, we not only acknowledge a scientific legacy but also reaffirm the importance of intellectual courage in the face of societal resistance. His life reminds us that progress is often born not just from innovation, but from the resilience of those who dare to think differently.

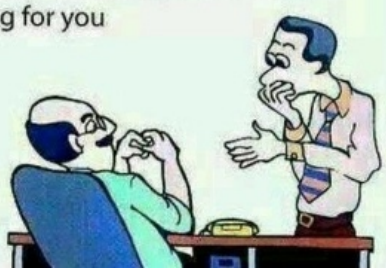
Jokes



BOSS to an employee:
Do you believe in life after Death?

EMPLOYEE:
Certainly not, there's no proof of it

BOSS: Well, there is now
After you left early yesterday to go to your uncle's funeral, he came here looking for you

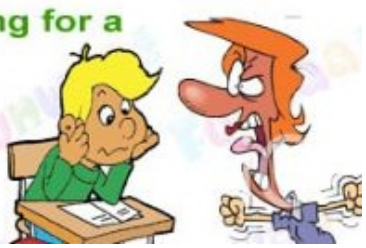


Maths Teacher: What is a line?

Pappu: A line is a dot that's going for a walk.

Teacher: Then what are parallel lines?

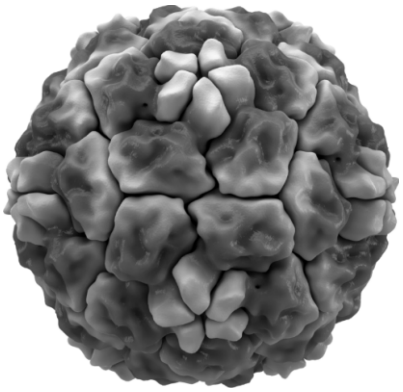
Pappu: A dot going for a walk with his Girlfriend!



JOKE OF THE DAY

Mom: Reema, have you seen my newspaper?
Reema: Mom, newspapers are old school! Why don't you use the tablet to read?
Mom: How am I supposed to use the tablet to kill a fly?

Rhinovirus



The **rhinovirus** is a positive-sense, single-stranded RNA virus belonging to the genus *Enterovirus* in the family *Picornaviridae*. Rhinovirus is the most common viral infectious agent in humans and is the predominant cause of the common cold.

The three species of rhinovirus (A, B, and C) include at least 165 recognized types that differ according to their surface antigens or genetics. They are among the smallest viruses, with diameters of about 30 nanometers. By comparison, other viruses, such as smallpox and vaccinia, are around ten times larger at about 300 nanometers, while influenza viruses are around 80–120 nm.

Rhinoviruses are transmitted through aerosols, respiratory droplets, fomites, and direct person-to-person contact. They primarily infect nasal epithelial cells in the airway and cause mild symptoms such as sore throat, cough, and nasal congestion. However, rhinovirus infection can cause more severe disease in infants, the elderly, and the immunocompromised. Rhinoviruses are also recognized as a major cause of asthma exacerbations.

As of April 2024, there are no FDA-approved vaccines or antiviral treatments for rhinovirus infection.

History

In 1953, when a cluster of nurses developed a mild respiratory illness, Winston Price, from Johns Hopkins University, took nasal passage samples and isolated the first rhinovirus, which he called the JH virus, named after Johns Hopkins. His findings were published in 1956.

In 2006, advancements in molecular testing techniques for identifying rhinoviruses in clinical specimens led to the discovery of rhinovirus C species in samples from Queensland, Australia and New York City, United States. The ICTV formally designated RV-C as a separate species in 2009.

Transmission

Rhinoviruses may be spread via airborne aerosols, respiratory droplets and from fomites (contaminated surfaces), including direct person-to-person contact. Rhinoviruses can survive on surfaces such as stainless steel or plastic for several hours. Airborne precautions are likely effective in reducing transmission, while other precautions such as hand-washing or cleaning surfaces with disinfectants are known effective in preventing rhinovirus transmission.

Signs and symptoms

Rhinoviruses are the primary cause of the common cold. Symptoms include sore throat, runny nose, nasal congestion, sneezing and cough; sometimes accompanied by muscle aches, fatigue, malaise, headache, muscle weakness, or loss of appetite. Fever and extreme exhaustion are less common in rhinovirus infection compared to influenza, but rhinovirus can cause lower respiratory tract infection and the pneumonia can, in young children, be fatal.

Epidemiology

Rhinoviruses can be detected year-round; however, the incidence of rhinovirus is higher in the autumn and winter, with most infections occurring between September and April in the northern hemisphere. The seasonality may be due to the start of the school year and to people spending more time indoors thereby increasing the chance of transmission of the virus. Lower ambient temperatures, especially outdoors, may also be a factor given that rhinoviruses preferentially replicate at 33 °C (91.4 °F) as opposed to 37 °C (98.6 °F). Other climate factors such as humidity may influence rhinovirus seasonality. Young children (<5 years old) experience a high rate of infection which can be detected in community surveillance studies of children up to 34% of the year. Phylogenetic analysis of rhinovirus strains in Nepalese infants revealed diverse lineages and patterns of virus circulation in low-resource settings. Those most affected by rhinoviruses are infants, the elderly, and immunocompromised people.

Pathogenesis

The primary route of entry for human rhinoviruses is the upper respiratory tract (mouth and nose). Rhinovirus A and B use "major" ICAM-1 (Inter-Cellular Adhesion Molecule 1), also known as CD54 (Cluster of Differentiation 54), on respiratory epithelial cells, as receptors to bind to. Some subgroups under A and B use the "minor" LDL receptor instead. Rhinovirus C uses cadherin-related family member 3 (CDHR3) to mediate cellular entry. As the virus replicates and spreads, infected cells release distress signals known as chemokines and cytokines (which in turn activate inflammatory mediators).

Infection occurs rapidly, with the virus adhering to surface receptors within 15 minutes of entering the respiratory tract. Just over 50% of individuals will experience symptoms within 2 days of infection. Only about 5% of cases will have an incubation period of less than 20 hours, and, at the other extreme, it is expected that 5% of cases would have an incubation period of greater than four and a half days.

Human rhinoviruses preferentially grow at 33 °C (91.4 °F), notably colder than the average human body temperature of 37 °C (98.6 °F), hence the virus's tendency to infect the upper respiratory tract, where respiratory airflow is in continual contact with the (colder) extrasomatic environment.

Rhinovirus A and C species viruses are more strongly associated with significant illness and wheezing, while rhinovirus B species are more commonly mild or asymptomatic.

Taxonomy

Maximum likelihood phylogenetic trees of enterovirus species A, B, C, D and rhinovirus A, B, C isolates from Latin America. The 5'UTR region is much more affected by recombination events than the VP4/VP2 coding sequence. The paraphyletic nature of "rhinovirus" is visible.

The International Committee on Taxonomy of Viruses (ICTV) defines rhinoviruses as three species within the genus

Enterovirus:

- *Enterovirus alpharhino* (*Rhinovirus A*)
- *Enterovirus betarhino* (*Rhinovirus B*)
- *Enterovirus cerhino* (*Rhinovirus C*)

Types

Prior to 2020, enteroviruses (including all rhinoviruses) were categorized according to their serotype. In 2020 the ICTV ratified a proposal[28] to classify all new types based on the genetic diversity of their VP1 gene. Human rhinovirus type names are of the form **RV-*Xn*** where *X* is the rhinovirus species (A, B, or C) and *n* is an index number. Species A and B have used the same index up to number 100, while species C has always used a separate index. Valid index numbers are as follows:[2]

- Rhinovirus A: 1, 1B, 2, 7–13, 15, 16, 18–25, 28–34, 36, 38–41, 43–47, 49–51, 53–68, 71, 73–78, 80–82, 85, 88–90, 94–96, 100–108
- Rhinovirus B: 3–6, 14, 17, 26, 27, 35, 37, 42, 48, 52, 69, 70, 72, 79, 83, 84, 86, 91–93, 97, 99, 100-104
- Rhinovirus C: 1–57

Structure

Rhinoviruses have single-stranded positive sense RNA genomes of between 7200 and 8500 nucleotides in length. At the 5' end of the genome is a virus-encoded protein and, as in mammalian mRNA, there is a 3' poly-A tail. Structural proteins are encoded in the 5' region of the genome and non structural at the 3' end. This is the same for all picornaviruses. The viral particles themselves are not enveloped and are dodecahedral in structure.

The viral proteins are translated as a single long polypeptide, which is cleaved into the structural and nonstructural viral proteins.[29]

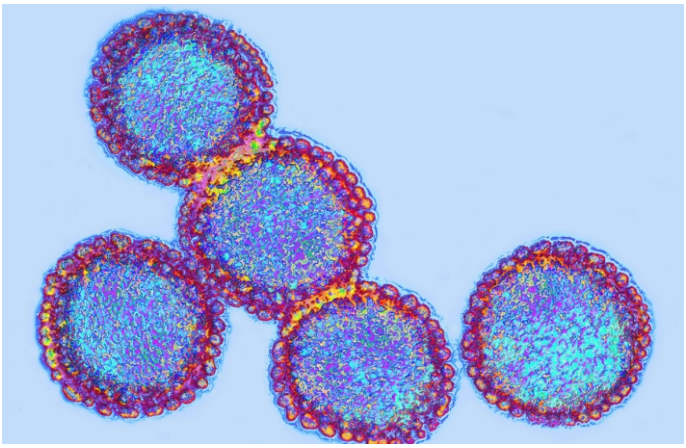
The structure of the virus was determined in 1985 using x-ray crystallography by researchers at Purdue University and the University of Wisconsin led by Michael Rossmann. The virus was crystallized forming cubic crystals with four virus particles in each unit cell (space group $P2_13$, no. 198), similar to a cubic close-packed arrangement.

Human rhinoviruses are composed of a capsid that contains four viral proteins, VP1, VP2, VP3 and VP4. VP1, VP2, and VP3 form the major part of the protein capsid. The much smaller VP4 protein has a more extended structure, and lies at the interface between the capsid and the RNA genome. There are 60 copies of each of these proteins assembled as an icosahedron. Antibodies are a major defense against infection with the epitopes lying on the exterior regions of VP1-VP3.

Prevention

Human rhinovirus can remain infectious for up to three hours outside of a human host. Once the virus is contracted, a person is most contagious within the first three days. Preventative measures such as regular vigorous handwashing with soap and water may aid in avoiding infection. Avoiding touching the mouth, eyes, and nose (the most common entry points for rhinovirus) may also assist prevention. Droplet precautions, which take the form of a surgical mask and gloves, are the method used in major hospitals.[44] As with all respiratory pathogens once presumed to transmit via respiratory droplets, it is highly likely to be carried by the aerosols generated during routine breathing, talking, and even singing. In order to prevent airborne transmission, droplet precautions are insufficient, and routine airborne precautions are necessary.

Viruses under the super microscope: How influenza viruses communicate with cells



Researchers at the HZI and the Medical Center – University of Freiburg uncover new mechanisms for influenza viruses to enter cells

Influenza viruses are among the most likely triggers of future pandemics. A research team from the Helmholtz Centre for Infection Research (HZI) and the Medical Center - University of Freiburg has developed a method that can be used to study the interaction of viruses with host cells in unprecedented detail. With the help of their new development, they have also analyzed how novel influenza viruses use alternative receptors to enter target cells. The results were recently published in two papers in the journal Nature Communications.

Viruses have no metabolism of their own and must therefore infect host cells in order to replicate. Contact between the virus and the cell surface is a crucial first step, which can also prevent infections if entry into the cells is blocked. "The interaction with a host cell is dynamic and transient for influenza viruses. In addition, associated processes occur at the nanoscale, requiring super-resolution microscopes for a more precise investigation. Using conventional approaches, it has therefore not been possible to investigate this important first contact in more detail," says Prof. Christian Sieben, head of the junior research group "Nano Infection Biology" at HZI, explaining the challenge the team has faced.

In collaboration with Prof. Mark Brönstrup's department "Chemical Biology" at HZI, his team has developed a universal protocol to investigate how viruses communicate with host cells. To do this, the scientists immobilized viruses individually on microscopy glass surfaces. Cells were then seeded on top. In conventional experiments, the viruses are added on top of pre-seeded cells. "The advantage of our 'upside-down' experimental setup is that the viruses interact with cells but do not enter them - the critical moment of initial cell contact is thus stabilized and can be analyzed," says Sieben.

Using the example of a seasonal influenza A virus, the researchers used high-resolution and super-resolution microscopy to show that contact between the virus and the cell surface triggers a cascade of cellular reactions. First, the cellular receptors accumulate locally at the virus binding site. This is due to the fact that the receptors move more slowly through the cell membrane near the binding site and are therefore more abundant locally. Subsequently, specific cellular proteins are recruited and finally the actin cytoskeleton is dynamically reorganized.

However, the researchers applied their method not only to an established influenza A model, but also to a novel influenza strain of animal origin: the H18N11 virus, which is found in bats in Central and South America. Unlike most influenza viruses, which bind to glycans - i.e. carbohydrate chains on the cell surface - for infection, the H18N11 virus has a different target. "This virus binds to MHC class II complexes - protein receptors that are typically found on certain immune cells," says Dr. Peter Reuther, research group leader from the Institute of Virology of the Medical Center – University of Freiburg. He is studying the cell entry of bat-derived H18 influenza A viruses.

Using single-molecule tracking, the researchers were able to show for the first time that MHCII molecules cluster specifically on the cell surface upon contact with the virus - a process that is essential for the virus to enter the cell. The teams from Braunschweig and Freiburg have thus characterized a new model of influenza A infection: the binding to MHCII as an alternative receptor and the associated dynamic reorganization of the cell surface. "The finding that influenza viruses do not bind exclusively to cellular glycans opens up new perspectives for research into these pathogens," says Reuther. "Particularly in view of their zoonotic potential, it is crucial to better understand these alternative receptors."

The virus-cell binding step is also the focus of the EU project COMBINE, which was launched at the beginning of 2025 and is coordinated by HZI researcher Sieben. In COMBINE, scientists from five European countries are investigating the virus entry process of newly emerging viruses, especially those with pandemic potential. "This process is a potential target for antiviral therapies. The methodology we have developed to investigate the virus entry process can be applied to many other viruses," says Sieben. The new results not only provide detailed insights into the biology of influenza viruses. They also provide a methodological basis for investigating the entry mechanisms of potential pandemic pathogens in a more targeted manner - and thus identifying new targets for antiviral therapies.

Can vitamin B12 damage kidneys? 5 things to know before regularly taking this supplement

Vitamin B12 is often seen as a go-to nutrient when people feel low on energy, focus, or stamina. It's found in multivitamins, energy shots, and even trendy injections at wellness clinics.

What Does Vitamin B12 Do?
Vitamin B12 plays a key role in forming healthy red blood cells, maintaining proper nerve activity, and supporting overall brain health. Since the body doesn't make it on its own, we need to get it through food like dairy, eggs, meat, or supplements. Many people, especially vegetarians or older adults, turn to B12 supplements to meet their needs. However, this does not imply that more is always preferable, even if something is "essential".

Can Vitamin B12 Supplements Harm Your Kidneys?
People with diabetic kidney disease experienced faster decline in kidney function when given high doses of B12 and B6 compared to those who received a placebo. **B12 supplement side effects** may be more serious in people with pre-existing kidney conditions. While healthy kidneys can flush out excess B12 since it's water-soluble, those with impaired kidney function might struggle to eliminate it, leading to unwanted buildup in the blood.

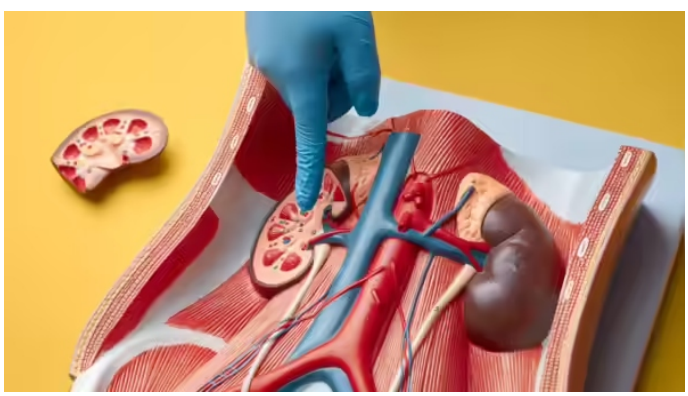
1) Why Dosage Matters More Than You Think

Many B12 supplements available over the counter contain 500 to 1000 mcg per dose, way more than what your body needs. The recommended daily intake is only 2.4 mcg. That's a huge difference.

If your kidneys aren't working well, they may not clear this excess efficiently. As a result, the B12 accumulates in your body. This brings us to one of the lesser-known **long-term effects of B12 supplements:** potential stress on the kidneys, especially if taken in mega doses every day without checking blood levels first.

2)Risk of B12 Shots: Not Just a Wellness Trend
B12 injections are increasing in popularity among people looking to fight fatigue, improve skin tone, or manage weight. These injections, which skip the digestive system and go straight into the circulation, typically include high levels of vitamin B12.

But here's the catch: for people with high blood pressure, diabetes, or declining kidney function (which is common with age), this sudden load on the system can do more harm than good. **Daily B12 supplement risks** are often ignored when people start self-prescribing shots or buying them online without proper consultation.



Doctors usually recommend B12 injections only in cases of severe deficiency, like in people with pernicious anaemia or absorption disorders. If you're not diagnosed with these, regular B12 shots might be unnecessary and risky.

3)One Size Doesn't Fit All
As we age, two important things happen: we absorb less B12 from food, and our kidney function starts to decline. So, while an elderly person might truly need a supplement, it doesn't mean they should start taking high doses every day.

This is where tailored solutions matter. Instead of going for the highest dose available, options like low-dose B12 tablets under the tongue or fortified foods might be gentler and safer. Understanding the **daily B12 supplement risks** can help avoid complications, especially for older adults or those on multiple medications.

4)Watch Out for Drug Interactions

Another layer to the B12 story is how it interacts with other medications. For example, diabetes medication like metformin is known to lower B12 levels. To correct this, people often start taking supplements, sometimes in large doses. But what if the kidneys are already under strain from diabetes? Now there's a new problem.

Also, taking B12 along with high-dose vitamin C or long-term antacids can affect how your body absorbs it. This may result in either too little or too much B12 in your system, both of which can be harmful over time. Being aware of **B12 supplements' side effects** in combination with other drugs is important to avoid added stress on your kidneys.

5) **Consult your healthcare team**

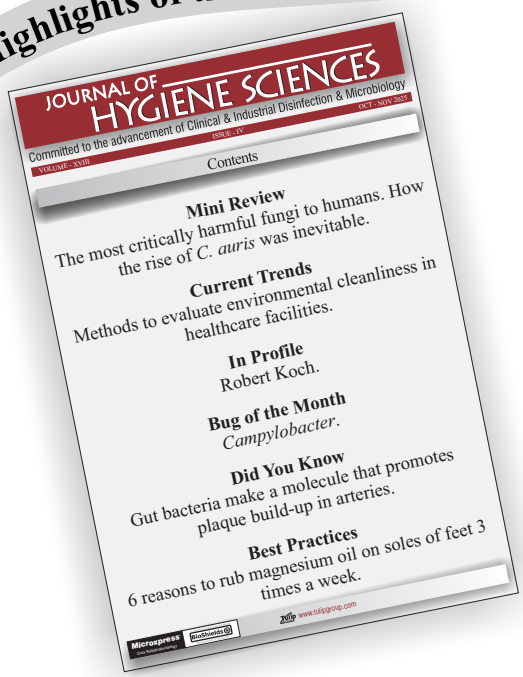
Before starting any new supplement regimen, including B12, it's essential to consult with a doctor or a specialist like a nephrologist (kidney specialist) or a pharmacist. They can assess individual risks, provide tailored advice, and ensure safe supplementation practices

How to Stay Safe with B12 Supplements

Here are a few practical tips if you're considering or already taking B12:

- **Get Tested First:** Before starting a supplement, check your blood B12 levels. Don't assume you're deficient.
- **Avoid Self-Dosing:** Especially with injections or high-dose pills. Talk to a healthcare professional first.
- **Start Low:** Use the smallest dose required. Fortified diets and low-dose oral supplements are frequently sufficient.
- **Monitor Regularly:** If you're on long-term supplements, keep checking your kidney function and B12 levels periodically.
- **Check for Interactions:** Look at your existing medications and discuss potential interactions with your doctor.

Highlights of the coming issue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