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The Relationships Among Routes of Transmissions, Environmental Factors Tolerance and Mutation Rates of Some RNA Viruses

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Abstract

In this study, an attempt was made to determine whether or not there is a connection between the pathogenesis of eight different RNA viruses (SARS-CoV-2, measles, mumps, seasonal influenza, avian influenza, HIV, hepatitis C, and poliovirus) and the environmental and physical conditions that surround them. These conditions included the viruses' ability to withstand heat and humidity, as well as the impact of ultraviolet radiation. The impacts of the acidity factor, the rate of transmission, and the size of the genome.

In addition, the viral mutation rates among the studied viruses were investigated. In general, the findings suggested that the following relationships exist: there is a direct relationship between virus susceptibility to infection and its heat tolerance; there is a positive relationship between virus susceptibility to infection and virus size; there is an inverse relationship between virus susceptibility to infection and high humidity; there is an inverse relationship between virus susceptibility to infection and the rate of mutation of the virus; and there is a positive relationship between virus size and the rate of mutation of the virus.

Keywords: RNA viruses, Route of transmission, Environmental tolerance, Mutation rate, Pathogenicity

Introduction

A virus is an infectious submicroscopic particle that only reproduces inside of live cells. All living things, including plants, animals, and microbes like bacteria and archaea, are susceptible to virus infection (1). They never include both DNA and RNA; instead, they only contain one form of nucleic acid. Since viruses lack ribosomes, mitochondria, or any other organelles, their cellular hosts are their only source of protein synthesis and energy (2). They can only reproduce inside the cells of the infected host. Protein and nucleic acid (the genome), two fundamental components of infectious viral particles, are required. Additionally, the envelopes of all enveloped viruses are made of lipid, and their glycoprotein polymers are made of carbohydrates (as well as that in the nucleic acid). A virus is referred to as an RNA virus if its genetic material is ribonucleic acid (RNA) (3). Usually, the nucleic acid is single-stranded RNA (ssRNA), but double-stranded RNA (dsRNA) is also possible (4). The common cold, influenza, SARS, MERS, SARS-CoV-2, dengue virus, hepatitis C, hepatitis E, west Nile fever, Ebola virus disease, rabies, polio, mumps, and measles are notable human illnesses brought on by RNA viruses. RNA serves as the genetic building block of RNA viruses (5). A protein capsule that encases the genetic material is present. Some viruses have a covering for the capsid called an envelope. RNA viruses replicate themselves in the cytoplasm of their host after infection. RNA viruses' error-prone reproduction results in modest genome sizes. Since viral RNA polymerases can't perform DNA polymerases' proofreading functions, RNA viruses often have far higher mutation rates than DNA viruses (6). It is challenging to create effective vaccinations against RNA viruses because of their genetic variety. In the more basic viruses, the nucleocapsid is made up of the capsid, the protein covering the nucleic acid, and the single molecule of nucleic acid that it encloses. A protein core is encased by the capsid in some of the more sophisticated viruses, whereas a lipoprotein envelope surrounds the capsid in other viruses. The capsid is made up with capsomers, which are morphological components connected by noncovalent connections (7). Electron microscopy often allows for the visualization of individual capsomers, which are made up of one or more polypeptide molecules. The majority of viruses are 20–500 nm in size (8).

In this work, an attempt was made to determine the association between the rate of mutation and the transmission of eight RNA viruses (SARS-CoV-2, measles (MEV), seasonal influenza (SEV), mumps (MUV), avian influenza (H5N1), human immunodeficiency virus (HIV), hepatitis C (HCV), and polio (POV).

SARS-CoV-2 (COVID-19)

A novel coronavirus is a new strain of the coronavirus, and it had not been discovered prior to the outbreak that was reported in Wuhan, China, in 2019 (8-9). The acronym " SARS-CoV-2 has been assigned to this virus. The virus that causes that disease is now referred to by its formal name, SARS-CoV-2. This particular virus is classified under the Nidovirales order and is a member of the Coronaviridae family (10). Under an electron microscope, coronaviruses have the appearance of a crown due to the presence of spike glycoproteins on the envelope of the virus (11). These viruses are very small, measuring between 65 and 125 nanometers in diameter. Coronaviruses have positive-sense RNA genomes that are single-stranded and between 26 and 32 kbp in size. The virus particles contain four key structural proteins (9-12). These particular proteins are referred to as the spike (S), membrane (M), envelope (E), and nucleocapsid (N) proteins (Figure 1).

Route of transmission of SARS-CoV-2

The human SARS-CoV-2 is most commonly spread from person to person through respiratory droplets

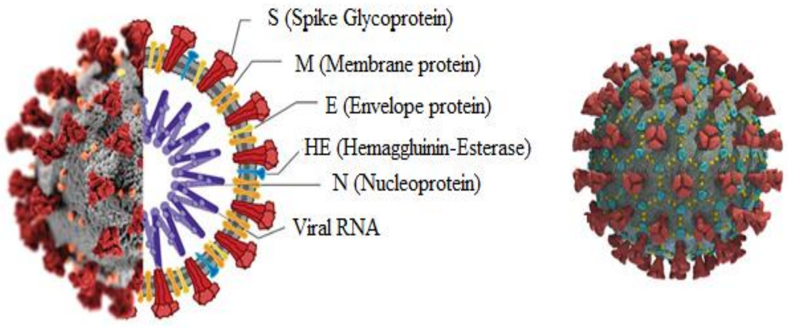

Figure 1: SARS-CoV-2 (a- structure, and b- image) (9).

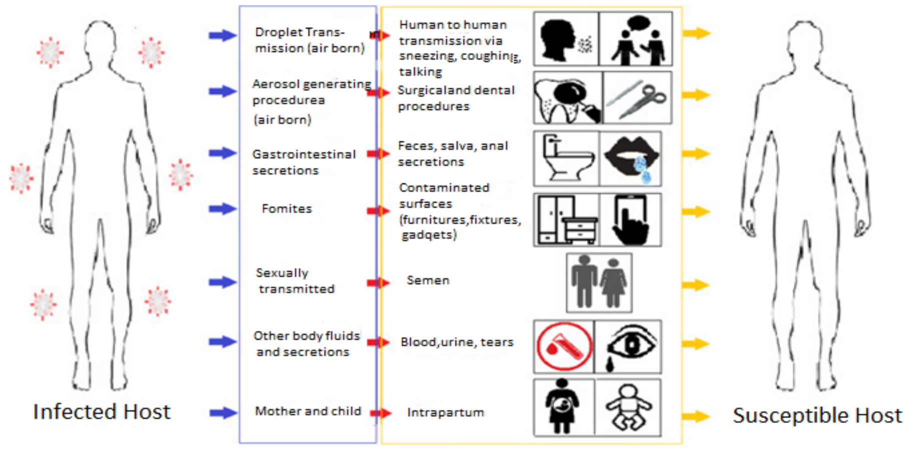

Figure 2: Route of transmission of COVID-19 (13).

that are produced by activities such as breathing, sneezing, coughing, etc., as well as through direct skin-to-skin contact (direct contact with an infected subject or indirect contact through hand-mediated transfer of the virus from contaminated fomites to the mouth, nose, or eyes) (Figure 2) (13-15).

Environmental Tolerance of SARS-CoV-2

Thermolability and effects of humidity on SARS-CoV-2

It was found that the new coronavirus was still able to replicate in cold weather, which led French researchers led by Professor Remi Charrel to conclude that the SARS-CoV-2 coronavirus is able to withstand high temperatures even when heated to as high as 60 degrees Celsius (140 degrees Fahrenheit) for an hour. This conclusion was reached after it was discovered that the new coronavirus was still able to replicate in cold weather. The surface stability of the virus was studied by researchers at room temperatures of both 4 degrees Celsius and 30 degrees Celsius. They discovered that the virus can remain infectious on surfaces for approximately the same amount of time regardless of whether the environment is hot or cold (10-12). A measurement of the amount of water vapor in the air in comparison to the total amount of water vapor that the air is capable of holding is referred to as relative humidity (RH). In general, cooler air can only hold a smaller amount of water vapor than warmer air can. At temperatures between 22 and 25 degrees Celsius and relative humidity levels between 40 and 50 percent, the SARS-CoV-2 virus can maintain its viability on smooth surfaces for more than 5 days. Virus viability, on the other hand, decreases precipitously (>3 log10) with increasing temperature and relative humidity (e.g., 38°C and RH of >95%) (16). In environments with low temperatures and low humidity, the SARS-CoV-2 is more likely to remain stable (9-10). Studies suggest that an increase in humidity can improve the body's ability to fight off infection; that the coronavirus deteriorates more quickly at close to 60% relative humidity than at other levels; and that exposure to dry air can result in a significant increase in the number of tiny coronavirus particles that travel further and penetrate deeper into the lungs (16-18).

Effects of ultraviolet radiation on SARS-CoV-2

The use of UVC-LED was shown to be an effective method for disinfecting surfaces that were contaminated with SARS-CoV-2 and were found on personal items (19-21).

Effect of pH changes on SARS-CoV-2

An acidic pH raises the risk of SARS-CoV-2 infection and increases the likelihood of death from COVID-19 (22). pH has a significant impact on the severity of SARS-CoV-2 infection (23). When incubated at either 4 or 33 degrees Celsius, the human coronavirus infectivity stability was at its highest at a pH of 6.0, and the pH was more pronounced at 33 degrees Celsius (24). After an incubation period of 14 days at temperatures of 22, 33, or 37 degrees Celsius, all trace of viral contagiousness was eradicated; however, at a temperature of 4 degrees Celsius, the infectivity of the virus remained more or less unchanged. At a temperature of 25 degrees Celsius, SARS-CoV-2 was able to maintain its viability for 3–5 days in dried form but only for 7 days in solution. The virus is viable for approximately six days at pH (19-21).

Spread rate of COVID-19

It is believed that two to fourteen days will pass between the time of exposure and the onset of symptoms (also known as the incubation period). Early reports of the epidemic showed exponential growth across the globe, with rates ranging from 0.19 to 0.29 per day (epidemic doubling times between 2.4 and 3.7 days). Close contacts of a COVID-19 patient were at risk of infection at a rate of 0.48 (95% confidence interval: 0.39–0.58) per case. This number would have been significantly higher, at 0.62 (95% confidence interval) (25). if isolation had not been implemented.

Genome size and mutation of SARS-CoV-2

The RNA viral genome of SARS-CoV-2 is the longest of any known virus, measuring between 26 and 32 kilobytes in length. The genome of SARS-CoV-2 shares approximately 82% sequence identity with the genome of MERS-CoV, and the essential enzymes and structural proteins of both viruses share >90% sequence identity (26). It was discovered that the nucleotide mutation rate of the entire SARS-CoV-2 genome was 6.677 x 10-4 substitution per site per year, and the nucleotide mutation rate of the S gene was 8.066 x 10-4 substitution per site per year, which was considered to be at a medium level in comparison to other RNA viruses (27). Because SARS-CoV-2 mutates so slowly, there is reason to be optimistic about the possibility of creating vaccines that are both effective and durable against the virus. Some research showed that the mutation rates of coronaviruses are between 10-5 and 10-3 substitutions per nucleotide site per cell infection (s/n/c) (28). This is despite the fact that the virus has mechanisms that act as a proofreading system.

Measles Virus

Measles is an acute viral infection that affects the entire body and is characterized by a rash, fever, and respiratory involvement. Measles virus, which is a member of the family of paramyxoviruses, is the causative agent of MEV. It is spherical, much like a ball, and the exterior is covered with an envelope. The matrix lies beneath the envelope and is composed of a protein that is referred to as M (Figure 3) (29). The matrix acts as a glue, connecting the envelope of the virus to the interior of the virus (30). The RNA genome of the measles virus consists of approximately 16,000 nucleotides and is encased in an envelope that contains lipids and is derived from the host cell. N, which is a nucleocapsid protein, serves as a shield for the viral genome (31). Other proteins found in the virus include a large protein denoted by the letter L and a phosphoprotein denoted by the letter P. Both of these factors play a role in the process by which the measles virus replicates (32). They contribute to the copying of the genome and the production of new viral proteins. The Morbillivirus Enterovirus (MEV) is a paramyxovirus belonging to the genus Morbillivirus. It ranges in size from 120 to 250 nm in diameter and has a genome consisting of single-stranded, antisense RNA (33). This virus has the appearance of enveloped, pleomorphic spheres that range in diameter from 100 to 300 nanometers. Virions have something called an inner helical nucleocapsid, which is a coiled helix of RNA and protein. The spikes of hemagglutinin and fusion glycoprotein can be found on the envelope. Nucleic acid: RNA virus that is no segmented, single-stranded, and has a negative sense (34).

Route of Transmission of Measles Virus

Measles virus lives in the mucus that is produced in an infected person's nose and throat. It is possible to pass it on to other people by coughing and sneezing. Other people can become infected if they breathe the contaminated air or touch the infected surface and then touch their eyes, noses, or mouths after making contact with the infected surface (35).

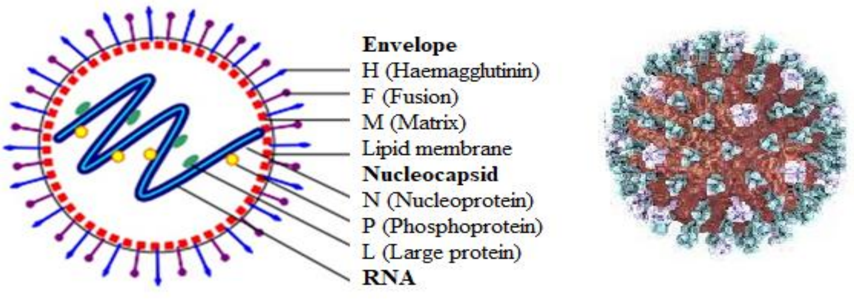

Figure 3: Measles virus (a- structure, and b- image) (29).

Because of how contagious measles is, if one person has it, up to ninety percent of the people in close proximity to that person who do not have immunity to the disease will also become infected. People who are infected with measles can pass the disease on to others from four days before the rash appears until four days after it has appeared. Measles virus has the potential to remain viable in the air for up to two hours. Measles is not a zoonotic virus (36-37).

Environmental Tolerance of Measles Virus

Thermolability and effects of humidity on measles virus

Temperature and relative humidity are the two most important environmental factors that determine whether or not the morbillivirus enterovirus will survive in the air. The incidence of measles is decreased by both hot and cold temperatures, and a low RM is a risk factor for the morbidity associated with measles (38). The relative risk of measles incidence decreased as the temperature rose either above or below the normal range of 18–20 degrees Celsius. The measles virus is temperature-sensitive, and its chances of survival are marginally higher at 15 degrees Celsius than they are at 20 degrees Celsius. It was discovered that the incidence of measles had a negative correlation with RM (39). As a result, there is a correlation between an increase in relative humidity and a decrease in the number of cases of measles. Temperature is a very important factor to consider as a parameter, as temperatures in the range of approximately 30 degrees Celsius are minimally required for the host cell's maintenance metabolism and even for the production of viruses. According to researches, the half-life for inactivation at 37°C or 32°C is just 1 hour, while the half-life for inactivation at room temperature or at 4°C is significantly higher (37). According to research, the ability of the measles virus to survive is highly dependent on the relative humidity of its environment, and the virus is able to thrive in environments with low RH. Therefore, there is a correlation between an increase in the relative humidity and a decrease in the incidence of measles (39).

Effects of UV radiation on measles virus

UV radiation can kill the measles virus easily (40-41).

Effects of pH changes on measles virus

Measles virus is extremely sensitive to shifts in environmental conditions that are not neutral (pH 7.2). At pH levels greater than 10 or 5, it was reported that the virus was completely rendered inactive (42). When the pH was between 7 and 8, the virus titers were stable, but when the pH was between 5 and 7 or 8 and 9, the virus was inactivated by one to two log10 units. At pH 4.4, 3.8, and 2.4 of 2.8 log10 units, an inactivation level that was comparably high was discovered; this level represented a loss of 80% (43). In this particular instance, it has been hypothesized that the observed virus inactivation was brought about by the aggregation of particles brought about by changes in pH. It was discovered that MEV could not survive in environments with pH levels lower than 6.0 and that the infectivity of the virus dropped by 0.5 log values when it was exposed to pH levels that were slightly acidic (41).

Spread rate of measles virus

Because of how contagious measles is, if one person has it, up to ninety percent of the people in close proximity to that person who do not have immunity to the disease will also become infected (37,44).

Genome size and mutation rate of measles virus

Measles virus has a genome that is 15.9 Kbp in size. It ranges in size from 120 to 250 nm, has a genome composed of single-stranded, antisense RNA, and is closely related to the canine distemper and rinderpest viruses. The length of the MEV genome is typically measured to be 15,894 nucleotides, and it encodes a total of eight proteins: six structural proteins (N, P, M, F, H, and L), as well as two nonstructural proteins (V and C) (45). Within this range can be found the average estimate for the measles virus, which is 1.2 x 10-4 per replication. According to the findings of other studies, the mutation rate of the measles virus is approximately 9 x 10-5 per base per replication, and the genomic mutation rate is approximately 1.43 (44).

Mumps Virus

Rubella is a member of paramyxoviruses family. Mumps is an infectious disease that is caused by a virus. The mumps virus is extremely contagious and is the root cause of outbreaks of respiratory illnesses (46). The full-blown mumps virion has the shape of an irregular spherical ball and a diameter that can range anywhere from 90 to 300 nanometers. Figure 4 shows that the nucleocapsid is encased in an envelope that is composed of three layers and has a thickness of approximately 10 nm (47). The mumps virus exists as pleomorphic particles that are composed of a helical ribonucleocapsid core that is surrounded by a lipid envelope that is derived from the host cell. The mumps virus is a member of the family Paramyxoviridae and the genus Rubella virus. Its genome is made up of a single-stranded, nonsegmented RNA with a sense that is opposite to its own. The majority of virus capsids can be classified as either helical or icosahedral, whereas DNA is a double helix made up of nucleotides. The genome of the mumps virus is encased by N proteins, which together form a flexible, loosely coiled helical ribonucleoprotein (RNP) complex. This complex is comprised of the genome being surrounded by a nucleocapsid, which RdRp is bound to (48). Ribonucleotides are encased in a lipid membrane that functions as an envelope. This membrane has two distinct types of spikes on its surface, which correspond to the HN and F glycoproteins, respectively. M proteins are located on the interior surface of the envelope, and they link the envelope to the RNP (49).

Route of Transmission of Mumps Virus

Direct contact with saliva or respiratory droplets from the mouth, nose, or throat is the primary means by which mumps is transmitted. It is possible for an infected person to spread the virus by doing any of the following: 1) coughing, sneezing, or talking; 2) sharing items that may have saliva on them, such as water bottles or cups; and 3) participating in close-contact activities with other people, such as playing sports, dancing, or kissing (50). Mumps can be spread by an infected person from a few days before their salivary glands begin to swell to up to five days after the swelling begins (51). The incubation period for the disease is a few days.

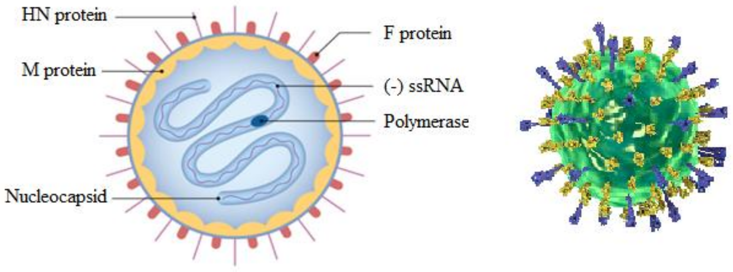

Figure 4. Mumps virus (a- structure, and b- image) [47].

Environmental tolerance of mumps virus

Effects of thermolability and humidity on mumps virus

The research deal with cases of mumps in children younger than 15 years old and variations in the weather in Fukuoka, Japan, from the years 2000 to 2008. It then used time-series analyses to determine how weather variability affected mumps cases, taking into account seasonal variations, inter-annual variations, and temporal variations caused by two large epidemics in 2001 and 2004–2005 (52). The weekly number of mumps cases increased by 75% (95% confidence interval: 4.0-11.1) for every 1°C increase in average temperature, and it increased by 14% (95% confidence interval: 0.5-2.14) for every 1% increase in RM. The percentage increase was highest in the age group 0–4 years old, and it tended to decrease with increasing age as the population got older. The number of reported cases of Mumps in children significantly increased as both the average temperature and the relative humidity rose (51).

Effects of UV radiation on mumps virus

UV light has been shown to be highly effective at deactivating the mumps virus. The exposure of the epidermis to ultraviolet B causes a stimulation of the release of several cytokines into the circulation. It has been demonstrated that exposure to ultraviolet-B radiation raises systemic levels of cytokines such as IL-1 and IL-6, both of which are known to stimulate the replication of viruses (53).

Effects of pH changes on mumps virus

Study demonstrated the stability of mumps virus at pH< 9 and above pH 4 and aggregation was observed at pH> 9 (51).

Spread rate of mumps virus

Studies on the transmission of mumps in households have reported secondary attack rates of anywhere from 33% to 48% among children and adolescents who had no previous history of the disease. The incubation period for mumps typically lasts between 16 and 18 days, but it can last anywhere from 12 to 25 days (53).

Genome size and mutation rate of mumps virus

The genome of the mumps virus is 15.3 Kbp in size and encodes 8 proteins. It is extremely contagious and has been linked to outbreaks of respiratory diseases. The genome of the mumps virus is 15,384 nucleotides long, and the 12 mumps genotypes can be differentiated from one another based on the SH and HN gene sequences (49,54). It is estimated that the F, SH, and HN genes, which are used to differentiate genotypes, undergo genetic mutations at a rate of 0.25, which translates to an average of 10-3 substitutions per site per year. This is considered to be a very low mutation rate for an RNA virus (54).

Seasonal Influenza virus

The structure of the influenza virus exhibits a certain amount of diversity on a cellular level. The virion's constituent particles have a typical round or ovoid shape, and their diameters range anywhere from 80 to 120 nanometers (55). In addition, filamentous forms of the virus can sometimes be observed, and the length of the filamentous structures is frequently greater than 300 nm. On the exterior, it possesses a layer of lipid membrane that is similar to an envelope and was derived from the host cell in which it replicated (56). The envelope is covered with structures that look like spikes that are formed from the glycoproteins HA and NA. It is a viral infection that can spread from person to person and affects the respiratory system. There are three primary groups that can be used to categorize the influenza viruses that can infect humans: A, B, and C. Influenza type A (Figure 5), also known as the "swine flu," is an infection that can be dangerous and lead to widespread outbreaks. Influenza A viruses, which include avian influenza viruses (AIVs), have an envelope, are pleomorphic, and have eight distinct RNA genomic segments with sizes ranging from 890 to 2341 nucleotides each (33).

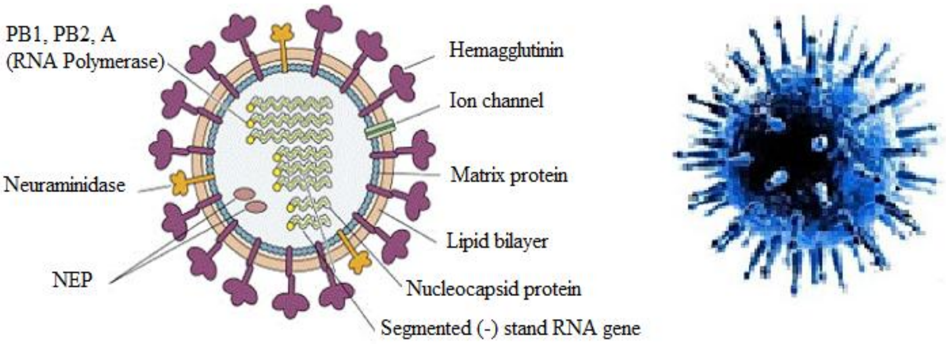

Figure 5: Seasonal flu virus (a- structure, and b- image) (33).

Route of Transmission of Seasonal Influenza virus

It is obviously, there are three ways in which humans can become infected with the influenza virus: 1) by droplets, these particles can settle on the mucous surfaces of the upper respiratory tract, such as the mouth and the nose, and can cause irritation. They can be breathed in, but at more than 10 micrometers in size, they cannot enter the lungs. 2) by droplet nuclei (aerosols), these particles are able to travel all the way down to the lower respiratory tract because they are so small (less than 5 micrometers). In addition, they might settle on the surfaces of the URT. 3) by particles that can be transferred to the mucous membranes of the upper respiratory tract either directly or indirectly through the contact with a contaminated object or person. This type of transmission is referred to as the contact transmission. In a nutshell, the influenza virus can be passed from person to person through direct contact with an infected person, indirect contact with contaminated objects (also known as fomites, such as toys and doorknobs), or through the inhalation of virus-laden aerosols (57).

Environmental Tolerance of Seasonal Influenza Virus

Effects of humidity and temperature on seasonal Influenza virus

According to the research that was conducted, at humidity levels of 23%, between 70 and 77% of the influenza viral particles were able to cause infection one hour after the coughing simulation (58). Only 14% of the virus particles were able to infect the host when the humidity levels were raised to 43%. After being released into the humid air for 15 minutes, the majority of the influenza particles became inactive and were no longer infectious. At high humidity levels, "the virus just falls apart," according to the researcher. The risk of influenza incidence was significantly increased, according to the findings of other studies, when daily temperatures were in the range of 0–5 degrees Celsius and the relative humidity was either low (30%–40%) or high (70%) (59).

Effects UV radiation on seasonal Influenza virus

A new study that was conducted by the Center for Radiological Research at Columbia University Irving Medical Center (CUIMC) demonstrates that continuous low doses of far ultraviolet C (far-UVC) light can kill influenza viruses in the air without causing any damage to human tissue (60).

Effects of pH changes on seasonal Influenza virus

When influenza viruses come into contact with acid-buffering solutions at a pH of 3.5, they are quickly rendered inactive. Researchers demonstrate that a human influenza virus is able to replicate more successfully in human airway cells when using a hemagglutinin that is pH-stable (61).

Spread rate of seasonal Influenza virus

There is a wide range of variation in the percentage of people who become ill from the Influenza, anywhere from 3% to 11%. When it came to influenza A, adults showed an attack rate of 3.4%, while children experienced an attack rate that was 8.8% higher than that of adults. After influenza viruses infect a person's respiratory tract, symptoms typically start to appear one to four days later, but this time frame can range anywhere from one to four days (62).

Genome size and mutation rate of seasonal Influenza virus

Influenza A virus is a member of the family Orthomyxoviridae and produces virions that range in size from approximately 80 to 120 nanometers and have an RNA genome that is approximately 13.5 Kbp in length. The approximate 13,500-letter sequence that is contained within the genome of the influenza virus can be deciphered by performing a full genome sequencing. There are eight different RNA gene segments in the A and B strains of influenza, which are responsible for seasonal epidemics (63).

The influenza virus has an extremely high mutation rate because the influenza viral RNA-polymerase represents the lack of proofreading function. Thus, the integration of faulty nucleotides often occurs during the viral replication process with a rate of 10^{-3} to 10^{-4}, which results in high mutation rates. The influenza virus has a high mutation rate, and this low replicative fidelity contributes to its capacity for rapid evolution.

The SEV has a mutation rate of almost 50 mutations per year. Evolutionary rates for the NS genes of human influenza A and B viruses have been reported in the range of 1.8×10^{-3} to 2.2×10^{-3} and 0.45×10^{-3} to 0.8×10^{-3} nucleotide substitutions/site/year, respectively, on the basis of nucleotide sequencing of circulating viruses. Influenza viruses have 2 to 8 substitutions per 1000 sites per year. Further, the segmented genome enhances the evolutionary speed of the virus by exchange (reassortment) of RNA gene segments between virus strains infecting the same host. Seasonal flu mutates roughly four times as fast as SARS-CoV-2. the fact that the seasonal flu mutates so quickly is precisely why it is able to invade the vaccine (64).

Avian Influenza Virus

Orthomyxoviridae is the family to which the H5N1 highly pathogenic avian influenza (HPAI) virus belongs to (65). It is an enveloped virus that has an eight-segmented, single-stranded RNA genome that has a negative polarity and a total length of approximately 13.5 kilobytes (Figure 6). The genes for polymerase basic 1 (PB1), polymerase basic 2 (PB2), polymerase acidic (PA), hemagglutinin (HA), nucleoprotein (NP), neuraminidase (NA), matrix (M), and nonstructural (NS) are located in each of the eight segments. All of the H5N1 viral genome sequences that have been deposited in the GenBank database up until this point have originated from individual virus isolates (66).

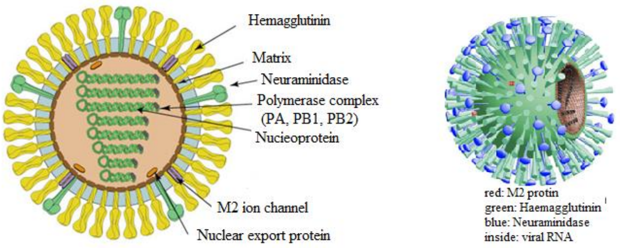

Figure 6: H5N1 virus (a- structure, and b- image) [66].

Environmental Tolerance of H5N1 Virus

Thermolability and effects of humidity on H5N1 virus. The H5N1 avian influenza virus can replicate in almost every part of a bird that has been infected with it. Avian influenza viruses are able to survive in contaminated raw poultry meat, and as a result, they can be passed from person to person through the marketing and distribution of contaminated food products, such as fresh or frozen meat (67).

The H5N1 virus can live in the faeces for at least 35 days when the temperature is 4 degrees Celsius, and at least six days when the temperature is 37 degrees Celsius. It has also been demonstrated that the virus is able to survive on surfaces for a number of weeks at ambient temperatures. The virus seems to flourish at lower temperatures, with low humidity, in the absence of sunlight, and on particular surfaces, such as glass and steel (68). The virus may have a half-life of up to two months in the soil and in the feces of chickens. The H5N1 virus loses its ability to cause infection after 30 minutes at 56 °C and after 1 day at 28 °C; however, it is able to survive at 4 °C for more than 100 days (69).

There is a negative correlation between temperature and the persistence of AIV H5N1. At a temperature of 4 degrees Celsius, the virus was able to live for more than a hundred days; however, it was rendered inactive after 24 hours at 28 degrees Celsius and after 30 minutes at 56 degrees Celsius (70,71).

Infections with H5N1 happen with the greatest frequency in a humidity range of 60–70%; in terms of atmospheric pressure, the concentration of H5N1 cases was at 990–1020 kPa (72).

Effects of UV radiation on H5N1 virus

In the past, it has not been demonstrated that ultraviolet radiation can deactivate H5N1 viruses in a timely manner. This is due to the fact that data have demonstrated that an exposure to a UV source for 45 minutes is not sufficient for the complete deactivation of HPAI strain A. Even after being exposed to UV light for a period of four hours, the virus' infectious potential was not entirely eliminated. Other contributing factors in UV-mediated viral destruction include the distance from the source of light as well as the shallowness of the exposed suspension. Because of this, UV light is only effective at killing microbes that are present in the air or on the surface of the material (72,73).

Effects of pH changes on H5N1 virus

It was observed that the H5N1 virus lost its viability after being exposed to pH 1, 3, 11, and 13 for a period of six hours, whereas it maintained its viability at pH 7 throughout all contact times (6, 12, 18, and 24 hours). It was at pH 5 for 18 hours before it became inactive [74], during which time it had maintained its virulence. At a pH of 9, the virus can maintain its infectious potential for more than 24 hours. According to the findings of another study, the H5N1 virus lost its infectious potential at pH levels that were lower than 5, but it remained viable after 18 hours when the pH was 5.

However, the virus maintained its infectivity at pH 5 (18 hours), pH 7 (more than 24 hours), and pH 9 (more than 48 hours). Acidic pH (1-3) and basic pH (11–13) can kill the virus after 6 hours of contact time; however, basic pH (11–13) and acidic pH (11–13) can also kill the virus (71).

This virus does not infect humans easily, and spread from person to person appears to be unusual.

Genome size and mutation rate of H5N1 virus

The genome of H5N1 is an RNA virus with a single strand and a negative polarity. It is about 13.5 kb in length in its entirety (74,75).

During the course of a natural spillover infection, H5N1 viruses produce putative human-adapting mutations. Many of these mutations can be found at a frequency of more than 5% within the host. According to the unrestricted version of Fouchier's study, the H5N1 super strain had to go through five different mutations before it could be spread from ferret to ferret through the air. Two of the five mutations are found quite frequently in wild strains of H5N1, whereas a third mutation has only ever been found in H5N1 in a single instance (76).

Human Immune Deficiency (HIV) Virus

Human immunodeficiency virus, also known as HIV, is a virus that targets the immune system of the body. Because HIV virus attacks and destroys CD4 cells, untreated HIV can result in the development of AIDS (acquired immunodeficiency syndrome), a condition that is chronic and may pose a risk to one's life (77).

The mature HIV particle is round, with a diameter of about 100 nm, and it has an outer lipid membrane that serves as its envelope. The genome is made up of two separate strands of single-stranded RNA. It possesses a linear, dimeric, ssRNA+ genome that is 9.75 kilobases in size (78). There are some proteins contained within the envelope that assist the virus in invading the next host cell. The attachment of the virus to the CD4 receptor on the host cell is facilitated by a protein known as gp120. The other protein, known as gp41, is responsible for assisting the virus in penetrating cells and fusing with the cell membrane (gp stands for glycoprotein, which is a protein with a carbohydrate attached; the number, 41 or 120, tells how big the protein is). The viral matrix can be found inside of the envelope. The viral core can be found contained within the matrix. The viral genome and the enzymes that are necessary for the virus to replicate in the host cell are both stored in the core of the virus (Figure 7) (79,80).

Route of Transmission of HIV Virus

HIV can be passed on from one person to another through the transfer of a number of different bodily fluids, including blood, breast milk, sperm, and vaginal secretions. HIV can also be passed on from a mother to her child while she is pregnant or during delivery. People are not likely to become ill from engaging in everyday activities such as kissing, hugging, shaking hands, or sharing personal items, food, or water with one another (81).

Environmental Tolerance of HIV Virus

Thermolability and effects of humidity on HIV virus

HIV is very susceptible to infection when it is outside of the body. It sustains damage very quickly and either stops functioning or perishes. Temperature is one of the factors that can determine whether or not HIV can survive outside of the body; HIV remains active in cold environments, but it cannot survive in hot environments. The virus is rendered incapable of reproduction as a result of the sun's ultraviolet light, which causes it to be damaged. In general, the longer it will take for all of the HIV virus to become inactive, the higher the level of the HIV virus that is present in the fluid (82). The concentration of active viruses in a virus sample is reduced when it is dried.

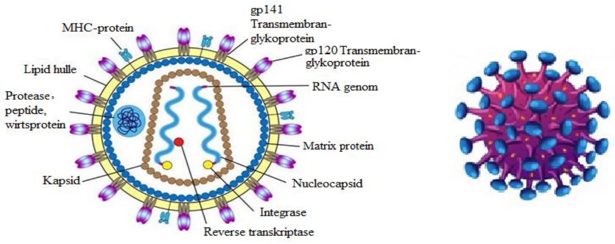

Figure 7: HIV- AIDS virus (a- structure, and b- image) [79].

Effects of UV radiation on HIV virus

It has been demonstrated that certain types of light sources can activate the HIV gene and/or promote viral replication. The most effective activator was UVC light. Even though their effects were less pronounced, UVB, sunlight, and PUVA were all able to activate HIV. On the other hand, neither y-radiation nor UVA triggered HIV activation. There is evidence that exposure to UVC raises levels of HIV-inhibitory cytokines like interleukin, and there is evidence that HIV is resistant to high temperatures in the range of 50–70 degrees Celsius (83).

Effects of pH changes on HIV virus

It has been determined that the HIV-1 particles that are free of cells are extremely sensitive to acidity. They begin to gradually lose their infectious potential between pH 7.4 and 6.0, but this process becomes irreversible below pH 6.0, and subsequent neutralization is unable to restore lost infectious potential. HIV is able to live in environments with a pH between 7 and 8, but it is rendered inactive when the pH of its surroundings changes by even a small amount (84).

Spread rate of HIV virus

Having receptive anal or vaginal sex with an HIV-positive individual who is acutely infected with the virus carries a greater risk of transmission than receptive vaginal sex, which carries a risk of up to 2% (the equivalent of 1 transmission per 50 exposures). Having receptive anal or vaginal sex with an HIV-positive individual carries a risk of over 20% (the equivalent of 1 transmission per 5 exposures) (85).

Genome size and mutation rated of the HIV virus

The genome of HIV is 9.75 Kbp in size and is monopartite, linear, and dimeric ssRNA+ [86]. HIV is known to have one of the highest mutation rates ever recorded. Our immune systems (and the people who develop vaccines) are simply unable to keep up with the rapid pace at which HIV evolves because the surface molecules of the virus are a constantly moving target. HIV accumulates mutations that change the shape of its surface proteins as it replicates within a single infected individual. This allows HIV to evolve right out from under the antibodies that are produced by the victim's immune system. In a similar manner, as the virus is spread from one individual to another, it undergoes further evolution and transforms into a wide variety of strains, each of which has surface proteins that are uniquely shaped. Researchers in the medical field face a difficult task because there are so many different strains of the virus that are circulating through human populations (86,87). It has been discovered that the HIV-1 virus undergoes mutations at a rate of 104 to 105 mutations/base pair/cycle (m/bp/c), which translates to 0.1–1 mutations per synthesized genome. Therefore, HIV-1 mutates approximately 10,000–100,000 times faster than the genomic DNA of eukaryotic organisms. It is hypothesized that such mutations occur as a result of the inability of viral reverse transcriptase to accurately translate RNA into DNA. This hypothesis is based on the observation that viruses frequently undergo mutations. In point of fact, the rates at which these spontaneous mutations occur are the primary factors that determine the genetic diversity and evolutionary trajectory of RNA viruses (88).

Hepatitis C Virus

Hepatitis C virus is a single-stranded RNA virus that is approximately 9,600 nucleotide bases in length. It is enveloped, has a positive sense, and has a sense of direction. It belongs to the Flaviviridae family and is specifically identified as being of the Hepacivirus genus. The HCV particles have a spherical shape and range in size from 40 to 80 nm in diameter. They have a heterogeneous size distribution. Lipoproteins are frequently found associated with HCV particles that are still whole. The E1 and E2 glycoproteins make up the HCV envelope glycoproteins, and these two glycoproteins are noncovalently associated with one another as a heterodimer (Figure 8) (89). This

E1/E2 glycoprotein heterodimer is encased in the host-derived lipid membrane to create the HCV envelope. Together, these two components form the HCV. In addition to playing a role in endosome-lipid membrane fusion and assembly, the envelope proteins also participate in the binding of host receptors. Comparatively, the length of the E1 protein is approximately 192 amino acids, whereas the length of the E2 protein is approximately 363 amino acids. The HCV capsid, also referred to as the HCV core, is the protein shell that encapsidates and protects the HCV RNA. It is also known as the HCV capsid. The HCV capsid is a spherical structure that varies in size and is heterogeneous. Its diameter is approximately 30 nm. The hepatitis C virus (HCV) capsid is combined with the HCV RNA to form the nucleocapsid (90,91).

Route of Transmission of Hepatitis C Virus

The majority of people who contract HCV do so as a result of receiving parenteral exposures to infectious blood or other body fluids that contain blood. Injection drug use and being born to a mother who is infected with the human cytomegalovirus (HCV) are both potential exposures (92). Sharing personal items that have been contaminated with infectious blood, such as razors or toothbrushes, unregulated tattooing, receiving donated blood, blood products, and organs, and needle-stick injuries in healthcare settings are all additional ways that HCV can be transmitted. Although these methods are less common, HCV can still be transmitted through them. Sex with an HCV-infected person is an inefficient means of transmission, although HIV-infected men who have sex with men have an increased risk (93).

Environmental Tolerance of Hepatitis C virus

Thermolability and effects of humidity on hepatitis C virus

It was discovered that HCVcc in culture medium could survive at 37°C and at room temperature (RT, 25±2 °C) for a total of 2 and 16 days, respectively, but that the virus could remain relatively stable at 4°C for at least 6 weeks without suffering a significant loss of infectivity. HCVcc was sensitive to heat and became inactive in the culture medium in 8 minutes when incubated at 60 degrees Celsius and 4 minutes when incubated at 65 degrees Celsius. However, in order to eradicate HCVc infectivity it took forty minutes when the temperature was set to 56 degrees Celsius (94,95). The level of relative humidity and temperature had a negative correlation with HCV survival. When there is a decrease in the relative humidity, HCV has a greater chance of surviving (95).

Effects of UV radiation on the hepatitis C virus

Because the transfection of viral particle-associated RNA into permissive cells did not initiate RNA replication, it was observed that heat and UV treatment of HCV particles caused direct damage to the viral genome. In addition, the HCV envelope was damaged as a result of thermal incubation at 80 degrees Celsius, which made the viral capsid more susceptible to proteolytic digestion.

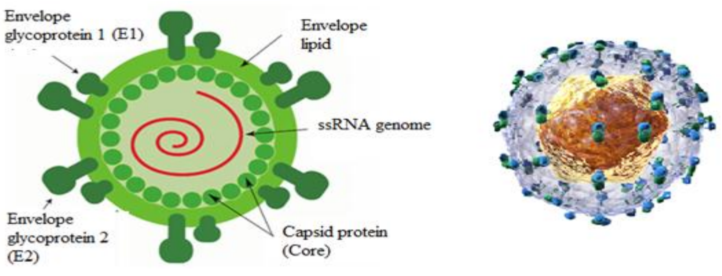

Figure 8: HCV virus (a- structure, and b- image) (89).

Irradiating HCVcc with UV light (wavelength = 253.7 nm) at an intensity of 450 µm/cm for two minutes was all that was necessary to successfully inactivate the virus (96).

Effects of pH changes on the hepatitis C virus

The viral envelope glycoproteins undergo a conformational change when they are subjected to an acidic pH, which results in the premature exposure of the fusion peptide and a reduction in the infectious potential of the virus (97).

Spread rate of the hepatitis C virus

The virus had a half-life that ranged from more than 7 days when the relative humidity was low and the temperature was 5 degrees Celsius to approximately 2 hours when the relative humidity was extremely high and the temperature was 35 degrees Celsius (98).

Genome size and mutation rate of the hepatitis C virus

The hepatitis C virus is an enveloped virus that has a single-stranded, positive-sense RNA genome that is less than 9.6 kbp in size [98]. The mutation rate of HCV, which is estimated to be 10-4 substitutions per site and round of replication, is among the highest for RNA viruses, including retroviruses, and it would appear to be high enough to generate all of the genetic variation that can be found in this virus (>15,000 spontaneous mutations for > 90% genome) (98).

Polio Virus

The polio virus, which is the infectious agent that causes poliomyelitis, is a serotype of the species Enterovirus C, which belongs to the family Picornaviridae. Poliomyelitis is also known as polio. There are three different types of POV virus, designated as types 1, 2, and 3. The RNA genome and the protein capsid are the two components that make up POV. A single-stranded, positive-sense RNA (+ssRNA) genom that is approximately 7500 nucleotides in length makes up the genome. The diameter of the viral particle is approximately 30 nm, and it has icosahedral symmetry. The poliovirus is considered by many to be the simplest and most significant virus. This is due to the fact that its genome is relatively small, and its composition consists of nothing but RNA and an icosahedral protein coat that encapsulates the virus (99,100). (Figure 9).

Route of Transmission of Polio Virus

The poliovirus is extremely contagious and spreads from individual to individual through close contact. It is transmitted through direct contact with an infected person's feces (poop) or the droplets produced by a sneeze, cough, or other respiratory secretion; you can become infected if you come into contact with the stool or droplets from an infected person on your hands and then put them in your mouth (101). Only human beings can become infected. A person who has been infected with the virus can pass it on to others both immediately before and up to two weeks after the onset of symptoms. The virus can survive in the feces of an infected person for several weeks at a time. It is possible for people who do not have symptoms to pass the virus on to others and cause illness in those people; the virus has been shown to be present in soil, sewage, and infected water (102).

Environmental Tolerance of Polio Virus

Thermolability and effects of humidity on polio virus

At temperatures around room temperature, the poliovirus can live for a few weeks. POV is resistant to being eliminate by typical disinfectants used in laboratories, such as alcohol. The virus can be quickly eradicated by being exposed to temperatures of at least 50 degrees Celsius, being autoclaved, or being burned. Formaldehyde in dilute solutions are sufficient to deactivate it in a short amount of time. As a disinfectant, it is recommended to use a bleach solution of 0.5%. The poliovirus is most successful in conditions with relative humidity levels that are higher than 85% (103).

Effects of pH changes on polio virus

Poliovirus is resistant to an extensive pH range, ranging from 3 to 9, and it is able to traverse the digestive tract while maintaining viability at the more acidic pH found in the stomach (104).

Effects of UV radiation on polio virus

Pulsed ultraviolet light treatment, which represents an alternative to continuous-wave ultraviolet light treatment, was shown to be effective in the inactivation of both the poliovirus and the adenovirus (105).

Spread rate of polio virus

A person who has been infected with the polio virus can pass it on to others both immediately before and up to two weeks after the onset of symptoms. The virus can survive in the feces of an infected person for several weeks at a time. In unsanitary conditions, it has the potential to contaminate both food and water.

The poliovirus is highly infectious; the seroconversion rate among susceptible household contacts of children is close to 100 percent, while the seroconversion rate among susceptible household contacts of adults is greater than 90 percent (106).

Genome size and mutation rate of polio virus

The poliovirus is considered to be the archetypal picornavirus. It is a non-enveloped RNA virus with a positive polarity and a genome that is approximately 7.5 kilobases in size. The length of the genome of the poliovirus 1 (PV1) strain 558 is 7,445 nucleotides (107). The poliovirus has an exceptionally high potential for both the rate of mutation and the rate of replication. It's possible that polio virus evolved through a process involving the generation of point mutations, followed by genetic drift and selection (108). According to the findings of several different studies, the mutation rate of polioviruses is approximately 3 x 10-2 mutations/synonymous site/year in the gene that codes for a viral protein. When there is insufficient coverage in a community, the vaccine virus may be able to circulate, mutate, and over the course of 12 to 18 months, cause paralysis (109). This is an extremely rare occurrence, but it can happen when there is insufficient coverage.

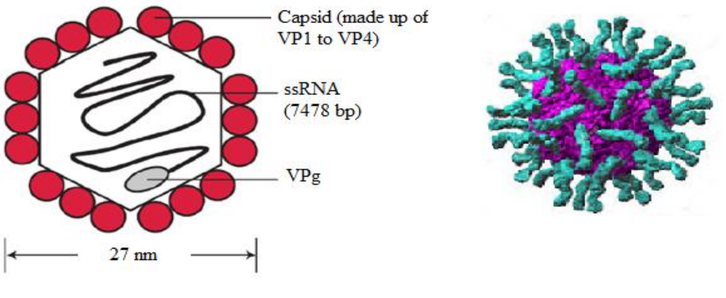

Figure 9: Polio virus (a- structure, and b- image) (99).

Discussions

Route of transmission.

Almost all RNA viruses are transmitted by ingestion, inhalation, or direct contact; just two are transmitted by vectors, (6). COVID-19 (SARS-CoV-2), MEV, MUV, and SEV viruses are transmitted mainly through inhalation of airborne droplets generated by breathing, sneezing or coughing. H5N1 spreads through contact with the virus-infected live or dead birds (the transmission from birds to humans is infrequent). HCV and HIV are transmitted through the exchange of various bodily fluids (blood, semen, and vaginal secretions). POV is transmitted by contamination with the feces of an infected person, it enters the body through the mouth. Mostly viruses that are transmitted through the respiratory system are the most likely candidates to cause a pandemic, which is expected to turn into an endemic epidemic over time.

Thermolability.

Most of the RNA viruses under study are active at temperatures between (18-25°C) and are killed by high heat. SEV and H5N1 are active at low temperatures (0–5°C) and (4–10°C), respectively. SARS-CoV-2 is characterized by being heat tolerant in the range (of 4-60°C), and this increases its ability to spread rapidly. The heat tolerance of the viruses under study can be arranged in the following order, Table 1: SARS-CoV-2> MEV> MUV> H5N1> SEV= HIV> HCV> POV

Relative Humidity.

There is no clear relationship between the studied RNA viruses' activity and the moisture level, where SARS-CoV-2 is effective in dry conditions and stable at RM of 40-50%. MEV, SEV, and HCV is effective at low levels of RH. MUV, H5N1, and Polio viruses are active at a high humidity level. drying lower the viral concentration of HIV active virus. There is an opinion that at high RH, evaporation from exhaled particles is limited, respiratory droplets settle out of the air, and transmission is blocked, Ultraviolet light. Ultraviolet light has been shown to be an effective method for disinfecting surfaces contaminated with most types of RNA viruses found on personal items, and low doses of UVC light can kill airborne viruses without harming human tissue.

pH or Acidity.

The RNA viruses under study showed their ability to infect at a very low or very high pH, and they offer stability at pH values that range approximately between (4-9). AIDS virus showed survives best at a pH of ~7 and becomes inactive when the environment is even just a little more or less acidic. SARS-CoV-2 could be stable in the broadest range of pH (2–13) compared to other studying viruses. The acidity tolerance of the viruses under study can be arranged according to the following order, Table 1: SARS-CoV-2> POV> MEV> SEV= H5N1= HCV> MUV> HIV

Speed rate of transmission:

From Table 1, it can be seen that the speed of spread of viruses under study among humans is as follows: POV> SARS-CoV-2> MEV> MUV> SEV> HIV> HCV> H5N1R0: Calculating a virus' R0 is helpful early on in an outbreak because a number higher than 1 means it is capable of causing an epidemic. Some researchers have considered the new coronavirus variants are as contagious as the measles virus (R0= 12-18), according to a measure of infectiousness known as "R0." Others considered 5 versions of the Omicron coronavirus variant to be more transmissible than measles, or that it has a basic reproduction number, or "R-naught" (R0= 18.6), [37]. The rest of the viruses under study have R0 similar in values, except for the MUV and POV

Table 1. Summary of modes of transmission, environmental tolerability and mutation rate of COVID 19, measles, seasonal influenza, mumps, avian influenza, HIV/AIDS, hepatitis C, and polio viruses.

RNA virus	**SARS-CoV-2**	**MEV**	**MUV**	**SEV**	**H5N1**	**HIV**	**HCV**	**POV**
Rout of transmission	- Direct contact, through droplets generated by breath. sneezing, coughing, - Indirect contact, through a transfer of the virus from contaminated fomites to the mouth, nose, or eyes)	- Infected people can spread measles through coughing and sneezing - Can live for up to 2 hours in an airspace - Animals do not get or spread measles	- By direct contact with respiratory droplets from the mouth, nose, or throat - Can be spread from a few days before salivary glands begin to swell up to 5 days after the swelling begins	- By direct contact with infected individuals; by contact with contaminated objects (called fomites, such as toys, doorknobs	-All infection cases in people have been associated with close contact with infected live or dead birds, with H5N1 - The disease cannot be spread through thoroughly cooked food	- Via the exchange of a variety of body fluids such as blood, breast milk, semen, and vaginal secretions - From a mother to her child during pregnancy - Cannot infect through (kissing, hugging,)	- By blood and occurs primarily through injecting drug use, and less frequently through sex with an infected partner, occupational exposure, and maternal-fetal transmission	- Spreads through person-to-person contact. - It lives in the throat and intestines. - Only infects people. It enters the body through the mouth - Lives in an infected person's feces for many weeks
Environmental tolerance								
Thermolability	4-60°C (stable at 22-25°C) - Decayed with increased temp.	- Not active in hot or cold temp., (stable at 18–20°C)	- Rapidly inactivated by heat.	- Active in the low temp. (0-5°C)	- Active in the cold (4-10°C) specially 4°C. Killed by heat	- Active in the cold - Killed by heat	- Active in room temp. 25 ± 2°C	- Active at room temp. 25°C
Relative Humidity (RH)	- Active in cold and dry condit. - Stable at RH of (40-50%). Decayed at RH>50%	- Active at low relative humidity (RH)	- Activity increased significantly with increased RH	- Active at low RM 30-40% or high 70% RH	- Active in a humidity range of (60–70%)	- Drying lowers the viral conc. of the active virus	- HCV survives better at low levels of RH	- Survives best at a higher RH of above 85%

UV effect	- UVC is an effective method for disinfecting CoV-2-contamina-ed surfaces	- UV radiation can kill the measles virus easily	- Rapidly inactivated by UV light	- Low doses of far UVC light can kill airborne flu viruses without harming tissues	- Only microbes on the surface of the material and in the air are killed by UV light	- UV light in sunshine damages the HIV virus	- UVC light (λ= 253.7 nm) with an intensity of 450 W/cm^2 inactivates HCV within 2 min.	- Pulsed UV-light treatment proved successful in the inactivation of POV
pH effect	- Stable at pH 6.0 (4 or 33°C) - With pH (2-13), the virus is viable for ~6 days	- The stability of MEV is at pH < 5 or >10 (5- 10) - Active at pH between (7-8)	- The study demonstrat-ed the stability of MUV at pH >4 or <9 (5-8)	- Stable at pH (5.0 to 5.3) - SEV viruses are inactivated in acid soln. of pH (3.5)	- H5N1 is inactive at pH < 5 or >10 but remained viable even at pH (5.6 to 6.0)	- HIV survives best at a pH of ~7 and becomes inactive when pH > 7 or < 7	- HCV infectivity was not affected at pH values ranging between (4- 9)	- Stable in a wide range of pH (3–9)
Speed rate	- On average, a COVID-19 case infects 0.48 (95%) close contacts, and with no isolation, 0.62 (95% CI)	- MEV is one of the most conta-gious of all infectious diseases with > 90% attack rates among susceptible close contacts	- Secondary attack rate range from (33-48%) among children and adolescents	- For influenza A, adults exhibited an attack rate of 3.4% and children's attack rate of 8.8%	- Human-to-human transmiss-ion of H5N1 viruses is limited	- A trans-mission risk of up to 2% for receptive vaginal sex and over 20% for receptive anal sex.	- The risk of transmis-sion after a needlestick or sharps injury is 0-10% and 6.1% from the source with HCV RNA	- Spreads among children ~100%, and > 90% adults - Spreads before and up to 2 weeks after symptoms appear

viruses, which has a higher value (R0= 10-12 and 5-7) respectively. The values of R0 for the viruses under study can be arranged according to the following order, Table 1: SARS-CoV-2> MEV> MUV> POV> SEV> H5N1> HIV> HCV

Genome size.

Genome size correlates positively with the studied RNA virus's speed rates of transmission or infectivity, therefore, the highest infectivity and widespread virus is the Coronavirus, which has the largest genome, and the least is the Polio virus, which has the smallest genome, [99,100]. The size of the viruses under study is arranged in the following order, Table 1: SARS-CoV-2> MEV> MUV> SEV= H5N1> HIV> HCV> POV

Mutation rate.

RNA viruses mutate faster than DNA viruses, single-stranded viruses mutate faster than double-strand viruses. RNA viruses are unstable due to their higher mutation rate, and evolve so quickly that their surface molecules are a constantly moving target and our immune systems (and vaccine developers) simply can’t keep up. Generally, mutation frequencies in RNA viruses typically range between 10^{-4} and 10^{-6} per site per replication, (6). The studied viruses have a high mutation rate because of the intrinsic error rate of RNA polymerase and the lack of proofreading mechanisms. Thus, the integration of faulty nucleotides often occurs during the viral replication process with a rate range of 10^{-3} to 10^{-6}, which results in high mutation rates. However, coronaviruses do not mutate and evolve quickly, because they possess a built-in “proofreading” mechanism to prevent mutations as they make copies of themselves while growing and multiplying in our cells, which results in a low mutation rate compared to other viruses. On average, the coronavirus accumulates about two changes per month in its genome. Most changes don't affect the virus's behavior, but a few may change the disease's transmissibility or severity (3). The mutational rate of SARS-CoV-2 gives hope for the potential development of effective long-lasting vaccines against the virus. Cuevas and colleagues quantify the HIV-1 genome-wide rate of spontaneous mutation in DNA sequences from peripheral blood mononuclear cells. For HIV-1. On the other hand it was revealed a mutation rate of 4×10^{-3} per base per cell for HIV-1genome (98). the highest reported mutation rate for any biological entity. When they sequenced plasma-derived HIV-1, they found the mutation rate was 44 times lower: “indicating that a large fraction of viral genomes is lethally mutated and fail to reach the plasma, and the authors showed that the viral reverse transcriptase accounts for only 2% of mutations, with the remaining 98% of viral genetic variation resulting from editing by host cytidine deaminases. Researchers found that low-level cytidine deaminase editing is “more abundant in rapid progressors, suggesting that failure of cytidine deaminase to inactivate the virus by hypermutation may promote HIV-1 intrapatient diversity and pathogenesis.” The mutation values for the rest of the studied virus’s range between the mutation values of COVID-19 and HIV viruses, according to the following sequence, Table 1: SARS-CoV-2> SEV= H5N1> HUV> POV> MEV> HCV> HIV

Conclusion

According to research on the relationships between some RNA viruses' susceptibility to infection and their transmission patterns, environmental tolerance, and rate of mutation, there is a direct correlation between their susceptibility to infection and heat tolerance, an inverse relationship between their susceptibility to infection and high humidity, a positive relationship between their susceptibility to infection and virus size, and an insignificant relationship between their susceptibility to infection and virus size. Inverse relationships exist between virus genome size and mutation rate, virus genome size and high humidity tolerance, virus genome size and spread rate, virus genome size and heat tolerance, virus genome size and mutation rate, virus genome size and high humidity tolerance, virus genome size and high humidity tolerance, and virus genome size and high humidity tolerance. Because RNA-polymerase stands for the absence of a proofreading function, the little virus mutates more frequently and more swiftly than the large virus and multiplies quickly. Therefore, a low rate of integration of defective nucleotides occurs during viral replication, and this low replicative fidelity aids in the virus's ability to evolve quickly. It cannot be distinguished by immune cells in the body, and the body does not produce immunity against it because the receptors that allow it to enter cells vary. It takes a while to spread. These characteristics make the viruses more likely to spread locally (they are endemic). The recipients that the large virus inserts into the cell do not change because they have a built-in "proofreading" mechanism to prevent mutations as they make copies of themselves while growing and multiplying in our cells. The large virus's multiplication inside the cell is low, so it mutates more slowly than the large virus. As a result, it may be possible to develop immunity to it. These characteristics make certain viruses candidates to spread globally and cause pandemics.

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