

Chapter 1

Introduction to Virus Outbreaks



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Abstract Human health is significantly affected time and again by emerging and re-emerging incidences of viral infections in spite of exceptional progress in the field of biomedical research. A great example of the same being recent pandemic of COVID 19 across the globe. Even reappearing outbreaks of chikungunya and dengue in tropical and sub-tropical regions, Zika virus outbreak in America and SARS, MERS and influenza A epidemics indicate the same. The natural reservoirs of human viruses are typically farm animals and occasionally the wild animals as well as arthropods. The key to understanding this emergence and re-emergence of these pathogenic viruses lies in the complex ‘host-pathogen-environment’ interaction. Selective pressure is put on these reservoirs due to changing human habits, increasing population density, stress on mother nature, poor sanitation and changing climate. For protection from these infections, new approaches like consumption of thoroughly cooked meat and animal products only are the most promising control measures. Although substantial progress has been made in human immunodeficiency virus and hepatitis virus control, the arbitrary disposition of evolving viruses and the occasional outbreaks critically restrain the prevention and control process.

Keywords Virus outbreak · Epidemic · Pandemic · Viral transmission

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1.1 Introduction

Human health is significantly affected time and again by emerging and re-emerging incidences of viral infections (Krause 1992). The emerging viruses which have just been introduced lately in a population are called novel etiological agents. Since the finding of human immunodeficiency virus (HIV) in 1980s began a global recognition of importance of more research in this field of novel pathogens. New outbreaks of infections in the past several decades has aided in detection of multiple varieties of very infectious viruses (Feng et al. 2008; Padgett et al. 1971).

Viruses are non-cellular pathogens in nature. They are all strict parasites occurring within a cell because their source of energy and basic composition is the host cell. The consequences of viral infection in humans and animals can vary from unapparent (no disease) to serious diseases and even death. While some cause acute infections in host body for a few days or weeks other viruses may result in a lifelong infection. A distinct characteristic of viruses which increases their perseverance is their ability to adapt quickly to changing environment and subsequently evolve (Gardner et al. 1971).

The effects of viral infection on cells individually can be seen in terms of changed cell functions and structures, even the overall health and fitness of the affected being is influenced noticeably. When a virus comes in contact with a cell one of these four outcome is likely:

- *Permissive (/Productive) infection*: In this case, viral proteins and nucleic acids are synthesised and virions are released.
- *Non-permissive infection*. The cell resists the infection, completely.
- *Abortive or non-productive infection*. The virus is able to enter the cell but before anything is synthesised the replication becomes irreversibly blocked at some step.
- *Latent infection*. It is the condition when although a viral genetic code is present in the cell, but only a few or no viral proteins are synthesised. It means that the virus can actively replicate under suitable conditions.

1.2 Types of Viral Diseases

Disease is caused as a consequence of tissue or organ injury. Viral infections can cause diseases which are acute, chronic or latent (Boldogh et al. 1996).

Acute diseases have a sudden onset which can last between a few days to several months, and the pathogen may be cleared, controlled or even cause the host death. Common cold is one of the many examples of acute viral infections.

Chronic diseases have a steady development and the treatment of can take years or a lifetime altogether. In some cases these infections can even cause death of the host but that is not always the case. Because of longer time taken for resolution, these infections are also called persistent infections. The virus is continuously produced and shed by the host. Hepatitis C virus (HCV), hepatitis B virus (HBV), and human

immunodeficiency virus (HIV) have a tendency to cause chronic infections in human beings.

Latent diseases is caused when viral genome is maintained without production of detectable virus. Chickenpox/Shingles virus is an example of virus that causing latent disease. Chickenpox is usually a mild infection, with characteristic pustules (like blisters), that are cured in around a week. Although, even long after the pustules have disappeared physically the virus remains in the body. These virus genomes sustain in the neurons silently, for decades. When this virus exits latency stage by producing blister-like lesions by travelling down to the skin via neuron network, a throbbing and weakening disease called shingles occurs in adults. These lesions in turn contain infectious virus capable of giving chickenpox to another host with low immunity (Mandell et al. 2009).

1.3 Origin of Viral Outbreaks

Viruses cannot replicate outside of living cells like most bacteria. About 80% of all viruses, known to cause human infections, mostly exist naturally in ‘reservoirs’ that are non-human like farm animals and poultry birds while some continue to be found in wild animals and some arthropods (Cleaveland et al. 2011). The zoonotic pathogens account for almost 60% of the ‘known’ infectious agents and 75% of ‘novel’ pathogens in human beings (Taylor et al. 2013; Woolhouse and Gowtage-Sequeria 2005; Kilpatrick and Randolph 2012). We have, however, limited understanding of these zoonosis and the varieties of such virus in known reservoirs. The information we have on domesticated animals maintaining a bunch of these viruses is little as well as data on wild animals hosting a number of viruses is insufficient (Cleaveland et al. 2007). The new influenza virus strains, human coronavirus (h) CoV, Hendra virus, Nipah virus and Zika virus are all associated with zoonotic transmission. Even the recent deadly outbreak of MERS-CoV was thought to be zoonotic as it was found to be genetically closer to bat CoV than any other hCoV (Corman et al. 2012). Data currently shows that bats harbour substantial varieties of CoVs that range from species-to-species and region-to-region (Anderson and Tong 2010).

In nature the existence of a virus is dependent on the perpetuation of a sequent infections, which is a chain of transmission of virus; the incidence of a disease is neither essential nor even beneficial. Although, clinical cases in fact give rise to more infectious viruses than unapparent cases, but the latter produces a larger number and, because there is no restriction on host movement it provides a great opportunity for viral dissemination. The survival of viruses in humans occurs through three different patterns according to epidemiologists, which is differentiated based on the use of reservoirs: acute self-limiting infections with no reservoir, persistent infections with a reservoir in humans, and involvement of an animal reservoir. A basic method is used by all viruses for survival (Table 1.1), and if by chance this system is obstructed for instance by abrupt fall in host species population because of some other infection of a weather event, then substitute pattern may be deployed (Klimpel 1996).

Table 1.1 Different patterns of survival used by viruses in nature

Infection pattern	Mechanism of survival	Examples of viruses
Acute self-limiting infection (lifelong immunity)	Reservoir absent; large population is needed with continuous chain of transmission	Polio, hepatitis A, enteroviruses, dengue, measles, mumps, rubella
Acute self-limiting infection (short-span immunity)	Reservoir absent; reinfection occurs, small-sized population needed	Rotavirus, influenza, coronaviruses, rhinoviruses, respiratory syncytial virus
Persistent infection (intermittent replication +/- shedding)	Reservoir in humans; lifelong source of virus- infected individuals	Varicella-zoster, Herpes simplex, other herpesviruses
Persistent infection (continuous replication)	Human reservoir; lifelong source of virus- infected individuals	Human immunodeficiency virus, Hepatitis-B, C, human papillomavirus
Zoonosis (human-human spread absent)	Endemic infection in animal reservoir and transmission to humans	Most arboviruses except dengue, yellow fever (urban cycle). Avian influenza, rabies, Hendra
Zoonosis (significant human-human spread)	Endemic infection in animal reservoir and transmission to and between humans	Marburg/Ebola, Hantaan, Nipah, dengue, yellow fever (urban cycle)

It is necessary to understand the clinical features as well as the disease pattern of an infection for structuring and actualizing its control programs. For instance, the reason behind successful eradication of smallpox was the understanding that an acute self-limiting infection where bulk of individuals show clinical symptoms was caused by a variola virus which had no animal reservoirs. A large portion of viral infections in humans fall into the acute self-limiting infection category. It is crucial to have optimal transmission and the viruses causing systemic infections with lifelong immunity need large dense populations to survive. While the ones causing surface infections with short- span immunity can even survive in smaller populations, and this ability to persist in limited population can be built up by the antigenic drift (Burrell et al. 2017).

1.3.1 Virus Infection Transmission

For transmission cycle to occur, the entry of virus into the body, its replication inside and shedding with successive spread to another host, is required. Viral transmission (Fig. 1.1) can take place horizontally or vertically, although mostly horizontal transmission occurs which is in between the individual at risk. Unlike most bacteria, different viruses use specified pathways for transmission. This selection is based on a number of factors like physical characteristics of the virus, course of shedding and other features of the pathogenic process. These ways are basically described by separate techniques used to enter the upper epithelial body lining by the viruses.

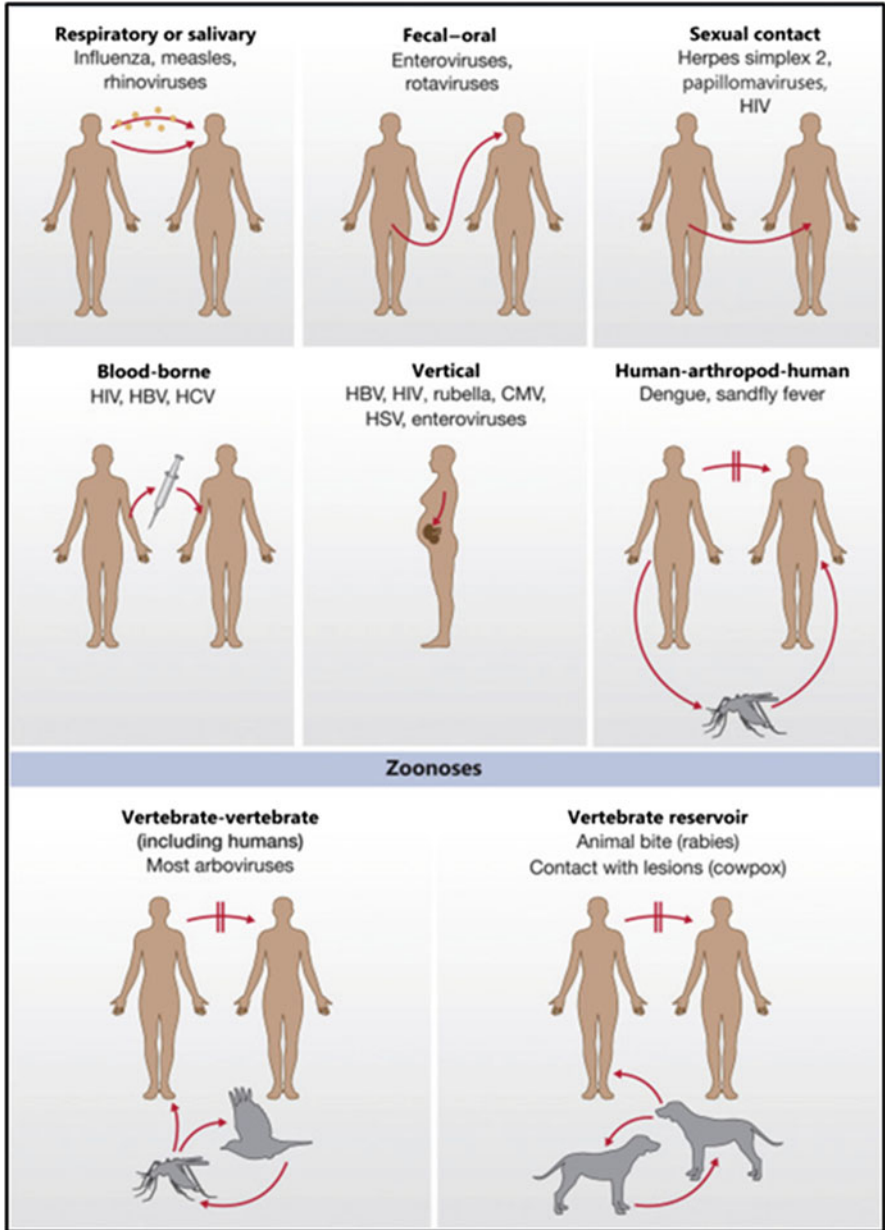


Fig. 1.1 Modes of transmission of human viral diseases. (Source: Burell et al., *Epidemiology of Viral Infections*. Fenner and White's *Medical Virology*, 2017)

Virus shedding takes place from one of the body surfaces or opening which was also included in the virus entry. The same body part is concerned with entry and exit of the virus in case of limited infections, but a considerable variation in manner of shedding is seen in case of generalized infections while few viruses shed from various sites like hepatitis B virus, HIV, and cytomegalovirus in semen, cervical secretions, milk, and saliva (Baron et al. 1996).

For transmission, the quantity of virus shed in secretion and excretion, is of great significance. Without transference of huge proportion of infected materials, little concentrations are almost inapplicable. However when the concentration of virus is very high, even tiny quantities like less than 1 μL , can transmit the infection.

1.3.2 Respiratory Route

Various viruses causing respiratory tract diseases are often shed as aerosols in the saliva or mucus from respiratory tract while talking, coughing and sneezing. Viruses are additionally shed from the respiratory tract in a few widespread diseases like measles, rubella and chickenpox. Some viruses are even shed into the oral cavity from infected lungs, nasal mucosa or salivary glands, and transmitted during salivary exchange. Aerosols are highly infectious in the preliminary stage of infection when the virus replication is at its pinnacle. While some individuals tend to be more infectious than the rest, it varies from person to person. The spread by respiratory route has three components:

- Small-droplets (<10 μm diameter): These cause quick sudden outbreaks and spread to far off contacts (>1.8 m). The droplets are likely settle in lower respiratory tract.
- Large-droplets (10 to 100 μm diameter): These cause slow onset, sporadic outbreaks with no huddling. The droplets settle on ground quickly so, transmission takes place between closer contacts (<~0.9 m). The particles rest in the upper airway.
- Fomites: When certain objects like napkins, medical devices and articles at home called fomites are contaminated with infectious aerosols and body fluids, they spread infection among close contacts of the host, often with poor hygiene (Box 1.1).

Box 1.1 Respiratory Tract Transmission Routes

Respiratory transmission is an effective and fast way to cause infection in large number of host contacts and to spread virus across the globe.

There are two methods:

(continued)

Box 1.1 (continued)

1. *Breathing in aerosols.* When breathing at rest, human beings usually filter about 600 L of air per hour.

Aerosols are formed while coughing and sneezing, lesser by talking.

Large droplets settle on the floor, while small droplets usually spread along but once dry the virus is inactivated; and therefore the atmosphere would not be infective for long.

The seasonality of these infections is affected significantly by increase in virus survival in cool temperature, variation in social activities like school vacations etc.

2. *Touching fomites.* Infection spreads through napkins, hands, surfaces and objects that have been contaminated by aerosols. Act of shaking individual's hands, touching fomites etc. gives a clear route for virus to reach another person's nose and mouth and enter their body (Bloom-Feshbach et al. 2013).

1.3.3 Gastrointestinal Route

Viruses of enteric system are shed via vomiting and faeces from the body. Larger the quantity of fluid released more is the environment contaminated. These kind of viruses are harder and more resilient, so they tend to survive outside the body for longer even in harsh conditions, than the respiratory viruses. Two transmission patterns are seen in this route:

1. Point-source outbreak: It usually happens when large number of people actually consume the contaminated water or food in events like parties etc. The sources are usually raw salad, shellfishes, or due to consuming unsafe water contaminated with sewage, and
2. Person-to-person spread: It usually occurs via faeco-oral route, gradually in homes which lack proper toilets, running water and other hygiene facilities that are more often poor and lack education.

1.3.4 Cutaneous Route

Healthy skin provides an inviolable hurdle for virus entry to the body. Virus shedding occurs at an almost insignificant rate from the skin so much so that even for people with blood-borne diseases their unbroken skin poses no threat of infection to their close contacts. Nonetheless, even minute skin scraping is a great opportunity for viral entry by direct contact. Infected blood from blood borne infections like hepatitis B can shed via scrapped skin and spread horizontally between individuals,

specifically those living in overcrowded, low socio-economic settings with a history of skin diseases. Although, these kind of infections are spread usually by various other ways like introduction of infected blood parenterally (through injections, sharing needles or blood transfusion).

Some poxviruses are also spread to humans from animals or vice versa due to contact with the skin lesions. While these lesions or rashes are formed in many diseases, viruses are not usually shed in infections like measles or picornavirus, flavivirus etc., but in herpesvirus infection, vesicular lesions are formed where virus is plenty in the lesion fluid. Even in this case, shedding by saliva and aerosols is more significant for transmission of the disease. A very important virus spread by cutaneous route is that of rabies virus transmission through bite of an infected animal on the skin.

1.3.5 Genitourinary Route

The genital secretions of found in semen and vagina contain various viruses. Transmission of infection by sexual route through mucosal contact is very effective as the virus remains moist and there is no need to survive outside the body for long. The spread by this route however takes more time than respiratory route because association with several contacts is slow. Research from HIV has demonstrated that transmission is increased when multiple consecutive partners are involved, when multiple consecutive mucosal abrasion are present, other infections also occur and the involved male is non circumcised. Some examples are HIV, HBV and herpes simplex type 2.

1.3.6 Blood-Borne Route

It is a significant route of disease spread both within an individual as well as between two individuals. Diseases like Hepatitis B, C, and D viruses and HIV all disseminated by blood transfusion, but now due to thorough testing of donated blood the risk has been significantly reduced. Transmission of blood borne infections due to shared needles and injecting paraphernalia is an issue amongst intravenous drug users in most of the countries today. Blood is also the most typical way how arthropods like sandflies, ticks and mosquitoes pick up viruses while having a blood meal from a host. Rarely, few arthropods like horseflies etc. even passively pass on viruses by interspersed blood meals from various hosts with contaminated mouth parts.

1.3.7 Ophthalmic Route

A common route of virus introduction into the body is through the eye by touching dirty fingers, using contaminated swimming pools, from non-sterilized ophthalmic articles or even aerosol droplets.

1.3.8 Milk-Borne Route

A number of viruses that are secreted in milk are transmitted to new born infants. There is also an added risk of introduction of an infectious agent to the infant while breastfeeding from an infected mother. Although, chances of this is a lot less than the risk of vertical transmission of an infection while childbirth. In countries and communities where malnutrition or infectious diseases are a common cause of infant mortality, breastfeeding may still be prescribed despite the added risk of transmission that it poses.

1.3.9 Vertical Transmission

This is the route of transmission of a virus from an infected mother to her embryo, foetus or the infant. It is a significant route of transmission across generations which enables the survival of virus in nature. There are three conditions where such a transmission occurs:

- Through the amalgamation of viral DNA directly into the germline DNA of the gametes and/or the fertilized egg,
- Through trans-placental transmission during pregnancy, and
- Through peri-natal or post-natal transmission by milk, saliva or other body fluids.

This transmission may cause severe infection, some congenital diseases or abnormalities or even death of the foetus and subsequent abortion in some cases. Vertical transmission establishes the infection into a new generation, in cases of hepatitis B and HIV diseases that are then competent to pass on the infection to subsequent birth cohorts for many more generations. For continuation of several arthropod-borne infections, vertical transmission in the vectors is very important (Knipe and Howley 2013).

1.4 Phases of an Outbreak

Several viruses continuously flow amongst animals, mostly birds. Although these viruses are capable of causing viral outbreaks theoretically, but in *Phase 1* of an outbreak there are no reported human infections caused by these viruses circulating amongst animals (Fig. 1.2).

In *Phase 2*, there is a potential epidemic threat from an animal virus circulating among domesticated or wild animals which has caused an infection in humans.

In *Phase 3*, although still no human-to-human transmission enough to sustain community-level outbreaks has been established but even so an animal or human-animal virus has caused sporadic cases or small clusters of disease in people. However in case of close contact between an infected person and an unprotected caregiver there may occur limited human-to-human transmission. The transmission under such constricted circumstances does not show that the virus has gained a level of transmissibility among humans required to cause an epidemic.

The *Phase 4* is said to begin once community- level outbreaks of human-to-human transmission of an animal or human-animal virus have been verified. A substantial increase in the risk of causing a pandemic is marked by the potential of a virus to cause perpetual disease outbreaks in the community. World Health Organisation should immediately be consulted if any country has verified or suspects such an episode such that the circumstances may be assessed together and a decision can be sought by the country that has been affected to implement a quick containment operation. While this phase stipulates a noteworthy hike in the risk of a pandemic it surely does not mean that a pandemic is certain in the future.

Phase 5 is finally where virus has spread via human-to-human route in at least two countries of one WHO region (Fig. 1.3). Although majority of the countries would not be affected at this time, the announcement of Phase 5 is a powerful indicator that a pandemic is absolute and there is just a short time to work out the organization, communication, and implementation strategy of the planned mitigation measures (World Health Organization 2009).

1.5 Factors Affecting Virus Outbreak

1.5.1 Transmissibility

The physical properties of a virus like the nature and extent of shedding from the viral body, social interaction among hosts etc. significantly affect the transmissibility of the virus. The human-to-human transmission is also enhanced by shedding of large volumes of infectious virions. Specifically in case of respiratory viruses, they are shed in high concentrations in a relatively shorter period of some days, as transmission to close contacts is almost certain when explosive aerosols are

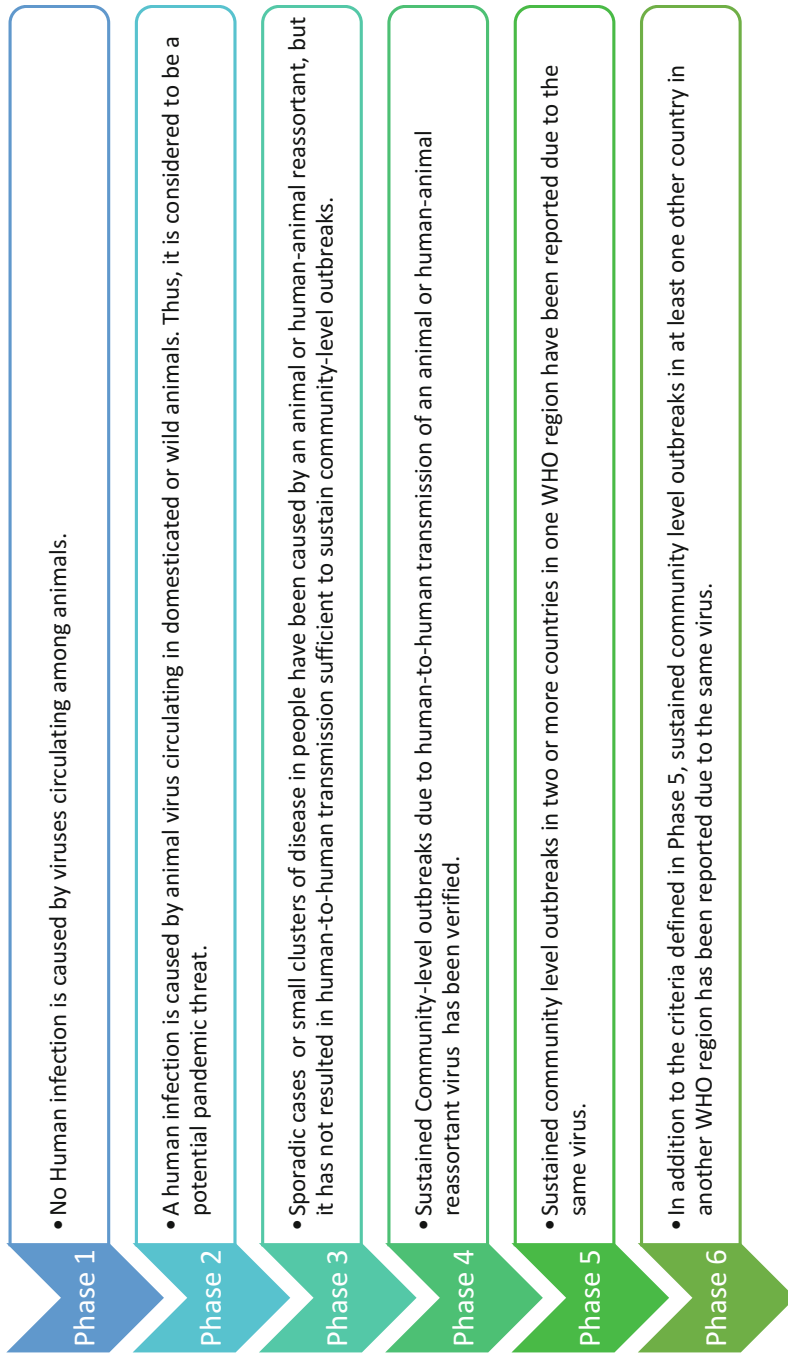


Fig. 1.2 Phases of an outbreak

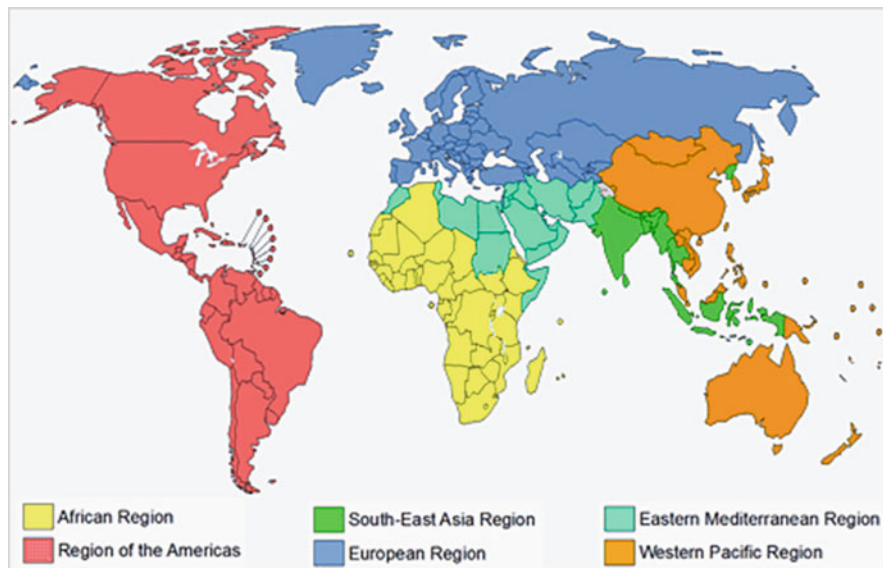


Fig. 1.3 Distribution of the WHO regions

produced while coughing or sneezing. The viral transmissibility of different influenza viruses between humans is determined by their complex use of varied receptors.

Enteric viruses are shed for a prolonged period of time (about a week or more) and released in high numbers in the faecal matter. Viruses also contaminate fomites, water, food and hands. The enveloped respiratory viruses are liable to change during summer or year-round in tropics. While several enteric viruses do not have an envelope and can survive in water, dust or on fomites for several days or even weeks like the hepatitis A and B viruses. However the transmission has slowed down with improved sanitary conditions, socio-economic status and educating the masses, shifting the prime age of acquiring the infections to an older age. Several infections give rise to clinical diseases in older age groups, therefore these improvements might affect the increase in clinical cases absurdly (Box 1.2).

Box 1.2 Impact of Sanitation Measures on Enteric Disease Transmission

In the twentieth century, as the level of hygiene and sanitation advanced in several places around the world, the dissemination of enteric virus infections became more and more inefficient increasing the usual age at which infection previously occurred. Subsequently, many individuals did not possess the acquired immunity for that infection by the time they reached adolescence. For some reason, the typical infection of poliovirus or hepatitis A in adults and

(continued)

Box 1.2 (continued)

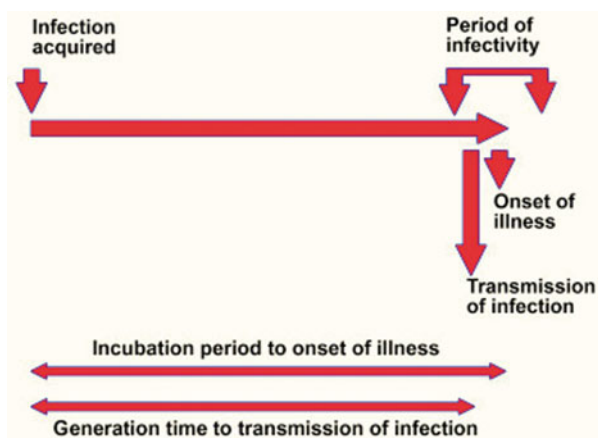
older children apparently cause a *clinical* disease, in comparison to infection at a younger age. The result of these improvements in living standards was an absurd *increase* in clinical diseases. It could be seen most notably in “virgin soil” epidemics that occurred long before the era of vaccinations, in outlying communities without previous viral exposure; where majority of fatalities were seen in adults. Ultimately, the countries having high standards of living started seeing a fall in the total numbers of clinical diseases and infections, when the virus circulation fell even more.

Figure 1.4 describes the various steps involved in progress of a typical acute transient infection. In several diseases like chickenpox and measles the individual becomes contagious a day or so before they become ill themselves. While for zoonotic infections either involving wild or domestic animal reservoirs, there always has to be close contact of humans with the animals or a transmission of virus by arthropod vectors.

1.5.2 Seasonality

The incidence of several viral infections shows a noticeable seasonal variation. The arbovirus infection transmission by mosquito or sand fly vectors occurs during summer season in the temperate regions when vector population is abundant and they are most active. While ticks transmit infectious diseases mostly in the spring or early summer season. It is also interesting to notice the seasonal variation in incidence of infections where humans are the only hosts.

Fig. 1.4 Sequence of infection transmission



The respiratory viral infections are however most prevalent during the winter months and to some extent in autumn or spring in temperate climates. An important characteristic of temperate climates is the annual winter outbreaks of severe respiratory viral infections in infants, even the epidemics of influenza occur mostly during the winter season. During the spring season most of childhood rash diseases that are spread by respiratory route reach the peak.

The seasonality in enteric virus infections actually depend on etiological agents like the incidence of majority of enteric infections is highest during the summers while calciviruses in fact have an irregular seasonal pattern and rotavirus infections occur predominantly during winters. Herpes virus infections are all spread through close contact with infected saliva or other body fluids. The incidence of these kind of infections and even other sexually transmitted diseases all show no seasonal dissimilarity of incidence. In the tropical areas however, summer and winter variation is in fact replaced by the wet and dry seasons that tend to govern the seasonality of diseases. Here the dry season typically witnesses a peak in incidence of measles and chickenpox followed by a swift fall with the beginning of the rainy season, while peak incidences of rhinovirus and influenza infections are noted during that season.

The seasonality mainly depends on biological as well as sociological factors viz. low humidity favours the survival of influenza, vaccinia and measles viruses, rhinoviruses, adenoviruses and polioviruses tend to survive better at high humidity. However, lesser temperatures and aerosols help viruses survive for longer periods. Seasonality also plays a major part in determining the host susceptibility to infections like nasal and oro-pharyngeal mucous lining changes due to smoke, heat or air conditioning. Even the social activities of the host affect the transmission opportunity of respiratory viruses. For instance in Antarctic and Arctic regions, the crowding behaviour into stuffy vehicles, cramped areas and buildings located in temperate regions, greatly enhance the respiratory virus transmission which cold weather alone cannot do. In the locations receiving monsoon rains early in summers, often the movement of people for social gatherings as well as daily life is significantly reduced. While this reduces the chances of getting infection from individuals of other locations, the opportunity of respiratory virus transmission among family members is greatly enhanced. Young children are the most remarkable sources of spreading the viruses in urban settings, because they interact with potential hosts at schools or neighbourhood playtime, and they lack acquired immunity for many infections at that age so they tend to shed considerably larger volumes of viruses than the adults.

1.5.3 Community Size

The viruses which cause acute self-limiting infections need dense and relatively larger at-risk population for their survival. If the prospective supply of vulnerable hosts is finished as a result of acquired immunity to re-infection, there are chances that such viruses might vanish altogether from the population. However, chronic

viruses can survive in a handful of population too by going from generation to generation. The community size critical for virus survival depends on its pattern of transmission as well as the time needed to develop immunity for it.

1.5.4 Host Immunity

Acquired immunity through either an earlier infection or a vaccination is very crucial in the viral disease epidemiology. For general infections the acquired immunity is exhibited primarily by IgG antibodies which is often permanent. Even when there are no repeated sub-clinical infections the immunity is acquired for life like in case of poliomyelitis and measles infections (Evans 1989).

In case of localised respiratory tract infections, the immunity is somewhat short spanned. Many varieties of rhinoviruses and some types of coronaviruses and enteroviruses can cause upper respiratory tract infections. The apparent continuous series of common colds infections in community actually indicate towards a chain of minor epidemics, which are all due to distinct varieties of virus. The antibody IgA present in nasal secretion provides defence against the chances of reinfection. While individuals do possess short spanned specific immunity, the immunity for other types of viruses does not exist. Majority of people catch the cold two to four times each year. For most of the respiratory viruses the span of shedding is short (few days to a week after symptoms appear) but for rhinoviruses this period can last up to 3 weeks even after common symptoms are unapparent. Research has shown that in order to maintain the herd immunity it is essential to have a continuous supply of vulnerable hosts for harbouring novel viral strains so that the diseases is maintained in nature and even repeat sub-clinical infections continue to occur (Fenner 1996).

1.5.5 Persistent Infections

Persistent viral infections amplify the perpetuation mechanisms of viruses even when they do not directly cause a clinical infection. The hosts carrying a persistent infection can shed the virus sporadically or continually in severe cases which in turn may re-establish the virus in a community where a lot of people were born after the last clinical episode of the infection was seen. Herpes virus survives by this mechanism in smaller populations. The disease causation, viral persistence and transmission pattern of viruses are not inevitably linked with each other. Consequently a persistent infections might have adverse effects on associated reservoir hosts but these are effective in continuing the infection chain. However, in case of sclerosing panencephalitis (SSPE) measles virus, the persistent infection in central nervous system is lethal for host but of barely any importance in virus propagation.

1.5.6 Non-Human Reservoirs

As in the case of zoonotic infections, a constant re-establishment of viruses from a non-human reservoir helps the virus in spreading in human population as well as it controls the distribution and the extent of viral infection in the community like arboviruses, rabies virus and hantaviruses. The level of infection in humans is dependent on the prevalence of the infection in the reservoir and the extent of contact with that animal reservoir. While planning for global eradication of any human viral infection it is important to consider the presence and the possible degree of animal reservoir (Beran 1994).

1.5.7 Arthropod Vectors

Transmission by arthropod vectors is one of the most composite transmission modes for viruses environmentally. Arbovirus is the term used to refer to a virus that has a vertebrate host and an intermittent blood feeding arthropod (typically mosquitoes or ticks) in its life cycle. The virus enters into the body of the vector via blood meal of a viremic individual or animal. The virus then replicates inside the gut initially and then moves to the salivary glands (Incubation period- extrinsic) depending on ambient temperature and the kind of virus. When the vector finally bites a vertebrate host to feast on its blood, the virus travels into the host bloodstream along with the salivary secretions of the vector. Additionally, mechanical contamination of vectors' biting parts ("flying pin") may also transmit the virus.

Vectors give viruses a straight opportunity to break the cross-species barrier because the vector will bite many organisms, like reptiles, mammals and even some birds, which are unlikely to ever come in each other's contact naturally. Some mammals or mostly birds are usually the vertebrate reservoir hosts that help in maintaining the virus in the vertebrate-arthropod-vertebrate life cycle. In very rare circumstances only humans are included in this virus maintenance cycle called the enzootic cycle until they voluntarily come in contact with the infected host (Parvez and Parveen 2017).

1.5.8 Hospital Acquired Infection

The spread of infection when an individual is in a hospital or clinic called the nosocomial transmission and "by the hand of the doctor" called iatrogenic transmission is a very important means for virus perpetuation in community. The best example of an iatrogenic and nosocomial infection was the lethal outbreak of Ebola virus in 1976 in Zaire. Other infections which propagate and maintain themselves in the community through hospitals spread are the common respiratory

viruses like influenza, chickenpox and respiratory syncytial virus infections in hospital settings. While infections from Hepatitis B and C viruses, and fewer cases of HIV, can also be spread by medical professionals, acupuncturists, tattoo artists, etc. to the general population and at the same time even the attending medical and laboratory staff are at risk of contracting the virus by contaminated needles stick and other similar injuries. There is an added risk of hospital spread of an infection due to collection of all cases at one place where even intrusive techniques and blood exposures are being done. So, even health professionals take appropriate preventive measures to protect themselves and others.

References

- Anderson LJ, Tong S (2010) Update on SARS research and other possibly zoonotic coronaviruses. *Int J Antimicrob Agents* 36:S21–S25
- Baron S, Fons M, Albrecht T (1996) Viral pathogenesis. In: Baron S (ed) *Medical microbiology*, 4th edn. University of Texas Medical Branch at Galveston, Galveston, TX. Chapter 45
- Beran GW (1994) Handbook of zoonosis. In: Section B. *Viral*, 2nd edn. CRC, Boca Raton, FL
- Bloom-Feshbach K, Alonso WJ, Charu V, Tamerius J, Simonsen L, Miller MA, Viboud C (2013) Latitudinal variations in seasonal activity of influenza and respiratory syncytial virus (RSV): a global comparative review. *PLoS One* 8(2):e54445. <https://doi.org/10.1371/journal.pone.0054445>
- Boldogh I, Albrecht T, Porter DD (1996) Persistent viral infections. In: Baron S (ed) *Medical microbiology*, 4th edn. University of Texas Medical Branch at Galveston, Galveston, TX. Chapter 46
- Burrell CJ, Howard CR, Murphy FA (2017) Epidemiology of viral infections. In: Fenner and White's *medical virology*. Academic Press, Cambridge, MA, pp 185–203. <https://doi.org/10.1016/B978-0-12-375156-0.00013-8>
- Cleaveland S, Haydon DT, Taylor L (2007) Overviews of pathogen emergence: which pathogens emerge, when and why? *Curr Top Microbiol Immunol* 315:85–111
- Cleaveland S, Laurenson MK, Taylor LH (2011) Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. *Phil Trans R Soc Lond B* 356:991–999
- Corman VM, Eckerle I, Bleicker T et al (2012) Detection of a novel human coronavirus by real-time reverse-transcription polymerase chain reaction. *Euro Surveill* 17:20285
- Evans AS (ed) (1989) *Viral infections of humans: epidemiology and control*, 3rd edn. Plenum Medical, New York
- Feng H, Shuda M, Chang Y, Moore PS (2008) Clonal integration of a polyomavirus in human Merkel cell carcinoma. *Science* 319:1096–1100
- Fenner F (1996) Epidemiology and evolution. In: Baron S (ed) *Medical microbiology*, 4th edn. University of Texas Medical Branch at Galveston, Galveston, TX. Chapter 48
- Gardner SD, Field AM, Coleman DV, Hulme B (1971) New human papovavirus (B.K.) isolated from urine after renal transplantation. *Lancet* 1:1253–1257
- Kilpatrick AM, Randolph SE (2012) Drivers, dynamics, and control of emerging vector-borne zoonotic diseases. *Lancet* 380:1946–1955
- Klimpel GR (1996) Immune defenses. In: Baron S (ed) *Medical microbiology*, 4th edn. University of Texas Medical Branch at Galveston, Galveston, TX. Chapter 50
- Knipe DM, Howley P (2013) *Fields virology*. Lippincott Williams & Wilkins, Philadelphia
- Krause RM (1992) The origin of plagues: old and new. *Science* 257:1073–1078

- Mandell G, Dolin R, Bennett J (2009) Mandell, Douglas, and Bennett's principles and practice of infectious diseases, 7th edn. Elsevier, Amsterdam
- Padgett BL, Walker DL, ZuRhein GM et al (1971) Cultivation of papova-like virus from human brain with progressive multifocal leucoencephalopathy. *Lancet* 1:1257–1260
- Parvez MK, Parveen S (2017) Evolution and emergence of pathogenic viruses: past, present, and future. *Intervirology* 60(1–2):1–7
- Taylor LH, Latham SM, Woolhouse MEJ (2013) Risk factors for human disease emergence. *Phil Trans R Soc Lond B* 356:983–989
- Woolhouse MEJ, Gowtage-Sequeria S (2005) Host range and emerging and re-emerging pathogens. *Emerg Infect Dis* 11:1842–1847
- World Health Organization (2009) Pandemic influenza preparedness and response: a WHO guidance document. World Health Organization, Geneva