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Mathematical Modeling and Dynamic System in Epidemiology

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Abstract

Pandemics have been happening on the planet since the days of yore. It is troublesome in the old pandemics however pandemics of the later beginning have passed on certain messages. These significant examples ought to be acknowledged sincerely for confronting all the more skillfully in the accompanying pandemics. They have raised a ruckus around town since early times. It has been found that Nepal did not have connectivity with the pandemics in the past. It is only in the Corona pandemic that Nepal has also been a part of the whole globe. Our investigation focuses on historical aspects of bio-epidemiological mathematical surveys. Historical background of epidemics, their parallel situation in Nepal, and their correlation with mathematical modeling have been provided. Different dynamical systems used in epidemiology are mentioned. This study has also emphasized on deterministic modeling, and different disease transmission rates applied to the population dynamics of infectious diseases.

Keywords: Compartmental model, transmission rate, nonlinear incidence, effective reproduction number.

Background

Introduction

Communicable diseases have been a great challenge to humankind since the beginning of human history. At present, we still have a deal with communicable diseases like measles, AIDS, Plague, Malaria, T.B., Dengue, SARI, and COVID-19. Millions of people die annually from these diseases and billions of others are infected. These diseases would be soon eliminated with the improvement in medical science care and awareness process. Communicable diseases caused by various microbes, pathogens or microorganisms have been a threat to public health (Martcheva, 2015). They are caused by pathogens and can be easily transmitted from one infected person to another non-infected person. The most common examples are influenza or flu, measles, rubella, HIV, mumps, malaria, and smallpox (Brauer et al., 2012; Waltman, 2013). The emergence and reemergence of infectious diseases have become a significant worldwide problem. So, a Proper understanding of disease transmission dynamics caused by existing and new pathogens facilitates devising prevention tools (Foppa, 2016; Dym, 2004). Prevention tools against the transmission of disease need to be developed. Implementation and proper use of these sophisticated tools against the microbes is another challenge. This article addresses some theoretical frameworks and intends to provide some basic information about the infection mechanisms

of microbes, their orientation, control mechanism and the role of mathematical models in the epidemiology (Murray, 2001; Banerjee, 2021).

Historical Background of Mathematical Modeling in Epidemiology

Epidemiological mathematical modelling's historical components were first inspired by historians' and academics' accounts of events like the Plague of Athens (430–428 BC). The scientific historian Thucydides (460–400 BC) gives the most accurate account, detailing the symptoms, transmission dynamics, and death toll. In his essay "On the Epidemics," Hippocrates (459–337 BC) outlined the variables influencing the spread of diseases at that time and how they propagated. The Bubonic Plague claimed the lives of 25 million individuals in Europe in the fourteenth century. During the Antonina Plague (165–180 AD), soldiers returning from Near Eastern operations carried either smallpox or measles back to the Roman Empire. Two Roman emperors perished in the plague that struck the Roman Empire, which also severely reduced its population and economy.

In the study of epidemiology, the severe effects of pandemic disease in Nepal were noteworthy. The Rigveda (c. 8000 BC), the Charaka Sahara (c. 700 BC) and various other Ayurvedic works up to 1600 AD, the Puranas (c. 200 BC to 750 AD), travelogues from visits to India, and certain British records are among the ancient literature that mentions human viruses (Sharma, 1951; Badshah et al., 2013). Humanity has always been concerned about infectious diseases from the dawn of time. Certain infectious diseases still pose a problem for us today (measles, AIDS, Plague, Malaria, T.B., Dengue, SARI, COVID-19). Each year, these illnesses took off the lives of millions of people while infecting billions more. Since last time, there has been a conviction that the development of antibiotics will soon lead to the elimination of infectious diseases.

Past Pandemic History and Their Parallel Situation in Nepal

Since the beginning of time, pandemics have happened all across the world. The ancient pandemics were harsh times, but subsequent pandemics have taught us some valuable lessons. In the study of epidemiology, the severe effects of pandemic disease in Nepal were noteworthy. Globalization, transportation infrastructure, and the influx of new people are the main factors in the spread of any infectious disease. There are several pandemics recorded in ancient history. In contrast to now, when COVID-19 circled the world, it was contained to a much narrower area because the earth was not as globally connected as it is now. Neither the Ramayana nor the Mahabharata make any reference to the pandemic (Pokharel, 2020; Sharma, 1951).

Prehistoric Pandemic in China

Ironically, the first pandemic is also thought to have occurred in China’s Yuhan province. Such a disaster is said to have happened in an archeological site known as Hamin Manga around 3000 BC. The finding of the skeletons of all age bunches proposes that it has not saved any individual old or youthful. There are a couple of different destinations exhumed which showed mass graves proposing that pandemics were the thing to take care of during those early times even. It is difficult to determine whether Nepal was inhabited prior to 5000 years. Future archeological exercises will decide it. The caves of Mustang, which are located in steep mountains and date back between 2000 and 3000 years,

have been the site of the earliest human settlement. Some sort of a pandemic priority prompted its relinquishment. The investigation of the inside caves recommends that individuals had progressed living. The arrangement of living, beds and different rooms with latrine offices features this reality (Pokharel, 2020).

Plague in Greece

Sthunko battled against Sovereign Ashok when he came to raise the Lumbini support point in the year 249 BC. It is said that the Kirat chieftain was badly defeated. In any case, after Ashok returned, Sthunko is said to have cut down the pony capital. Wang Huen Tse has composed that he had seen the Pony Capital when he came to Lumbini for 656. It doesn't exist now. An unhinged pursuit was made by the creator in Lumbini during the development of the Maya Devi sanctuary yet without any result. This may not anyway be valid given that Jitadisti is accepted to be a contemporary of Buddha who lived somewhere in the range of 623 and 543 BC. The events of 430BC in Nepal could theoretically be observed using a powerful telescope aligned with a planet 2450 light years away from Earth. This is because the light is as yet going to those planets from the Earth as of now. Science may one day make it a reality (Pokharel, 2020).

Plague in Rome

This is also known as the Antonine Plague. It is said to have occurred in the year 27 BC going all the way to 180 AD. It is said to be transmitted by the soldiers who had gone to fight in the war against Parthia. The Licchavi King Jaya Verma was ruling then in Nepal for 185 AD. We have very little information regarding what transpired during Jaya's regime.

Plague of Cyprian in Rome

In Rome, another plague struck, killing 5,000 people in a single day. It is believed that this incident occurred between 250 and 271 AD. Nepal is in the post-Jaya Verma era at this point. The famous leader of the Licchavi time frame Man Dev governed from the fifth hundred years. The first inscription, which is dated 464 AD, clearly identifies his rule. The period going before this time is obfuscated simply by stories and fantasies. Along these lines, we don't have a record of what happened most definitely.

Plague of Justinin

Sovereign Justinian was a renowned ruler. During his rule, the popular church of St Hagia was constructed. It is wonderful to the point that he is said to have gloated by saying that he had outperformed Solomon, the incredible Ruler. During his system, a plague shook Rome right from its spine in a 542 Promotion. Despite being sickened by the plague, the Emperor was able to recover. This plague is said to have ended the existence of 10% of the number of inhabitants on the planet in general. Vamana Dev, a Licchavi ruler, ruled Nepal at the time. This is portrayed in a stone spout in Sankhu known as Dagu Hiti. There is no reference to pandemics during this period in Nepal.

Black Death of 1343

There was a gap of precisely 800 years when the pandemic broke after quite a while. It is said that the West lost two-thirds of its population to this pandemic. It is hard to let whether know this pandemic went to Nepal. Be that as it may, individuals passed on in huge numbers in Nepal too. Nepal experienced several catastrophes during this time. A quake emitted around Barpak in the Gorkha region killing the supreme Ruler Ari Malla Dev a day later. Muslim ruler Shamshiddun went after Kathmandu Valley in a 1349 Promotion focusing on well-known strict places like Pim Bahal, Swoyambhu and Pashupati Nath. As a result, Pashupati Nath's Linga was split into three pieces. During this time, the Khash rulers also attacked the valley. In the year 1255, there was also a huge earthquake. Nepal was ruled by Abhya Malla at this time. He tragically passed away during the earthquake. His rule was likewise hit by starvation and pandemics. The general public then was exceptionally eccentric. Individuals accepted that transgression would be committed someplace other by individuals on account of affection and desire and God would answer it as infections. These could be the plague, smallpox, starvation and such. As a result, the epidemics were a blessing to the gods. At the point when horrendous starvation of the year 1231 struck Kathmandu, 33% to one-6th of the populace died. Lord Abhyaya Malla performed Laksahome and Mahasnana to pay tribute to God Pashupati to avoid the underhanded impacts of starvation and pandemics.

American Plague

It is believed that 90% of the native Intec and Aztec populations perished as a result of Europeans spreading the disease from their homelands. Due to the epidemic, the local population was unable to fight, so the 1519 million Spanish army led by Hernan Cortes and the 1532 million Spanish army led by Francisco Pizarro benefited. Ratna Malla was administered during this time in Kantipur. There is no reference to any plague happening in Nepal during this time.

Cocolitzli Epidemic

In Mexico and Central America, this epidemic started at 1545 and lasted until 1548. High fever brought about the passing of a few groups. This plague didn't come as far. In Nepal, Prana Malla was administered in Bhaktapur along with Jit Malla. Narendra Malla was in control in Kantipur. Festivals, songs, and dances are said to be favourites of his. He started numerous celebrations and moves in Kantipur. In Lalitpur, Vishnu Singh was the Ruler. Vishnu Singh is said to have developed the sanctuary of Bhringareswore. Nepal does not appear to have experienced any epidemics during this time. During this time, pandemics didn't happen in every one of the three realms in Nepal.

Great Plague of London, 1665 − 1666

In London, this plague is said to have killed 100000 individuals. The plague is said to have spread from bugs after interacting with plague-ridden rodents. This plague hit London twice: first, a baker's shop in London caught fire, destroying the building for four days. One average person had drawn in the consideration of the City hall leader towards this. However, the Mayor was rather caring to be sure when he trained to demand an old woman to pee in the fire to quench. Ruler Pratap Malla was controlled

in Nepal during this time. Kathmandu has not been the site of any epidemics. In any case, in Bhaktapur smallpox emitted and Jagat Prakash Malla passed on because of this pandemic in the year 1762. There is a whip demonstrating the far-reaching of cholera in the year 1761.

Great Plague of Marsellie

This plague was conveyed by a boat from the Mediterranean which came to Marseille. It lasted for a year from 1722. It ended the existence of nearly 100000 persons. On account of the London plague, bugs after interacting with plague-ridden rodents sent the sickness.

In Nepal, there was a plague in 1722 when Bhaskar Malla was administering in Kantipur. This is portrayed in the records of Father Frayer, a Christian Preacher. According to legend, the King allowed Dashain to be observed, which was prohibited in Bhaktapur and Lalitpur due to the leap year. A severe hunger struck the Kingdom. What's more, a bizarre illness was pervasive which would bring about enlarging throughout the long-term prompting passing. Lord Bhaskar Malla with his sovereigns and a few workers was made to remain in Kindol Bahal sort of isolation now. Meanwhile, one holy person said that the infection bend would level if individuals were welcomed for a banquet. The Lord acknowledged and the pandemic seemed to defrost a little. The Lord turned out to be exceptionally cheerful and went to the royal residence subsequent to leaving from a window. But the King contracted the disease and passed away quickly.

Russian Plague

Over the course of two years, and starting at 1770, this plague claimed the lives of nearly 100000 people. Individuals had been isolated on a huge scale. They revolted and emerged. They also killed Archbishop Ambrosius, who had asked people not to gather in the church for fear of contracting more diseases.

Nepal was ruled by Prithvi Narayan Shaha at the time. There is no mention of a serious epidemic during this time period in Nepal's history.

Philadelphia Yellow Fever

This scourge was spread by mosquitoes in Philapheldia that ended the existence of 5000 individuals in the year 1793. When the mosquitoes became inactive and instinctive after the winter, they vanished. Numerous people have been killed by smallpox in Nepal on a regular and unwelcome basis. Until a vaccine led to its complete abolition in Nepal, devotees fervently worshipped the Sitala Mai temple, which is now mostly deserted. A fairly unusual Lord Rana Bahadur Shaha, gave proper respect to a few sanctuaries and gave a lot of cash to the Brahmins wanting for the expedient recuperation of his contaminated sovereign. He obliterated a few such sanctuaries as well as grabbed the gift back from the ministers after his heartbreaker Kantimati kicked the bucket directly following the 1799 Smallpox plague.

Flu Epidemic

One million people were killed in Russia in the year 1889 in the city of St. Petersburg. By then, individuals were moving from one country to another through the ocean on a huge scale and it was liable for its spread in Europe and the remainder of the world.

Ruler Prithvi Beera Bikram was administered in Nepal during this time. Beer Shamsher was the State leader. He developed a water supply network which was known as Bir Dhara. This assisted with having unadulterated water instead of tainted one. Additionally, he established Bir Hospital, which provided individuals with limited opportunities. Cholera and smallpox were customary executioners then, at that point. However, the cholera flare-up of the year 1821 has been recorded as the principal occasion of its sort ever, it is right around a yearly peculiarity in Nepal. In 2009, 500 individuals lost their lives in Jajarkot and just in 2014, 500 individuals were impacted in Rautahat region.

Spanish Flu

This flu showed up in the outcome of WWII in the year 1918 and went on for a long time till 1920. This flu caused the deaths of many soldiers. It impacted 500 million individuals and one-fifth of this is said to have passed on. 15000 Nepali troopers had likewise participated in WWI and a considerable lot of them kicked the bucket. Some returned and went to their home. Be that as it may, there is no record of this influenza to have impacted anyone in Nepal. Tribhuvan was the Ruler and Chandra Shamsher was the Priminister of Nepal during this period.

Asian Flu

This influenza again began in China in the year 1957. It then spilled into Singapore prompting Hongkong lastly showing up in the US in the mid-year of 1957. Some 1.1 million individuals were dead with 1, 16, 000 ends in the US alone.

Nepal was a multiparty a majority rules system after People groups' Upheaval in 1950. Nepal was simply open to the rest of the world subsequent to staying shut to it for quite a while. Thus, this influenza doesn't appear to have gone into Nepal. Nonetheless, smallpox and cholera proceeded to destruction Nepal.

AIDS Pandemic and Epidemic

An estimated 35 million individuals died from AIDS in 1981. West Africa is intended to be its launchpad. With 40 million people living with HIV in Sub-Saharan Africa, it is widespread. Nepal was also impacted by this virus. It was widespread throughout the country, however it was particularly severe in western Nepal. It is thought to have been transmitted by Indian Nepali laborers.

H1N1 Swine Flu Pandemic

A novel H1N1 strain is what prompted this flu to begin in Mexico. Triple-reassortant H1N1 influenza with swine origins in April 2009, the first influenza pandemic of the twenty-first century was caused by a virus discovered to be a distant descendant of the 1918 "Spanish flu" virus. Half a million people died as a result of the 1.4 billion infections it caused. An intriguing feature of this flu was that, compared to all other pandemics where elderly individuals were prime targets, those under 65 were more

susceptible. Corona is likewise in this situation. Fortunately, Nepal was spared this flu. There isn't a single instance of this flu reported in Nepal.

West African Ebola Epidemic

The greatest Ebola outbreak since the virus's discovery in 1976 took place in West Africa between 2014 and 2016. There have been seven Ebola infection episodes since the disease's discovery. This episode had more cases and fatalities than any other. It began in Guinea and swiftly extended to the neighboring countries of Sierra Leone and Liberia. By July 2014, it had made its way to all three of these nations' capital cities, and in August of that same year, the World Health Organization had deemed the epidemic a Global Health Emergency. Specifically, the Ebola pandemic devastated West Africa, with an estimated 30,000 cases and 10,000 fatalities reported. It originated in Guiana.

Zika Virus

It is mosquitoes that spread the Zika virus. In 1947, a Rhesus macaque monkey in Uganda was the first known to have it. Human cases of pollution and disease were documented in several African nations throughout the 1950s. In Africa and Asia, various human diseases were discovered in the 1960s and 1980s. However, starting about 2007, cases of Zika virus have been documented throughout Africa, the Americas, Asia, and the Pacific. There have been Zika virus outbreaks during the past ten years, and these have been connected to a rise in the incidence of Guillain-Barre syndrome. During the 2015 severe pandemic in Brazil, an association was found between Zika infection sickness and microcephaly, a condition characterized by a smaller-than-average head size.

COVID-19

COVID − 19, caused by the novel coronavirus SARS − CoV − 2, emerged in late 2019 in Wuhan, China. It rapidly spread worldwide, leading to a global pandemic declared by the World Health Organization (WHO) on March 11, 2020. This disease spreads through respiratory droplets and has a wide range of symptoms, from mild respiratory issues to severe pneumonia and death. Its exact origin is still under investigation, but the virus is believed to have zoonotic origins, possibly linked to a seafood market in Wuhan where live animals were sold. It spreads mainly through close contact with an infected person via respiratory droplets when they cough, sneeze, or talk. It can also spread by touching surfaces contaminated with the virus and then touching the face. Symptoms can appear 2 − 14 days after exposure and vary widely with common symptoms: fever, cough, and shortness of breath and other symptoms: Fatigue, muscle or body aches, headache, new loss of taste or smell, sore throat, congestion, nausea, and diarrhoea (Hao et al., 2022).

The pandemic overpowered medical care frameworks around the world, causing deficiencies of clinical supplies, medical clinic beds, and medical services laborers. It prompted worldwide monetary interruption, with numerous organizations shutting, joblessness rates increasing, and critical effects on worldwide exchange and travel. The pandemic provoked lockdowns, social removing measures, remote work, and changes in day to day existence and social communications.

Quick turn of events and organization of immunizations have been basic in dealing with the pandemic. Antibodies like those created by Pfizer-BioNTech, Moderna, and Johnson and Johnson have been approved for crisis use, essentially lessening the seriousness of disease and transmission rates. While the pandemic's intense stage has died down because of inoculation endeavors and general wellbeing measures, Coronavirus keeps on flowing with occasional flare-ups and the rise of new variations. Endeavors are progressing to accomplish higher inoculation inclusion, foster medicines, and screen for new variations to oversee and in the end the pandemic.

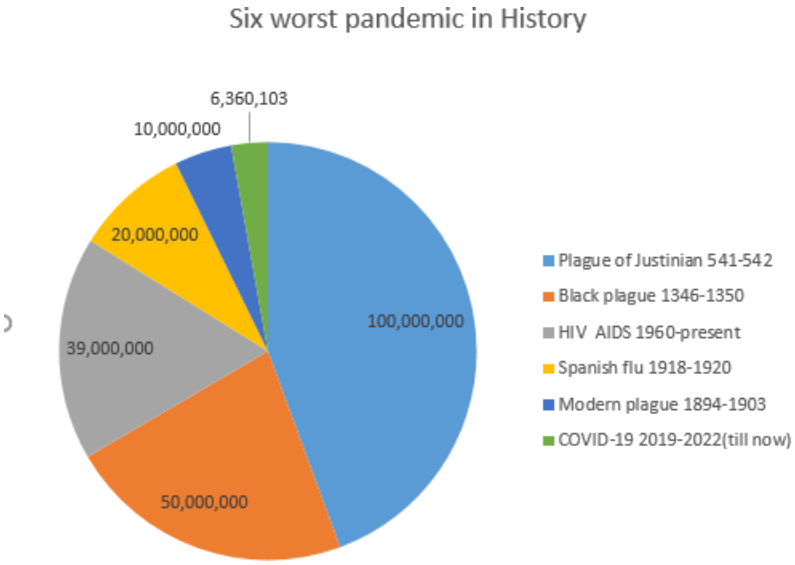

Figure 1: Six worst history

Learning for Nepal

One can finish up from the over that pandemics have pained the world from days of yore. Coronavirus has demonstrated the way that it can't be wished out of the present even in present day times that have been set apart by extraordinary advancements in science and innovation. Race is on for the advancement of the antibody however the possibilities seem remote soon. Over a year might be a sitting tight time for the achievement of a reasonable immunization. Nepal has been impacted by Smallpox and cholera since early times. It is likewise said to have been desolated by plague in 1347 when plague consumed 66% of the populace in the west. Be that as it may, whether this plague made a passage into Nepal or it was a neighbourhood still needs not be set in stone. In any case, smallpox and Cholera have been normal undesirable guests in Nepal.

There have been three-pronged methodologies embraced for the anticipation of such pandemics in Nepal. The first is customary. It comprises revering divine beings and goddesses. Sitala Mai goddess was venerated intensely for fixing smallpox till the antibody was created to counter the smallpox. The sanctuary of Sitala Mai used to be exceptionally packed however it seems abandoned now because of the immunization that has been viable to counter it. For the fix from cholera, Bagala Mai was adored hotly. Presently, the cholera has likewise died down because of the ac- cessibility of an immunization

and the Goddess is loved as a Goddess of Want. The subsequent one is semi-customary. It is about the utilization of Ayurvedic natural medication. This is more on account of cholera. The third one is the cutting-edge one which is set apart by the utilization of antibodies and medication. There was additionally the arrangement of isolation in the past in Nepal. Individuals experiencing such illnesses used to be put independently as was above all else Bhaskar Malla in the mid-eighteenth hundred years. The way of life of washing hands now and again after crap, when food additionally added to the anticipation of the spread of the infection. Also, Yoga would be a superior counteraction measure. These days individuals have been rehearsing Yoga and Yoga mindfulness as referenced by (Bhatta, 2024).

System in Epidemiology

Epidemic dynamics is an important method of studying the spread of infectious diseases. It is based on the specific property of population growth, the spread rule of infectious disease, and the related social factors etc. Dynamic systems in epidemiology are used to construct mathematical models reflecting the dynamic properties of infectious disease, to analyze the dynamical behaviour of the model so formed and to do some simulations. The research result helps predict the growth of infectious diseases, determine the key factors of the spread of infectious diseases and seek the optimum strategies for preventing and controlling the spread of infectious diseases.

Compartmental Models in Epidemiology

Most infectious disease dynamic models are based on the compartment structure of the diseases. First provided by Kermack and Mckendrick in 1927, the compartment structures for dynamic models are developed by numerous other biomathematicians in 1932. Those who recover from viral diseases such as influenza, measles, swine flu, and chikungunya develop immunity to the same virus. The SIR model can be used to describe these illnesses. Furthermore, those who recover from bacterial illnesses such as gonorrhea, the bubonic plague, tuberculosis, syphilis, etc. do not develop immunity and are susceptible to re-infection. The SIS model can be used to investigate the dynamics of these illnesses.

Fundamental Forms of Compartmental Models

Models without Latent Periods

In these models the infected individuals becomes infectious immediately (Martcheva, 2015). These models are as follows:

1. **SI Model:** In this model, the infectives cannot be recovered from infection. It is represented diagram 2. The model equations are:

Figure 2: SI model

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$$\frac{dS}{dt} = -\beta SI, \text{ and } \frac{dI}{dt} = \beta SI$$

2. **SIS Model:** In this model, the infectives individuals are recovered, but gain no immunity from infection. It is represented diagram. 3 The model equations are:

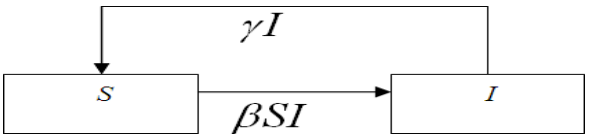

Figure 3: SIS model

$$\frac{dS}{dt} = -\beta SI + \gamma I, \text{ and } \frac{dI}{dt} = \beta SI - \gamma I.$$

3. **SIR Model:** In this model, the infectives obtain permanent immunity to the disease after recovered from infection. It is represented by diagram 4.

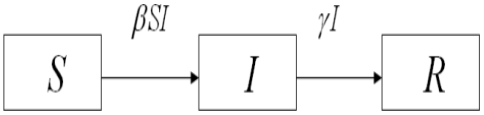

Figure 4: SIR model

$$\frac{dS}{dt} = -\beta SI,$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

$$= BI(S - \rho) \text{ where } \rho = \frac{\gamma}{\beta}$$

$$\frac{dR}{dt} = \gamma I$$

4. **SIRS Model:** In this model, the recovered individuals may have only temporary immunity after they recovered from infection. Diagram 5 represents this model.

The model equations are:

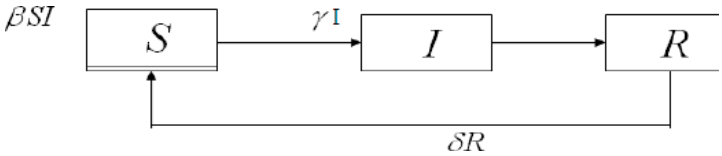

Figure 5: SIRS model

$$\frac{dS}{dt} = -\beta SI + \delta R,$$

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$$\frac{dI}{dt} = \beta SI - \gamma I$$

$$\beta I(S - \rho), \text{ where } \rho = \frac{\gamma}{\beta}$$

$$\frac{dR}{dt} = \gamma I - \delta R.$$

5. **SIRI Model:** In this model, the infectives individuals cannot obtain permanent immunity to the disease when they recovered from infection. Diagram 6 represent in model: The model

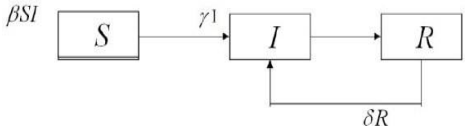

Figure 6: SIRI model

equations are:

$$\frac{dS}{dt} = -\beta SI\,, \frac{dI}{dt} = \beta SI - \gamma I + \delta R = \beta I(S - \rho) + \delta R, \text{ where } \rho = \frac{\gamma}{\beta}, \text{ and } \frac{dR}{dt} = \gamma I - \delta R.$$

6. **MSIR Model:** Babies are not born into the susceptible compartment for many illnesses, such as measles, but are instead immune to the illness for the first several months of their lives because of maternal antibodies (either through the placenta or through colostrum). This can be shown by including an M class (for maternally derived immunity) at the beginning of the model. It is represented by diagram 7.

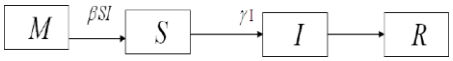

Figure 7: MSIR model

Models with Latent Periods

There is often a considerable period of time during which the affected person is infected but not yet contagious for many serious infections. In the course of this latent time, the person is in the exposed compartment (E) Martcheva (2015). The following are these models:

1. **SEI Model:** This model is represented by diagram 8

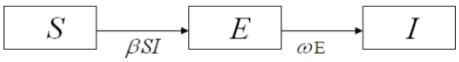

Figure 8: SEI model

2. **SEIR Model:** In this model the population is broken into four compartments: susceptible, exposed, infec- tious and recovered. This model is represented by diagram 9.

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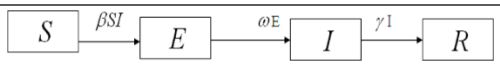

Figure 9: SEIR model

3. **SEIS Model:** In this model the population is broken into four compartments: susceptible, exposed, and infectious again susceptible. Diagram 10 represents this model.

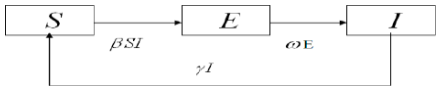

Figure 10: SEIS model

4. **SEIRS Model:** The population is divided into five compartments in this model: susceptible, exposed, infectious, recovered, and susceptible again. The representation of this model is diagram 11

5. **MSEIR Model:** The MSEIR model is used for epidemiological classes in cases of disease where the factors of latency period and passive immunity are present. Diagram 12 serves as a representation of this model.

   where M is births and passive immunity.

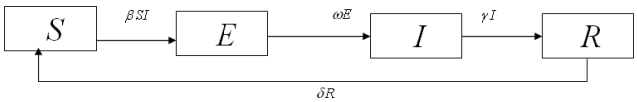

Figure 11: SEIRS model

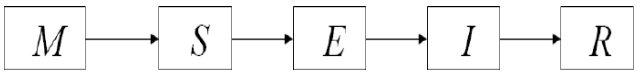

Figure 12: SEIRS model

Basic Concepts of Dynamical System in Epidemiology

Terms such as contact rate, adequate contact rate, infection rate, simple mass action incidence, standard incidence, saturation incidence, nonlinear incidence, monotonic incidence, basic reproduction number, threshold numbers, etc. are frequently encountered in an epidemiological dynamic system and are defined as follows:

Disease Transmission Rate

Infectious diseases can transmit by direct contact. The contact rate of infection, represented by P(N), is the number of people contacted by an infectious per unit of time. Depending on the population as a whole, N. The persons may become infected if they come into contact with an infectious susceptible. Assume that there is a β_0 probability of infection for every contact. Subsequently, the function $\beta 0N$ is

referred to as an adequate contact rate, denoting the degree of infection caused by the infectious agents and typically reliant on the bacterial or viral toxicity and environmental conditions.

The average rate at which susceptible individuals come into touch with infected persons per unit of time is known as the contact rate, and it is commonly represented by the symbol β.

$$\beta = \text{contact rate} \times \beta_0.$$

For example, if an average individual has 10 contacts per day and the probability of disease transmission per contact is 0.2(20%), then β =10 contacts/day × 0.2 transmission/contact = 2

This means that each susceptible individual is effectively exposed to the infection at a rate of 2 contacts per day.

To determine the unit of β, we need to consider the units of its components: the average number of contacts per susceptible individual per unit of time and the probability of disease transmission per contact. The unit of β will be a combination of these units. An average number of contacts per susceptible individual per unit time has units of ”contacts” per ”unit time” (e.g., contacts per day, contacts per week) and the probability of disease transmission per contact is a dimensionless quantity, as it represents a probability or a ratio. To obtain the unit of β, we multiply the units of the average number of contacts per susceptible individual per unit of time by the dimensionless unit of the probability of disease transmission per contact.

Example 1:

If the average number of contacts per susceptible individual per day is 10 contacts/day and the probability of disease transmission per contact is 0.3 (dimensionless), then β = Average number of contacts per susceptible individual × Probability of disease transmission per contact.

β = (10 contacts/day) × (0.3) = 3 contact per day.

Unit of β = (contacts / day) × (dimensionless) = contacts / day.

Example 2:

Average number of contacts per susceptible individual per week: 50 contacts/week Probability of disease transmission per contact: 0.2 (dimensionless)

β = Average number of contacts per susceptible individual × Probability of disease transmission per contact β = 50 contact / week × (0.2) = 10 contact pert week.

Therefore, unit of β = (contacts / week) × (dimensionless) = contacts / week. In both examples, the unit of β is the same as the unit of the average number of contacts per susceptible individual per unit of time. This is because the probability of disease transmission per contact is dimensionless, so it does not affect the units of β.

A higher β indicates a higher likelihood of disease transmission, assuming other factors remain constant. Factors such as population density, behaviour, interventions (like masks or social distancing), and immunity levels can influence β.

Time Dependency: β may not remain constant over time, especially during outbreaks or in response to interventions. It can change due to changes in behaviour, awareness, and public health measures.

Incorporation into Models: In infectious disease models, β is often used along with other parameters like the infectious period and the number of susceptible individuals to predict the spread of the disease over time. Models like the SIR (Susceptible-Infectious-Recovered) model use β as a key parameter to simulate disease transmission dynamics. Understanding and estimating the contact rate β is essential for assessing the risk of disease spread and for designing effective public health interventions to mitigate transmission.

Since diseases are only transmitted to susceptible by contact with infectives and the fraction of the susceptible with the population is $\frac{S}{N}$, then the mean adequate contact rate is $\beta_0 P(N)\, SI/N$. This rate is called an infection rate. Then the total new infectives in the infected compartment are $\beta_0 P(N)\, \frac{SI}{N}$, which is called an incidence of the disease.

Force of Infection

The force of infection (often denoted by λ is a crucial concept in the mathematical modelling of infectious diseases. It quantifies the rate at which susceptible individuals become infected. Essentially, it measures the risk of infection for a susceptible person per unit of time, based on the current epidemiological conditions.

Example 3:

There is a 1,50,000 population. out of which we only took 2000 samples for a survey of disease transmission and found that 100 were infected by communicable diseases in a year. How can we calculate the disease transmission rate per day?

To calculate the disease transmission rate per day from a given sample from a larger population, we need to use this information to estimate the transmission dynamics in the overall population. Given data:

Total population (N) = 1, 50, 000

Sample Size (n) = 2000

Infected Individuals in Sample (I_s): 100 Time Frame: 1 year (365 days)

Steps to Calculate the Transmission Rate Per day are:

1. To calculate the infection proportion in the sample:

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The infection rate within the sample to estimate the prevalence of the disease in the entire population is given by

Infection Rate in Sample = probability of infection $= \frac{I_S}{Samplesize} = \frac{100}{2000} = 0.05$. This means that 5% of the sample was infected over the year.

2. To estimate the total number of infected individuals in the population

   Assume the infection rate in the sample reflects the infection rate in the entire population. Estimated infected individuals in population = 0.05 × 150,000 = 7, 500

3. To estimate the force of infection

   It represents the rate at which susceptible individuals become infected per unit time. It can be approximated as:

   Force of infection= $\frac{Total\ Infected\ individuals}{n \times T}$ where T is time period (1 year = 365 days).

   Force of infection= $\frac{7500}{1{,}50{,}000 \times 365} = 0.000137/infections/person/day.$

4. To calculate the daily transmission rate

   We can determine the effective daily transmission rate (β) in the population by

$$\beta = \frac{force\ of\ infection}{\frac{S}{N}}$$

   Assuming the initial number of susceptible individuals S is approximately the total population N ($S(t) \to N$), especially at the beginning of the outbreak:

   $\beta = \frac{0.000137}{\frac{S}{N}} = 0.000137\ infections/persons/day$. This represents the transmission rate per individual per day. This calculation assumes that the infection rate in the sample is representative of the entire population.

   The transmission rate per year is 0.000137 × 365 = 0.050005

   If out of 1,50,000 people, 1,00,000 have been infected and only the remaining 50,000 are susceptible then the transmission rate is given by

   $\beta = \frac{force\ of\ infection}{\frac{S}{N}} = \frac{0.000137}{\frac{50{,}000}{1{,}50{,}000}} = 0.000411$ infections/person/day=0.150 infection/person/year.

5. To determine the basic reproduction Number (R_0)

If R_0 or the average number of secondary infections produced by one infected individual in a fully susceptible population is known or estimated, it can be used to calculate the transmission rate β

$R_0 = \beta \times D$ where D is the infectious period. If we assume $R_0 = 2$ (for influenza-like disease it is assumed to be approximately equal to 2) and an average infectious period of 5 days:

$\beta = \frac{R_0}{D} = \frac{2}{5} = 0.4$ This value of β is often derived from epidemiological studies and can be used if we have assumptions about R_0 and D

Practical Considerations:

Homogeneity: The assumption that the infection rate in the sample represents the entire population might not hold if there is significant heterogeneity in contact patterns.

Temporal Dynamics: The transmission rate can vary over time, especially if interventions are implemented. Here, the rate is considered uniform throughout the year.

Reporting and Bias: Ensure accurate reporting and consider potential biases in the sample. The daily transmission rate provides a measure of how rapidly the disease is spreading in the population, crucial for modelling the epidemic and planning public health interventions. The entire population is initially considered susceptible, which might not hold if there is existing immunity. Types of incidence used in disease modelling are:

Bilinear incidence

If the contact rate is proportional to the total population size i.e. $P(N) = kN$ then the incidence βIS, where $\beta = \beta_0 k$, is called the transmission coefficient. This type of incidence is called bilinear incidence or simple mass action incidence. the transmission rate β is often assumed to be proportional to the product of the susceptible and infectious populations. It is called "bilinear" because it is a product of two linear terms: S and I. Bilinear incidence models are used in epidemiology to study the dynamics of various infectious diseases, including influenza, HIV/AIDS, and sexually transmitted infections. Most of the standard epidemiological models used a bilinear incidence rate. In this incidence rate, it is assumed that the population is homogeneously mixed and it is normally used for airborne diseases. However, in case of large number of susceptible or population is not homogeneously mixed (i.e. heterogeneous mixing), it is not realistic to consider the bilinear incidence rate due to the number of susceptible with which every infective contact is limited within a definite time. It allows researchers to explore how changes in the size of the susceptible and infectious populations affect the spread of the disease over time and to evaluate the potential impact of interventions such as vaccination or behaviour change campaigns.

Standard Incidence

If the contact rate is constant i.e. $P(N) = k$ then the incidence $\beta \frac{SI}{N}$, where, $\beta = \beta_0 k$ is called the standard incidence. If S, I and N are several susceptible, infectious and total populations at time t, respectively, then $\frac{S}{N}$ and $\frac{I}{N}$ represent the susceptible and infectious fractions, respectively.

If β is the average number of adequate contacts of single susceptible with other members of the population per unit time, then $\frac{\beta I}{N}\beta$ is the average number of contacts with infectives per unit time of a single susceptible and $\beta\frac{I}{N}S$, that is, $\frac{\beta IS}{N}$ is the number of new cases per unit of time due to the S susceptible. Thus, $\frac{\beta IS}{N}$ is the rate at which the susceptible population becomes infected. This form of horizontal incidence is called the standard incidence (proportionate mixing incidence) because it is formulated from the basic principles.

The standard incidence rate adjusts the bilinear form by normalizing it for the total population N (usually the sum of susceptible, infected, and recovered individuals). This Incidence Rate $\beta S\frac{I}{N}$ is useful in models where the population size is large and possibly variable.

Comparative Analysis Bilinear and Standard Incidence Rates

1. Impact of Population Size: Bilinear incidence does not account for total population size. The incidence rate increases directly with S and I, potentially leading to unrealistic predictions in large populations because it assumes that every individual has the same likelihood of making contact.

2. Standard incidence accounts for total population size by dividing by N. As N increases, the effective contact rate per susceptible individual decreases, leading to more realistic dynamics in large populations.

3. Contact Dynamics: Bilinear incidence assumes a constant contact rate per individual, which can be unrealistic in densely populated areas where the contact rate should logically decrease as the population grows.

4. Standard incidence adjusts for the fact that in larger populations, each individual's chance of contact with any specific other person decreases, leading to a more accurate reflection of how diseases spread in real-world settings.

5. Disease Modeling Applicability: Bilinear incidence is more suited for small, isolated populations or in situations where contact between individuals is high and relatively unrestricted. Standard incidence is better suited for large, heterogeneous populations or for diseases where the probability of contact between individuals is diluted by the large population size.

6. Example Comparison: Let us compare the models using the same parameters: Transmission rate (β) = 0.3, recovery rate (γ) = 0.1 Initial populations S(0) = 999, I(0) = 1, R(0) = 0, total population N = 1000 For Bilinear Incidence:

   $$\frac{dI}{dt} = \beta SI - \gamma I = 0.3 \times 999 \times 1 - 0.1 \times 1 = 299.6$$

   For Standard Incidence: $\frac{dI}{dt} = \beta\frac{SI}{N} - \gamma I = 0.3 \times 999\frac{1}{1000} - 0.1 \times 1 = 0.1996$

The initial rate of increase in infections is much higher in the bilinear model compared to the standard incidence model, reflecting how bilinear incidence can overestimate infection rates in large populations.

Bilinear incidence is simpler and effective for small populations but may overestimate infection rates in large populations. Standard incidence provides a more accurate reflection of disease dynamics in large populations by considering the total population size, which adjusts the effective contact rate.

Choosing between these incidence rates depends on the size of the population being modelled and the specific characteristics of the disease being studied.

Saturated Incidence Rate

Saturated incidence rates are used in epidemic models when the transmission rate of the disease does not increase indefinitely with the number of infected individuals but instead levels off or saturates as infection levels rise. This can reflect real-world constraints such as limitations on the number of effective contacts due to behavioural changes, healthcare capacity, or other social factors.

Saturated incidence rates are often represented using Michaelis-Menten kinetics, also known as Holling type II functional response in ecological models. They are used to model scenarios where the incidence rate plateaus as the number of infected individuals becomes large, preventing the unrealistic assumption of unlimited growth in infection rate. The following are the reasons for saturated incidence rates.

1. **Behavioural Changes:** As the number of infected individuals increases, people might change their behaviour to reduce contact (e.g., practising social distancing, wearing masks, Yoga). This behaviour leads to a saturation effect where the rate of new infections does not continue to increase linearly with the number of infected individuals.

2. **Healthcare System Capacity:** When the number of infections is low, healthcare systems can manage and perhaps reduce transmission through effective isolation and treatment. As the number of infections grows, the healthcare system might become overwhelmed, limiting the effective contact rate, and leading to a plateau in new infections.

3. **Resource Limitations:** In the context of a controlled environment like a hospital or a care home, the number of new infections might be saturated due to limited interactions beyond a certain threshold.

4. **Biological Limits:** There can be biological constraints in disease transmission, for example, saturation in vector-borne diseases where the number of vectors or hosts is limited.

5. **Real-World Examples:**

    a. During the COVID-19 pandemic, the rate of new infections in many places showed signs of saturation as governments-imposed lockdowns, people adhered to social distancing guidelines, and healthcare systems reached capacity.

    b. Seasonal flu often shows saturation effects as public health measures, vaccination, and natural immunity reduce transmission rates at high levels of infection.

c. In HIV/AIDS disease, saturation in the incidence rate can occur as behaviour changes (like increased use of protection and awareness campaigns) take effect in high-prevalence areas.

The most commonly used saturated incidence rates are $\frac{\beta SI}{1+\alpha S}$ and $\frac{\beta SI}{1+\alpha I}$. If too many persons are infected in the population, they are not able to affect more susceptibles because of protective actions taken by susceptibles or by the crowding effect of infectives. In this kind of situation saturating incidence rate $\frac{\beta SI}{1+\alpha S}$ is used which tends to $\frac{\beta}{\alpha}$ as S tends to ∞, where α and β are positive constants. Capasso and Serio (Capasso & Serio, 1978) investigated another kind of saturated incidence rate g(I)S for epidemic models, where $g(I) = \frac{\beta I}{1+\alpha I}$ approaches to saturation level when I becomes large. In this incidence rate, the number of effective contacts between susceptibles and infectives may saturate at high infective levels due to crowding of the infected population and due to the protective actions taken by the susceptible population. These incidence rates are more reasonable and suitable for the real world than bilinear and standard incidence rates because of the involvement of behavioural change and crowding effect of the infective individuals and control the unboundedness of the contact rate by choosing the suitable value of α.

The saturated incidence rate introduces a nonlinear relationship between the susceptible and infected populations through the denominator (1+αI). As the number of infected individuals I increases, the term (1+αI) also increases, which slows down the rate of new infections. This creates a nonlinear saturation effect that limits the growth of new infections as I become large. Therefore, the saturated incidence rate is not bilinear because it involves a non-linear dependency on the number of infected individuals, leading to a more complex relationship between S and I than simple multiplication. This captures more realistic dynamics at high infection levels, and additional infections do not increase proportionally due to factors like reduced contact rates or competition for resources. So, it is nonlinear and captures saturation effects, making it different from a bilinear incidence rate, which assumes a direct proportionality between the susceptible and infected populations.

Nonlinear Incidence Rate

Nonlinear incidence rates in epidemiological models like the SIR and SIS models extend beyond the simple bilinear form and capture more complex interactions in disease transmission. These nonlinear rates can better represent various real-world scenarios where the rate of new infections does not increase proportionally with the number of susceptible and infected individuals. Liu et al. (1986, 1987) introduced a non-linear incidence rate of the form $\beta IpSq$ shows a much wider range of dynamical behaviours than do those with bilinear incidence rate βIS. These behaviours are determined mainly by p and β, and secondly by q. For these models, there may exist multiple equilibria in the feasible region and thus model becomes more general and informative. For more application of this incidence rate one can refer to (Dubey et al., 2015; Grigorieva et al., 2016; Wang et al., 2021).

Different types of nonlinear incidence rates commonly used in SIR and SIS models:

1. Saturated Incidence Rate: It is of the form $\frac{\beta SI}{1+\alpha I}$. This rate accounts for saturation effects α, where the infection rate slows as the number of infected individuals increases. It is useful in modelling

2. Scenarios where there is a limited capacity for infection spread due to constraints like contact opportunities or resources.

3. Holling Type II Incidence Rate: It is of the form $\frac{\beta SI}{1+\alpha S}$. Saturation depends on the susceptible population S, meaning that the infection rate slows as the number of susceptibles grows. This can occur if susceptible individuals develop some level of selfprotection or avoidance behaviour. Models scenarios where an increasing susceptible population leads to a decreasing probability of each individual becoming infected.

4. Beddington-DeAngelis Incidence Rate: It is of the form $\frac{\beta SI}{1+\alpha S+\gamma I}$. It is used in models where both overcrowding among susceptibles and competition among infected individuals affect the transmission dynamics.

5. Crowding Incidence Rate: It is of the form $\frac{\beta SI}{1+\alpha(S+I)}$. It accounts for the crowding effect where the total population influences the transmission rate, leading to a more realistic limitation in densely populated settings. it is relevant for densely populated regions where both infected and susceptible individuals compete for limited space or resources.

6. Nonlinear Transmission Function: It is of the form βSI_q where q is a positive constant. The exponent q allows for a more flexible modelling of infection rates. When q > 1, the infection rate grows super-linearly with the infected population, capturing scenarios with clustering or herd behaviour. When q < 1, the rate grows sub-linearly, modelling situations where each additional infected individual has a diminishing effect on new infections. It is useful in capturing complex interaction patterns that deviate from simple proportionality.

Nonlinear incidence rates in SIR and SIS models provide a richer framework for modelling disease dynamics by incorporating more realistic factors such as saturation effects, population interactions, and behavioural responses. They allow for more accurate predictions and better insights into the spread and control of infectious diseases compared to simple bilinear models.

Non-monotonic Incidence Rate

A non-monotonic incidence rate in epidemiological models like the SIR or SIS models captures the complex dynamics where the rate of new infections does not simply increase or decrease with the number of susceptible or infected individuals but can also exhibit peaks and troughs. This can model phenomena where the infection rate might increase up to a certain point and then decrease, reflecting various real-world scenarios such as behavioural changes, resource limitations, or public health interventions. Capasso & Serio (1978) proposed a non-monotonic incidence rate $g(I)S = \frac{\beta IS}{1+\alpha I^2}$ in which g(I) is non-monotonic, that is, g(I) increases when I is small and decreases when I gets large. In this incidence rate, βI measures the force of infection and $\frac{1}{1+\alpha I^2}$ describes the psychological or inhibitory effect from the behavioural change of the susceptibles when the number of infectives gets large. This is important because the number of effective contacts between infectives and susceptibles

decreases at high infective levels due to the quarantine of infectives or due to the protective measures by the susceptibles.

The general incidence rate $g(I).S = \frac{\beta I^P}{1+\alpha I^q}$ was given by Liu et al. (1986) and used by a number of authors (Moghadas & Gumel, 2002; Alexander & Moghadas, 2004; Khan et al., 2015). Here are some forms of non-monotonic incidence rates:

1. Logistic Incidence Rate: It is of the form $\beta SI(1-\frac{I}{K})$. This form incorporates a logistic growth factor $(1-\frac{I}{K})$ where K is carrying capacity for the infected population. Initially, as I increases, the infection rate βSI increases, but after reaching a peak, it decreases as I approach K. It is useful for modelling diseases where the infection rate is self-limiting due to factors such as resource constraints or limited contact opportunities as the infected population grows.

2. Oscillatory Incidence Rate: It is of the form $\beta SIsin(\omega I)$ The sinusoidal term $sin(\omega I)$ introduces periodicity in the incidence rate, reflecting situations where the infection rate oscillates to the number of infected individuals. The parameter ω controls the frequency of oscillations. It is used in scenarios where seasonal effects, periodic behavioural changes, or intervention measures cause fluctuations in the infection rate.

3. Threshold-Based Incidence Rate: It is of the form $\beta\ SI\frac{A}{B+I^2}$. It is suitable for modelling scenarios where public health interventions or behavioural changes significantly impact the infection rate when infection levels are high. Suitable for modelling scenarios where public health interventions or behavioural changes significantly impact the infection rate when infection levels are high.

4. Holling Type III Functional Response: It is of the form $\frac{\beta SI^2}{1+\alpha I^2}$. This form increases slowly when It is small, accelerates to a maximum rate, and then levels off or declines. This is similar to a ”sigmoidal” curve, indicating a more complex relationship between the susceptible and infected populations. It is often used to model predator-prey interactions but is applicable here to represent diseases where the rate of new infections accelerates with I up to a certain point before levelling off.

5. General Polynomial Form: It is of the form $\beta SI\left(1-\frac{I}{K}\right)^n$. This is a generalization where n controls the shape of the incidence curve. For $n = 1$, it reduces to a logistic form. Higher values of n can model more complex non-monotonic behaviours. It is useful in exploring various shapes of non-monotonic incidence functions in disease modelling.

Non-monotonic incidence rates in SIR and SIS models offer a powerful tool for capturing the complex dynamics of disease spread. They reflect realistic scenarios where infection rates can increase up to a point and then decrease, exhibiting peaks and troughs due to various internal and external factors. These models are crucial for accurately predicting and managing epidemic behaviours in complex environments.

Apart from the above-discussed incidence rates, several incident rates are investigated by researchers and provided detailed qualitative analysis of the models. in the case of the Yoga awareness model disease transmission rate can be considered as $\beta e - cM\ SI$, where M is Yoga awareness infected mass and c is constant (Bhatta, 2024).

Reproduction Number

Basic Reproduction

The number of secondary cases that a single infectious person causes in a susceptible community during the infection is known as the basic reproduction number. It is denoted by R_0 and is a key epidemiological metric used to describe the contagiousness or transmissibility of infectious agents.

If $R_0 < 1$ the infection will likely decline and eventually die out in the population. $R_0 = 1$ the infection will remain stable within the population, neither increasing nor decreasing significantly.

$R_0 > 1$, the infection will likely spread and cause an epidemic or pandemic if other conditions are favorable. For example, during the early stages of the COVID−19 pandemic, estimates of R_0 for the SARS −CoV−2 virus varied, but a common estimate was around 2.5−3. This means that, on average, one person infected with COVID−19 could be expected to infect about 2.5−3 other people in a population with no prior immunity or interventions (like social distancing, masks, or vaccines). It is given by

Reproduction Number (R_0) = (transmission rate per contact (β_0)) ×(average number of contacts per unit time (k))×(duration of infectiousness (D))

Let us use a hypothetical disease with the parameters

Transmission rate ($\beta = 0.02$ (2% chance of transmission per contact). Average number of contacts per day (k) = 10.

Duration of infectiousness (D) = 5 days.

We get $R_0 = 0.02 \times 10 \times 5 = 1$. In this example, $R_0 = 1$ means each infected individual, on average, would infect one other person, indicating the disease would remain stable in the population without growing or declining. It helps public health officials and policymakers to assess the potential for an outbreak, implement appropriate control measures, and allocate resources effectively to prevent or contain the spread of infectious diseases.

Effective Reproduction

The effective reproduction number is the average number of secondary infections produced by one infected individual in a population where some individuals may be immune, and intervention measures might be in place. It reflects the actual disease transmission at a specific time in the current state of the population, accounting for factors such as immunity, behavioural changes, and public health

interventions. It is denoted by Re and given by R0 = R0 × St, where St is the fraction of the population that is still susceptible at time t

Suppose the basic reproduction number for a disease is 3, and due to vaccination and previous infections, 60% of the population is immune. This means 40% of the population is still susceptible. Therefore, $R_e = 3 \times 0.40 = 1.2$ indicating that the disease is still spreading, but at a slower rate than it would in a fully susceptible population. In summary, R_e is a dynamic and more realistic measure of an infectious disease's transmissibility in a given population at a specific time, considering the effects of immunity, interventions, and behaviour changes. It helps to evaluate Interventions, to predict trends, and to inform strategies. Monitoring Re is crucial for effective disease control and prevention strategies.

Conclusion

This study emphasizes an understanding of historical aspects of epidemic diseases and deterministic modelling applied in epidemiology. Pandemic history and the situation in Nepal have been described and learning from them to improve our health system has also been discussed. Various compartmental epidemic models have been studied. Mathematical modellings of different infectious diseases are mentioned. The historical characteristics of the bio epidemiological mathematical survey are the main subject of this work. This work also highlights the important relationship between the dynamic features of particular epidemic diseases and mathematical modelling. It provides examples of each to illustrate the concepts of reproduction number and various disease transmission rates. There have also been descriptions of nonlinear, monotonic, and saturated incidence rates.

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