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Chapter 1

Classical results in mathematical epidemiology

Epidemics are typical examples of complex phenomena. A mathematical description of an epidemic needs to take into account its non deterministic nature, and, therefore, rely on probabilities. An epidemic process is, essentially, a stochastic process.

In the dawn of mathematical epidemiology, researchers studied the dynamics in time of probabilities regarding the states of individuals. This is the case of the study of life expectancy and smallpox by Daniel Bernoulli, and so is the famous contribution of Kermack y McEndrick with the SIR models. In both cases, the resulting dynamic equations correspond to the Master equation of the given stochastic processes.

1.1 A supersonic intro to Master equation

Introduction to Stochastic Processes and Markov Chains

A **stochastic process** is a mathematical model that describes the evolution of a random system over time. It's a collection of random variables indexed by time, and can be used to model a wide range of phenomena, from the stock market to the weather.

A **Markov chain** is a specific type of stochastic process that has the **Markov property**: the future state of the system only depends on the current state, and not on any of the previous states. This makes Markov chains particularly useful for modeling systems where the future is dependent on the present, but not on the past.

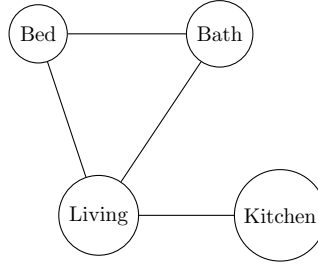
Formally, a Markov chain is a sequence of random variables X_0, X_1, X_2, \dots that take values in a finite or countable set S , where the probability of moving from one state to another depends only on the current state. That is, for all $n \geq 0$ and all states $i, j \in S$,

$$\mathbb{P}(X_{t+1} = j \mid X_t = i, X_{t-1} = i_{t-1}, \dots, X_0 = i_0) = \mathbb{P}(X_{t+1} = j \mid X_t = i).$$

Markov chains have many interesting properties and applications, including their use in modeling queueing systems, random walks, and machine learning algorithms.

Drunken man

A drunken man is moving around his own house, which is made of a Bedroom, a Bathroom, a Livingroom and a Kitchen, connected through doors as shown in the diagram:

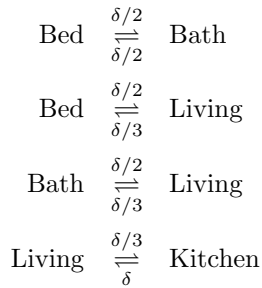


Everytime he enters a room, he chooses one of the doors in that room at random, and moves through it to the next room. The man starts in his bedroom.

1. What is the shortest path from the bedroom to the kitchen?
2. What is the largest path between bed and kitchen (without cycles)?
3. If the man keeps walking for a very long time, what amount of time he spends in every room?
4. What is the expected time that takes the man to visit the kitchen for the first time?

Solution in discrete time:

Let $\vec{p}(t) = (p_1, p_2, p_3, p_4)$ be the vector state defining the probabilities, at time t of finding the drunken man at (bed, bath, living, kitchen), respectively. This can be seen as the following set of stochastic decaying chemical reactions:



Consider the transition matrix

$$M = \begin{bmatrix} 1 - \delta & \delta/2 & \delta/3 & 0 \\ \delta/2 & 1 - \delta & \delta/3 & 0 \\ \delta/2 & \delta/2 & 1 - \delta & \delta \\ 0 & 0 & \delta/3 & 1 - \delta \end{bmatrix}$$

such that the evolution in discrete time of the Markov process is given by

$$\vec{p}(k+1) = M \cdot \vec{p}(k)$$

So the full evolution from $t = 0$ to t is given by

$$\vec{p}(k) = M^k \vec{p}(0)$$

Matrices as M , positive in their values, and such that every column add to 1, are known as stochastic matrices. It can be shown that the largest eigenvalue, in any stochastic matrix, is $\lambda = 1$, and its eigenvector is a probability vector.

In the current case, it is easy to prove, by substitution, that $\vec{p}_\infty = (2, 2, 3, 1)/8$ is such eigenvector. This implies that

$$\vec{p}_\infty = M \cdot \vec{p}_\infty$$

meaning that \vec{p}_∞ is the stationary solution of the process.

A simple generalization proves that an unbiased random walk on a graph has a steady state probability that only depends on the degree of each node, and its proportional to it.

Take home message: every discrete time homogenous in time Markov process corresponds to a transition (stochastic) matrix, whose $\lambda = 1$ eigenvector is the long time limit of the process.

Continuous time limit:

We can consider the scaling of this process as we divide the time scale in T units, and send $\delta \rightarrow \delta/T$. This process is known as continuous time limit, when $T \rightarrow \infty$. In this case

$$\vec{p}(k+1) - \vec{p}(k) = (M(\delta/T) - \mathbb{I}) \cdot \vec{p}(k)$$

resulting in

$$\frac{d\vec{p}}{dt} = (M(\delta) - \mathbb{I}) \cdot \vec{p}$$

Which also shows that the stationary solution is given by

$$\frac{d\vec{p}}{dt} = 0 \Rightarrow (M(\delta) - \mathbb{I}) \cdot \vec{p} = 0$$

which is the definition of the eigenvalue $\lambda = 1$ for matrix M .

Attention, this eigenvector might not be unique. If it is unique, it is the solution in the long time limit. If it is not unique, then the solution will depend on the initial conditions.

Master equation

The Master Equation is a fundamental tool in the study of stochastic processes, particularly in probability theory and statistical physics. It describes the time evolution of the probability distribution of a system in terms of transition rates between different states. In its simplest form, the Master Equation takes the form of a first-order differential equation:

$$\frac{d}{dt} P_i(t) = \sum_j W_{ji} P_j(t) - \sum_j W_{ij} P_i(t), \quad (1.1)$$

where $P_i(t)$ is the probability of the system being in state i at time t , and W_{ij} is the transition rate from state j to state i . The first term on the right-hand side of the equation represents the rate of transitions from all other states j to state i , weighted by the probability of being in state j . The second term represents the rate of transitions from state i to all other states j , weighted by the probability of being in state i .

Master equation can be seen as a continuity equation, or a mass conservation equation. In fluid dynamics, for instance, the rate of change of mass in a control volume must be equal to the net rate of mass flow into or out of the control volume. Mathematically, the continuity equation can be written as:

$$\frac{\partial \rho}{\partial t} + \nabla \cdot (\rho \mathbf{v}) = - \frac{d}{dt} \int_V \rho \, dV + \oint_S \rho \mathbf{v} \cdot d\mathbf{S} = 0$$

where ρ is the fluid density, \mathbf{v} is the velocity vector, $\frac{\partial \rho}{\partial t}$ is the rate of change of density with respect to time, and $\nabla \cdot (\rho \mathbf{v})$ is the divergence of the mass flux density vector.

In other words, the continuity equation states that the change in the amount of fluid within a control volume is equal to the net flow of fluid across the boundaries of that control volume. This principle is based on the law of conservation of mass, which states that mass cannot be created or destroyed, only transferred or transformed.

1.2 Daniel Bernoulli and smallpox

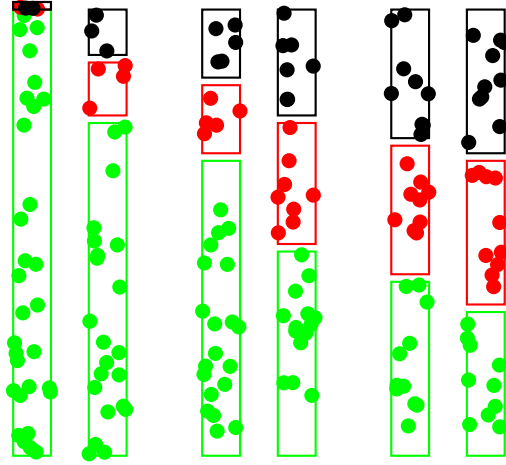
Daniel Bernoulli's study of the effect of smallpox in life expectancy and the possibility of inoculation as a vaccination strategy, a debated argument in the Royal Academy of Science in Paris, in the second half of the XVIII century.

Effect of inoculation in Smallpox, Royal Academy of Science, Paris, 1760, published in 1766

Scope: calculate the gain in life expectancy at birth if smallpox were to be eliminated as a cause of death. The model assumes a fixed amount of infections every year, so, it is not a model for the infection. Still, it deals with relevant questions in epidemiology.

The model of Bernoulli studied the age dynamics of the entire population in a country, as classified in three groups: susceptibles, immunes and dead¹. Susceptible was everyone that had not been in contact with the disease. Immune was everyone that had passed the disease. Dead were those that died, either from the disease or from other causes. Smallpox could either cause death, or cause life-long immunity. So, it is considered one of the SIR family of models, but rather, in this case, SR model.

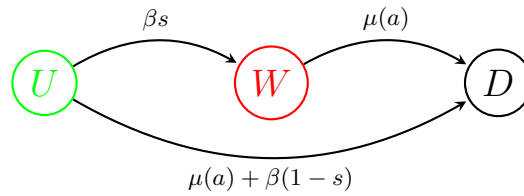
¹As you can see, there is no characterization of those that are currently infected. The reason for this is that the study considered large time scales, such that the actual infectious period was negligible in that scale, and people were considered to transition immediately between susceptible to immune.



Bernoulli imagined that people born in year $a = 0$, could go through the states (un-infected, immune, dead) with certain probabilities ² The dynamics relied on the following dynamic parameters:

- $\mu(a)$ the rate of death of all causes except smallpox, for an individual of age a
- β is the **force of infection**: the rate at which susceptible individual get infected
- $c = 1 - s$ the fraction of individuals infected that dies of smallpox.

The Markov chain is described graphically as this:



The relevant quantities (dynamic variables) of the model depend on the age of the persons a . They are:

- $u(a)$ the probability for an individual of being alive and susceptible (un-infected) at age a
- $w(a)$ the probability for an individual of being alive and immune at age a
- $d(a)$ is the probability of being dead at age a , but since probabilities add up to 1, and there are only three possible states, $d(a) = 1 - u(a) - w(a)$.

²The transitions between this states were described as a Markov chain, although Markov contribution came centuries later.

Considering the rates at which individuals can move from one state to the other, we can compute the contributions of each state in the Master equation. The dynamical system derived by Bernoulli is given by:

$$\begin{aligned}\frac{du}{da} &= -(\beta + \mu(a))u(a) \\ \frac{dw}{da} &= \beta(1 - c)\mu(a)u(a) - \mu(a)w(a)\end{aligned}$$

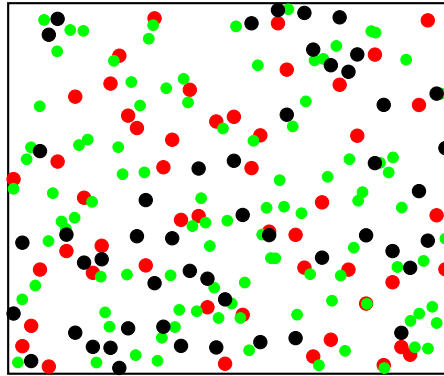
The interest of Bernoulli was comparing the total amount of persons alive at a given age $u(a) + w(a)$ in the case where $c = 0$ (people do not die of smallpox), and $c \neq 0$.

These equations are recognized as one of the first modern mathematical approaches to mathematical epidemiology. Their solution and Bernoulli conclusions is out of the scope of the present course (if interested see [?]). However, its interpretation and derivation are useful to introduce some probabilistic methods and epidemic concepts.

The Master Equation provides a powerful framework for analyzing a wide range of phenomena, including chemical reactions, population dynamics, and the behavior of complex systems. By solving the Master Equation, one can obtain the time-dependent probability distribution of the system, which can be used to calculate various statistical quantities of interest, such as the mean and variance of observables. The Master Equation can also be used to study the long-time behavior of the system, such as the emergence of steady-state solutions and the behavior of fluctuations around these solutions.

1.3 SIR compartmental models

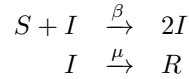
The SIR (Susceptible-Infectious-Recovered) model is a widely used mathematical framework for studying the spread of infectious diseases in populations. It was first introduced by Kermack and McKendrick in a seminal paper published in 1927. They used a system of differential equations to describe the time evolution of the number of individuals in each compartment, and derived an expression for the critical threshold of the disease, above which an epidemic occurs.



A first idealization of what actually happens in an epidemic, is to think of society as a gas of individuals. Each individual can be in one of the following states:

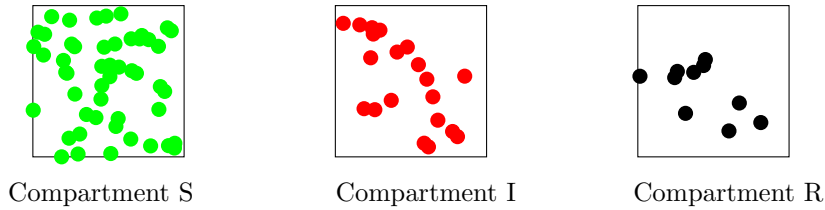
- **Susceptible** individuals, who are at risk of becoming infected;
- **Infectious** individuals, who are capable of transmitting the disease to susceptible individuals;
- **Recovered** individuals, who have either recovered from the disease and are immune, or have died from the disease, and are removed.

Under well mixed assumptions, these individuals-particles move around and interact with each other, occasionally. The process of contagion can be seen as a catalytic chemical reaction, in which the interaction of an Infectious individual with a Susceptible, can induce the latter to turn into infectious with a certain probability:

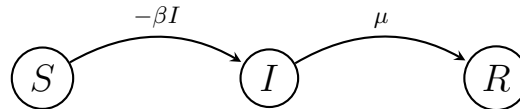


Simulation of this process in a computer, relies on Monte Carlo methods, more precisely in action diffusion equations and Gillespie algorithm, that we will study later.

A mean field probabilistic description can be made, by considering the population to be classified into three different compartments, according to their state:



The evolution of the epidemic corresponds to the process of changing individuals between compartments, with certain probabilities, as represented in the following diagram:



A key difference with the work of Bernoulli is that the **force of infection**: $\beta I(t)$ (the rate at which susceptible individuals contract the disease) is not fixed in time, but proportional to the amount of people infected in the population. This is typical for human-human transmitted diseases in a well mixed population, where the probability of getting infected depends on the chances of meeting

an infectious individual. This probability is proportional to the current amount of individuals in that state.

The dynamic variables for this model are:

- $S(t)$, the fraction of the population susceptible to contracting the disease
- $I(t)$ the fraction that is currently infected and infectious
- $R(t) = 1 - S(t) - I(t)$ the fraction that is recovered or deceased.

The Master equations for the dynamic parameters reads

$$\begin{aligned}\frac{dS}{dt} &= -\beta SI \\ \frac{dI}{dt} &= \beta SI - \mu I \\ \frac{dR}{dt} &= \mu I\end{aligned}\tag{1.2}$$

where, the parameters

- β is the transmission rate or the rate at which susceptible individuals become infected.
- μ is the recovery rate or the rate at which infected individuals recover and become immune.

The first equation describes the rate at which susceptible individuals become infected. It assumes that the rate of transmission is proportional to the product of the number of susceptible individuals and the number of infectious individuals, with the proportionality constant β .

The second equation describes the rate at which infected individuals recover. It assumes that the rate of recovery is proportional to the number of infectious individuals, with the proportionality constant μ . The term βSI represents the rate at which susceptible individuals become infected, and the term μI represents the rate at which infected individuals recover.

The third equation describes the rate at which individuals recover from the disease and become immune. It assumes that the rate of recovery is proportional to the number of infectious individuals, with the proportionality constant μ .

These equations are a set of coupled first-order ordinary differential equations, which means that the values of S , I , and R change over time according to the values of β , μ , and the initial conditions. The SIR model is a simple but powerful tool for studying the dynamics of infectious diseases and predicting the impact of interventions such as vaccination and social distancing.

1.3.1 Short time limit

In the early stages of an epidemic, when the number of infected individuals is small compared to the total population size, the SIR model can be approximated by a linear differential equation. Suppose that the initial number of infected individuals is small compared to the total population size, i.e., $I(0) \sim 1/N$, where N is the total population size. Then, the number of susceptible individuals can be approximated as $S(t) \simeq 1$, and the number of recovered individuals can be approximated as $R(t) \sim 1/N$.

Under these assumptions, the dynamics of the infected compartment can be approximated by the following linear differential equation:

$$\frac{dI}{dt} = \beta S(t)I(t) - \mu I(t), \quad (1.3)$$

where β is the transmission rate, μ is the recovery rate, and $I(t)$ is the fraction of infected individuals at time t . During the initial epidemic phase, when few cases still exists compared to the population size, we can approximate $S(t) = S(0) = 1$, and write the linear equation:

$$\frac{dI}{dt} = \lambda I(t), \quad \text{where } \lambda = \beta - \mu. \quad (1.4)$$

The solution to this linear differential equation is an exponential function of the form:

$$I(t) = I(0) \exp(\lambda t). \quad (1.5)$$

The exponential solution of the SIR model in the low prevalence linear phase shows that the number of infected individuals changes exponentially, growing when $\beta > \mu$, and decreasing when $\beta < \mu$. This exponential growth phase is characteristic of the beginning of an epidemic, when the number of infected individuals is small compared to the total population size and the epidemic has not yet reached a significant proportion of the population.

The basic reproduction number R_0 , is the expected number of new infections created by every single infected individual, in the early exponential phase of an epidemic. Since μ is the recovery rate, $\tau = 1/\mu$ is the expected duration of the infection on an individual. Since β is the rate of new cases, $R_0 = \beta\tau = \beta/\mu$ is the average number of new cases created by an infected individual during the period in which it is actively infectious. Notice that the criteria $\lambda \leq 0$ is equivalent to $R_0 \leq 1$.

1.3.2 Infinite time limit

Properties of SIR equations. Since the total amount of studied subjects remain invariant, only their categories changes, $S(t) + I(t) + R(t) = 1$. This can also be proven by adding (1.2). Furthermore, since

$$S'(t) + I'(t) = -\mu I(t)$$

is a decreasing non negative function, it converges to a limit $S_\infty + I_\infty$ in $t \rightarrow \infty$. Moreover, it is easy to see that I_∞ can only be zero, so $S + I \rightarrow S_\infty$.

Integration of the previous equation results in

$$1 - S_\infty = \mu \int_0^\infty I(t) dt$$

The first number $S_0 + I_0 = 1$ comes from assuming that all the population is originally in the states S or I.

Dividing the equation for $S'(t)$ by $S(t)$, we get

$$\frac{d \log S(t)}{dt} = -\beta I(t) \quad \text{that integrating} \quad \log(S_\infty/S_0) = -\beta \int_0^\infty I(t) dt$$

This results in a relation for the final amount of susceptible individuals

$$\log(S_0/S_\infty) = \frac{\beta}{\mu}(1 - S_\infty) = R_0(1 - S_\infty)$$

that is called the *final size relation* for the epidemic. If the variables are kept extensive $\tilde{S}(t) = NS(t), \tilde{I}(t) \dots$, the final relation reads

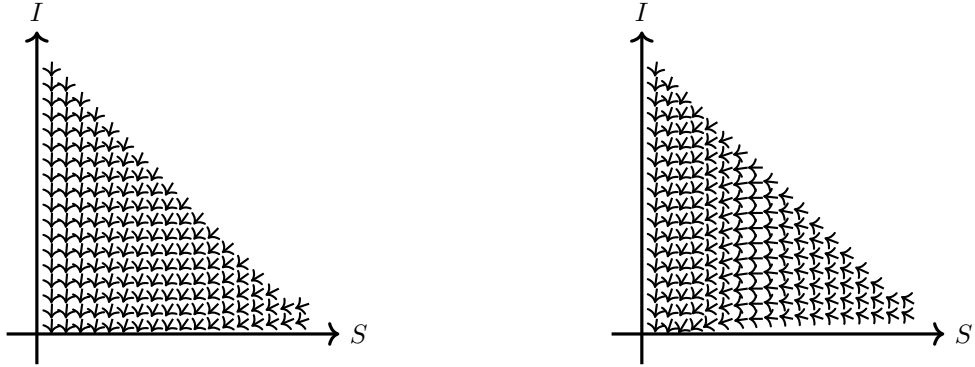
$$\log(\tilde{S}_0/\tilde{S}_\infty) = R_0(1 - \frac{\tilde{S}_\infty}{N}) \quad (1.6)$$

which is a useful relation between the basic reproduction number and the final size of the epidemic.

The total number of infected people in the population is given by $N - \tilde{S}_\infty$, and the fraction $1 - S_\infty$ or $1 - \frac{\tilde{S}_\infty}{N}$ is called the *attack rate*, although it is not a rate, but a fraction.

Another conclusion from (1.6) is that $S_\infty > 0$, and so, in every epidemic there is always a fraction of the population that will not be infected.

While the recovery rate μ is generally simple to estimate from studies on infected individuals. However the contact rate $\tilde{\beta} = \beta/N$ is much harder. Direct calculation of β , or of $R_0 = \beta/\mu$ can be difficult, while a post facto analysis of the epidemic through serological studies, can provide estimation for S_∞ , and through (1.6), to R_0 and β .



A similar procedure as the one we carried, but taken up to time t instead of ∞ , results in a parametric relation between $S(t)$ and $I(t)$:

$$I(t) + S(t) - \frac{\mu}{\beta} \log S(t) = 1 - \frac{\mu}{\beta} \log S_0 \quad (1.7)$$

This trajectories correspond to the flux lines in the diagram of I vs S , using the vector defined by the right hand side of (1.2), normalized.

The maximum number of infectives at any time is the number of infectives when the derivative of I is zero, that is, when $S = \mu/\beta$. This maximum is obtained by replacing this value into (1.7) as

$$I_{max} = I_0 + S_0 - \frac{\mu}{\beta} \log S_0 - \frac{\mu}{\beta} + \frac{\mu}{\beta} \log \frac{\mu}{\beta}$$

1.4 SIS model and epidemic thresholds

1.5 Percolation an epidemics

1.6 And beyond

Chapter 2

Monte Carlo simulation of epidemics

So, epidemics are stochastic processes. Some analytical results exist, for the simplest of cases, as the SIR or SIS dynamics. In more complex cases, simulations are the only tool at hand to study their dynamics.

As we mentioned in last chapter, epidemics can be seen as a set of stochastic reactions, turning healthy people into infectious, and turning them into recovered, at certain rates. Next we discuss how to simulate such types of processes in a computer.

2.1 Rates and exponential distributions

Exponential distribution

As you shall remember (see appendix) from your probabilities class, the exponential distribution is the continuous time limit of a process in which you flip a coin as many times as needed until you find the first “head” coming out ¹. It is clear from the nature of this process, that if a head has not shown up after we toss the coin 5 times, then the probability that it will appear in the next x shots, is the same probability that it had in any x shots, regardless of the fact that we already shot it 5 times. This results in a very important property of exponential distributions: they do not have memory.

The probability density of the exponential distribution is

$$f(x) = \lambda e^{-\lambda x}$$

The cumulative distribution function is therefore

$$F(x) = \int_{-\infty}^x f(x) = 1 - e^{-\lambda x}$$

(defined only for $x > 0$, with $F(x) = 0$ otherwise), and the mean value $\langle X \rangle = \frac{1}{\lambda}$.

¹In appendix of this chapter, you will find a brief reminder of what conditional probability means, and the deduction of the continuous exponential distribution as a limit of a Bernoulli process, if you are interested to check.

memorylessness of exponential random variables

Let's look at an interesting case. Let's prove that the exponential distribution has no memory. This means the following:

$$P(X > s + t | X > s) = P(X > t)$$

This property is called memorylessness because the probability of $X > s + t$ given that $X > s$ is independent of the value of s . Using the definition of conditional probability,

$$P(X > s + t | X > s) = \frac{P(X > s + t, X > s)}{P(X > s)}$$

But $P(X > s + t, X > s) = P(X > s + t)$, since the set $\{x : x > s + t\} \subset \{x : x > s\}$. Note that here the notation $P(A, B)$ corresponds to the probability of both A and B occurring simultaneously, that is, $P(A \cap B)$. Therefore,

$$P(X > s + t | X > s) = \frac{P(X > s + t)}{P(X > s)}$$

The probabilities $P(X > a) = 1 - F(a)$, so $P(X > s + t) = e^{-\lambda(s+t)}$ and $P(X > s) = e^{-\lambda s}$, from which

$$P(X > s + t | X > s) = \frac{P(X > s + t)}{P(X > s)} = \frac{e^{-\lambda(s+t)}}{e^{-\lambda s}} = e^{-\lambda t} = 1 - F(t) = P(X > t)$$

thus concluding the proof.

This property of the exponential density has the following physical implication. The decay of a radioactive atom is a process that occurs with exponential probability. The lack of memory of this probability means that regardless of the past lifetime of an atom, its probability of decaying in the future is always a decreasing exponential.

Law of the minimum of independent exponential variables:

Another fundamental result for the following discussion is the following property of exponential r.v.:

$$\begin{cases} X_i \sim \text{Exp}(\lambda_i), \\ Y_i = \min_i X_i, \\ Z_i = \text{argmin}_i X_i \end{cases} \Rightarrow \begin{cases} Y \sim \text{Exp}(\sum_i \lambda_i) \\ \mathbb{P}(Z = i) = \frac{\lambda_i}{\sum_j \lambda_j} \end{cases}$$

The proof of which is left to the interested student.

2.2 Gillespie's Stochastic Simulation Algorithm

The Gillespie algorithm is a stochastic simulation algorithm that is often used to simulate chemical reaction systems. It simulates the time evolution of a reaction system by randomly selecting which reaction will occur next and when it will occur.

Consider the following chemical reaction system:



where A , B , C , and D are chemical species, and k_1 , k_2 , and k_3 are reaction rate constants.

The interpretation of this list of reactions is the following. We have a system with a certain ammount of elements $X = (n_A, n_B, n_C, n_D)$ at time t . At any moment in time, any of the three reactions can take place. Let us call ΔX_r the vector of the effect that reaction r has on the system, in this case:

$$\Delta X_1 = \begin{pmatrix} -1 \\ 0 \\ 1 \\ 0 \end{pmatrix} \quad \Delta X_2 = \begin{pmatrix} 0 \\ 0 \\ -1 \\ 2 \end{pmatrix} \quad \Delta X_3 = \begin{pmatrix} 1 \\ -1 \\ 0 \\ -2 \end{pmatrix}$$

So, the stochastic process is actually defined by a sequence of growing times $t_{k-1} < t_k$ at which reactions occur, and the index of the reaction occurring:

$$\mathcal{P} = \{(t_1, r_1), (t_2, r_2) \dots\}$$

such that the stochastic variable is given by

$$X(t) = X(t_{k-1}) + \Delta X_{r_k}$$

where k is such that $t_{k-1} < t < t_k$. In other words, the t_k are the moments in which reactions occur, and at those points, the state of the system changes from $X(t)$ to $X(t + dt) = X(t^+) = X(t) + \Delta X_{r_k}$.

So, simulating such stochastic process amounts to defining a way to generate the stochastic squence \mathcal{P} . Furthermore, this process is a Markov process, meaning that the values of (t_k, r_k) depend only on the current state of the system $X(t_{k-1}^+)$.

2.2.1 Gillespie's algorithm

To simulate this system using Gillespie's algorithm, we first define the propensity functions for each of the three reactions:

$$a_1 = k_1[A][B] \tag{2.4}$$

$$a_2 = k_2[C] \tag{2.5}$$

$$a_3 = k_3[D]^2[B] \tag{2.6}$$

where $[A]$, $[B]$, $[C]$, $[D]$, and $[E]$ are the concentrations of the corresponding chemical species.

We then randomly select the time τ until the next event occurs and the reaction that will occur next. The probability that reaction i will occur next is given by:

$$P_i = \frac{a_i}{\sum_j a_j} \tag{2.7}$$

where the sum is taken over all reactions.

Once we have determined which reaction will occur next, we update the concentrations of the chemical species according to the stoichiometry of the reaction. This is summarized in the algorithm 2.2.1.

This example illustrates how Gillespie's algorithm can be used to simulate the time evolution of a simple chemical reaction system. By defining the propensity functions and randomly selecting the next event, we can simulate the stochastic behavior of the system and gain insight into its dynamics.

Algorithm 1

Gillespie SSA

```

1:  $t = 0$ 
2:  $X(0) = X_0$  ▷ Set initial state
3: while  $t < t_{end}$  do
4:    $\text{Update}(a_i)$  ▷ Calculate each reactions propensity
5:    $a_0 = \sum_i a_i$  ▷ Total propensity
6:   for  $i = 1 \dots K$  do ▷ Compute probabilities for every reaction
7:      $P_i = \frac{a_i}{a_0}$ 
8:    $r_1 \sim U(0, 1)$  ▷ Generate random numbers,  $r_1$  and  $r_2$ 
9:    $r_2 \sim U(0, 1)$ 
10:   $\tau = \frac{1}{a_0} \ln \frac{1}{r_1}$  ▷ Time until the next reaction
11:   $k = i | P_{i-1} < r_2 < P_i$  ▷ Next reaction with probability  $P_i$ 
12:   $X(t + \tau) = X(t) + \Delta X_k$ 
13:  Set  $t = t + \tau$ 

```

2.3 Practical Python class

In this practical class we will program the Gillespie algorithm for stochastic simulations in Python. I will share with you a Jupyter Notebook.

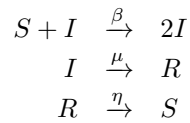
Once the algorithm is running, we will test it in three different models:

1. Drunken man model
2. SIR model
3. SIS model

In the first two cases, we will compare the results of the simulation with the known analytical solution.

2.4 Practical Homework

Consider the model SIRS, as follows:



1. Make a simulation of a system of $N = 1000$ individuals with parameters $\beta = 0.02$, and $\mu = \eta = 0.01$. Use as initial condition $(n_S, n_I, n_R) = (N/3, N/3, N/3)$.
2. By visual inspection in your previous simulation, define a value of T where the system is at equilibrium (approximately not changing).
3. Study the long time behavior of this system for a range of values of $\beta \in (0.001, 0.002, 0.003, \dots, 0.009, 0.01, 0.011, 0.012, \dots, 0.02)$