How does Infectious Disease Propagate?

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1 Introduction

1.1 Topic Relevance

There are many problems that exist in this world that require in-depth research to understand and solve. One such issue is the study of how infectious disease propagates; epidemiology. There are diseases that are deadly enough that they cause international concern with regards to population loss and economic loss. If an outbreak occurred in Canada, it would cost the country an estimated $8.3 billion (CAD) which translates to $8.7 billion (CAD) in today's money. (Diener et al., 2016). Typically, such deadly diseases have high mortality rates. For some perspective, the H1N1 pandemic in 2009 caused 152K – 575K deaths within the first year with mortality rates of 80% for ages 65 and younger and 80% - 90% for people older than 65 (CDC, 2019). Even influenzas that are not as deadly as H1N1 have serious consequences. In a 2018 article by The New York Times, it is claimed that about 80 000 Americans had flu-related deaths in the 2017 winter (McNeil, 2018). The plague has remerged again, where it was reported that two people in China contracted the Pneumonic plague (Wee et al., 2019). Considering the high mortality rates of some deadly diseases and that there is an increasing global interest in urbanization, it is thus crucial to carry out an in-depth analysis of how airborne infectious disease propagates in metropolitan areas.

1.2 Modelling Methods

Classically, epidemiology studies are modelled using the SIR (Susceptible, Infected and Recovered) model. The SIR model was developed by Kermack and Mckendrick in 1927 and is still a widely accepted model for epidemic assessment (Satorras, 2015). There have been recent updated versions of the SIR model for example Gakkhar and Negi proposed the SIRS (Susceptible, Infected, Recovered and Susceptible) model in 2008 while Lekone and Finkenstädt proposed the SEIR (Susceptible, Exposed, Infectious and Recovered) in 2006. There have also been more advanced modern models such as the 8-state ICSAR (Ignorance, Information Carrier, Information Spreader, Information Advocate, Removal) model by Zhang (2014). However, even with all these newer or updated models they either take long times to simulate a result or they do not account for static and dynamical factors such as the influences to propagation due to public transit, confined spaces, risk sources, biological

effects and environmental influences (Zhang, 2016). By considering both static and dynamical factors, there will be a comprehensive risk analysis of airborne infectious disease propagation.

As anticipated, Zhang (2016) did their comprehensive risk analysis using the popular SIR model by including these static and dynamical factors. In addition to the comprehensive analysis, a sensitivity analysis will be discussed to look in more detail the impacts of public transportation and risk source locations have on disease propagation. For example, the effects of a subway system on disease transport and the implications of workplaces and homes as an incubator for disease growth.

2 Flow Diagram and Equations

2.1 Flow diagram and comprehensive risk

Fig. 1 shows the flow diagram of how the infectious disease propagates in a 1 km x 1 km grid where the grid represents a specific environment. It is possible to think of the left j grid, as the environment before the infectious disease takes place; hence the t – 1 time step. As one would assume, there are $N_{inf}(t-1,j)$ number of infected individuals, $N_{non}(t-1,j)$ number of non-infected individuals and $N_r(t-1,j)$ number of recovered individuals in grid j. Once the disease is introduced in this environment, there will be $\Delta N_{inf}(t,j)$ number of non-infected that will now be infected and $\Delta N_r(t,j)$ number of infected who have now recovered. Acknowledging that people do not stay in one place for long, the newly infected, non-infected and recovered individuals will establish the current j grid on the right (Fig. 1) when the disease is in play. Following the same logic, people will leave the newly established j gird and move into the i grid where the movement is denoted as (t, j, i) (Fig. 1). Subsequently, people from i grid can move into j grid which is denoted as (t, i, j) (Fig. 1).

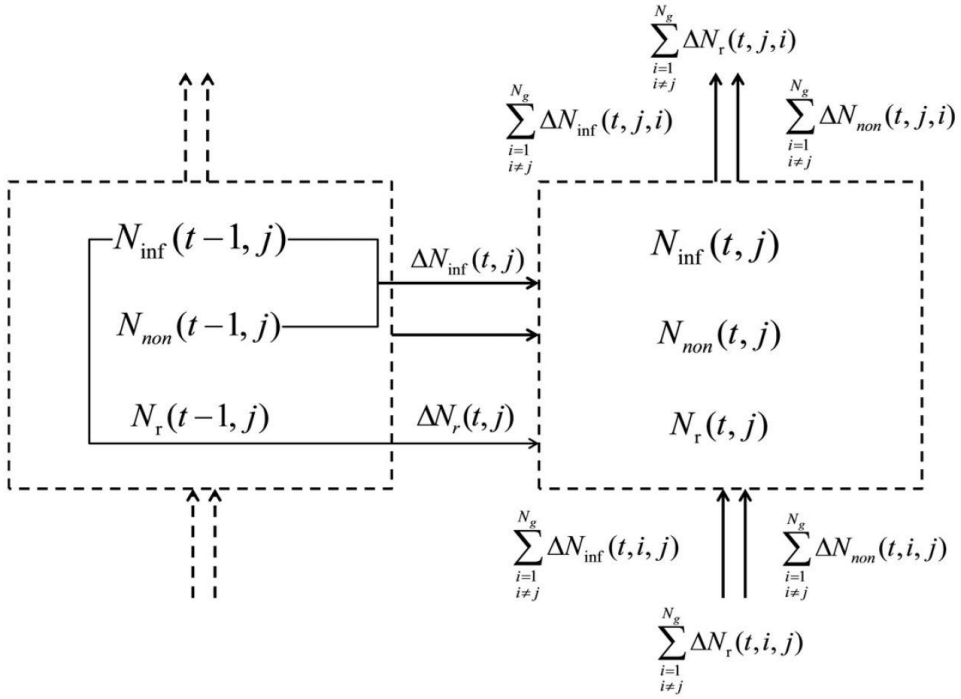

Figure 1: Infectious disease propagation through ith and jth grids (Zhang, 2016).

As a result of Fig. 1, $N_{inf}(t,j)$, $N_{non}(t,j)$ and $N_r(i,j)$ can be calculated using equations [1] – [3] (Zhang, 2016):

$$N_{inf}(t,j) = N_{inf}(t-1,\, j) + \Delta N_{non}(t,\, j) - \Delta N_r(t,\, j) + \sum_{\substack{i=1\\ i\neq j}}^{N_g} \Delta N_{inf}(t,\, j,\, i) - \sum_{\substack{i=1\\ i\neq j}}^{N_g} \Delta N_{inf}(t,\, i,\, j) \quad [1]$$

$$N_{non}(t,j) = N_{non}(t-1,\, j) + \Delta N_{inf}(t,\, j) + \sum_{\substack{i=1\\ i\neq j}}^{N_g} \Delta N_{non}(t,\, i,\, j) - \sum_{\substack{i=1\\ i\neq j}}^{N_g} \Delta N_{non}(t,\, j,\, i) \quad [2]$$

$$N_r(t,j) = N_r(t-1,\, j) + \Delta N_r(t,\, j) + \sum_{\substack{i=1\\ i\neq j}}^{N_g} \Delta N_r(t,\, i,\, j) - \sum_{\substack{i=1\\ i\neq j}}^{N_g} \Delta N_r(t,\, j,\, i) \quad [3]$$

Equations [1] – [3] are formulated by using the concept of mass conservation; where the positive terms can be thought of as things entering the j grid whilst the negative terms are things leaving the j grid. The units of N whether it be for infected, non-infected or recovered is (Zhang 2016):

$$[N] = \frac{infected\ person}{\min \cdot grid}$$

As asserted before, we are interested in finding a comprehensive risk analysis of airborne infectious disease in a metropolis. Thus, the comprehensive risk is defined as ΔN_{inf}. The comprehensive risk is displayed as such (Zhang, 2016):

$$\Delta N_{inf} = \frac{N_{inf}(t-1,\, j)\cdot N_{non}(t-1,\, j)\cdot \sigma I \cdot f(T)\cdot f(RH)}{R_B(t-1,\, j)} + N_h(t,\, j) \quad [4]$$

2.2 Viral infectivity

A variable in eq. [4] worth mentioning is the viral infectivity (I). Viral Infectivity is the infectivity rate that depends on the virus or disease and its particle size.

2.3 Temperature influence

The temperature distribution, f(T), is given by (Zhang, 2016):

$$f(T) = 1.34775 + 0.13811\cos(0.1509T) + 0.29952\sin(0.1509T) \quad [6]$$

2.4 Humidity influence

The humidity influence is expressed by eq. [7] (Zhang, 2016):

$$f(RH) = 1 = 0.005 \cdot RH \quad [7]$$

2.5 Personal biological resistance

Personal biological resistance (R_B), seen in eq. [4] is found using eq. [7] (Zhang, 2016):

$$R_B = G \cdot f(A) \cdot \frac{f(E)}{S \cdot C} \quad [8]$$

Eq. [8] includes the following parameters: age (A), chronic illness status (C), education level (E), gender (G) and smoking status (S) (Zhang, 2016). Someone's age influences airborne diseases as a result the function that describes its influence is shown in eq. [9] (Zhang, 2016):

$$f(A) = 0.906 + 0.0084A - 0.00014A^2 \quad [9]$$

Likewise, education dictates a person's ability to fend off the infectious disease either through voluntary vaccination or avoiding sources of infectious disease (Zhang, 2016). Thus, the education degree (in years) a person has is expressed as:

$$f(E) = 0.5759 \cdot e^{-\left(\frac{E-12.09}{11.33}\right)^2} \quad [10]$$

Before the infectious disease outbreak, if an individual does not have pre-existing chronic illnesses, they are said to have higher resistance to infectious disease compared to those who have pre-existing illnesses (Zhang, 2016). However, chronic illness is influenced by gender, where someone lives and their age. As such, it is postulated that values for chronic illness probability (C_1) vary depending on the factors stated above (Zhang, 2016).

One of the factors that influence the probability of contracting a chronic illness is air pollution. Particulate matter that has a typical diameter of 10 μm or smaller are the air pollutants that are the most common particulate in recurring respiratory diseases (Zhang, 2016). Research done by Ezzati and Kammen on the influence of air pollution is summarized in eq. [11] (Zhang, 2016):

$$f(PM_{10}) = \begin{cases} 1 & 0 \le PM_{10} \le 200 \\ 1.65 & 1.65 \le PM_{10} \le 200 \\ 1.87 & 1.87 \le PM_{10} \le 200 \end{cases} \quad [11]$$

Thus, the comprehensive risk equation's chronic illness (C) is found using eq. [12] (Zhang, 2016):

$$C = C_1 \cdot f(PM_{10}) \quad [12]$$

Finally, the last variable to consider in eq. [8] is the smoking status (S). Smoking cigarettes supresses an individual's immune system and as a result the individual will be more susceptible to infectious diseases. Therefore just like chronic illness, S-values take up different values for different factors: gender, age and location either urban or rural (Zhang, 2016).

2.6 Risk sources

There is an extra term in eq. [4], $N_h(t, j)$, that is known as the number of non-infected individuals that got infected in the hospital. This term contains information about how different factors influence the comprehensive risk analysis: factors such as hospital size, total number of hospitals, relative intimacy of patients in the hospital, probability of isolation, etc. Zhang formulates this term as follows (Zhang, 2016):

$$\triangle N_{inf}(t,j) = \frac{\beta N_{inf}(t,j) \cdot N_{non}(t,j) \cdot \sigma I}{\overline{R_B(t,j)}} + \frac{D_h(j)}{\sum_{i=1}^{N_h} D_h(i)} \quad [13]$$

The $\Delta N_{inf}(t,j)$ term indicates the risk the hospital possess in grid j at time t. β is the probability an infected individual will be put in isolation at a hospital.

2.7 Study area

The metropolatian area the study was carried out on was Beijing, China (Zhang, 2016). With a population size of about 20 million people it has a very high population density for rural and urban areas: 550 people/km^2 and 9000 people/km^2 respectively (Zhang, 2016).

2.8 Initial assumptions

In this study, the disease that causes the outbreak is assumed to be some diverse influenza that does not discriminate in any way; it targets everyone and not just attacks a certain population (Zhang, 2016). The virial infectivity is assumed to be 1/100 000 where 1 person is infected out of 100 000 people (Zhang, 2016).

2.9 Overall remarks

As it can be seen, eq. [4] – [13] is how Zhang intends to incorporate the various static and dynamical factors that influence disease propagation: temperature, humidity, education, gender, age, smoking status, etc.

3 Results

3.1 Personal biological influence

Fig. 2 was graphed using eq. [8] (Zhang, 2016). When analysising someone's personal biology, it is evident in Fig. 2a that the rural areas have the greatest personal biological resistance compared to the urban city of Beijing. This means that the rural areas have the greatest resistance to the disease than the city does. However, in Fig. 2b, the risk the disease poses is much higher in the city meaning that people in the city are at a higher probability of getting infected. This end result is due to the higher population density of urban areas and the higher probability of physical contact with other people; infected or not (Zhang, 2016). It is thus important that a city or country prioritize improving their peoples personal biological resistances in areas with high risk to mitigate disease propagation.

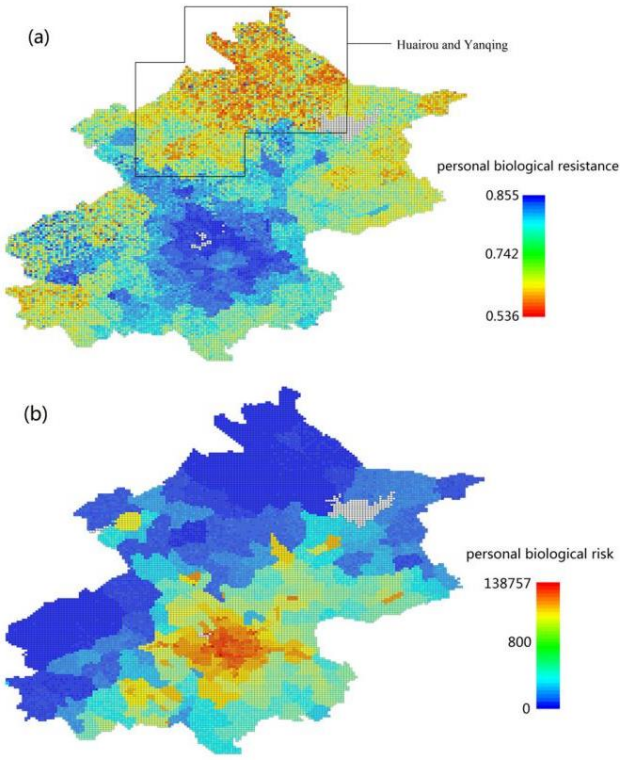

Figure 2: Distribution map of personal biological influences: (a) shows the personal biological resistances to disease; (b) shows the personal biological risk to disease. (Zhang, 2016)

3.2 Temperature – Humidity Infleunce

Using eq. [6] and [7], results in the distribution map seen in Fig. 3 (Zhang, 2016). This distribution map shows that the infectious disease propagation is the least affected by temperature and humidity in urban areas than rural (Northern) areas. If temperature - humidity was the only factored considered, then rural areas should focus on mitigation measures such as better ventilation and heat/humidity diffusion methods to lower the spread of the infectious disease.

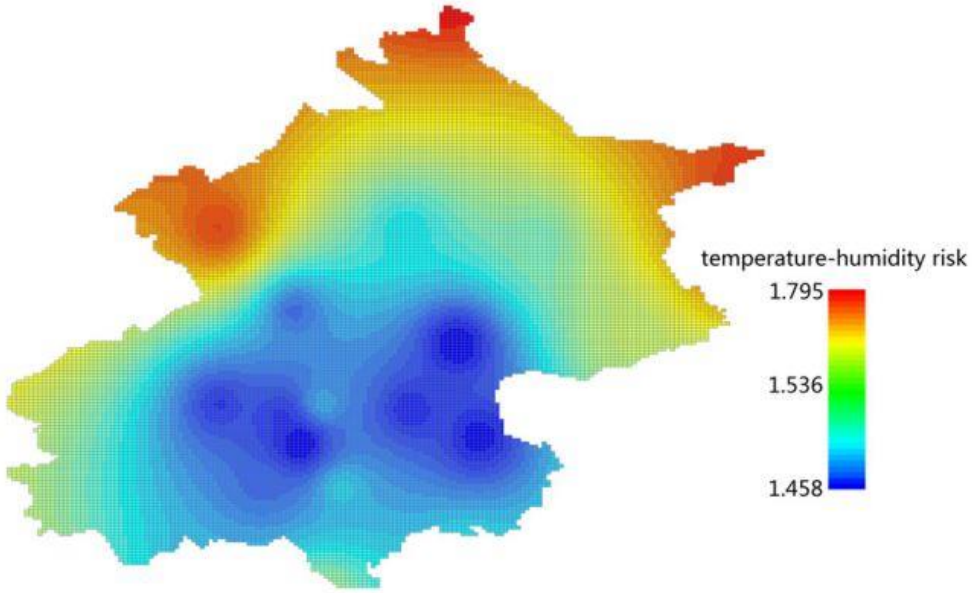

Figure 3: The temperature – humidity risk distribution map of Beijing (Zhang, 2016).

3.3 Overall comprehensive risk

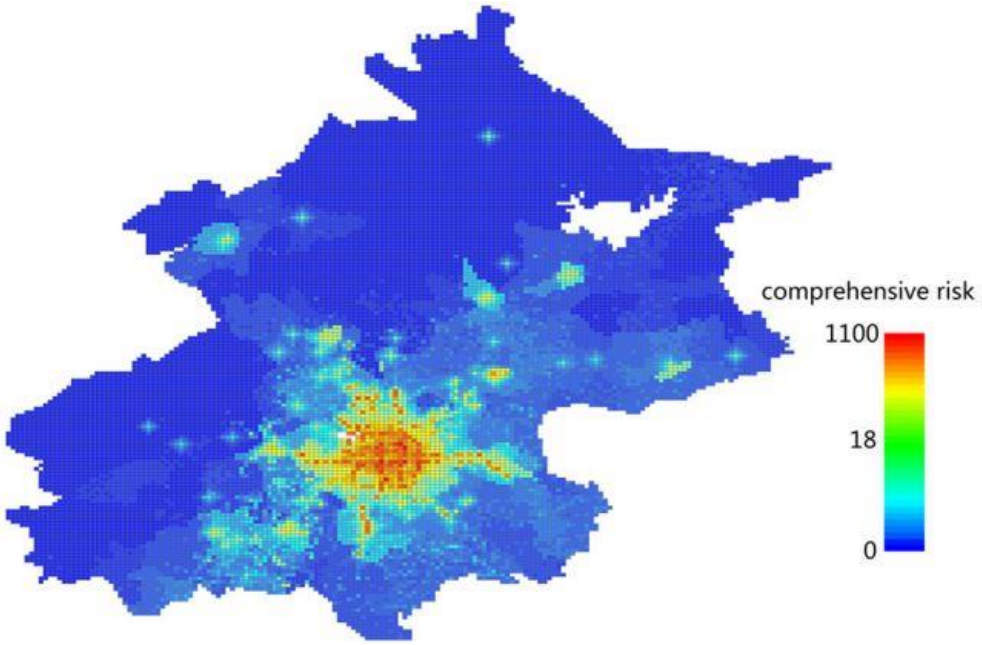

Figure 4: Comprehensive risk distribution map, incorporating all factors, for airborne infectious disease propagation (Zhang, 2016).

If all factors that could influence how an infectious disease propogates are considered, then eq. [4] would result in the distribution map seen in Fig. 4 (Zhang,

2016). As expected, the city centre has the highest risk to airborned infectious disease and as it spreads towards the rural areas, the risk discreases. This conclusion is thus very useful for governments in determing where they should allocate resources to maximize mitigation of infectious disease propagation.

3.4 Sensitivity Analysis of Key Factors

3.4.1 Influence of different public transportation and locations

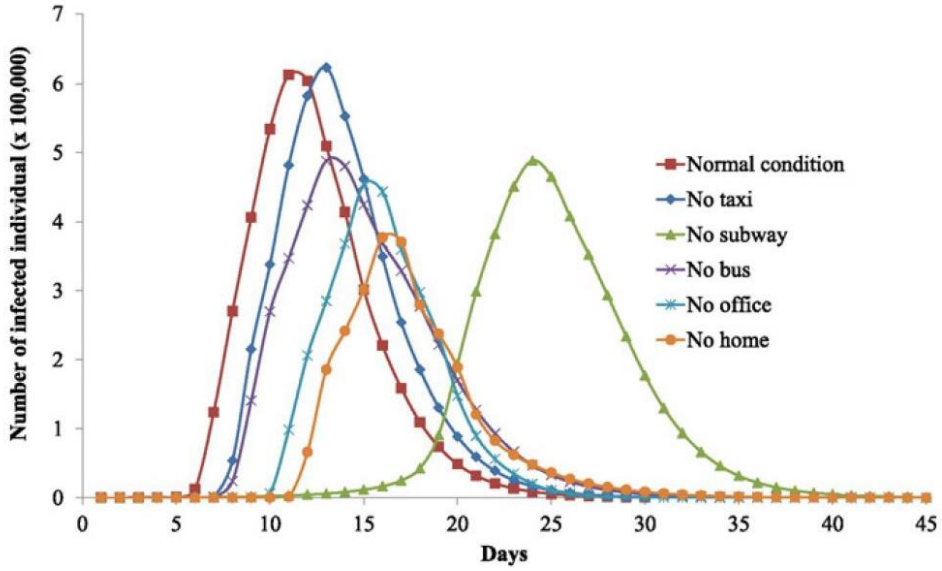

Figure 5: Sensitivity analysis of different forms of transportation and risk sources (homes & offices) have on disease propagation (Zhang, 2016).

An interesting analysis done by Zhang is that when the Beijing subway system is removed as a influence on infectious disease propagation, it results in the green curve seen in Fig. 5. The green curve shows that without the subway system that the number of infected is reduced and that the time it takes for someone to get infected is prolonged compared to the red curve when the subway was included as an impact. Considering that subway systems like the Toronto Transit Commission (TTC) in Toronto spans great distances in a short time, then it makes sense that the transit system acts like a catalyst in disease propagation by lowering travel times and travel distances. Another interesting curve worth mentioning is the orange curve; when residential areas (homes) are removed from the equation. As seen in Fig. 5, the removal of homes greatly lowers the number of infected and also prolongs the time it takes for someone to get infected;

however not as prolonged as the green curve. A hypothesis of why homes have such a high influence is due to the closer vicinity of individuals within a confined space; this closer proximity makes disease transfer much easier and faster.

3.4.2 Sensitivity analysis of government response time

Ana analysis was done on how government response time affects infectious disease propagation. It showed that if the government responded to the outbreak within 3 days compared to 7 days, the number of infected decreased significantly from 70 000 infected to 30 000 infected, respectively (Zhang, 2016). In addition, the time it took for people to get infected increased from 6 days to get infected to about 12 days to get infected, respectively (Zhang, 2016). Thus to have a greater mitigation of the impacts of airborne infectious disease, is to have an immediate response to the outbreak and to begin mitigation procedures as quickly as possible.

4 Conclusion

Overall, there are four key factors that dictate infectious disease propagation: environmental influences, personal biological influences, risk sources and active population flow. Research has shown that certain secondary factors act as a catalyst to the movement of disesases within a metropolis; secondary factors such as subway systems and the close confined space of residential areas. In fact, sensitivity analysis (Fig. 5) show that taxi's have the least influence of disease spread. This can be extrapolated to say that cars, in general, play a very insignificant role in disease transmission. As a result, mitigation measures such as stopping or better managing of all public transit would greatly lower the rate of spread of disease within a metropolis. Seeing that homes act like an incubator for disease spreading, measures should be put in place to lower personal contact, in the time of disease spread. Hospitals are another risk source environment where infected and non-infected are congregate into one area, further increasing the risk of spread. Thus, making it key to have proper hospital managing to isolate the infected and have strict quarantine procedures to lower the risk of spread. As like any other kind of natural disaster, immediate response by government and emergency responders is crucial at mitigate the scale and severity of the epidemic. In summation, there was overwhelming evidence showing that city centres pose the most risk for disease spread.

5 Other Research

5.1 Computational fluid dynamics

Further research can be done to use computational fluid dynamics (CFD) on certain systems to see the impacts of how diseases develop within that system and to see what kind of treatments could be done. For example research done by Acuna et al. aim to apply CFD to the cardiovascular in the attempts to see how diseases grow in it. They analyse fluid dynamical concepts such as pressure gradients, shear stresses on the walls of the heart and bloody velocity profiles (Acuna et al., 2018). One of the important features of CFD software is that it allows physicians to implement patient-specific information in their treatment procedures (Acuna et al., 2018); indicating that in the future, CFD may play a key role in diagnosing and treating patients. In an article by Stefania et al. studies how to use CFD to model hemodynamic variations of atrial fibrillation within the heart (2016). Atrial fibrillation is caused by heart diseases specifically targeting valvues.

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