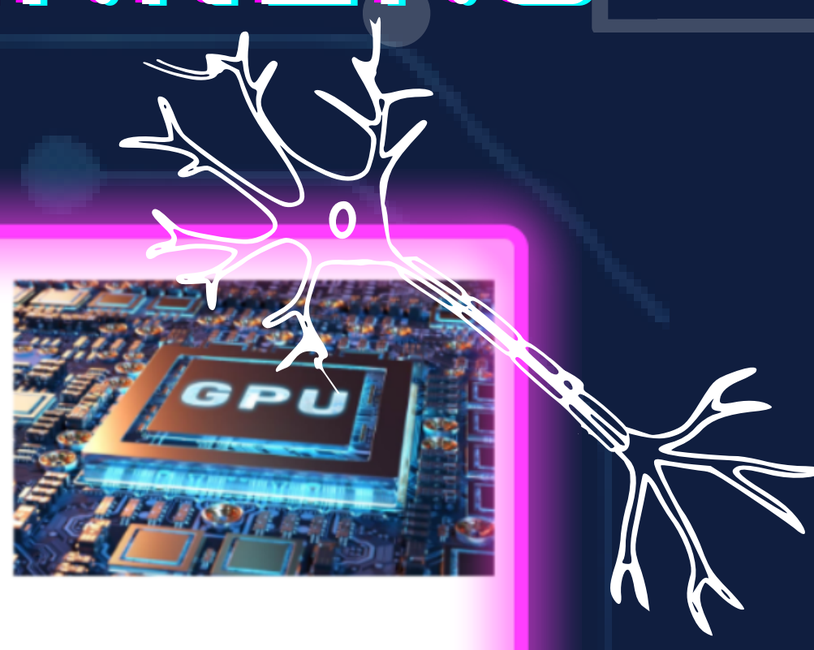
NEURAL NETWORK STRUCTURES: LOGISTIC THINKING AND PATTERNIST THINKING APPROACH

But what is neural networks?
Neural network, are computing systems inspired by the biological neural networks i.e. neurons



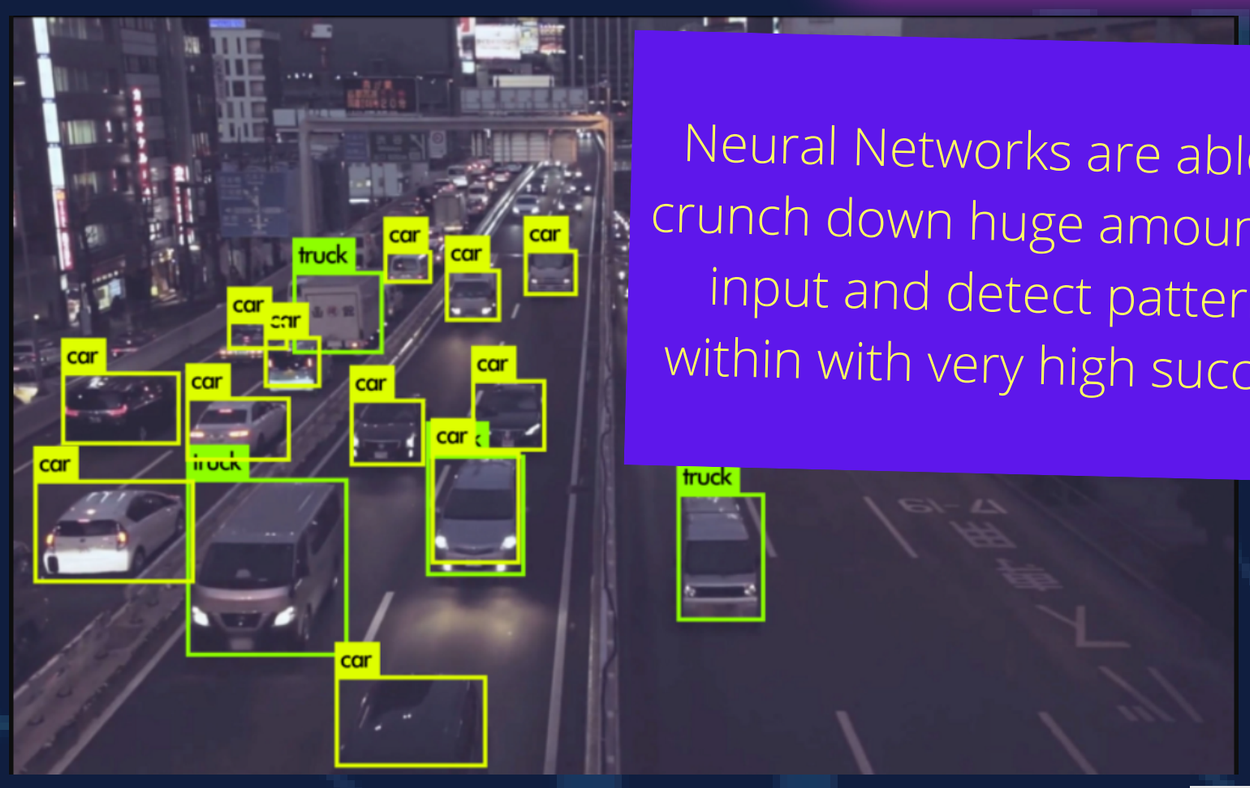
Why bother with Neural Networks?

NN tasks:
- Matrix Multiplication
- Gradient Decent

→ Parallel Computing

→ Faster training time
→ Faster calculation time

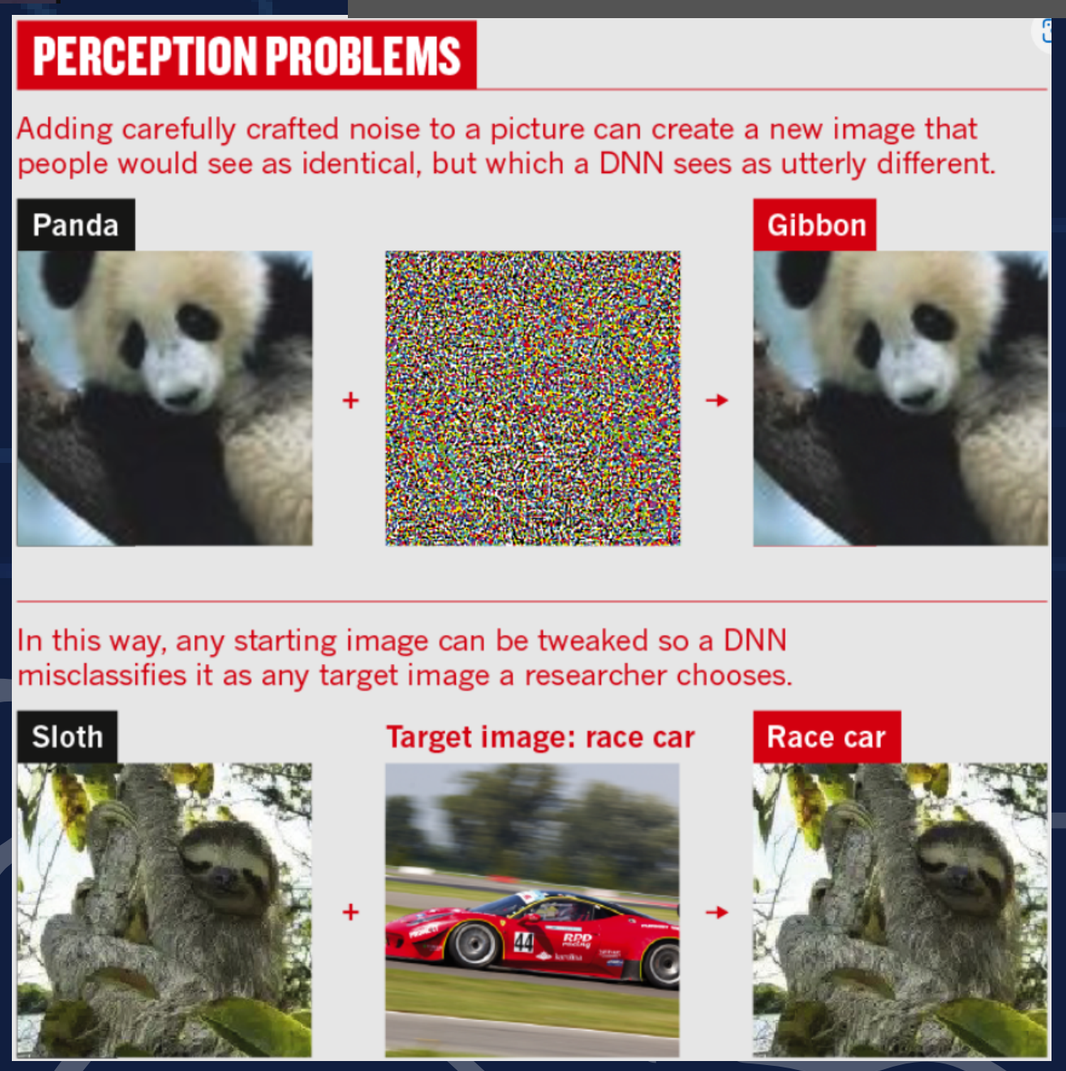
→ Better Accuracy, more reliable program

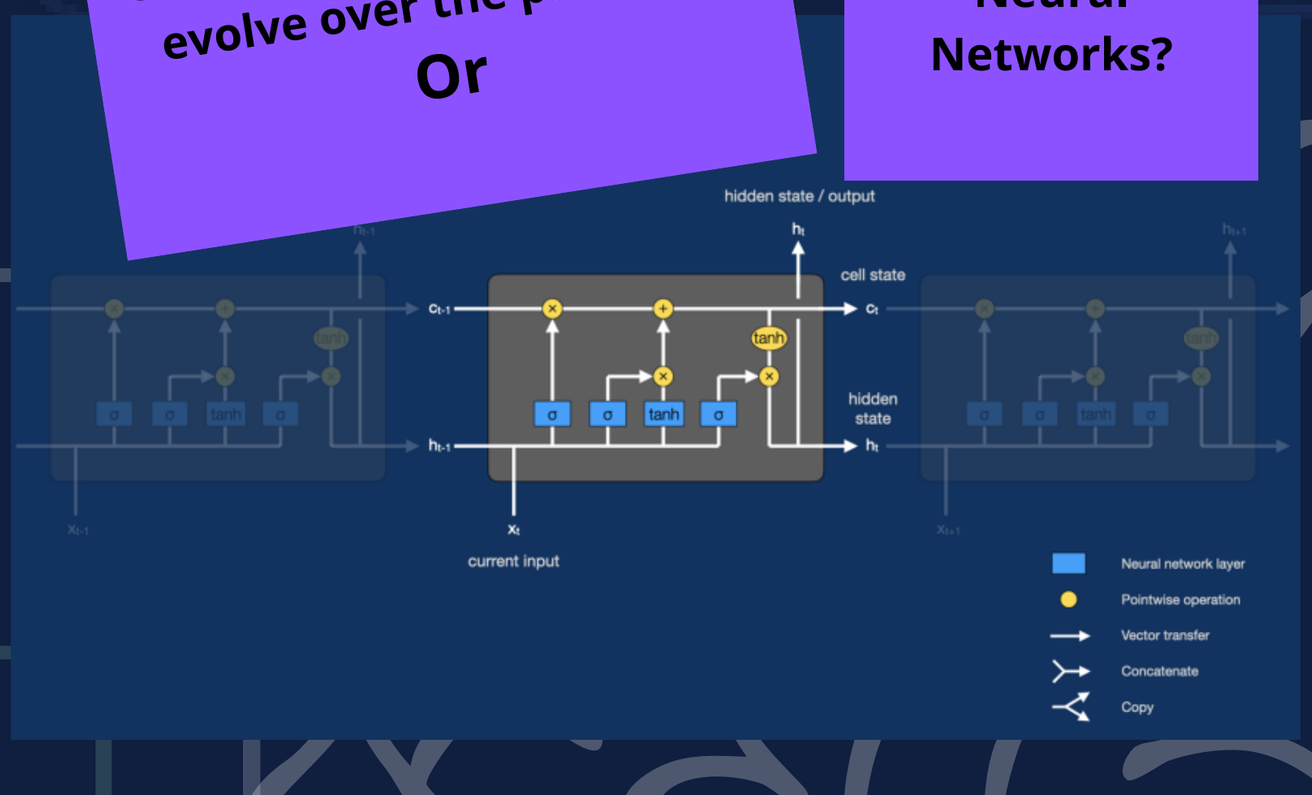


Neural Networks are able to crunch down huge amounts of input and detect patterns within with very high success.

A concerning problem is that Neural Networks is so easy to trick, which begs the question. Where is the fine line between functioning and malfunctioning for it

Is this a problem specified to current models and we will evolve over the problem
Or
Is there a problem in the Basis of how the Neural Networks?





Zhang Shuo
17203839
Supervisor: Associate Prof Dr. Ng Kok Haur

Forecasting Volatility using financial models

01 Abstract

In the financial markets, the volatility is always associated with monetary policies, exchange rate and global events. As a result, the volatility in financial market is often unpredictable and fluctuant. Nowadays, there are many range-based volatility measures and return-based volatility measures widely used in the world. we propose Parkinson (PK) and Open-to-close (CO) measures to estimate the volatility of the Standard and Poor's 500 (S&P500) between 2011 and 2015 and use Conditional autoregressive (CARR) model to fit the volatilities and obtain the in-sample forecasts and out-of-sample forecasts. Also, different error distributions are used in this paper to illustrate the differences of Akaike Information Criterion, *MSE* and *MAD* between different volatilities and choose the best in-sample forecast and out-of-sample forecast based on these criterions. The results illustrate that the PK measure is a better measure in fitting the volatility and generalized gamma distribution is a better choice as error distribution.

02 Problem statement

The trend of stocks is hard to measure and predict because it is always associated with many factors, such as political and economic factors, industry and sector factors and company performance etc. As a result, there are many different types of volatility models that have been invented by researchers to better forecast the stock price as it is a valuable and reliable model to predict the trend.

03 Objectives

1. To use CARR model measure to fit different volatility measure (CO) measure and (PK) and analyse the result by comparing the Akaike Information Criterion.
2. To generate the in-sample models and out-of-sample forecasts by using the CARR model with different volatility measures.
3. To obtain the different in-sample models and out-of-sample forecasts by using three different error distributions for one particular volatility measure..

04 Methodology

CARR model

$$r_t = \lambda_t \varepsilon_t \quad ,t=1,2,\ldots,T,$$

$$\lambda_t = \beta_0 + \sum_{i=1}^{p} \beta_{1i} r_{t-i} + \sum_{j=1}^{q} \beta_{2j} \lambda_{t-j}$$

Open-to-Close measure

$$\sigma^2{}_{co,t} = (C_t - O_t)^2$$

C_t is the logarithmic closing price on date t,
O_t is the logarithmic opening price on date t

Parkinson measure

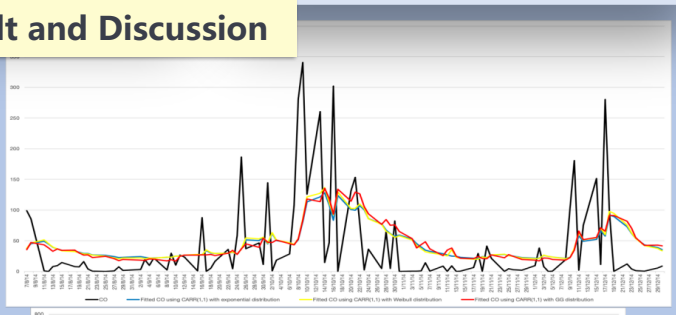
$$\sigma^2{}_{pk,t} = \frac{(H_t - L_t)^2}{4ln2}$$

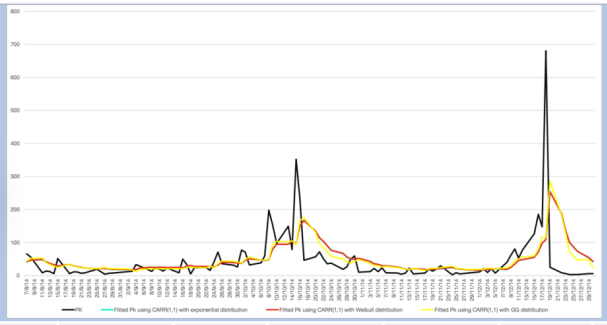
H_t is the logarithmic highest price on date t
L_t is the logarithmic lowest price on date t.

05 Result and Discussion

AIC	8584.430	7963.730	*7961.437*	8619.234	8857.800	*8444.606*
MSE	13602.95	13483.04	*13482.19*	8911.428	8887.981	*8693.496*
MAD	55.32747	55.66942	*55.52548*	39.32484	39.86525	*39.00179*

the CARR model with the generalized gamma distribution of error term generates the lowest AIC value with 7961.437 and 8444.606 for the in-sample forecasts of the CO and PK values respectively. according to MSE and MAD, we find that the generalized gamma distribution in the setting of CARR model obtains the best in-sample forecasting performance as well.





06 Conclusion

PK measure is a more accurate and efficient measure when we want to estimate the volatility, for out-of-sample forecasts, PK measure is doing better in forecasting, because the MAD is smaller compared with MAD of CO measure. This situation also happens in in-sample model, and it is obvious that the MSE and MAD of PK measure are both smaller than those of CO measures.

the generalized gamma distribution is a better choice as of the error distribution, for in-sample models the CO measure fitted by CARR model with generalized gamma distribution has the smallest AIC, MSE and MAD, and the PK measure fitted by CARR model with generalized gamma distribution has the smallest AIC, MSE and MAD as well.for out-of-sample forecasts, the CO measure fitted by CARR model with generalized gamma distribution has the smallest MAD and the PK measure fitted by CARR model with generalized gamma distribution has the smallest MSE and MAD

Loss function	Exponential	Weibull	Generalized gamma	Exponential	Weibull	Generalized Gamma
MSE	4489.355	4434.53	4495.282	6281.451	6291.081	6087.369
MAD	45.13196	44.87622	44.86203	38.30963	38.46638	35.81122

Table demonstrated the MSE and MAD values of the out-of-sample forecasts. For the CO index, we find the CARR model with Weibull distribution generates the best performance under the MSE measure with 4434.53, while it is the generalized gamma distribution that generates the lowest MAD value with 44.86203. For the PK index, the generalized gamma distribution outperforms the others under both the MSE and MAD criteria with 6087.369 and 35.81122 respectively.

07 References (not all references included)

[1] Engle, R. F. (1982). Autoregressive conditional heteroscedasticity with estimates of the variance of United Kingdom inflation. Econometrica, 50, 987–1007.
[2] Bollerslev, T. (1986). Generalized autoregressive conditional heteroskedasticity. Journal of Econometrics, 31, 307–327.
[3] Taylor, S. J. (1986). Modelling financial time series. Chichester: John Wiley and Sons.
[4] Li, H., & Hong, Y. (2011). Financial volatility forecasting with range-based autoregressive volatility model. Finance Research Letters, 8, 69–76.

ACCELERATED NUMERICAL METHOD FOR SOLVING BRAIN TUMOR MODEL

SHIVANESH SIVAKUMAR
171817 73/1

INTRODUCTION

This mathematical project is to compare the Gauss-Seidel and Successive Over Relaxation method to see which method is more effective to study the dynamic of brain tumor

THE HEAT EQUATION

Professor J.D. Murray was the first one to investigate the challenge of monitoring the growth of an invading glioma in the early 1990s. He came up with a conservation equation to solve the problem. He came up with an equation:

$$\frac{\partial c}{\partial t} = (D\nabla^2 c) + \rho c$$

D = diffusion coefficient
p = proliferation rate.

OBJECTIVES

1. To discretise the brain tumor growth model's function using backward time centered space (BTCS) and form a linear system.
2. To determine the growth of the infiltrating brain tumor by using the Successive Over Relaxation (SOR) method in solving the generated system of the linear equations from the heat equation
3. To investigate the performances of the Successive Over Relaxation (SOR) method with the Gauss-Seidel (GS) method in solving the generated linear system.

METHODOLOGY

- Backward Time Centered Space
- Gauss-Seidel
- Successive Over Relaxation

RESULTS

We will discretise the heat equation using the BTCS scheme and form a linear system. Then, solve the linear system using Gauss-Seidel and Successive Over Relaxation method by using the Matlab software since the mesh size is bigger. Calculate the results for 5,10,15 days. The results for 5 days is shown below:

Table 4. 1 Numerical results for $s = 5$ days

N	Methods	k	Time	C_{MAX}
30x30	GS	2712	112.25	4364
	TSGS	1623	16.28	4355
	SOR($w = 1.94$)	996	9.36	4364
60x60	GS	10045	126.16	4413
	TSGS	5640	25.08	4412
	SOR($w = 1.94$)	3833	16.88	4413
120x120	GS	36714	203.32	4439
	TSGS	19047	53.38	4424
	SOR($w = 1.94$)	13498	24.3	4439
240x240	GS	132766	1975.5	4443
	TSGS	62038	194.33	4420
	SOR($w = 1.94$)	48226	100.71	4443

DISCUSSION

The number of iterations and the computational time by the SOR iterative method dropped drastically compared to the GS and TSGS iterative methods. Note that the fewer the number of iterations and the smaller the computational time, the better the performances are. Therefore, as shown from all the figures above, the computational results reveal that the SOR iterative method is more convenient than the GS and TSGS iterative methods.

CONCLUSION

The BTCS discretisation method was successfully developed and used in the heat diffusion equation. The growth of the infiltrating brain tumor was determined by using the Successive Over Relaxation (SOR) method in solving the generated system of the linear equations from the heat equation We also include a comparison of GS, TSGS, and SOR, suggesting that the SOR system is more effective than the GS and TSGS method.

UNIVERSITY OF MALAYA

NAME: OOI MING YANG
MATRIK NO.: 17202815/1

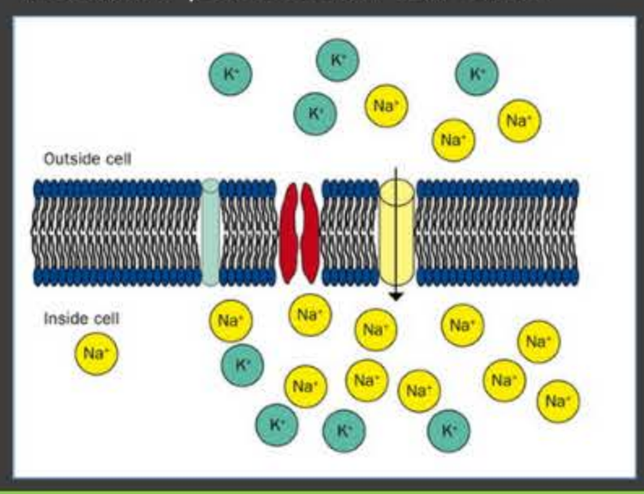
SUPERVISOR: DR MUHAMAD HIFZHUDIN BIN NOOR AZIZ

SIN3015 MATHEMATICAL SCIENCE PROJECT

DETERMINING THE EFFECTS OF ELECTROPHYSIOLOGICAL HETEROGENEITY IN VENTRICULAR CELLS ON CHANNEL BLOCKS

INTRODUCTION

Understanding the effects of this heterogeneity is important, particularly as it can modulate response to drug application. In this work, we conduct a computational study on populations of rabbit cardiomyocytes in order to investigate the effects of variability in cellular electrophysiology to drug blocking of the rapid and slow component of the delayed rectifier potassium current.



This is the cross section of an isolated ventricular cell and ions can passes through the protein channels and creates ionic current in and out of the cell. The voltage across the membrane is the action potential (AP) of the cell.

MOTIVATION & PROBLEM STATEMENT

Conducting the experiment protocol to investigate the effect of cardiac drug on different ventricular cell is very expensive and difficult to be done in large scale. Hence using an existing mathematical model to replicate the real life experiment is important so that we can push on the research on the effect of cardiac drugs due to electrophysiological variability.

OBJECTIVES

1. To investigate the underlying mechanism of cardiac electrophysiological variability.
2. To quantify the correlation between the ionic current and action potential's biomarkers.
3. To study the effects of electrophysiological variability on the response to cardiac drugs in ventricular cells.

SCOPE OF STUDY

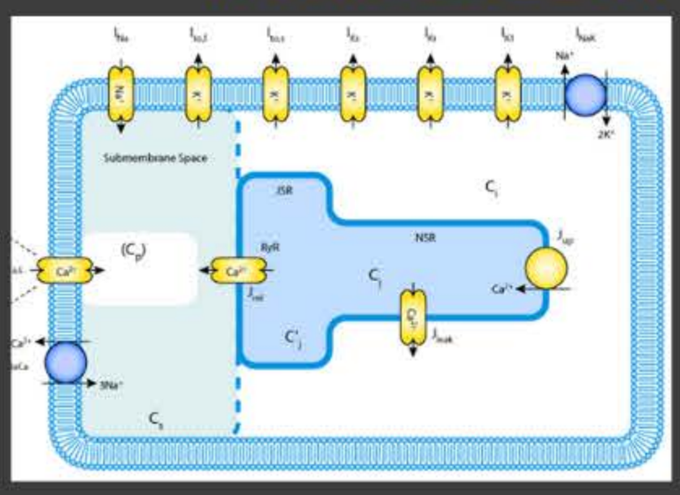
- limit the study within individual isolated ventricular cells
- limit the variability between cell to cell is the density of ionic protein channels

METHODOLOGY

We will use the model developed by Mahajan et al. (2008) to do drug simulation on ventricular cell.

$$\frac{dV}{dt} = -(\sum I_{ionic} + I_{stim})/C_m$$

$$\frac{d\mathbf{y}}{dt} = S(V,\mathbf{y})\frac{d\mathbf{w}}{dt} = R(V,\mathbf{w})$$



METHODOLOGY (CONT.)

1. Generate Parameter Sets

Use Latin Hypercube Sampling to generate parameters that scale from 0.1 to 2

2. Generate Model Population

Simulate control cases for each parameter set by putting them in the model

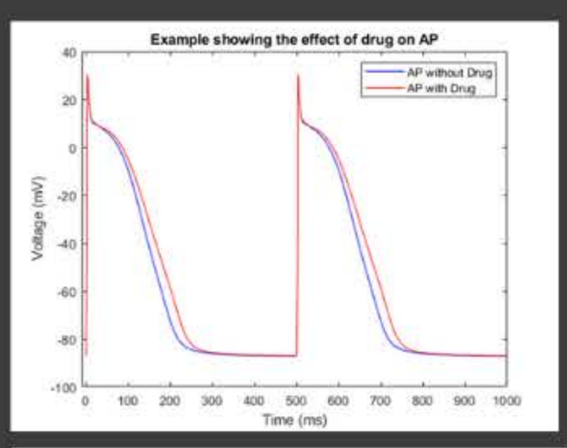
3. Calibrate Model Population

Remove illogical AP and fit the population based on experimental data

4. Drug Simulation

Do drug simulation model with IKr channel blocked at 95% and find the partial correlation coefficient of conductance and biomarkers

RESULT AND DISCUSSION




```
Median of Delta: 21.905174
Total Responses: 294
Total Hyper Responses: 16
Total Normal Responses: 278
Total HR Proportion : 5.442177 %
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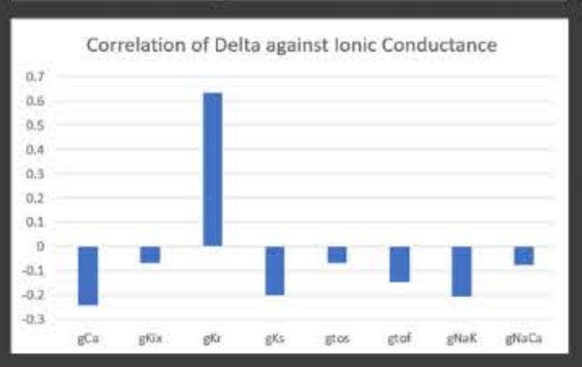


For normal responses, the action potential is prolonged because the IKr channel is blocked, the repolarization phase becomes slower.

Among the model populations, 16 out of 294 models exhibit hyper response towards the drug, which is about 5.44% of the total proportion. For the normal response, the median of delta is about 21.91ms, this verifies the observation above.

Out of all ionic conductance, gKr has the highest correlation with delta, this is because we have use the 95% IKr channel blocker.

CONCLUSION

We have understood the variability of electrophysiological behaviour is due to the different in protein channel that affect the ionic current from cell to cell, found out the correlation between the ionic current and AP biomarkers, where we verify each of the ionic current's role in the AP, and discovered the potential side effect where 5.44% of the population exhibits hyper response towards the drug, and among the normal responses, the median of δ is 21.91ms.

The use of mathematical modelling and numerical simulation can ease the prediction of effect of some drugs and at the same time cut the cost for the research work because we do not have to carry out large scale experimental protocols.

Selected Reference:

Britton, O. J., Bueno-Orovio, A., Van Ammel, K., Lu, H. R., Towart, R., Gallacher, D. J. and Rodriguez, B., 2013. 'Experimentally calibrated population of models predicts and explains intersubject variability in cardiac cellular electrophysiology', Proceedings of the National Academy of Sciences 110(23), E2098–E2105.

EXTENDED SEIR MODEL TO AID DEVELOPMENT OF COVID-19 CONTROL STRATEGIES IN KLANG VALLEY

BY SAFA MALIK

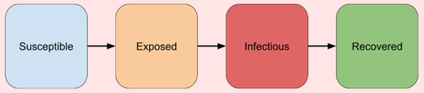
Introduction

COVID-19

- Infectious disease caused by newly discovered coronavirus.
- First reported in Wuhan, China in December 2019.
- Symptoms include fever, cough and shortness of breath
- Infected over 492 million individuals and caused 6 million deaths.

Compartmental Models

- Mathematical models define real world situations in mathematical form such equations.
- SEIR models are commonly used
- Can forecast the progression of infectious diseases
- The SEIR model is a common compartmental model



Objectives

To determine the impact of control strategies, such as screening and isolation, in curbing the spread of the virus.

To observe the effect of vaccination.

To perform mathematical analysis of the model.

Mathematical Analysis

The reproduction number or the R_0 value represents the number of new infections produced by a single infectious individual in a completely susceptible population.

- $R\_0 > 1$: Each infection causes multiple new infections leading to an exponential increase
- $R\_0 < 1$: Each existing infection causes less than one new infection

Using next-generation matrix method, we get the following for our compartmental model:

$$R\_0 = \frac{\beta\varepsilon\gamma\nu * test * (\kappa-1)(q-1)\big(p+\sigma(1-p)\big) * scr^2}{(scr+\alpha*se)(\varepsilon*scr*test+\ \varepsilon\gamma*scr*se+\varepsilon\gamma*se*test-\ \varepsilon\Lambda*scr*test+\gamma\Lambda*scr*test-\varepsilon\gamma*p_{asy}*scr*se)}$$

The following epidemic equilibrium points are found by setting the derivatives to 0:

$E_1 = \left(0,0,0,0,0,\frac{(\delta-\gamma)(\Lambda-1)N}{\beta\delta\gamma\nu\tau(\kappa-1)},0,0,0,0,0\right)$ when $\delta > \gamma$

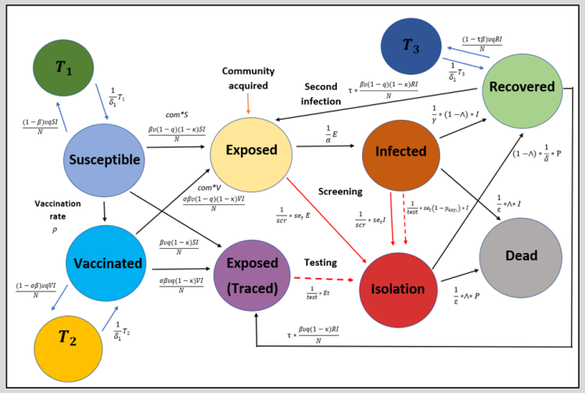
$E_2 = (0,0,0,0,0,0,0,0,0,0,0)$

We construct the Jacobian matrix and find the eigenvalues at each point
For both equilibrium points we get $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8, \lambda_9 < 0$ and $\lambda_{10}, \lambda_{11} = 0$.

The points are stable but not asymptotically stable

Proposed Extended SEIR Model

S – Unvaccinated population who has not been infected
V – Vaccinated population who has not been infected
E – Exposed population who has not been traced
Et – Close contact traced population who will test positive.
I – Population capable of transmitting the disease
P – Population that has been tested positive
R – People that have recovered
D – Population that died as a result of contracting the disease.
T_1**–** Traced close contacts from susceptible population that have tested negative.
T_2 **–** Traced close contacts from vaccinated population that have tested negative.
T_3 **–** Traced close contacts from recovered population that have tested negative



Parameter	
β	Probability that susceptible becomes infectious per contact
υ	Average number of contacts per day per case
q	Proportion of close contacts traced per day
κ	Proportion of exposed persons who performed effective precautions
ρ	Vaccination rate (rate of people who are vaccinated)
com	Daily per capita (per person) community infection
σ	Vaccine inefficacy
$\frac{1}{\alpha}$	Rate of progression from exposed to infectious (α is latent period)
scr	Screening interval (days)
$\frac{1}{\gamma}$	Recovery rate of infectious individuals (1/ infectious period)
δ	Duration of isolation or quarantine (days)
se	PCR sensitivity
$test$	Time from onset of infectiousness to testing (days)
$1-p_{asy}$	Proportion symptomatic
τ	Rate of second infection
Λ	Mortality rate
ε	Average days until death

Differential Equations

$$\frac{dS}{dt} = \frac{1}{\delta_1}T_1 - \frac{\beta\upsilon(1-q)(1-\kappa)SI}{N} - \frac{(1-\beta)\upsilon qSI}{N} - \frac{\beta\upsilon q(1-\kappa)SI}{N} - \rho S - com*S$$

$$\frac{dE}{dt} = \frac{\beta\upsilon(1-q)(1-\kappa)SI}{N} + \frac{\sigma\beta\upsilon(1-q)(1-\kappa)VI}{N} - \frac{1}{scr}*se_tE - \frac{1}{\alpha}E + \tau*\frac{\beta\upsilon(1-q)(1-\kappa)RI}{N} + com*S + com*V$$

$$\frac{dI}{dt} = \frac{1}{\alpha}E - \frac{1}{scr}*se_tI - \frac{1}{test}*se_t\left(1-p_{asy_i}\right)I - \frac{1}{\gamma}*(1-\Lambda)*I - \frac{1}{\varepsilon}*\Lambda*I$$

$$\frac{dP}{dt} = \frac{1}{test}*se_t\left(1-p_{asy_i}\right)I + \frac{1}{scr}*se_t(E+I) + \frac{1}{test}*Et - \frac{1}{\delta}*(1-\Lambda)*P - \frac{1}{\varepsilon}*\Lambda*P$$

$$\frac{dT_1}{dt} = \frac{(1-\beta)\upsilon qSI}{N} - \frac{1}{\delta_1}T_1$$

$$\frac{dR}{dt} = \frac{1}{\gamma}*(1-\Lambda)*I + \frac{1}{\delta}*(1-\Lambda)*P - \tau*\frac{\beta\upsilon(1-q)(1-\kappa)RI}{N} - \tau*\frac{\beta\upsilon q(1-\kappa)RI}{N} - \frac{(1-\tau\beta)\upsilon qRI}{N} + \frac{1}{\delta_1}T_3$$

$$\frac{dV}{dt} = \frac{1}{\delta_1}T_2 - \frac{\sigma\beta\upsilon(1-q)(1-\kappa)VI}{N} - \frac{(1-\sigma\beta)\upsilon qVI}{N} - \frac{\sigma\beta\upsilon q(1-\kappa)VI}{N} + \rho S - com*V$$

$$\frac{dD}{dt} = \frac{1}{\varepsilon}*\Lambda*I + \frac{1}{\varepsilon}*\Lambda*P$$

$$\frac{dT_2}{dt} = \frac{(1-\sigma\beta)\upsilon qVI}{N} - \frac{1}{\delta_1}T_2$$

$$\frac{dT_3}{dt} = \frac{(1-\tau\beta)\upsilon qRI}{N} - \frac{1}{\delta_1}T_3$$

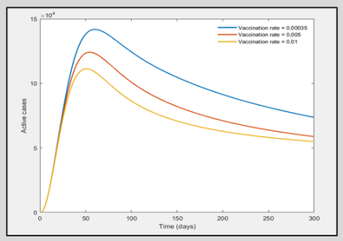
$$\frac{dEt}{dt} = \frac{\beta\upsilon q(1-\kappa)SI}{N} + \frac{\sigma\beta\upsilon q(1-\kappa)VI}{N} + \frac{\tau\beta\upsilon q(1-\kappa)RI}{N} - \frac{1}{test}*Et$$

Assumptions

- The entire population of Klang Valley is divided into 11 compartments.
- The transmission will be monitored for 92 days.
- Natural births and deaths have minimal impact on progression of the disease, so they are not considered in the model.
- Testing will only be conducted on symptomatic individuals.
- Screening involves testing the entire population at fixed intervals irrespective of whether they exhibit any symptoms.
- Individuals infected by the disease and traced will move to move to exposed traced compartment (Et).
- Those who are not traced will move to the Exposed (E) compartment.
- The susceptible and vaccinated individuals that have been traced and tested negative will move to T_1 and T_2 respectively.
- The recovered population can be exposed to the disease again with a lower transmission rate.

Parameter Sensitivity Analysis

- We examine the effect of changing parameter values.
- Altering more sensitive parameters will lead to large changes.
- We can understand the association between compartments and variables.
- This is also a way of testing the model as we may uncover unexpected relationships.



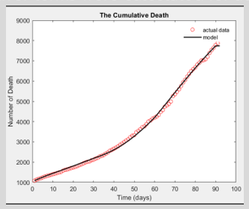
Effect of changing vaccination rate on the active cases:
- When the vaccination rate is 0.00035 $day^{(-1)}$, the peak number of active cases is approximately 14.5×10^4 cases.
- When the vaccination rate is 0.01 $day^{(-1)}$, the peak number of cases reduces to 11×10^4 cases.

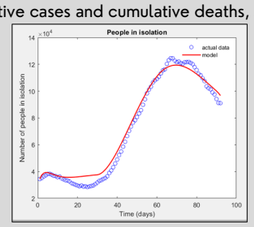
As vaccination rate increases, more individuals move from the susceptible to vaccinated compartment. Since the transmission rate is lower, they are protected from being infected which reduces the number of active cases and herd immunity is achieved quicker.

Optimisation

Data such as active cases, vaccination rates and cumulative deaths was collected from **Klang Valley** which consists of Selangor, Kuala Lumpur and Putrajaya, for the months June to August, 2021. We wish to fit the model to the real data.

Optimisation is performed using Nelder-Mead algorithm.
We estimated the parameters β and Λ.
The built-in function 'fminsearch' is used to find the minimum of the error function.
Error terms were calculated for active cases and cumulative deaths, using residual sum of squares.





$$\Lambda = \begin{cases} 0.0125 & ,1 \le t \le 30 \\ 0.0113 & ,30 < t \le 60 \\ 0.0133 & ,60 < t \le 92 \end{cases} \qquad \beta = \begin{cases} 0 & ,1 \le t \le 30 \\ 0.0446 & ,30 < t \le 60 \\ 0.0399 & ,60 < t \le 92 \end{cases}$$

- The transmission rate increases in July 2021, which may be due to the low vaccination rate and lack of adherence to the movement control order.
- Since the cases increased, the enhanced movement control order was introduced in areas of Klang Valley.
- This along with more people receiving vaccination may have reduced the transmission rate for August 2021.

Conclusion

- COVID-19 has had a significant impact worldwide.
- We constructed an extended SEIR model to monitor the progression of the disease, predict the likely conclusion and assist in developing public health measures.
- Data was collected from Klang Valley to estimate the transmission and mortality rates.
- In order to prevent such high active cases and deaths, more screening and higher vaccination rates required. Parameter sensitivity analysis showed that these had an impact on the active cases and cumulative deaths.
- Policymakers need to consider appropriate screening intervals and vaccination rates by estimating the costs etc.

As shown during the mathematical analysis, the R0 value is dependent on several parameters such as proportion of close contacts traced. The aim is for the R0 to be less than 1, so measures such as efficient contact tracing will help minimise the impact of the virus.