PhD Diary Entry for week beginning October 15th 2018

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1 Tasks

1.1 TODO Read Introduction to Diffusion Modelling

- http://www.mathematica-journal.com/2012/03/diffusion-modeling/
  - This could be v.good to try and translate into python as an exercise / practice

1.1.1 TODO Revise Partial Diff Equations

- https://www.math.uni-leipzig.de/~miersemann/pdebook.pdf
- http://tutorial.math.lamar.edu/Classes/DE/TheHeatEquation.aspx

1.2 TODO Random Walk / Diffusion tutorials

- http://courses.washington.edu/matlab1/Lesson_18.html
- http://www.math.cmu.edu/~shlomo/courses/BioSystems/Lectures/RandomWalk.pdf

1.3 Euler's method

1.3.1 Differential equations revision

- $y'' + 2y' = 3y$
- $f''(x) + 2f'(x) = 3f(x)$
- $\frac{d^2y}{dx^2} + 2\frac{dy}{dx} = 3y$

Solution to diff equation: function(s). Whereas algebraic equations have a number or set of numbers as their solution

1.3.2 Derivatives

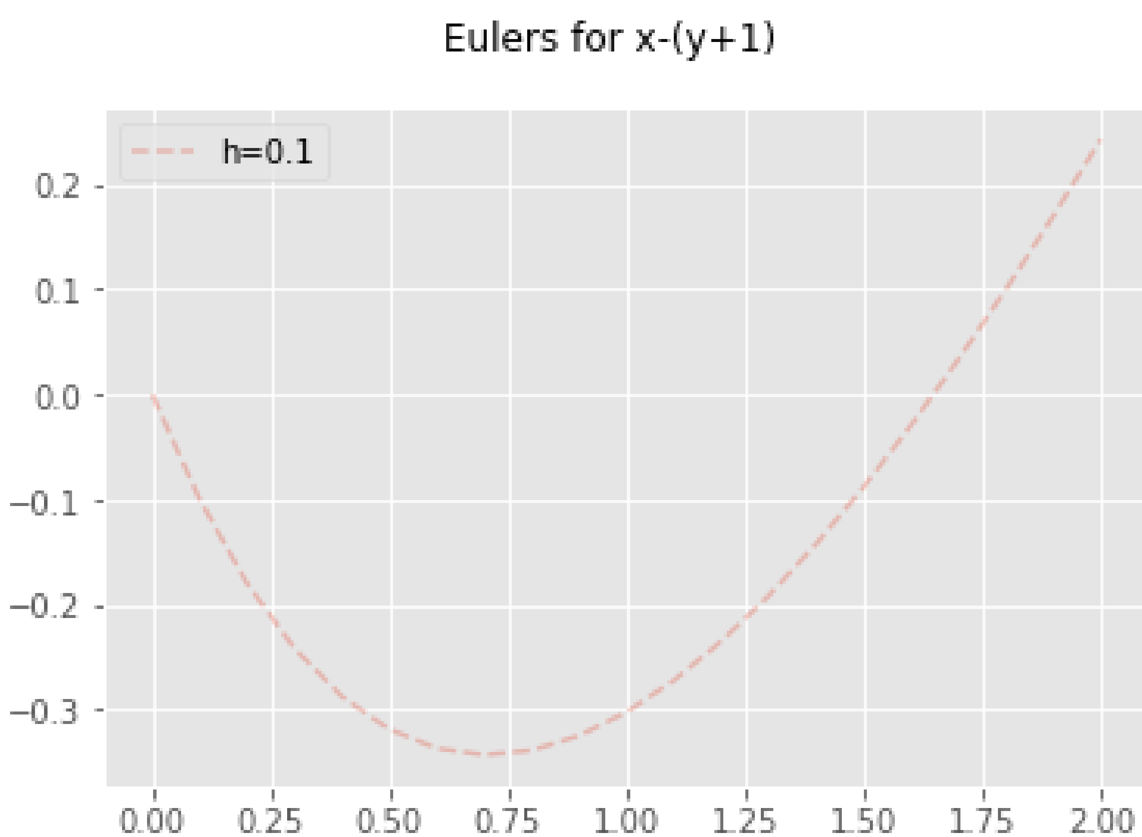
- refers to instantaneous rate of change
- $\frac{dy}{dx}$
- $f'(x)$ A.K.A. slope of tangent line at x

1.3.3 My implementation of Euler's method:

```python
%matplotlib inline
import numpy as np
import matplotlib.pyplot as plt
import numpy as np

plt.close('all')
def solve(h, f, t0, t1):
    y0 = 0
    x = np.arange(t0, t1+(t1 % h), h)
    y = np.zeros(len(x))
    y[0] = y0
    for i in range(1, len(y)):
        y[i] = y[i-1] + (h * f(x[i-1], y[i-1]))
    plt.plot(x, y, '--', label='h={0}'.format(h), alpha=0.3)
    return (x, y)
x, y = solve(0.1, lambda x, y: x-(y+1), 0, 2)
plt.suptitle('Eulers for x-(y+1)')
plt.legend()
```

```
<matplotlib.legend.Legend at 0x120073080>
```



2 Interesting

The figure:1 is sourced from [6]

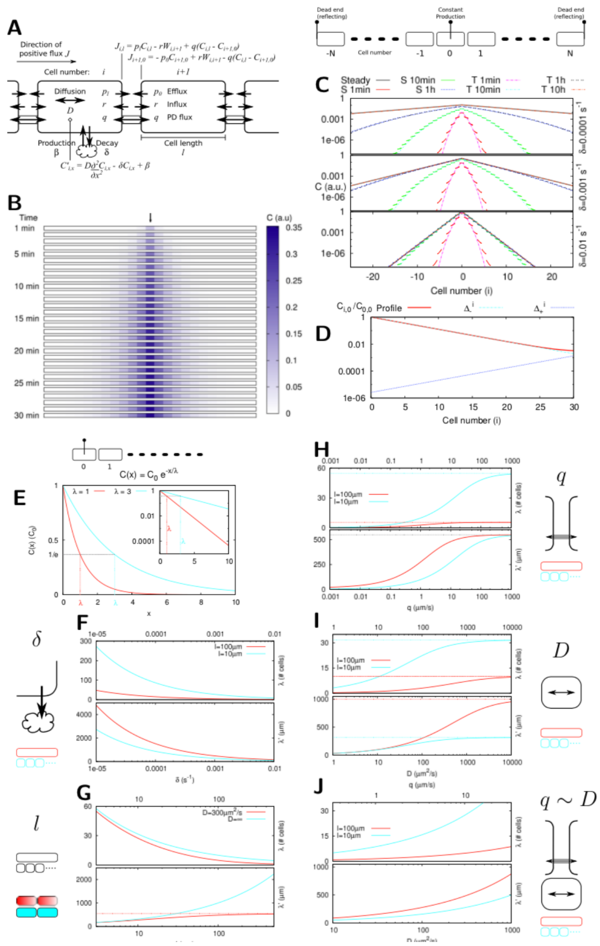


Figure 1: Basic properties of symplastic transport. A: model outline. All fluxes through the walls are modelled as effective permeabilities (with units m/s). Concen- trations are given as Ci,x in cell i at location x and Wi,i+1 in the wall between cell i and i + 1. Model parameters: decay constant σ , cell length l, effective wall permeability q and diffusion constant D. B: time series for a 1D tissue with a single producing cell (with rate β/volume = 2σ a.u.) in the middle (q = 1m/s, = 0.001s1, l = 100m, D = 300m2/s). C: Dependence of profile steepness and time scales on σ (other parameters as in B). Simulation profiles are indicated with S, analytical predictions with T. D: Example steady state profile (solid red) with source left and reflecting wall right. This is the sum of two exponential functions (dashed): a decreasing one (cyan) and approxi- mately the continuation of its reflection on the wall (blue). E: The steepness of the con- centration profiles can be expressed using the characteristic length, the length over which the concentration drops with a factor 1/e (0.37). This can be expressed in number of cells (λ) or physical length (m; λ)

3 Papers to read

3.1 TODO Glutamate triggers long-distance, calcium-based plant defense signaling [1]

3.2 TODO Necrotrophic Pathogens Use the SA Signaling Pathway to Promote Disease Development in Tomato [2]

3.3 TODO Callose biosynthesis in arabidopsis with a focus on pathogen response: what we have learned within the last decade [3]

3.4 TODO Regulation of solute flux through plasmodesmata in the root meristem [4]

4 Paper review for Morris group meeting

4.1 A single fungal MAP kinase controls plant cell-to-cell invasion by the rice blast fungus [5]

- The fungus they have used is *Magnaporthe oryzae* and it effects rice
- When this fungus gets into cells, it expands and seeks to colonise as many cells as possible. It does this by way of tendril like appendages called hyphae
- Whilst this process is happening the attacker secretes effectors that try and suppress host defence responses
- The fungus seeks out "pit fields" which are plasmodesmata rich sites and the process repeats

4.2 What is the hypotheses

In English:

- Inhibiting a single enzyme (kinase) prevents fungal infections from spreading through plant cells

4.3 What gap do they aim to fill

- The aim of this research is to find methods of reducing the 30% of rice crop which is lost annually to blast disease (aforementioned fungus)

4.4 How did they design the experiments to address their questions

4.4.1 Infection in relation to PD restrictions

- The experiment started with ultrastructural analysis of cells infected by pathogens
- The analysis confirmed that fungal hyphae were present between cells
- They noticed that the infection sites' plasmodesmata were still open at 27hrs post inoculation

- Callose deposition was noticed at 30hrs as it formed around invasive hyphae
- The hypothesis proposed is that the infection was suppressing / clearing PD before penetrating into neighbouring cells
- To test whether fungus could manipulate PD's SEL (size exclusion limit) two different mCherry (flourophone) molecules were bombarded at infected tissue and again at uninfected tissue results below:

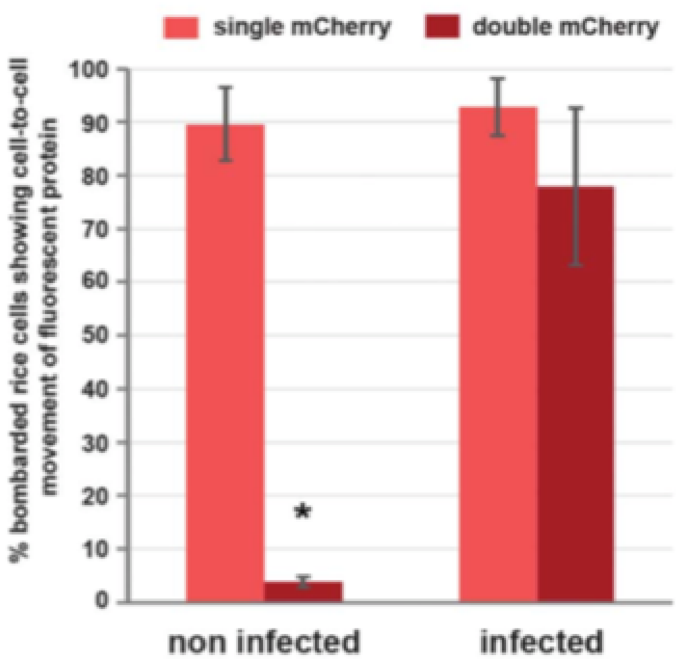


Figure 2: mCherry at infected cells

- This showed that infection was clearly having an effect on PD being able to regulate the SEL
- And that PD were not reacting until around 25~ hrs post invasion

4.4.2 Regulatory mechanism of fungus

- Pmk1 - A MAPK (mitogen activated protein kinase) was identified which is essential for infection development
- PMK1 null mutants cannot infect plant leaves
- The experiment used a conditionally activated Pmk1
  - Using a chemical genetic approach
  - They generated an analog sensitive allele of PKm1 (by using magic)
- They then used this mutant, allowing PMK1 to be active at the start of the infection phase and then suppressing it
- This treatment blocked invasion of adjacent epi-dermal cells, resulting in the infected cells becoming filled with fungal hyphae

  - The morphology of the hyphae appeared unaffected
  - This was tested in both another rice cultivar and barley

4.4.3 Finding M. oryzae genes affecting infection

- RNA-seq was performed on the pmk1 mutants
- The results showed that 1457 fungal genes differentiated
  - 11.5% of the total protein-encoding genes
- 715 genes were up-regulated and 742 were down-regulated

4.5 What were the results?

- That pmk1 could contain the key to deactivating fungus' ability to infect new cells

4.6 What is the significance of the results

4.7 Strengths/Weaknesses

Table 1: Strengths/Weaknesses of Paper

Strengths	**Weaknesses**
Experimental design seemed en point	They still have 1400+ genes to narrow down
	Details on RNA-seq seem pretty sparse

5 Questions

- How do plants handle multiple attacks at once, does it struggle more at a 1:1 ratio or is it slightly easier to fend off multiple attacks as it's already producing defence chemicals/signals?
- In [6] chapter 2, figure 2.1 How does one even start to form the equations for movement

References

[1] Masatsugu Toyota, Dirk Spencer, Satoe Sawai-Toyota, Wang Jiaqi, Tong Zhang, Abraham J. Koo, Gregg A. Howe, and Simon Gilroy. Glutamate triggers long-distance, calcium-based plant defense signaling. *Science*, 361(6407):1112–1115, September 2018. 00003.

[2] Taha Abd El Rahman, Mohamed El Oirdi, Rocio Gonzalez-Lamothe, and Kamal Bouarab. Necrotrophic pathogens use the salicylic acid signaling pathway to promote disease development in tomato. *Molecular plant-microbe interactions: MPMI*, 25(12):1584–1593, December 2012.

[3] Dorothea Ellinger and Christian A. Voigt. Callose biosynthesis in Arabidopsis with a focus on pathogen response: What we have learned within the last decade. *Annals of Botany*, 114(6):1349–1358, October 2014. 00050.

[4] Heidi L. Rutschow, Tobias I. Baskin, and Eric M. Kramer. Regulation of Solute Flux through Plasmodesmata in the Root Meristem. *Plant Physiology*, 155(4):1817–1826, April 2011. 00077.

[5] Wasin Sakulkoo, Miriam Osés-Ruiz, Ely Oliveira Garcia, Darren M. Soanes, George R. Littlejohn, Christian Hacker, Ana Correia, Barbara Valent, and Nicholas J. Talbot. A single fungal MAP kinase controls plant cell-to-cell invasion by the rice blast fungus. *Science*, 359(6382):1399–1403, March 2018. 00004.

[6] E. E. Deinum. *Simple Models for Complex Questions on Plant Development.* PhD thesis, s.n., S.l., 2013. 00010.