PhD Diary Entry for week beginning October 8th 2018

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October 19, 2018

Contents

1 General TODOs

1.1 TODO Talk to R and J about trying to diagramatically explain data flow.

1.2 TODO Study up on Fick's laws of diffusion

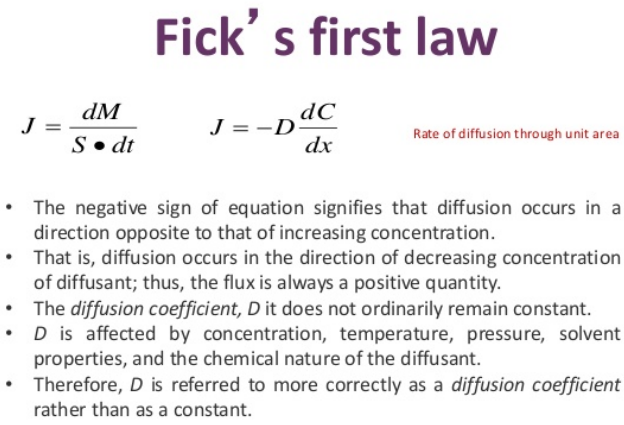

Figure 1: Diffusion

1.3 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

1.3.1 TODO Revise Partial Diff Equations

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

2 Paper Reviews

2.1 TODO Understanding Plant Immunity as a surveillance system to detect invasion [1]

2.1.1 Intro

- Crop production needs doubled by 2050 by some current estimates
- A lot of work has been done on conceptual models for plant pathogen systems
  - Details in the models vary

  - Though they all agree in the observation that plants mainly rely on innate immune systems that are largely controlled by encoded receptors that identify invasion

- It's important to note that models:
  1. Are generalisations and therefore incomplete
  2. Increasing the details of a model decreases its general applicability
  3. Multiple models can be used to explain the same phenomenon
  4. All models should be continually challenged via experimentation to advance scientific knowledge
- Models are full of limitations and narrowly define molecular plant-invader interactions
  - Additionally do not integrate experimental data from diverse systems
- Introduces the *Invasion Model*

2.1.2 Advances in explaining the plant immune system

- Talks about gene-for-gene hypothesis
- And how other research suggests "general elicitors" from microbes which could **not** be used to determine race specificity or were detected by multiple plant species
  - In contrast to pathogen Avrs that induced responses only on particular varieties of a host species
  - These seeminly different observations were regarded as disparae phenomena
  - The identification and characterisation of general elicitors and their receptors in vertebrate immunity helped to further refine the concept in plant immunity
- Charles Janeway postulated the concept of conserved microbial ligands for innate immunity to account for lapses in the conceptual model of vertebrate immunity
  - He reasoned that microbes possess pathogen-associated molecular patterns (PAMPs)
  - That are recognised by host patter recognition receptors (PRRs) as nonself
  - He anticipated that PRRs perceive microbe-derived conserved general structural patters that are critical for the organism and require significant changes to avoid recognition
- PAMP is noted to be a misnomer, as it often concerns molecules present in both pathogenic and nonpathogenic organisms
  - Hence why we now use the term MAMPs (microbe-associated molecular pattern)
- Talks about zigzag model (see figure in key words/phrases)

2.1.3 Limitations and incongruities in the MAMP-effector dichotomy

- Continued research into plant microbe-interactions have identified a number of concerns over the MTI-ETI dichotomy

- Issues are raised with the conceptual layout of the model in distinct phases governed by discrete responses
- A primary concern is the relationships between:
  - MAMPs and Effectors
  - PRRs and R proteins
  - MTI and ETI
- These relations also ignore DAMPs
- The zigzag model does not acknowledge the number, kinetics and combined action of multiple receptor ligands that govern these interactions
- Additionally, pathogen perception and response are illustrated over an ambiguous spatial and temporal frame, obscuring the model's intention to represent evolution or a particular cellular encounter
- The zigzag model also doesn't account for previous life history events of the hsot or invader prior to the interaction, which may influence the outcome of said interaction

2.2 TODO Simple models for complex questions on plant development (Thesis) [2]

2.2.1 Chapter 2

- To form different tissues, cells need to differentiate
- Plant cells mostly can't move
- Requirements for coordination of developmental and functional needs
- To do this cells use a number of signalling molecules
- Animals use different systems for communication of inter-cellular information
  - A Delta-notch system is one example
- Protein-Protein interactions are obviously impossible between plants, unlike in animals
- Plants can achieve results using a "non-cell-autonomous proteins" method
  - i.e. Protein signals that are transcribed in one cell and affect transcription in another
  - Like a factory which outsources some of its labour?
- These proteins move symplastically through PD channels
- Symplastic transport comes in two forms:
  1. Generic: the passive movement of all sufficiently small molecules; this is non-targeted and is diffusion driven
  2. Targeted: targeted symplastic transport is a container term for a variety of different mechanisms that allow symplastic movement of molecules that depend on it
- Virus' create their own "movement proteins" to facilitate the crossing via targeted symplastic movement

  - This process maybe involve structural alterations of the plasmodesmata, which affect the non-targeted transport properties
- Molecules moving by non-targeted symplastic transport may include plant hormones, small RNAs and small proteins
  - Providing they are small enough
  - This means that they fit the "size exclusion limit" (SEL)
  - The SEL is developmentally regulated and varies among different tissues
- The aperture of plasmodesmata is controlled through the deposition and degradation of callose
  - by callose synthase (Ca1S aka GSL gene family) and β-1,3-glucanase respectively
  - Further regulation takes place by other factors too [7]

2.3 DONE Modelling advection nad diffusion of water isotopologues in leaves [3]

- `Not relevant`

3 Papers to read

3.1 TODO Necrotrophic Pathogens Use the SA Signaling Pathway to Promote Disease Development in Tomato [4]

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

3.3 TODO Callose biosynthesis in arabidopsis with a focus on pathogen response: what we have learned within the last decade [6]

4 Key words/phrases

4.1 PRR

- [Host] Pattern recognition receptors
- Used to recognise PAMPs as "nonself" materials

4.2 MTI

- MAMP-triggered immunity
- Because of the redundancy of PAMPs as an idea, this is preferred

4.3 Effectors

- Are proteins expressed by plant pathogens
- They aid infection of specific plant species
- Are central to understanding complicated interplay between plants and their pathogens

4.4 ETS

- Effector-Triggered Susceptibility

4.5 ETI

- Effector-Triggered Immunity

4.6 MTI-ETI

- A juxtaposed relationship between MTI-ETI is often misunderstood
- For example, some would believe that the processes are independent and therefore not at a similar level

4.7 DAMPs

- Damage associated molecular patterns

4.8 Kinase

- Is an enzyme that catalyses the transfer of phosphate groups from high-energy, phosphate donating molecules to specific substrates

4.9 Dichotomy

- A divison or contrast between two things that are or are represented as being opposed or entirely different
- e.g. 'a rigid dichotomy between science and the DUP'

4.10 Advection

- The transfer of heat or matter by the flow of a fluid

5 Questions

- Regarding DAMPs and general damage control there must be some overlap or at least connection in the pathways that ultimately lead to repair or maintenance
- Are plants/cells aware of other cells around them. i.e. if there was a "perfect" invasion and a pathogen prevented the cell sending any signals and it just "stopped" communicating, would the border cells know something was up and try to correct?
  - Similarly, if you perfectly removed a group of cells, a plant repairs itself what's this communication method?
- Bacteria have been attacking plants for millions of years, why haven't they won, yet we fight them with anti-biotics for a century and they're looking like they'll overcome pretty quickly
- Two identical plants, control conditions, infected with same pathogen, is it likely for them to develop a similar response or is there a level of ambiguity in the choices made to protection, prevention and developing immunity?
- If a crab eats a plant, does the plant think its a fungal attack?
- Do many attackers perform prolonged, dormancy based attacks or is it an all out attack most of the time?
- During an attack, is it possible to something like bacteria to adapt during the attack and to adapt, or is that too small a timescale?
  - If so is this a potential concern for any kind of analysis, that is to say should we expect "experimental tactics" from the invaders?
- Things that the plant makes, are they somehow signed by the cell to prove authenticity or how do receptors / w/e know that it isn't foreign
- Is the MAMPs response like a magnet i.e. how mechanical is the process, like molecules naturally drawn together v.s. a more fluid process of decision making and evaluation of the attack
- Mutants with bad callose deposition reportedly don't make it much further than embryo stage, what other functions than PD does it affect?

References

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

[3] Matthias Cuntz, Jérôme Ogée, Graham D. Farquhar, Philippe Peylin, and Lucas A. Cernusak. Modelling advection and diffusion of water isotopologues in leaves. *Plant, Cell & Environment*, 30(8):892–909, August 2007. 00099.

[4] 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.

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