

Example lecture notes

I have produced this document in response to an e-mail from a student, who sent me their own lecture notes to help me answer a query.

I was quite disturbed by how little the student had written and how almost everything I said about hypotheses, and about the experimental approaches taken to answer them, was entirely missing.

Staff in general have been becoming increasingly concerned about students apparently learning less and less from lectures, something that seems to have grown with the use of computers in the lecture hall. Many colleagues assume this is because the screens of those computers actually show Facebook etc., but another possibility is that using keyboards restricts rapid note-taking. Significantly, the notes sent to me were on a computer file.

I have therefore played the role of a student for my own lecture, taking notes pretty much as I would have taken them if I had been sitting with you. The only difference is that I used loose paper for this exercise to make photography easier, whereas I use hard-backed books for taking notes in real lectures (we staff still attend many lectures, of course, as part of our lives as researchers and scholars).

The photos of the 12 pages represent the level of detail all staff would expect you to capture in any typical lecture. My real undergrad lecture notes (which I still have on the shelves of my office) look very similar. Apologies for the writing – making rapid lecture notes has this effect – but the purpose of these notes is not to help you revise directly, but to illustrate what is expected.

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January 2017

Comments for organization

Website: gdgi.ana.ed.ac.uk/courses/bms2/

↗
has lecture slides.

↳ [bms2/bms2.html](#)

Generation of organized structures

- blueprint-driven
- self-organizing

↖ lecturer seems to have a bee in his bonnet about this!

Evidence against blueprint:

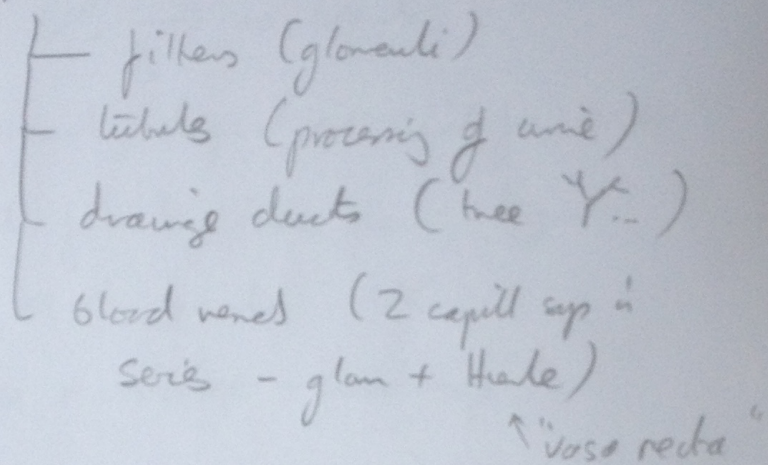
human fingerprints
of Manó-3 twins

— genetic 'fingerprint' same
— literal fingerprint NOT.

< J For Ident 112:58(1) >

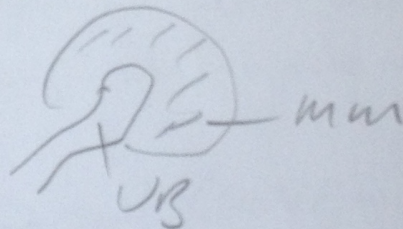
Rest of lecture with address in "model system" —
human (?-germ man?) kidney.

Kidneys :- complicated structures

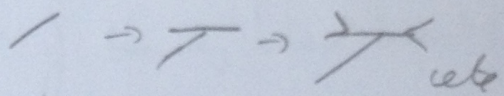


Development of Ks:

- 1) Start - Ureteric bud (epithelium, blood vessel) + Metanephrosic mesoderm



- 2) Bud branches



(happens in culture too - cool mania!)

How is branching controlled?

Isolated UB → no branch.

Does branching depend on signal from MM?

→ look for lig tree pairs, ligated in MM recipe: UB

(use a database - Goodmap?)

↑ No "GOODMAP"

→ see: mmr ^o _o - GDNF.

UB/Ret (= GDNF recipe)

Test: Interrupt signal (i: GDNF blocks branching: chl ab does not)

Give ectopic signal (GDNF or chl protein on same kind of bead): lots of branching where bead is.

Summing this page -

MM makes GDNF; causes UB to branch.

There are lots of other signals known -

- pro-branch
- pro growth anti branch
- "keep out".

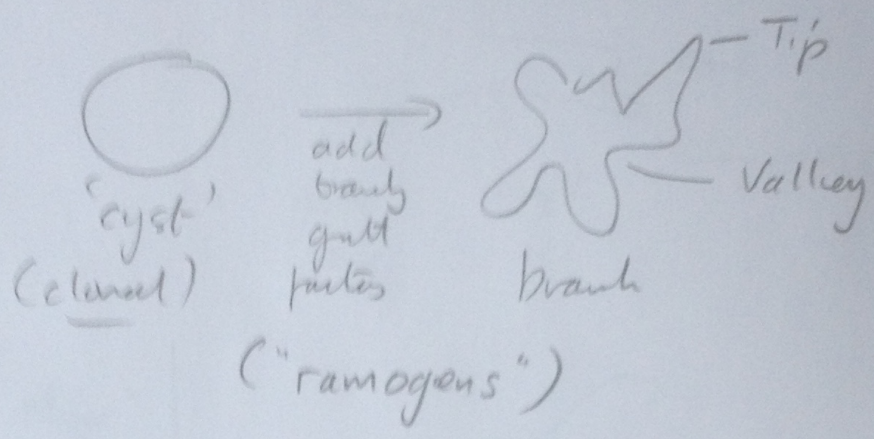
Feedback: UB causes MM to diff to nephros (the filter - & tube thing!)

So MM touched by UB is no longer MM - no longer makes GDNF, now makes anti-branch stuff (BMP, TGFβ &c)

So Self-ageing: Virgin MM attracts UB but once pruned does not attract any more, so nephron fun = not interesting!

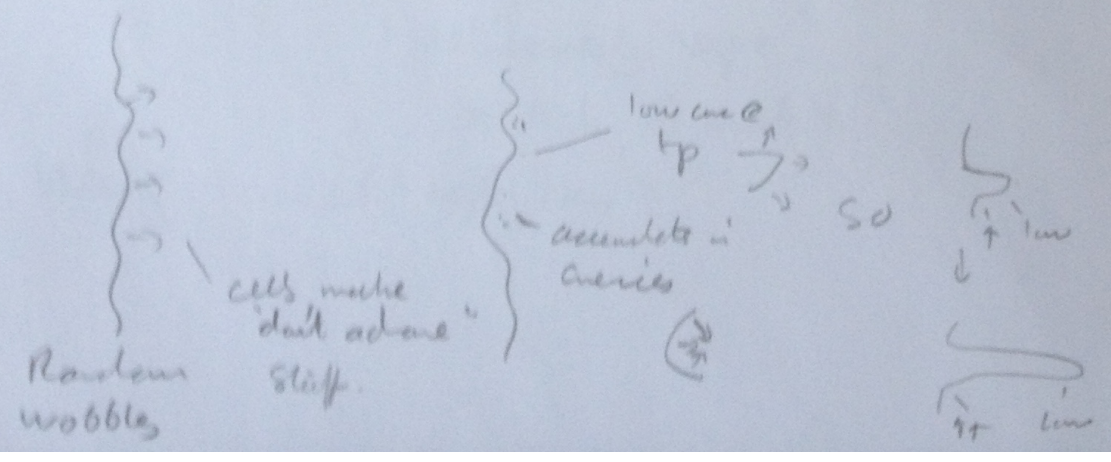
(no lecture being deliberately ironic??)

More Q - how does branching happen (at a cell level)?



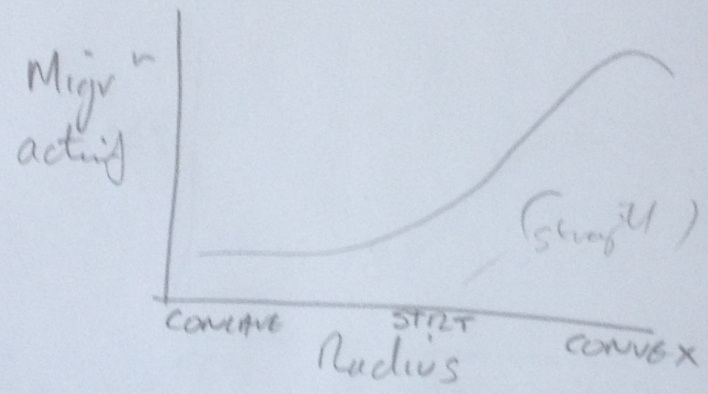
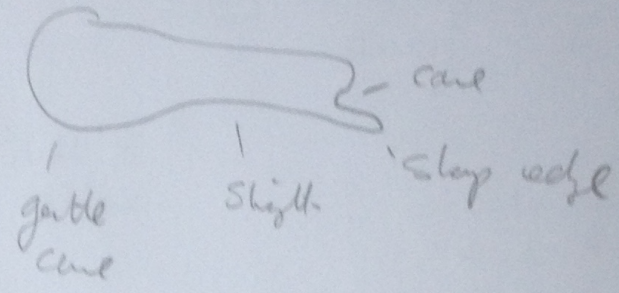
① — All cells start same, why end up doing different things?
 (Lecturer kept using phrase 'symmetry breaking')
 ↗ Look up ***

Hypothless (Cieloshe Nelson if need to look up)



Test

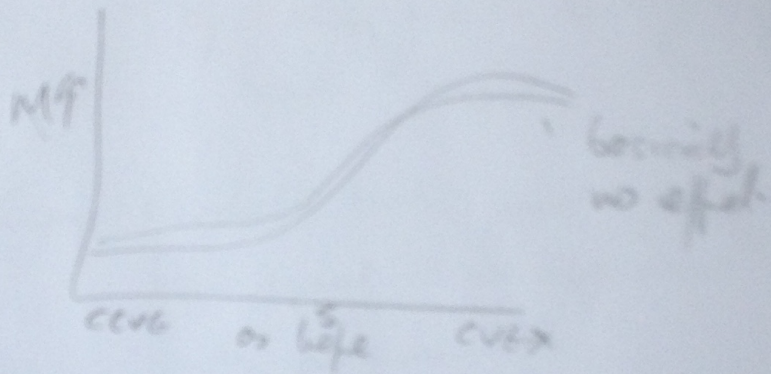
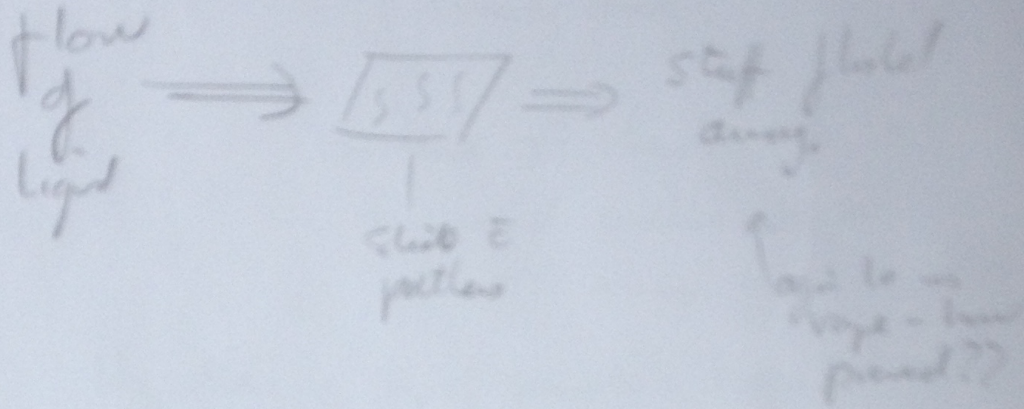
Cells grow on every island



→ What Nelson predicted
 (? - he did not do any quant. analysis - was he being deliberately vague?
 * Ash @ end *

Wang - comet = # cars =

Test of the suggested inlet idea:



Conclude \Rightarrow Nelson wrong re mech
right re correlation.

Recall Wheel?

- Uniform. Tension??

New Q

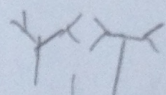
How does the tree (URB) spread out? ⑧

Evidence \neq blueprint; constant
in varied culture cond^{ns}
→ always kinda sensible

Hypothesis [tips senesce "horrid"
tips always grow in dirⁿ of
least "horrid"

Comp Model looks good - get
tree spread; \bar{c} no info about
angles programmed in.

Assay: Attempted collision



what happens?
→ avoid (odd steps as a
result)

Screen (drugs; look for ^{result} collisions)

→ Inhibit TGF β family \Rightarrow collisions.

More selective utilitarians (material
'greenish')

↪ Look up **

⇒ Looks like it's BMP7 ↪ Look up *

a' BMP7

- collus between trees
- collus within trees.
- do see a cone gradient.

* - Worry - he did not show what happens
to MM / replus - could all
of this be induced? - ask
@ end of time.

New Q

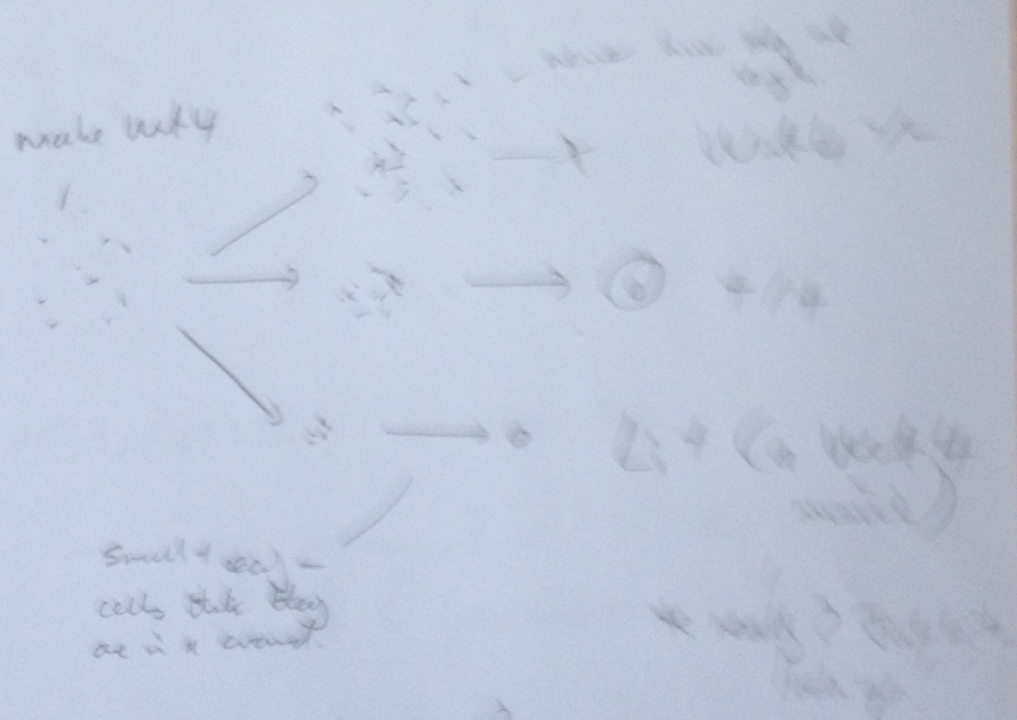
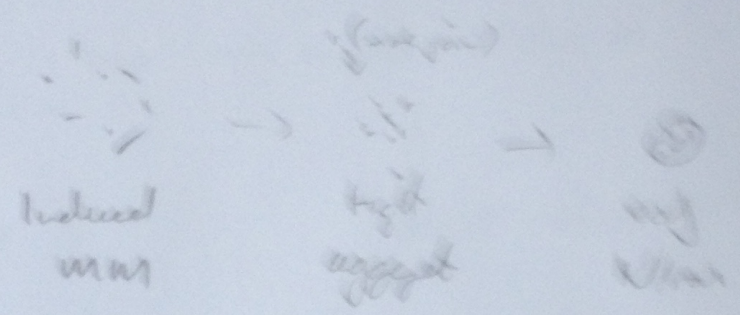
Why do replus form ~~at~~ near branches?

- induced by Wnt-9b secreted
from branches.

(MM dies if no UB tree)

Q how do cells make a large signal aggregate to make a response.

[drifted for a moment a look up of they need to!]

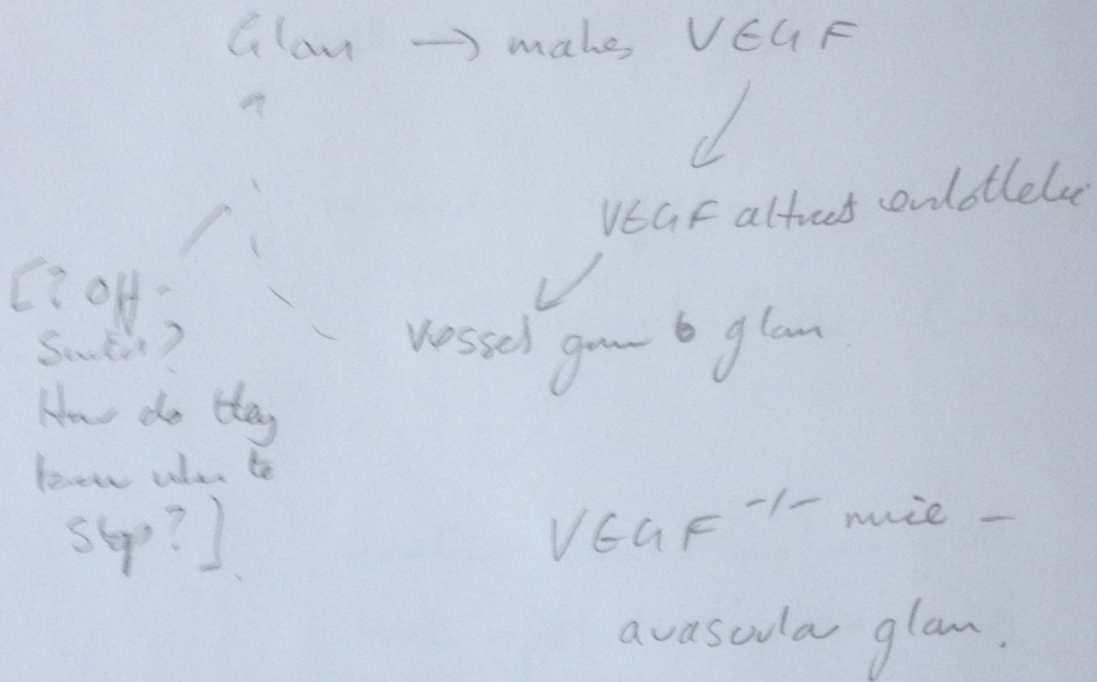


ref to Davis 2000
molecular biology of the cell

→ "Quorum Sensing"

New Q

— How do cells find glomeruli?
↑
filtes.



→ OVERALL SUMMARY /

— Self organization by eedg of
signals & feedback.

Loose ends (he wants more about the
series)

NOT
EXAMINABLE

Does this scale up?

Models → cells → people → society?

Big received Hofstadter D
Gödel Escher Bach



Lecturer said this was the
most important thing he read
as an undergraduate.

* Own thoughts - can be same thing
he said of ecosystem agent?
end = business? Chat & Hang out
coffee?