CHAPTER 1: CELLS AND TISSUES


Page 14: Answer to thought question: Malformation of the endoplasmic reticulum and Golgi apparatus is thought to underlie one type of inherited spastic paraplegia – for more information, see Rismanchi et al. (2008) Hum Mol Genet 17 p.1591.

CHAPTER 3: MEMBRANES AND ORGANELLES

Page 43: In the second edition we gave the thickness of the lipid bilayer as between 5 and 10 nm. However estimates of biological membrane thickness have been declining over the years. Mitra et al. 2004 (PNAS 101 p4083) measured the thickness of endoplasmic reticulum, Golgi, basolateral and apical plasma membranes of rat hepatocytes as, respectively, 3.85 ± 0.04 μm, 3.95 ± 0.04 μm, 3.56 ± 0.06 μm, and 4.25 ± 0.03 μm, so in this edition we state the typical thickness as 4 nm.


CHAPTER 4: DNA STRUCTURE AND THE GENETIC CODE


Page 63: Medical Relevance 4.3: For a description of the different mutations in the FMO3 gene found in different families, see Treacy et al. (1998) Human Molecular Genetics 7 p.839.

Page 64: It is in various ciliates that we find UAA and UAG coding for glutamine rather than stop. For more information, see Sánchez-Silva et al. (2003), Current Biology 13 p.442.

CHAPTER 5: DNA AS A DATA STORAGE MEDIUM

Page 77: In Depth 5.2 BLASTN and BLASTP are some of the many tools provided by the US National Center for Biotechnology Information. This link goes directly to the BLAST tools.

CHAPTER 6: TRANSCRIPTION AND THE CONTROL OF GENE EXPRESSION


Page 95: Medical Relevance 6.1: For more information on hyperforin’s action see Moore et al. (2000) Proc Natl Acad Sci USA 13 p.7500.

Page 96: Medical Relevance 6.2: This box is based on Mick et al. (2001) Molecular Endocrinology 15 p. 575. It is surprising to find aldosterone, a mineralocorticoid, acting through the glucocorticoid receptor but the evidence presented is strong.

CHAPTER 7: RECOMBINANT DNA AND GENETIC ENGINEERING

Page 113: Medical Relevance 7.1: See the video “DNA microarrays” and read Stoughton (2002) Scientific American 286 p.44.


CHAPTER 8: MANUFACTURING PROTEIN

Page 128: Example 8.1: For more on formyl methionine and the immune system see Web Text Box 8.1: More on the irritating formyl methionine: why doesn’t our immune system attack mitochondria?

The discovery of cells in the nose that respond to N-formyl methionine peptides is described in Riviere et al. (2009) Nature 459 p. 574.


Well formed, barrel shaped proteasomes are characteristic of eukaryotic cells. Proteasomes are found in bacteria (e.g. Sharon et al. (2007) *Journal of Biological Chemistry* 282 p.18448) but in most bacteria the components are not so obviously organized into a discrete barrel-shaped machine.

## CHAPTER 9: PROTEIN STRUCTURE

Page 141: Example 9.1: The medical name for the disease caused by failure of ROMK is Bartter’s syndrome type II. Our discussion of how the salt bridges are necessary to keep the pore in ROMK patent is based on Leng et al. (2006) *PNAS* 103 p.1982.


Discussion of the farming practices that led to the disease outbreak is based on Bradley et al. (2006) Folia Neuropathol. 44 p.93 and p.102.
This is also the source of vCJD deaths up to 2005 inclusive. Here are data on later years.

### CHAPTER 10: INTRACELLULAR PROTEIN TRAFFICKING

**Page 166: In Depth 10.1:** To see trafficking movies, we recommend Part 1 of Jennifer Lippincott-Schwartz’ online seminar “Breakthroughs in Intracellular Fluorescent Imaging” at iBioSeminars.

Other sites to look at are:

- [http://mcbi.ouhsc.edu/clarkelab/movies_bead_uptake.html](http://mcbi.ouhsc.edu/clarkelab/movies_bead_uptake.html)
- [http://www.nimr.mrc.ac.uk/research/tom-carter/movie-gallery](http://www.nimr.mrc.ac.uk/research/tom-carter/movie-gallery)
- [http://vimeo.com/887388](http://vimeo.com/887388>

**Page 176:** For more on Arf see *Web Text Box 10.1, Arf’s sticky finger.*

### CHAPTER 11: HOW PROTEINS WORK

**Page 182:** For more on how proteins operate as catalysts, see *Web Text Box 11.1, Enzyme catalytic strategies.*

**Page 184:** For more on cytochromes and their different colors, see *Web Text Box 11.3, A cytochrome ABC.*

**Page 187: Medical Relevance 11.3:** For more on how we study the effect of drugs on enzymes, see *Web Text Box 11.2, Analyzing enzyme kinetics: finding out how drugs work*
CHAPTER 12: ENERGY TRADING WITHIN THE CELL


Page 196: For more on cytochromes, see Web Text Box 11.3, A cytochrome ABC.

Page 196: For a listing of the names used for various members of the electron transport chain, see Web Text Box 12.2, Alternative names for components of the electron transport chain.

Page 201: In Depth 12.2: See animation of ATP synthase’s operation that includes laboratory data showing rotation of a fluorescent tag attached to the rotor.

CHAPTER 13: METABOLISM


CHAPTER 14: IONS AND VOLTAGES

Page 231: The statements we make here concerning the resting voltage of glial cells remain the dogma, although the truth is no doubt more complicated. If the membrane of a cell is permeable only to potassium, then if the extracellular
potassium concentration is changed the membrane voltage will change such that $V_m$ is proportional to $(RT/F) \log_e [K]$ (see In Depth 14.2). We call such behaviour “Nernstian”. Kuffler (1967, Proc Roy Soc B 168 p.1) shows in Figure 8 salamander optic nerve glia with a resting voltage of -89 mV and a perfect potassium Nernstian slope. For an example of a recent paper in which the glia show a highly Nernstian behaviour with $V_m \sim E_K$, see Tritsch et al. (2007) Nature 450 p.50.


There are in fact two isoforms of the sodium, glucose cotransporter. Initial uptake of glucose is by SGLT2 with a stoichiometry of one glucose molecule for each one sodium ion. More distally, cells express SGLT1, with a stoichiometry of 2 Na$^+$ to 1 glucose, to take up glucose against a greater concentration gradient. We state that cytosolic glucose is about 40 mM in kidney cells.

We know of no measurements of this, and make an estimate of 40 mM by analogy with intestinal uptake which uses the same carrier systems and where measurements of cellular glucose (which will have been contaminated with extracellular fluid) gave a value of 36 mM (Leese (1974) Nature 251 p.512). We are very grateful to David Bender, Ted Debnam and Robert Unwin of University College London for their helpful input on this question.


CHAPTER 15: INTRACELLULAR SIGNALING

Page 260: The mechanism by which PDGF activates its receptor is described in Chiara et al. (2004) Journal of Biological Chemistry 279 p. 19732. The best studied receptor tyrosine kinase is the epidermal growth factor receptor, where the mechanism of activation is somewhat different – see Zhang et al. (2006) Cell 125 p.1137.


CHAPTER 16: INTERCELLULAR COMMUNICATION


CHAPTER 17: MECHANICAL MOLECULES

Page 291: Medical Relevance 17.1: For more detail on Listeria and its use of the host cell’s actin see Web Text Box 17.2, How Listeria uses actin polymerization to pass between host cells.

Page 291: For more on the lamin intermediate filaments and how mutations in lamins cause premature aging, see Web Text Box 17.5, Lamins, disease and aging.


CHAPTER 18: CELL CYCLE AND THE CONTROL OF CELL NUMBER

Page 298: For more on the replication of the centrosome see Web Text Box 18.1, Poles apart: centrosome duplication through the cell cycle.

Page 305: Medical Relevance 18.2: For more on cousin marriage in the Pakistani immigrant population in Britain see Gadher, Morgan and Oliver, Sunday Times, February 10th 2008 and Paul and Spencer (2008) PLoS Biology 6 (12) e320. We calculate the eightfold value from the statement in the PLoS article “data from the English West Midlands suggest that British Pakistanis account for only ~4.1% of births, but about 33% of the autosomal recessive metabolic errors recorded at birth”. Thus in 100 births in the general population there are N cases, in 4.1 births in the Pakistani population there are N/3 cases, therefore in 100 births in the Pakistani population there are \((100/4.1) \times (N/3) = 8N\) cases.

Page 305: A critical experiment in the discovery of the cyclinB/CDK1 system is described in Web Text Box 18.2, A factor in dividing cells triggers mitosis in interphase cells.
Page 306: For more on ATM, see Web Text Box 18.3, What is Ataxia Telangiectasia?

Page 306: For more on contact inhibition, see Web Text Box 18.4, Control of cell number: why are we bigger than a mouse and smaller than an elephant?


CHAPTER 19: THE CELL BIOLOGY OF THE IMMUNE SYSTEM

Page 325: Medical Relevance 19.1: For more on monoclonal antibodies, see Web Text Box 19.2, Making monoclonal antibodies.

Page 325: Answer to thought question: For more on the multiple tricks used by viruses to evade the immune system see Loch and Tampé (2005) Pflügers Archiv 451 p. 409.

CHAPTER 20: CASE STUDY: CYSTIC FIBROSIS


Page 334: Answer to thought question: The hypothesis that an abnormal pH in the Golgi lumen impairs the glycosylation of mucus proteins was proposed in Barasch et al. (1991) Nature 352 p.70.

GLOSSARY Links

Page 346: For more on ATM, see Web Text Box 18.3, What is Ataxia Telangiectasia?