helical structure respectively, and the role of H-bonding in protein structure was known from the work of Linus Pauling. But the beauty of the work of Watson and Crick lies in the fact that they used the available information to come up with the double-helical structure of DNA, which explained the three cardinal attributes of the genetic material, namely replicability, mutability and information storage. Having all the information available, others also could have come up with the same model, but none did. Is it a fault of Watson and Crick that they did, while others did not? Rosalind Franklin could have had genuine grievances and disappointments in her career, but I feel it is somewhat uncharitable to project her as an ungracious person in defeat.

There are a few errors, historical and scientific, in the early part of the book dealing with Stone Age genetics. Some are listed below in the hope that they might be rectified in future editions.

1. Watson, Crick and Wilkins got the Nobel Prize in 1962, not in 1958 (p. 10).

2. It is odd to have Mendel use the terms 'genotype' and 'phenotype'. These words were coined by the Danish botanist, Wilhelm Johannsen in 1909, i.e. 25 years after Mendel's death (p. 11).

3. The term 'transforming principle' was coined by Frederick Griffith and not Avery, who along with McLeod and McCarthy identified it as DNA (p. 17).

4. The classical work of Gierer and Schramm on tobacco mosaic virus could have been included to show that in some viruses RNA is the genetic material (pp. 11–20).

5. Joshua Lederberg discovered mating in *Escherichia coli*, but it was William Hayes who identified the causative agent as the F (Fertility) factor (p. 41).

6. In $F^- \times$ Hfr crosses there is no transfer of the F factor from the donor to the recipient, whereas in $F^+ \times F$ crosses there is. (The former is a chromosome transfer process and the latter is a plasmid transfer process. The small frequency of chromosome transfer which Lederberg detected in the latter is due to the presence of a few Hfr cells in the donor population; p. 42.)

7. A minor typo. It should be English language, not English literature (p. 61).

The conversational style found in the book is odd. The characters are imagined to be engaged in informal coversation. Therefore, it is natural to speak to one another using shortened first names such as Jim, Bill, Fred, etc. But they talk using last names and that too without any prefix such as Mr, Dr or Prof, etc. It is neither informal nor formal. This aspect needs to be given attention to, in future editions. In summary, this book is the outcome of an interesting and novel idea of presenting information. Its usefulness could be greatly enhanced if some illustrations are provided and adequate attention is paid to small details, some of which are listed above.

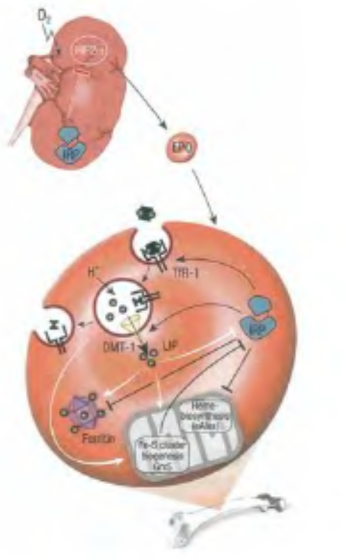
R. JAYARAMAN

R.H. 35, Palaami Enclave,
Reserve Line,
New Natham Road,
Madurai 625 014, India
e-mail: aarjay007@rediffmail.com

Annual Review of Nutrition, 2008. R. J. Cousins, Dennis H. Bier and Barbara A. Bowman (eds). Annual Reviews, 4139 El Camino Way, P.O. Box 10139, Palo Alto, California 94303-0139, USA. Vol. 28. 480 pp. Price not mentioned.

The first and last chapters in this volume make for absorbing reading. In a recent symposium that reflected on the greatest discovery in nutrition during the last 30 years (albeit in a small select audience), the winner was the discovery of the role of folic acid in preventing birth defects[1]. It is fitting that the prefatory chapter in this issue is by Irwin Rosenberg, whose elevating journey in translational science involved the elucidation of the nutritional importance of folate; and the translation of that science into policy through folate fortification. The diet–heart hypothesis, which for so long only involved research into the role of dietary fat, now includes research into many other nutrients, prime among which are the B vitamins, with their link to homocysteine; Rosenberg's research in this area is exemplary. The last chapter is no less absorbing, dealing as it does with bioethical considerations in nutrigenomics. The transfer of technology between borders is much faster than the resolution of resulting ethical difficulties. Specific to nutrition, there are issues surrounding the development of cohorts with biobanks of biological samples that could be used to define biomarkers of risk of later disease. How are these samples to be stored and used? Who would guard their (and the donor's) interests? How will commercial exploitation be handled in a way that benefits all society? These are questions that we must address, and while some solutions are presented, more discussion is needed in a specific national context, that may create more nuanced approaches. Another issue relates to the 'unhealthy quest for health' with the seemingly unattainable summit of the nutrigenomically personalized diet, and the ethical dilemmas that are related to such information. Thus far, the promise held out by early breakthroughs in genotyping and informatics has not been borne out; there are far more unknowns relating to the phenotype and environmental aspects, than are known. In the present condition, it seems ever more likely that the simple exhortation to 'eat more fruits and vegetables' will be the effective way forward to a healthy diet for all, rather than a complex individualized paradigm. Population rather than individual specific strategies are far more suitable from the current nutrigenomic state of the art, and the review of these issues is well presented[2].

India has a burgeoning obese population[3], as do other countries, and this is related to a positive energy balance due to too little physical activity or too much food energy intake. Weight reduction strategies that seek the creation of a negative energy balance through increased energy expenditure, such as exercise have not been successful in the long term,



The iron-responsive element/iron-regulatory protein (IRE/IRP) regulatory network in erythropoiesis.

since behavioural alteration is not a trivial exercise. An area that has received attention in the past, as an energy 'wasting' mechanism that would hopefully lead to a negative energy balance, is mitochondrial coupling efficiency. Studies on brown adipose tissue have shown that its mitochondria exhibit a decrease in coupling efficiency, leading to increased energy dissipation in the form of heat[4]. In this volume, Harper *et al.* assess the existence of decreased mitochondrial coupling efficiency in the body, as a target for obesity interventions[5]. While this phenomenon appears, in small studies, to explain why some dieters lose weight and others do not, the fact is that when decreased coupling efficiency occurs in peripheral tissues, there are far too many side effects. Therefore, the pursuit of this strategy has many pitfalls, which are well reviewed. In the same vein, the increase in obesity has also been attributed to the increase in consumption of processed foods and artificial sweeteners. The regulation and trafficking of the apical GLUT 2 glucose transporter in the enterocyte is multifaceted, involving local paracrine and endocrine factors. The ability of high sugar diets to permanently locate GLUT 2 transporters in the apical part of the enterocyte leads to higher sugar absorption, with consequences for obesity and insulin resistance. An interesting part of this review[6] relates to the description of intestinal sweet-taste receptors that seem to increase the number of apical GLUT 2 receptors and thereby increase glucose absorption, relating once again to the use of sugar or sweeteners in modern dietary products and the acute consequences of their use. Those who are interested in genetic approaches to obesity and weight gain, which are represented by quantitative trait loci, will find the chapter on systems biology approaches with mouse models interesting[7]. With obesity, insulin resistance is also of interest, and the current teaching is that type-2 diabetes mellitus is caused by a combination of peripheral insulin resistance as well as pancreatic islet β-cell dysfunction[8]. The chapter on β-cell function reviews the role of insulin in feedback regulation of the β-cell, as well as of insulin resistance on β-cell function[9]. New concepts are discussed, and as in any good review, many questions are also raised.

There is some continuity with specific themes in the previous year's volume. The role of malonyl CoA as a central player in the role of fatty acids in regulating food intake through central mechanisms[10], finds continuity with the review of its role in peripheral tissues through its inhibition of carnitine palmitoyl transferase, and its own regulation[11]. The role of hepcidin and other regulatory elements in the body's iron homeostasis is also well presented[12] and relevant to those who deal with the problem of iron deficiency anaemia, which is rampant in India. There has been interest in the definition of daily essential amino acid requirements and the importance of protein quality[13]. The link between protein intake and bone health is important, since osteoporosis is beginning to appear as a major problem in Indian populations[14]. The consensus appears to be that increased protein intake is linked to increased bone mineralization. Mechanisms linking amino acid levels to calcium absorption and changes in the hormonal milieu to favour bone growth are also discussed, and are well placed in the context of the chapter on mechanisms of calcium absorption in the intestine[15].

There is a great deal of interest in the birth weight of Indian babies[16], in terms of immediate infant health and morbidity, as well as in terms of the role foetal programming and birth weight may have on future adult chronic disease[17]. A recent study showing that lower maternal cholesterol levels have an impact on reducing birth weight, and the link between low cholesterol and microcephaly[18], is pertinent in the context of the chapter on the source of foetal and embryonic cholesterol[19]. In early or mid-pregnancy, it is difficult to predict the risk of a mother having a small baby, but the better indications come from maternal weight gain during pregnancy. However, how much weight gain is optimal, and how does antenatal counselling impact this? These questions are reviewed well in the chapter on maternal weight gain by Olson[20]. There is also excitement about epigenetic effects on foetal development and the course of further development into adulthood linked to the predisposition to chronic disease. Epigenetics refers to a process by which the expression of genes is changed through chemical processes, in a stable manner, without changing the actual DNA sequence. This neo-Lamarckian form of (epi)genetic alteration would probably be most effective during early foetal development and gametogenesis, and includes histone alterations and methylation of the genome. In particular, histone alteration is thought to be associated with the increase in lifespan associated with caloric restriction, and many other phenomena linked to cancer[21]. It is difficult to unravel these effects in human models and to clearly define the role of maternal nutrition in this process, although there are several leads[22]. Nutrient–gene interactions do exist; some through the targeting of specific nuclear receptors, and others through the epigenetic process. For example, abnormal glucocorticoid levels are known to be associated with the metabolic syndrome, and maternal protein restriction has been shown to upregulate genes encoding the glucocorticoid receptor in mice[23]. Methyl transfer linked to gene methylation is also dependent on thiol (methionine) metabolism and its regulation, in this volume, reviewed in linkage with liver disease[24], is also reviewed through the prism of cystic fibrosis[25]. These are all interesting reading for different perspectives. Overall, this collection of reviews is eclectic, covering a wide range of issues pertinent to today's nutrition, and as always, well written.

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ANURA KURPAD

St John's Research Institute,
St John's National Academy of Health Sciences,
Bangalore 560 034, India
e-mail: a.kurpad@iphcr.res.in

Annual Review of Cell and Developmental Biology, 2008. Randy Schekman, Larry Goldstein and Janet Rossant (eds). Annual Reviews, 4139 El Camino Way, P.O. Box 10139, Palo Alto, California 94303-0139, USA. Vol. 24. 652 pp. Price not mentioned.

The topics reviewed in this volume highlight recent trends in the fields of cell biology and developmental biology. Reviews on cell division, gene regulation and evolution, neuronal circuitry, cell–cell interactions, cell polarity and intracellular transport are likely to be of special interest to workers in the field. In the 'Perspective' chapter honouring his pioneering work on light microscopy, Shinya Inoué gives an engaging account of his early experiments on polarized light microscopy of dividing cells, which proved the existence and function of the mitotic spindle.

There has been a surge of interest in the past decade in understanding the intricacies of mitosis and meiosis, which has been made possible by the convergence of different conceptual approaches. The molecular mechanism and role of the cohesin complex in sister-chromatid cohesion is discussed by Koshland and coworkers. A key mechanism that drives mitosis to completion is the rapid degradation of certain mitotic proteins by the anaphase-promoting complex (a multisubunit E3 ubiquitin ligase), and Pesin and Orr-Weaver review the regulation of this process. Bhalla and Dernburg describe the various mechanisms of pairing and synapsis of homologous chromosomes during meiosis in different organisms. An important aspect of chromosome biology is the maintenance of large clusters of genes in a transcriptionally active or silent state. McStay and Grummt describe how active and silent rDNA clusters are distinguished by their pattern of DNA methylation, specific histone modifications and distinct nucleosome positions; furthermore, heterochromatin formation together with transcriptional silencing help maintain the structural integrity of nucleoli and genetic stability of rDNA repeats.

An enormous amount of information is now available on genome sequences, transcript profiles (transcriptomes) and protein profiles (proteomes) of a large number of organisms and of specific tissue or cell types. This has enabled scientists to use a 'systems biology' approach to understand biological systems as a whole. Methods to identify gene regulatory networks in plants from large datasets are highlighted by Long and colleagues. It is also now possible to examine the genetic basis and evolution of diverse aspects of cellular physiology that are seen across many species in nature. Different strategies adopted by placental mammals for evolution and diversification of genes involved in placental development such as appearance of new genes, use of alternative promoters and multigene families are described by Rawn and Cross. The evolutionary trends of coloration patterns are reviewed by Protas and Patel. An unusual aspect of animal development is the ability of some species to regenerate large parts of the body, and Brockes and Kumar deal with the evolutionary origins and functional aspects of this process. This review which includes important aspects of progenitor or stem cell functions during regeneration, as well as a review on spermatogomal stem cells by Oatley and Brinster are likely to be of interest to those in the field of stem cell biology. An inevitable outcome of normal growth and development is the process of ageing. Genome-wide comparisons between species suggest a significant conservation between longevity pathways in yeast and multicellular organisms. This newly emerging area of work is reviewed by Steinkraus and colleagues.

Colouration differences in cichlids. In the Great Lakes of East Africa, there are almost 2000 species of cichlids that have evolved relatively recently with a wide variety of colour patterns (images are courtesy of R. Roberts).

A central question in neurobiology that continues to dominate research in the field is how synaptic connections are established in the brain during development. Early models proposed the requirement for 'chemical tags' that allowed specific neuronal circuits to be established. Subsequently, a large number of recognition molecules have been discovered, and include those that confer properties of either adhesion or repulsion, both of which play crucial roles in specifying neural circuitry. Hattori and colleagues describe the properties of the Dscam family of cell adhesion molecules; alternative splicing at the *Drosophila* locus can potentially generate tens of thousands of Dscam1 isoforms and this exquisite diversity is essential for neural circuitry. During post-natal development, neural circuits are strengthened by sensory experiences that trigger specific signalling pathways between the nucleus and the synapse, as discussed in the review by Cohen and Greenberg. The formation of synapses occurs through long and intricate steps, and Jin and Garner highlight the molecular mechanisms of presynaptic differentiation.

Cell–cell and cell–matrix interactions are involved in the basic aspects of cell adhesion, locomotion and polarity, which are in turn crucial for cell differentiation and development. Takai and colleagues present a detailed account of the properties of nectins, which are key molecules involved in the formation of cell–cell