Cell Cycle Control; Edited by C. Hutchinson and D.M. Glover, Oxford University Press, New York, 1995. xv + 304 pp. £29.50 (pb). ISBN 019 9634106.

Cdc2 was identified by yeast geneticists nearly twenty years ago. At that time, cell cycle was just an emerging and virgin field of research with no need at all for a textbook. Our field has changed drastically since sisters of cdc2, the 'cyclin-dependent kinases' (CDK), were discovered and a family of cyclin companions was also identified. Cell cycle research is now a multidisciplinary field, and every emerging new question is enlarging the audience of CDKs.

The editors C. Hutchinson and D. Glover conclude the preface of their book with the following sentence 'We are conscious, as we go to press, of the important recent findings that we have been unable to cover as the field moves on, but nevertheless we hope that this book gives a timely overall perspective on current cell cycle research'. To my point of view, the answer is absolutely yes. The major difficulty of writing a textbook in a field that is moving as fast as the cell cycle has during the last decade, is perfectly avoided by the judicious and careful choice of the topics that are covered. The editors, who are leaders in the field, have commissioned chapters that have been written by experts in various aspect of cell cycle research. Indeed, this book not only provides a full coverage of all topics, but is enlightening the most fascinating advances of recent work. In addition, sufficient background is provided with each of the ten chapters to allow the reader to find easily the sources of primary data and to search for supplementary material.

The first chapter is an introduction to cell cycle control written by L. Hartwell, who initiated genetic work on budding yeast cell division cycle mutants in the early 70's. The next one by S. Reed, C. Hutchinson and S. MacNeill is a more technical introduction to the concepts and the experimental models that have made major contributions to cell cycle research. The following two chapters, by S. Reed and by S. MacNeill and P. Fantes are dedicated to the review of our current knowledge of the molecular mechanisms of control at the G1-S phase and the G2-M phase transitions in budding yeast and fission yeast, respectively. Chapter 5, by G. Basi and G. Draetta deals with questions arising from the study of the cdc2 kinase structure-function relationships, and also addresses the role and regulation of this kinase in vertebrate cells. So many kinases, so many cyclins, why? This is one of the most fascinating questions that is addressed in chapter 6 of this book, by J. Pines and T. Hunter. In chapter 7, J. Blow's contribution is an excellent up to date review of the regulation of S-phase. Chapter 8, by A. Zetterberg and O. Larsson, is dedicated to the study of cell growth control in mammalian cells.

One of the major advances of the last few years has been the discovery of several lines of evidence for connecting the molecular mechanisms of cell cycle control and cancerogenesis. This evidence is clearly reviewed in chapter 9 by E. Lees and E. Harlow. The last chapter, by H. White-Cooper and D. Glover beautifully illustrates how useful and powerful multicellular organisms can be to investigate specific aspects of cell cycle control in connection with developmental programs.

These ten chapters are of equal very good quality and have been efficiently compiled, making this a real textbook and not just a juxtaposition of specialized reviews. Scientists who do not follow the cell cycle literature on a daily basis, newcomers to the field and graduate students, all have great difficulty merging and assembling the mass of data that has been accumulated in the recent past. I am very much convinced that they will enjoy and greatly profit reading this book. So, it was time for this new excellent *Cell Cycle Control* textbook.

Bernard Ducommun

Cell Adhesion and Human Disease; Edited by J. Marsh and J.A. Goode, Ciba Foundation Symposium 189, Wiley, Chichester, 1995. ix + 243 pp. $49.95 (hb). ISBN 0-471-95279-6.

Adhesion molecules participate in a large number of cellular functions and activities. They contribute to normal biological processes and disease states such as cancer (invasion and metastasis), inflammatory disorders (rheumatoid arthritis and autoimmune diabetes) and cardiovascular diseases (heart attack and stroke). This Ciba Foundation Symposium volume contains the papers presented at a recent meeting on Cell Adhesion and Human Disease. Each paper is followed by a very interesting discussion. The meeting focused on blood cell (both leukocyte and platelet)-vessel interactions, but also dealt with adhesion molecules in skin and solid tumors. This volume presents current research on selectins and their ligands, immunoglobulin-related molecules, integrins, cadherins and other adhesion molecules. It also considers the therapeutic potential of adhesion molecule antagonists in inflammatory and malignant diseases. It should be of interest to investigators of adhesion molecules, as well as immunologists, hematologists and tumor biologists.

Following a stimulating introduction by R.O. Hynes, chairman of the symposium, adhesion molecules mediating blood cell-vessel interactions are explored in several chapters and discussions spread throughout the volume. Adhesion molecule knockouts, presented in two papers, beautifully demonstrate the biological relevance of these molecules. Wagner presents the P-selectin (CD62P) knockout and demonstrates the importance of this selectin in early leukocyte rolling along vascular endothelium and the subsequent leukocyte extravasation at inflammatory sites. Kwee et al. present E-selectin (CD62E)- and VCAM-1 (CD106)-deficient mice. Participation of E-selectin in neutrophil extravasation was less notorious than that of P-selectin. In contrast to the selectin knockouts, VCAM-1-deficient mice were not viable, indicating the relevance of this molecule during development. A human deficiency of the selectin ligand sialyl Le X (Sle^x, CD15s), known as leukocyte adhesion deficiency (LAD) II, is described by Etzioni et al. As in LAD I ($\beta 2$ integrin-, CD11/CD18- or Leu-CAM-deficiency), these patients suffer from recurrent bacterial infections. Endogenous protein-associated carbohydrate ligands for E-selectin are explored by Patel et al. The authors identify sialyl-di-Lewis X as the endogenous ligand.

Participation of adhesion molecules in detrimental inflammatory reactions is also explored following administration of blocking monoclonal antibodies. Winn et al. concentrate on $\beta 2$ integrins and L (CD62L)- and P-selectins in ischemia/reperfusion injury (stroke, myocardial infarction, hemorrhagic shock, etc.), while Rothlein and Jaeger summarize the effect of anti-ICAM-1 (CD54) antibody in animals and humans (allograft rejection and rheumatoid arthritis). Both groups report protective effect of the antibody treatments. Two chapters describe progress in leukocyte $\alpha\beta 1$ integrin (CD49d/CD29) and its ligands fibronectin and VCAM-1. Elices reviews the protective effect of antibodies to $\alpha 4$ in several animal models of allergy and chronic inflammatory diseases, and introduces a low molecular weight inhibitor of this integrin based on the LDV sequence of fibronectin. Humphries et al. dissect the mechanisms of VCAM-1 and fibronectin binding to $\alpha 4\beta 1$, and particularly the peptide sequences participating in this and other integrin-ligand interactions. Moreover, Hogg explores the binding sites of the integrin $\alpha L\beta 2$ (CD11a/CD18) for its ligand ICAM-1 and describes that, in addition to the I-domain, domains V and VI of αL also bind ICAM-1. Binding of von Willebrand factor to specific platelet membrane receptors and its relevance for platelet adhesion and thrombus formation at sites of vascular injury are summarized by Ruggeri, including the importance of shear forces.

Up-regulation of vascular adhesion molecules in inflamed skin, a process which parallels leukocyte infiltration, is presented by Barker. The skin, because of its accessibility, is an ideal organ to study adhesion molecule expression in inflammatory disorders such as psoriasis and atopic dermatitis. Stanley describes two blistering skin diseases (pemphigus foliaceus and pemphigus vulgaris) as illustrative examples of defective cell-cell adhesion in the epidermis. These patients' autoantibodies to desmogleins, desmosomal glycoproteins members of the cadherin gene superfamily, block adhesion of the epithelial cells.

Adhesion molecules in human solid tumors are explored in three