As part of the Cancer Drug Discovery and Development series, this book aims to explain basic biological processes and signalling pathways and to link that knowledge to the anticancer drugs associated with those pathways. Each chapter begins with a detailed introduction to the basic science of the specific process, pathway or drug family before entering into their clinical applications. The book is divided into six parts and, although not officially stated, these roughly divide into two halves. The first half deals with the mechanisms of apoptosis and other modes of cell death; telomeres and senescence; and the DNA damage response. In addition to apoptosis, other modes of cell death dealt with within this half of the book include alternative cancer therapy targets such as autophagy, anoikis and mitotic catastrophe. The clinical aspects of the earlier chapters nicely links to the second half of the book, which is more pharmacologically/therapeutically orientated with three parts dealing with tumour resistance and sensitisation, established cancer therapies and developing cancer therapies. A flavour of the chapters in this half of the book include, 'perturbation of cellular functions by Topoisomerase II inhibitors' and 'monoclonal antibodies in lymphomas'. With some 28 chapters, the book covers a wide range of topics within the title’s remit, from information on the mechanisms of apoptosis to antimetabolite and tyrosine kinase inhibitor therapies.

The book is generally well written and extremely informative. The information within is as up to date as can be expected from this kind of book, but the reader should bear in mind the speed at which these fields are rapidly developing. The book engagingly details the history of each area and the main studies involved in forming that history. This gives the reader a broad understanding of the background to the particular area of research or therapy being discussed. On the whole, chapters are well referenced and provide easy access to the relevant literature for further reading. My only disappointment in the book was the under-representation of senescence: only a couple of the 28 chapters are wholly concerned with senescence and there are limited references to senescence in additional chapters.

I have certainly learnt a lot from reading this book. Having been previously solely involved in basic research, I have now moved into a translation research cancer centre. I therefore found the information on the therapeutic applications of what I had previously studied in terms of cell/organism function very useful. At £104 perhaps this book might be a little too expensive for an individual’s collection, but I would have no hesitation in recommending it to a laboratory/clinical group or library for purchase.

A Young1

1Cellular Senescence and Tumour Suppressors Laboratory, Cancer Research UK, Cambridge Research Institute, Robinson Way, Cambridge CB2 0RE, UK

Apoptosis, Senescence and Cancer provides insight into established practices and research into apoptosis and senescence by thoroughly examining novel and emerging techniques and research in the fields of cell death pathways, senescence growth arrest, drugs and resistance, DNA damage response, and other topics which still hold mysteries for researchers. The volume is divided into six easy to follow sections. The first is Apoptosis and Alternative Modes of Cell Death, followed by chapters on Telomeres and Telomerase, Senescence, Genomic Instability and Tumorigenesis. The third part covers DNA Da Apoptosis, Senescence and Cancer (Cancer Drug Discovery and Development). Post author. By. Post date. December 28, 2017. Get IT free here. http://nitroflare.com/view/617F13484B2EFA3/1617376213.pdf. â† © Problem-Free Diabetes: Controlling Diabetes With the Help of the Power of Your Metabolism â† Memory in a Social Context: Brain, Mind, and Society. Cancer is one of the scenarios where too little apoptosis occurs, resulting in malignant cells that will not die. The mechanism of apoptosis is complex and involves many pathways. The abundance of literature suggests that targeting apoptosis in cancer is feasible. However, many troubling questions arise with the use of new drugs or treatment strategies that are designed to enhance apoptosis and critical tests must be passed before they can be used safely in human subjects. 1. Introduction. This in turn, may help in the development of drugs that target certain apoptotic genes or pathways. Caspases are central to the mechanism of apoptosis as they are both the initiators and executioners. There are three pathways by which caspases can be activated. In cancer, the apoptosis cell-division ratio is altered. Cancer treatment by chemotherapy and irradiation kills target cells primarily by inducing apoptosis. Hyperactive apoptosis[edit]. On the other hand, loss of control of cell death (resulting in excess apoptosis) can lead to neurodegenerative diseases, hematologic diseases, and tissue damage. It is of interest to note that neurons that rely on mitochondrial respiration undergo apoptosis in neurodegenerative diseases such as Alzheimer’s[88] and Parkinson’s.[89] (an observation known as the â€œInverse Warburg hypothesisâ€ [90][81] ). Â “Apoptosis in the development and treatment of cancer”. Carcinogenesis. 26 (2): 263â€“70. doi:10.1093/carcin/bgh283.