Preface

Cancer is one of the most feared diseases of the twentieth century, and it is spreading even further with the passage of time and the rise in incidence in the twenty-first century. The situation is so dire that one in every four people is at risk of developing cancer during their lifetime. The number of new cancer cases registered in India each year exceeds 11 lakhs; however, this figure is higher than 14 million worldwide. Oncologists have looked into a variety of treatment options that are targeted at the rapidly multiplying mutated tumour cells, including surgery, radiation, chemotherapy, and immunotherapy. In terms of chemotherapy, many agents that are developed by researchers for the effective elimination of tumors. Each tumors show genotypic difference; hence it is difficult to treat with single chemotherapeutic agent to all the cancer types. There are variety of drug targets that are available for the development of new drugs. In this book, was aimed to consolidate the drug targets of various cancers. Cancerous cells use HSP70s to repress numerous apoptotic pathways, regulate necrosis, bypass the cellular senescence programme, interfere with tumour immunity, promote angiogenesis and enhance metastasis. Cancer "addiction" to HSP70 is explained by its direct involvement in most cancer hallmarks, including tumour survival and proliferation. HSP70 has many stages during its catalytic cycle, suggesting it can multi-function in malignant cells. Tumor cells actively release HSP70 into the extracellular environment, affecting patient life. Because of its therapeutic importance, small molecule inhibitors of the HSP70 machinery were created. The tumour microenvironment characterizes the non-cancerous cells in the tumour and has enabled researchers to analyze the behavior and response of cancer cells to a treatment process; it comprises of a tissue that may have predictive implications for tumour behavior and response to therapy. These include fibroblasts, immunological cells, and the cells that make up the blood vessels. It also contains the proteins produced by all of the cells present in the tumour that promote the proliferation of the cancer cells. It will be critical for finding cell or protein targets for cancer prevention and treatment reasons if changes in the tumour microenvironment are monitored utilizing its molecular and

cellular profiles as the tumour advances. It is hoped that immunotherapy-based cancer medicines would significantly reduce the burden of personal suffering and community costs associated with cancer in the future decades. We now have solid proof of efficacy for two immunotherapeutic approaches, checkpoint inhibition and cellular therapy with autologous ('self') chimeric antigen receptor T cells (CAR T cells), which promise even faster progress as they are refined and combined with existing conventional therapies and each other. The PI3K/AKT/mTOR signaling system is hyperactive or changed in many cancers and affects cellular activities such as survival, proliferation, growth, metabolism, angiogenesis, and metastasis. The PI3K/AKT/mTOR system is regulated by several upstream signaling proteins and collaborates with multiple downstream effectors, particularly the RAF/MEK/ERK pathway. Limitations in available targeted therapy drugs and obstacles posed by tumour heterogeneity across cancer types highlight the need of targeting PI3K, AKT, and mTOR pathway changes in tailored treatment strategies. We offer a comprehensive PI3K/AKT/mTOR network that illustrates the complicated interplay between compensatory mechanisms. Aberrant Wnt/β-catenin signaling is frequently found in malignancies, particularly colorectal cancer (CRC), and is essential for carcinogenesis. Approximately 80% of CRC cases have adenomatous polyposis coli gene mutations, while half of the remaining instances have Wnt/β-catenin signaling pathway alterations. Unsurprisingly, the Wnt/β-catenin signaling pathway may be a useful therapeutic target for CRC. Several Wnt/β-catenin signaling pathway inhibitors have been developed for CRC treatment, however none have been approved for use in oncology. Of the cytokines linked to cancer, interleukin-6 causes many of the cancer hallmarks via the Janus kinase/signal transducer and activator of transcription 3 signaling pathway. Also, IL-6-mediated JAK/STAT3 signaling pathway dysregulation is linked to various human solid cancers, including colorectal cancer (CRC). A new approach to treating CRC involves modulating the IL-6/JAK/STAT3 signaling pathway. When cells degrade their own waste, they engage in what is known as utophagy. This process results in the production of energy and macromolecular precursors while also allowing cells to maintain a constant level of cell

components and function. It has been hypothesized that cancer therapy should target autophagy's conflicting and context-dependent roles in cancer. Interventions to both promote and inhibit autophagy have been proposed. As a result, the therapeutic targeting of autophagy in cancer has been criticised on occasion and is now considered controversial. Throughout this Review, we propose a path forward for the successful targeting of autophagy by better understanding the context-dependent roles of autophagy and by leveraging contemporary approaches to clinical trial design to achieve this goal. The current review highlights existing therapeutic tactics and the quest for potential therapeutic options to treat CRC. This book comprised of ten chapters and each chapter discussed in detail about the various cancer drug targets.

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