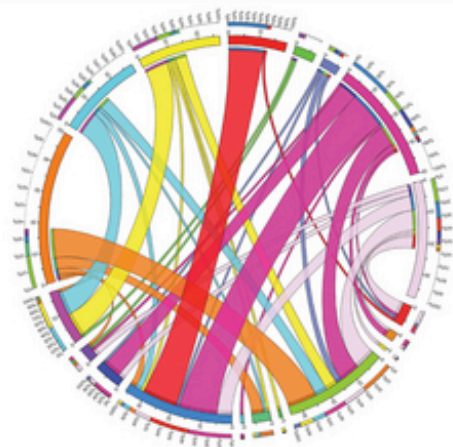
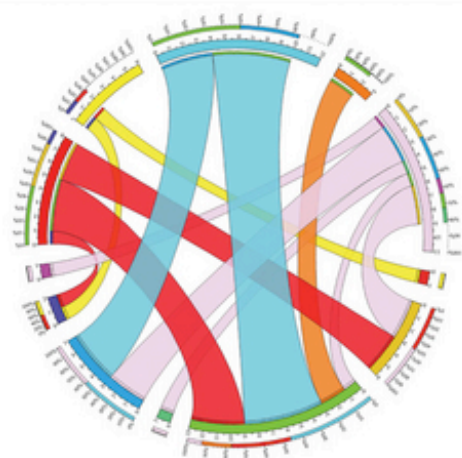


THE GENETIC BASIS OF
**HAEMATOLOGICAL
CANCERS**

EDITED BY **Sabrina Tosi & Alistair G. Reid**



WILEY

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Preface

The haematological malignancies are a complex group of neoplastic diseases, linked by their origin in bone marrow-derived cells. Since the discovery of the Philadelphia chromosome, in the 1960s, as the pathognomonic marker of chronic myeloid leukaemia, the field of haematological malignancy has provided several important paradigms for the direct contribution of causal genetic lesions to the initiation of human cancer.

The subsequent leap in our understanding of leukaemia and lymphoma pathogenesis via a variety of molecular and cytogenetic abnormalities that disrupt normal cellular processes has challenged traditional approaches to disease classification and transformed both the diagnosis and management of patients. The characterization of tumour cells by genetic methods is now regarded as being as important as the traditional morphological approach to diagnosis. This trend is being accelerated by the introduction of monoclonal antibody therapy and by novel drugs designed to target specifically the molecular abnormalities responsible for the development of the tumour. Somatic genetic changes therefore increasingly define not just the diseases themselves, but the way in which an individual patient should best be treated and monitored.

With the following chapters, compiled by leading researchers in the field, we aim to provide a summary of current knowledge on the contribution of genetic and epigenetic lesions to the biology and management of haematological malignancies. A unifying factor of these biologically diverse diseases is the recent explosion of information on hitherto unrecognized molecular lesions arising from the application of novel next-generation sequencing technologies. In most diseases, these newly identified aberrations are already contributing to improved stratification and, in some cases, showing early promise as therapeutic targets. It is hoped that further functional analysis of recurrent lesions will permit the development of additional therapies targeted against critical oncogenic drivers. Although the majority of recurrent changes appear to have been identified, there remains scope for further refinement of this knowledge with studies of larger cohorts,