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Cancer Genomics

From Bench to Personalized Medicine



Cancer Genomics From Bench To Personalized Medicine

Ryan D. Morin, Stephen B. Montgomery



Cancer Genomics From Bench To Personalized Medicine:

Cancer Genomics Graham Dellaire, Jason N Berman, Robert J. Arceci, 2013-11-21 Cancer Genomics addresses how recent technological advances in genomics are shaping how we diagnose and treat cancer Built on the historical context of cancer genetics over the past 30 years the book provides a snapshot of the current issues and state of the art technologies used in cancer genomics Subsequent chapters highlight how these approaches have informed our understanding of hereditary cancer syndromes and the diagnosis treatment and outcome in a variety of adult and pediatric solid tumors and hematologic malignancies The dramatic increase in cancer genomics research and ever increasing availability of genomic testing are not without significant ethical issues which are addressed in the context of the return of research results and the legal considerations underlying the commercialization of genomic discoveries Finally the book concludes with Future Directions examining the next great challenges to face the field of cancer genomics namely the contribution of non coding RNAs to disease pathogenesis and the interaction of the human genome with the environment Tools such as sidebars key concept summaries a glossary and acronym and abbreviation definitions make this book highly accessible to researchers from several fields associated with cancer genomics Contributions from thought leaders provide valuable historical perspective to relate the advances in the field to current technologies and literature

Cancer Genomics Michael Fraser, Alejandro

Berlin, Veronique Ouellet, Fred Saad, Robert G. Bristow, 2013-11-21 Prostate cancer CaP is the most commonly diagnosed malignancy in men in the Western world In North America more than 275000 men are diagnosed annually whereby approximately 1 in 6 men will be diagnosed with CaP in their lifetime and 1 in 34 men will die from castrate resistant metastatic disease Unfortunately current clinical prognostic factors explain only a proportion of the observed variation in clinical outcome from patient to patient Furthermore over treatment of indolent and low risk cancers leads to inappropriate morbidity following radiotherapy or surgery As such better predictors of individualized prognosis and treatment response are urgently needed to triage patients to customized and intensified CaP treatment Recent developments in next generation sequencing have made it possible to identify prognostic and predictive signatures based on genomic profiles Herein we review the recent genetic data pertaining to prostate cancer carcinogenesis progression castrate resistance and metastases We discuss the genetic basis of CaP progression from localized to systemic disease e g point mutations copy number alterations and structural variants and important considerations for CaP biology including intra and inter prostatic heterogeneity multifocality and multiclonality TMPRSS2 ERG and other ETS family gene fusions and the role of the tumor microenvironment e g hypoxia and the contribution of cancer associated stroma Finally we focus on the use of genomic markers as prognostic factors for local failure and for systemic disease as novel risk stratification tools in triaging patients to existing treatment options and ultimately the potential of genomics for the identification of molecular targets for CaP therapy We conclude by summarizing selected outstanding questions in CaP biology that can be addressed effectively through

international cooperation between genome sequencing projects such as The Cancer Genome Atlas TCGA and the International Cancer Genome Consortium ICGC

Cancer Genomics Daniel Gaston, Carman Giacomantonio, 2013-11-21

Colorectal cancer CRC is the third most common form of cancer and a leading cause of cancer related mortality in both men and women particularly in Western and developed nations The high mortality rate has been attributed to the fact that colon cancer is often diagnosed at a late stage Three primary subtypes of CRC have been described based on their molecular pathology and their underlying genetics chromosomal instability CIN microsatellite instability MSI and CpG island hypermethylation Over the last 30 years molecular and genetic studies have determined a number of key genetic pathways that are subverted in CRC including those involving APC KRAS and the p53 tumor suppressor More recently high throughput genome wide studies have begun to characterize the broader genomic features of CRC These high throughput studies provide an ever expanding and increasingly complex view of the molecular underpinnings of CRC The chief goal of these studies being the identification of new therapeutic targets as well as the definition of prognostic and diagnostic biological markers of CRC Here we highlight the key genetic and molecular pathways underlying CRC as well as more recent insights into this disease uncovered by genomic studies

Cancer Genomics David Malkin, 2013-11-21

Cancer is the most common cause of disease related death in children beyond the newborn period Most cancers are thought to arise sporadically however classical studies of well defined familial cancer associations known as cancer predisposition syndromes CPS together with emerging work arising from new high resolution genomic platforms have confirmed that at least 25% of childhood cancers result from hereditary factors The spectrum of cancers found in the diverse array of known hereditary cancer syndromes is vast Similarly the number of genes linked to these syndromes continues to expand This chapter explores the genotype phenotype correlations in several defined cancer predisposition syndromes that primarily affect children In particular a selection of syndromes that are caused by germline mutations in classical tumor suppressor genes RB1 TP53 WT1 and oncogenes RET syndromes associated with congenital developmental anomalies Beckwith Wiedemann syndrome Gorlin syndrome and an emerging syndrome associated with microRNA processing DICER1 provide examples of the heterogeneity that these syndromes exhibit The chapter concludes with a discussion of the clinical impact of genetic testing and clinical surveillance for early cancer detection

Cancer Genomics Katayoon Kasaian, Yvonne Y. Li, Steven J.M. Jones, 2013-11-21

Advances in high throughput sequencing technologies have enabled cost effective sequencing of a single human genome at an unprecedented rate facilitating scientific endeavours never imagined possible before These improvements have transformed the field of cancer genomics allowing the complete molecular characterization of individual cancer genomes However the promise of unveiling the complexity of cancer has lent itself to yet another level of complexity the task of managing and integrating the massive amount of data that is generated as part of such experiments There is a need to manage and store large sequence datasets such that they can be accessed and shared readily but more importantly

there is a need for their thorough and efficient analysis. Developments and improvements in computer hardware and processing power have eliminated the data storage and access issues. Additionally, bioinformatic algorithms and software designed specifically for the analysis of cancer genomic data are now able comprehensively to profile the mutations in a cancer sample to provide a probability score for their role as disease drivers and to identify potential actionable targets. Although the functional validation of putative driver mutations will remain a necessity, continued improvements in sequencing technologies and analysis tools promise to provide increasingly reliable computational analysis of cancer genomes.

Cancer Genomics Janet E. Dancey, 2013-11-21. Many hope that the promise that omics science holds for medicine will be realized through the development of better biomarkers for patient management. However, the development of omic technologies magnifies the issues and challenges of performing high quality biomarker studies. The complexity of these technologies and of the resulting high dimensional data require rigorous technical, statistical, bioinformatics, laboratory and clinical procedures to develop, evaluate and validate these tests. A number of considerations are key to the design and successful execution of genomic biomarker studies. These include the assessment of: 1. the complexity and heterogeneity of cancer samples; 2. the quality and quantity of tumor specimens; 3. the potential bias inherent in the assays; 4. the availability of appropriate controls or standards; 5. technical validation of assays and bioinformatics analysis; and 6. the validity of the final interpretation of results. In this chapter, we review these key considerations required to design and conduct biomarker studies that will yield results that can confidently inform future clinical research and clinical practice.

Cancer Genomics Elizabeth J. Perlman, Peter Hohenstein, 2013-11-21. Wilms tumors are pediatric tumors of the kidney found in young children with a mean age of 40 months. Genes found mutated in Wilms tumors have essential functions in the early stages of nephron development. Different subsets of Wilms tumors can be recognized via genetic and transcriptomic analysis, and ontological analysis of expression patterns suggests different developmental origins for different tumor classes. A better understanding of normal early kidney development will help the design of new therapeutic approaches for different classes of Wilms tumors.

Cancer Genomics Daniel A. Morgenstern, Meredith S. Irwin, 2013-11-21. Neuroblastoma is one of the most common childhood tumors and has a remarkably diverse pattern of presentation and clinical behavior. Current management approaches rely on risk stratification using clinical, pathological and increasingly genetic factors. This chapter explores the current state of knowledge of the genetic factors behind neuroblastoma and discusses how these may impact on treatment. Both segmental chromosomal abnormalities including loss of 1p or 11q or gain of 17q and changes in individual genes such as MYCN amplification, mutations in ALK and ATRX have been implicated in neuroblastoma pathogenesis. Recent whole genome approaches have identified multiple genetic variants involving LMO1, BARD1, LIN28B, NBPF23 and others that may predispose to neuroblastoma, while germline mutations in ALK and PHOX2B are associated with rare familial cases of neuroblastoma. The roles of mRNA gene expression profiling, microRNAs that regulate protein translation from mRNA and

epigenetic modifications such as DNA methylation in neuroblastoma are also discussed Incorporation of subsets of these genomic factors into risk stratification will ultimately lead to more personalized treatment for neuroblastoma patients

Cancer Genomics Zhaolin Xu,2013-11-21 Lung cancer remains the leading cause of cancer related death worldwide Although surgical resections of these tumors are considered as one of the most effective treatments most lung cancer patients present at an advanced stage of the disease at the time of diagnosis and are not candidates for surgical resection Overall the prognosis of lung cancer is very poor and the 5 year survival rate is only about 16 % which has not significantly changed in the past several decades Therefore seeking new directions of treatment for this most deadly disease becomes crucial Recent development in the understanding of the molecular pathogenesis of lung cancer has led to new strategies of treatment Development of lung cancers is thought to be driven by gene mutations in most if not all cases Detailed analysis at the molecular level to identify these gene mutations or alterations in lung cancer provides the insight for understanding the disease and is fundamental for establishment of personalized targeted therapy Personalized targeted therapy based on particular gene mutations has shown to be effective and is believed to be one of the new directions of the treatment in dealing with this disease In modern oncology there is an increasing need to facilitate the development and implementation of biomarkers based on known gene mutations alterations in clinical practice and identification of new gene mutations alterations through high throughput DNA sequencing technology to enter a new era of personalized targeted therapy for lung cancer patients

Cancer Genomics Francesca Cottini,Kenneth C. Anderson,Giovanni Tonon,2013-11-21 Multiple myeloma MM is a clonal proliferation of abnormal plasma cells in the bone marrow BM associated with a monoclonal protein and end organ damage MM originates from a pre malignant condition called monoclonal gammopathy of undetermined significance MGUS and can progress to an extramedullary disease termed plasma cell leukemia PCL which invades the bloodstream MM cells manifest a wide spectrum of genomic abnormalities creating a strong intertumoral heterogeneity Historically MM patients have been divided into two subgroups hyperdiploid cases with 46 chromosomes and non hyperdiploid cases However the introduction of novel technologies such as fluorescence in situ hybridization FISH array comparative genomic hybridization aCGH and sequencing techniques is helping to unveil the complexity of MM genomes In particular MM cells present recurrent translocations which deregulate known oncogenes such as CCND1 FGFR3 MMSET c MAF and MYC numerous copy number variations CNVs including deletion of chromosome 13 deletion of chromosome 17p13 and amplification of chromosome 1q21 and various somatic mutations in genes involved in cancer proliferation RAS BRAF FGFR3 protein homeostasis and RNA processing FAM46C DIS3 XBP1 and LRRK2 NF B signaling histone methylation and tumor suppression TP53 This chapter will summarize our current knowledge of the MM genomic field focusing on the different types of abnormalities and their relationship with the phases of disease

Cancer Genomics Hye-Jung E. Chun,Jaswinder Khattra,Martin Krzywinski,Samuel A. Aparicio,Marco A. Marra,2013-11-21 Cancer results from accumulated

mutations in the genome Sequencing is an accurate method to detect mutations Second generation sequencing technology commonly referred to as next generation sequencing technology enables rapid efficient and affordable DNA sequencing and is transforming the scale and scope of cancer research The technology is sufficiently flexible and affordable to allow sequencing of many cancer genomes and thus facilitates both sequencing of samples from large patient cohorts and during disease progression in individual cancer patients The high depths of redundant sequence coverage that can be obtained using some second generation sequencing technologies along with sequencing reads amplified from single DNA molecules facilitate detection of subclones of cells in tumors Large scale genome sequencing of hundreds or even thousands of cancer samples is being conducted by several groups that aim to identify and characterize cancer driver mutations Goals of such work previously infeasible with Sanger sequencing instruments are to use this information to improve cancer prognosis diagnosis and therapeutic decision making The speed of data analysis is rate limiting and investigators are struggling to accommodate and interpret the data deluge produced by second generation technologies In this chapter we discuss cancer properties that are revealed by sequencing and the implication of such properties in experimental design and data interpretation We describe past current and upcoming sequencing technologies and the application of second generation sequencing technologies in cancer genomics Finally we discuss the impact of second generation sequencing technology in shaping personalized medicine

Cancer Genomics Robert J. Arceci, Jason N. Berman, Soheil Meshinchi, 2013-11-21 On first consideration acute myeloid leukemia AML represents a nearly insurmountable challenge in terms of understanding it at the molecular level in large part because of its immense heterogeneity as well as its variability across different age groups In addition while significant progress has been made in the overall survival of subsets of patients with AML many continue to show little progress in terms of positive treatment outcomes Cytogenetic and initial molecular studies have resulted in the ability to stratify patients into specific risk categories that predict favorable intermediate and poor risk outcomes However these categories are limited in their ability to predict accurately how individual patients will respond to therapy and have not resulted in the ability to treat effectively patients with specific treatments They have however resulted in excluding hematopoietic stem cell transplantation for patients with favorable risk disease Genome wide analysis promises to improve both treatment and outcomes The initial studies using whole exon or whole genome sequencing identified mutations in several novel genes that surprisingly were involved in regulating DNA methylation and chromatin structure Subsequently mutations were found in genes encoding transcription factors signaling pathway modulators and genes involved in RNA splicing Further analyses have identified mutations in key elements of miRNAs Genome wide methylation studies have highlighted key patterns that track with specific cytogenetic and gene mutations Such epigenetic studies have led to the use of treatments directed to altering chromatin structure and DNA methylation These treatments remain targeted specifically at specific enzymatic components of chromatin structure and function but their key molecular consequences remain unclear

and clinical responses unpredictable RNA sequencing has led to the identification of both novel pathways of leukemia cell survival and unexpected fusion transcripts which may ultimately be therapeutically targeted

Cancer Genomics Cécile Le Page, Anne-Marie Mes-Masson, Anthony M. Magliocco, 2013-11-21 The tissue microarray TMA is a recent technology developed to increase the performance of molecular profiling in cancer research by rapidly performing large scale studies while reducing experimental variables and conserving priceless tissue samples The technology allows on a single glass slide the parallel analysis of protein or genes in an array composed of hundreds of paraffin embedded cored tissue specimens TMAs can be evaluated with immunohistochemistry fluorescent dye assays and in situ hybridization ISH While strictly a research and screening tool TMAs enable the rapid investigation of normal and cancer tissues and are especially useful for cancer biomarker validation studies In oncology the ability to couple TMA results to clinical parameters is especially useful for the development of clinically useful prognostic and predictive biomarkers derived from genomics studies This chapter will introduce the readers to the main technical aspects of TMA construction and the methods of TMA analysis particularly in the context of breast and ovarian cancers Important points for consideration in order to avoid the common pitfalls of the technology will be presented and a particular focus will be provided on quality control and analysis of TMA

Cancer Genomics Shahrad Rod Rassekh, Colin J.D. Ross, 2013-11-21 In the last 50 years the cure rate for children with cancer has increased from 5 10% to over 80% today However this dramatic improvement in outcome has come at a significant cost Approximately 40% of children suffer irreversible life threatening or long lasting toxicities that are caused by their medications during cancer treatment Recent technological advances in genomics have opened the door to potentially identifying genetic differences between individual children that may explain their different responses to cancer therapies The goals of the emerging field of cancer pharmacogenomics are to identify the genomic factors responsible for individual differences in drug response specifically drug effectiveness and susceptibility to adverse drug reactions Pharmacogenomics helps to explain why one child treated with the same medication as another child may respond well to therapy while another patient may not respond or worse may develop a life threatening adverse drug reaction The focus of this chapter is to explore recent developments in the pharmacogenomics of pediatric cancer with a focus on serious adverse drug reactions Specific drugs of focus include thiopurines methotrexate cisplatin vincristine and anthracyclines Several pharmacogenomic tests are currently available to provide dosing recommendations and the number of these tests is expected to increase significantly in the future There is a strong immediate need for international collaboration to advance this research to reduce the occurrence of severe adverse drug reactions in pediatric oncology

Cancer Genomics Louise Parker, 2013-11-21 The number of people diagnosed with cancer each year will almost double to 21 million cases worldwide by 2030 because of the aging population Studies of the human genome have demonstrated that as few as 5 10% of adult cancers are due to genetic inheritance Over 90% of cancers are due to endogenous or exogenous exposure to chemicals and radiation many of these

carcinogenic exposures to tobacco smoke for example are avoidable and thus the consequent disease is preventable The exposome is the concept which captures all carcinogenic exposures across the lifetime quantified by direct biomarker assessment Exposomics is in its infancy but if progress such as has occurred in the Human Genome project occurs in this field it has the potential greatly to enhance our understanding of the complex interactions and mechanisms of action of environmental exposures In particular evidence is increasing that cancer is as much a disease of the epigenome as the genome and that many of these environmental factors act to alter gene expression through changes in the epigenome Exposomics will enable us better to avoid carcinogenic exposures to limit the carcinogenic effect of these exposures and to identify potential new targets for developmental therapeutics Cancer Genomics Amal M EL-Naggar,Gabriel

Leprivier,Poul H Sorensen,2013-11-21 Soft tissue sarcomas STSs are a diverse group of uncommon mesenchymally derived malignant tumors and commonly present as an asymptomatic mass almost anywhere in the body The most important steps in the diagnosis and therefore subsequent management of STSs include adequate tumor biopsies for proper histologic evaluation including immunohistochemical studies and detection of disease extension and radiological imaging Although STSs are relatively uncommon they are typically high grade and if diagnosed at an advanced stage survival rates for such patients are poor Although important insights into STS pathogenesis and new molecular diagnostic tools have emerged prognosis for sarcoma patients with metastatic disease has failed to improve Identifying those factors that contribute to STS metastasis that could be targeted therapeutically would have a tremendous impact on survival in these diseases Here we summarize characteristics of some of the more common STS subtypes as well as known molecular alterations and their roles in sarcomatogenesis *Cancer Genomics* Matthew Herder,Conrad V Fernandez,2013-11-21 Large scale cancer genomics

research projects raise a range of ethical and legal issues We focus on two emerging themes challenged within genomic research 1 issues surrounding the return of research results to research participants whether in summary or individualized form and 2 issues connected to the commercialization of research including the patenting of biomedical discoveries and biological materials We examine current thinking in these areas and highlight areas for further exploration **Cancer Genomics** Angela Greco,Claudia Miranda,Maria Grazia Borrello,Marco A. Pierotti,2013-11-21 Thyroid cancer represents the most common endocrine malignancy and its incidence is increasing The majority of thyroid tumours 90 95% originate from the follicular epithelial cells it includes several histotypes and related variants characterized by different biological and clinical behavior such as differentiated papillary and follicular carcinoma PTC and FTC and poorly differentiated and anaplastic carcinoma PDTC and ATC Medullary thyroid carcinoma MTC represents a small fraction of thyroid tumors and originates from the parafollicular C cells PTCs feature a frequent deregulation of the MAPK pathway as RET PTC TRK RAS or BRAF oncogenes have been found in about 70% of cases FTCs are associated with RAS mutations and PAX8 PPAR oncogenic chromosome rearrangements PDTCs and ATCs display some alterations occurring in differentiated tumors as well as other

alterations specific of late tumor stages MTC carry RET or RAS mutations Here we summarize the molecular alterations detected in each thyroid tumor type their role in the process of thyroid carcinogenesis as well as how high throughput gene expression analyses have contributed to the study of this malignancy **Cancer Genomics** Ryan D. Morin,Stephen B. Montgomery,2013-11-21 Identifying gene expression changes in cancer provides opportunities to identify biomarkers that can be informative in regard to risk and in the choice of treatment options In recent years advances in sequencing have provided not only a quantitative measure of gene expression but the resolution of diverse species of RNA alternative transcripts and allele specific expression As well such data have revealed novel sequences such as those from pathogens mutations resulting in amino acid differences and fusion transcripts resulting from translocations and other structural alterations each of which can inform the development of novel treatment strategies or potential preventive measures In this chapter we will discuss how transcriptome sequencing is conducted and analyzed and provide examples that illustrate its utility in studying cancer samples **Cancer Genomics** Jason N. Berman,Priscilla P.L. Chiu,Graham Dellaire,2013-11-21 Genome wide association GWA studies and tumor specific epigenome transcriptome and genome sequencing projects are generating an ever growing list of susceptibility alleles as well as putative gain and loss of function gene mutations associated with cancer These genetic changes ultimately need to be validated to determine their contribution to the initiation progression and likelihood of treatment response for various cancers The bottle neck is no longer obtaining sequence data or completion of the GWA studies but rather the ability efficiently to validate candidate genes identified by these projects In vivo studies in animal models are the gold standard for validation of these candidate drivers and modifiers of cancer Furthermore once a gene product or molecular pathway has been validated as playing an important role in the development or progression of cancer animal models provide the necessary preclinical data for evaluation of the efficacy and toxicity of new therapeutics targeting that gene or pathway As such animal models play an essential role in cancer research by facilitating the translation of genomic discoveries into preclinical studies that precede new targeted therapies for cancer In this chapter we will discuss vertebrate and invertebrate animal models as they apply to cancer genomics as well as key technologies employed In particular we will focus on the use of murine and zebrafish human tumor xenografts and transgenic models

Unveiling the Magic of Words: A Review of "**Cancer Genomics From Bench To Personalized Medicine**"

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