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Causes and biology: Cancers and its markers

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Abstract

Cancer is a disease of the cell. This rather simple statement implies an enormous complexity when attempting to identify efficacious anticancer agents. One of the major issues associated with anticancer research is that traditional targetdirected strategies are confronted with the essentiality of the function of the target in healthy cells. Inevitably, targeting proteins that have essential functions are likely to lead to chemical entities with narrow therapeutic windows and significant toxic effects. An additional challenge is the unstable epigenetic and genetic status of cancer cells, undergoing multiple mutations, gene copy alterations, and chromosomal abnormalities that have a direct impact on the efficacy of anticancer agents at different stages of the disease ^[2]. All these aspects make cancer drug discovery extremely difficult and have led to poor clinical approval success rates compared to other therapeutic areas.

Therefore, individualized therapy is paramount for improving of cancer treatment. The development of rationalized and individualized therapy is reliant on the identification of the specific biomarkers, validation of the biomarkers to identify the therapeutic targets, and drug development against the identified.

Keywords: Biomarkers, Cancer Cells, Cancer Therapy, Cancer Targets

1. Introduction

Cancer is a gathering of sicknesses including irregular cell development with the possibility to attack or spread to different parts of the body. These stand out from benevolent tumours, which don't spread to different parts of the body ^[1]. Possible signs and side effects incorporate a bump, strange dying, delayed hack, unexplained weight reduction, and an adjustment in gut movements. Tobacco use is the cause of about 22% of cancer deaths. Another 10% are due to obesity, poor diet, lack of physical activity, and excessive drinking of alcohol. Other factors include certain infections, exposure to ionizing radiation and environmental pollutants. In the developing world, 15% of cancers are due to infections such as Helicobacter pylori, hepatitis B, hepatitis C, human papillomavirus infection, Epstein–Barr virus and human immunodeficiency virus. , Colorectal cancer, Non-Hodgkin lymphoma, Prostate cancer, Lung cancer, stomach cancer ^[2]. Cancer starts when cells change abnormally. Cancer is when abnormal cells divide in an uncontrolled way. Some cancers may eventually spread into other tissues. There are more than 200 different types of cancer. 1 in 2 people in the UK will get cancer in their lifetime. Thanks to research many people are cured (Figure 1).

Cancer grows as cells multiply over and over. Cancer starts when gene changes make one cell or a few cells begin to grow and multiply too much. This may because a growth called a tumour. Some cancers can spread to other parts of the body ^[3]. A primary tumour is the name for where a cancer starts. Cancer can sometimes spread to other parts of the body – this is called a secondary tumour or a metastasis. Cancer and its treatments can affect body systems, such as the blood circulation, lymphatic and immune systems, and the hormone system. Most cancers start due to gene changes that happen over a person's lifetime. More rarely cancers start due to inherited faulty genes passed down in families ^[4]. Genes, DNA and cancer Genes and inherited cancer risk. Cancer can sometimes come back. Many cancers are cured. But in some people cancer can return ^[5]. Some cancers can't be cured but treatment is often able to control them for some years. Cancers are divided into groups according to the type of cell they start from. They include, Carcinomas, Lymphomas, Leukaemia's, Brain tumours, Sarcomas. Stages and grading of cancer. Staging and grading give an idea of how quickly a cancer may grow and which treatments may work best. The stage of a cancer means how big it is and whether it has spread. Grading looks at how abnormal the cancer cells ^[6].

2. Material and Methods

Cancer biomarkers can be DNA, mRNA, proteins, metabolites, or processes such as apoptosis, angiogenesis or proliferation. The markers are produced either by the tumor itself or by other tissues, in response to the presence of cancer or other associated conditions, such as inflammation ^[7]. Such biomarkers can be found in a variety of fluids, tissues and cell lines. "A biological molecule found in blood, other body fluids, or tissues that is a sign of a normal or abnormal process, or of a condition or disease. A biomarker may be used to see how well the body responds to a treatment for a disease or condition. Also called molecular marker and signature molecule ^[8]. Diagnostic (screening) biomarker, Prognostic biomarker, Stratification (predictive) biomarker.

Biomarkers play a key role in the diagnosis and management of patients with cancer, and are important for fulfilling the promise of precision medicine in oncology ^[9]. However, although numerous biomarkers have been shown to have clinical validity, many have not undergone rigorous testing to demonstrate clinical utility so that they can be appropriately incorporated into clinical care ^[10]. This review article highlights the characteristics of a good biomarker and the steps required to demonstrate clinical utility, and gives examples of both successful established biomarkers and promising new tissue-based and circulating biomarkers on the horizon ^[11]. Circulating tumor cell, circulating tumor DNA, clinical utility ^[12].

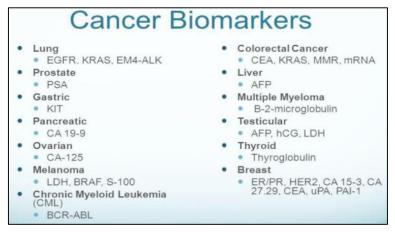


Fig 1: Cancer Biomarkers

A Biomarker is the organic particle found in blood, other body liquids, or tissues that is an indication of a typical or anomalous process, or of a condition or disease. A biomarker might be utilized to perceive how well the body reacts to a treatment for a malady or condition. Likewise called molecular marker and mark particle. Cancer biomarkers are arranged by their diverse capacities ^[13]. Biomarkers that Trigger Cells to Grow and Multiply Abnormally, Biomarkers That Support a Treatment's Cellular or Molecular Action, Biomarkers That Disrupt a Treatment's Cellular or Molecular Action, Detecting and Measuring Biomarkers to Develop a Personalized Anticancer Treatment Plan. Genomic Transcriptomic biomarker, Metabolomics biomarker, biomarker, Drug activity markers, Imaging biomarker, Surgical oncology is the branch of surgery applied to oncology; it focuses on the surgical management of tumors, especially cancerous tumors. Surgery is used to diagnose, stage and treat cancer, and certain cancer-related symptoms ^[14]. Surgeons have performed thousands of procedures and will discuss appropriate surgical options that meet your individual needs. Childhood cancers are different from adult cancers ^[15]. The Advanced Surgical Recovery Program (ASURE) is designed to help patients recover from surgery more auickly and with fewer complications. Lymphadenectomy. ASURE is intended to improve surgical outcomes and enhance the patient experience before, during and after surgery, while also reducing patients' overall hospital stay. Pancreaticoduodenectomy, Thyroidectomy, Appendix surgery. Childhood growth (otherwise called paediatric malignancy) is disease in a kid. Paediatric oncology is the branch of medication worried about the determination and treatment of disease in kids. A pediatric oncologist spends significant time in research and treatment for growths that create in babies, little children, youngsters, youths and adolescents. This is one reason why there is a requirement for pediatric oncologists who are prepared in treating the two youngsters and growth. Numerous pediatric oncologists additionally represent considerable authority in hematology, which is the investigation and treatment of sicknesses identified with the blood. These specialists are in some cases alluded to as pediatric oncologists/hematologists, Neuroblastoma, Wilms tumour, Pediatric Neuro oncologist, Hepatoblastoma and hepatocellular carcinoma^[5]. A haematologist-oncologist is a physician who specializes in the diagnosis, treatment and/or prevention of blood diseases and cancers such as iron-deficiency anemia, hemophilia, sickle-cell disease, leukemia and lymphoma.^[16] This physician is trained in haematology the study of blood and oncology the study of cancer ^[17]. Hematologist-oncologists do not usually treat operable cancers such as prostate cancer, but specialize in treating blood cancers, such as Hodgkins and non-Hodgkins lymphomas, leukemias and multiple myelomas., Haemophilia, Bone marrow disease, Anticoagulation therapy, Blood transfusion. Breast cancer begins in the cells of the breast ^[18]. A harmful tumor is a gathering of growth cells that can develop into and annihilate close-by tissue. It can likewise metastasize to different parts of the body. Cells in the breast some of the time change and never again develop or act ordinarily. These progressions may prompt non-malignant bosom conditions, for example, atypical hyperplasia and sores ^[19]. They can likewise prompt non-carcinogenic tumors, for example, intraductal papillomas. Breast cancer screening, Breast reconstruction, Lobular carcinoma, Abortion breast cancer hypothesis ^[20].

3. Results

Cancer pharmacology ncorporate investigations of the fundamental mechanism of signal transduction related with cell multiplication and apoptosis, the mechanism of activity of anti-neoplastic specialists, the outline and revelation of new medications, essential components of DNA repair and DNA harm resilience and the advancement of novel techniques for quality treatment. Human cancer cell lines, Genetic manipulation of cancer, malignant transformation, Cancer cell proliferation (Figure 3).

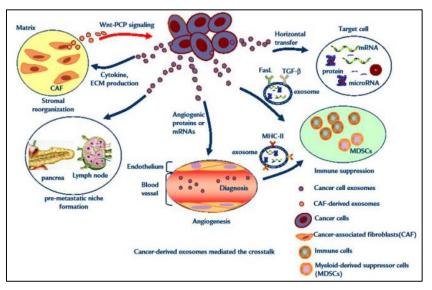


Fig 3: Cancer derived exosomes mediated the cross link

Cancer is a disease caused by genetic changes leading to uncontrolled cell growth and tumour formation. The basic cause of sporadic (non-familial) cancers is DNA damage and genomic instability. A minority of cancers are due to inherited genetic mutations. Most cancers are related to environmental, lifestyle, or behavioural exposures. Cancer is generally not contagious in humans, though it can be caused onco viruses and cancer bacteria. The term by "environmental", as used by cancer researchers, refers to everything outside the body that interacts with humans. The environment is not limited to the biophysical environment (e.g. exposure to factors such as air pollution or sunlight), but also includes lifestyle and behavioural factors. Over one third of cancer deaths worldwide (and about 75-80% in the United States) is potentially avoidable by reducing exposure to known factors. Common environmental factors that contribute to cancer death include exposure to different chemical and physical agents (tobacco use accounts for 25-30% of cancer deaths), environmental pollutants, diet and obesity (30-35%), infections (15-20%), and radiation (both ionizing and non-ionizing, up to 10%). These factors act, at least partly, by altering the function of genes within cells. Typically many such genetic changes are required before cancer develops. Aging has been repeatedly and consistently regarded as an important aspect to consider when evaluating the risk factors for the development of particular cancers. Many molecular and cellular changes involved in the development of cancer accumulate during the aging process and eventually manifest as cancer.

3.1. Heredity

Although there are over 50 identifiable hereditary forms of cancer, less than 0.3% of the population are carriers of a cancer-related genetic mutation and these make up less than 3-10% of all cancer cases. The vast majority of cancers are non-hereditary ("sporadic cancers"). Hereditary cancers are primarily caused by an inherited genetic defect. A cancer syndrome or family cancer syndrome is a genetic disorder in

which inherited genetic mutations in one or more genes predisposes the affected individuals to the development of cancers and May also cause the early onset of these cancers. Although cancer syndromes exhibit an increased risk of cancer, the risk varies. For some of these diseases, cancer is not the primary feature and is a rare consequence.

Many of these syndromes are caused by mutations in tumour suppressor genes that regulate cell growth. Other common mutations alter the function of DNA repair genes, oncogenes and genes involved in the production of blood vessels. Certain inherited mutations in the genes BRCA1 and BRCA2 with a more than 75% risk of breast cancer and ovarian cancer. Some of the inherited genetic disorders that can cause colorectal cancer include familial adenomatous polyposis and hereditary non-polyposis colon cancer; however, these represent less than 5% of colon cancer cases. In many cases, genetic testing can be used to identify mutated genes or chromosomes that are passed through generations. Multiple colon polyps within the colon of an individual with familial adenomatous polyposis.

3.2. Cancer Syndromes

Ataxia telangiectasia, Bloom syndrome, BRCA1 & BRCA2,Fanconi anemia, Familial adenomatous polyposis, Hereditary breast and ovarian cancer, Hereditary non-polyposis colon cancer, Li-Fraumeni syndrome, Nevoid basal cell carcinoma syndrome, Von Hippel-Lindau disease, Werner syndrome, Xeroderma pigmentosum.

3.3. Physical and Chemical Agents

Particular substances, known as carcinogens, have been linked to specific types of cancer. Common examples of nonradioactive carcinogens are inhaled asbestos, certain dioxins, and tobacco smoke. Although the public generally associates carcinogenicity with synthetic chemicals, it is equally likely to arise in both natural and synthetic substances. It is estimated that approximately 20,000 cancer deaths and 40,000 new cases of cancer each year in the U.S. are attributable to occupation. Every year, at least 200,000 people die worldwide from cancer related to their workplace. Millions of workers run the risk of developing cancers such as lung cancer and mesothelioma from inhaling asbestos fibers and tobacco smoke, or leukemia from exposure to benzene at their workplaces. Cancer related to one's occupation is believed to represent between 2-20% of all cases. Most cancer deaths caused by occupational risk factors occur in the developed world. Job stress does not appear to be a significant factor at least in lung, colorectal, breast and prostate cancers.

3.4 Inflammation

There is evidence that inflammation itself plays an important role in the development and progression of cancer. Chronic inflammation can lead to DNA damage over time and the accumulation of random genetic alterations in cancer cells. Inflammation can contribute to proliferation, survival, angiogensis and migration of cancer cells by influencing tumor microenvironment. Individuals with inflammatory bowel disease are at increased risk of developing colorectal cancer

3.5 Viruses

HPV is the most common virus that infects the reproductive tract. Infection can lead to the development of cervical cancer in women. Viral infection is a major risk factor for cervical and liver cancer. A virus that can cause cancer is called an oncovirus. These include human papillomavirus (cervical carcinoma), Epstein-Barr virus (B-cell lymphoproliferative disease and nasopharyngeal carcinoma), Kaposi's sarcoma (Kaposi's sarcomaand primary effusion herpesvirus lymphomas), hepatitis B and hepatitis C viruses (hepatocellular carcinoma), and Human T-cell leukemia virus-1 (T-cell leukemias). In Western developed countries, human papillomavirus (HPV), hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most common oncoviruses. In the United States, HPV causes most cervical cancers, as well as some cancers of the vagina, vulva, penis, anus, rectum, throat, tongue and tonsils. Among high-risk HPV viruses, the HPV E6 and E7 oncoproteins inactivate tumor suppressor genes when infecting cells. In addition, the oncoproteins independently induce genomic instability in normal human cells, leading to an increased risk of cancer development. Individuals with chronic hepatitis B virus infection are more than 200 times more likely to develop liver cancer than uninfected individuals. Liver cirrhosis, whether from chronic viral hepatitis infection or alcohol abuse, is independently associated with the development of liver cancer, but the combination of cirrhosis and viral hepatitis presents the highest risk of liver cancer development

4. References

- 1. Doll R, Peto R. The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. Journal of the National Cancer Institute. 1981; 66:1191-1308.
- 2. Bernstein C, Prasad AR, Nfonsam V, Bernstein H. DNA Damage, DNA Repair and Cancer. In Tech, 2013.
- Stewart BW, Wild CP, eds. "Cancer Etiology". World Cancer Report 2014. World Health Organization, 2014, 16-54.
- 4. Whiteman David C, Wilson Louise F. The fractions of cancer attributable to modifiable factors: A global

review. Cancer Epidemiology. 2016; 44:203-221.

- 5. Kravchenko J, Akushevich I, Manton KG. Cancer mortality and morbidity patterns from the US population: an interdisciplinary approach. Berlin: Springer, 2009.
- Irigaray P, Newby JA, Clapp R, Hardell L, Howard V, *et al.* Lifestyle-related factors and environmental agents causing cancer: an overview. Biomedicine & Pharmacotherapy = Biomedecine & Pharmacotherapie, 2007.
- 7. Anand P, Kunnumakkara AB, Kunnumakara AB, Sundaram C, Harikumar KB, *et al.* Cancer is a preventable disease that requires major lifestyle changes. Pharmaceutical Research. 2008; 25:2097-2116.
- 8. Heikkilä K, Nyberg ST, Theorell T, Fransson EI, Alfredsson L, *et al.* Work stress and risk of cancer: metaanalysis of 5700 incident cancer events in 116,000 European men and women. BMJ. 2013; 7:346: f165.
- 9. Sasco AJ, Secretan MB, Straif K. Tobacco smoking and cancer: a brief review of recent epidemiological evidence. Lung Cancer. 2004; 451(2):S3-9.
- Cooke A, Fergeson J, Bulkhi A, Casale TB. The Electronic Cigarette: The Good, the Bad, and the Ugly. The Journal of Allergy and Clinical Immunology. In Practice. 2015; 3:498-505.
- 11. Ferguson LR, Chen H, Collins AR, Connell M, Damia G, *et al.* Genomic instability in human cancer: Molecular insights and opportunities for therapeutic attack and prevention through diet and nutrition. Seminars in Cancer Biology. 2015; 35:S5-S24.
- Maltoni CF, Holland JF. Chapter 16: Physical Carcinogens. In Bast RC, Kufe DW, Pollock RE, *et al.* Holland-Frei Cancer Medicine (5th ed.). Hamilton, Ontario: B.C. Decker, 2000.
- Robbins basic pathology. Kumar, Vinay, Robbins, Stanley L. (Stanley Leonard), 1915-2003. (8th ed.), 1944.
- Roukos DH. Genome-wide association studies: how predictable is a person's cancer risk? Expert Review of Anticancer Therapy. 2009; 9(4):389-92. Doi:10.1586/era.09.12. PMID 19374592.
- 15. Cancer and the Environment: What you need to Know, What You Can Do. NIH Publication No. 03-2039: National Institutes of Health. Cancer develops over several years and has many causes. Several factors both inside and outside the body contribute to the development of cancer. In this context, scientists refer to everything outside the body that interacts with humans as 'environmental, 2003.
- 16. World Cancer Report. World Health Organization, 2014, 1:1.
- Cancer Fact sheet N°297. World Health Organization, 2014. Retrieved 10 June 2014.
- Hodgson S. Mechanisms of inherited cancer susceptibility. Journal of Zhejiang University. Science. 2008; 9:1-4.
- 19. World Cancer Report. World Health Organization, 2014, 5:5. ISBN 9283204298.
- Ames BN, Gold LS. Paracelsus to parascience: the environmental cancer distraction. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis. 2000; 447:3-13.