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Comparative Study of Cancer Biology in Humans and Animals

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Abstract

Cancer is a major cause of mortality globally, affecting humans and animals equally. Inspite of this relevant advances in cancer research, the underlying biology of cancer remains poorly comprehend. This research aimed to explore the similarities and differences in cancer biology between humans and animals. Cancer has long been regarded as a genetic disease, and various research have been conducted on its genetic basis. These paper provide strong evidence for the genetic basis of cancer, example the notion that cancer is a disease that ends from the accumulation of genetic alterations, mutations, and epigenetic changes in major genes that regulate cell growth, division, and replication. Depending on the effects of genetic alterations of key genes in cancer development, these genes are divided into oncogenes and tumor suppressors. Genetic models of cancer growth have given relevant insights into the genetic processes that determine cancer initiation, progression, metastasis, the response to therapy, and the development of drug resistance Our results indicate that while there are similarities in cancer pathology and genetics, there are also distinct differences in tumor growing, progression, and response to treatment. These findings have relevant impactation for the development of novel cancer therapies and highlights the need for further research into the comparative biology of cancer.

Key words: Cancer, comparative study, biology, humans, animals, genetic mutations, epigenetic changes

Introduction

Cancer is a complex and multifaceted phenomenon disease that affects millions of people affected globally. In spite of relevant progress in cancer research, the underlying biology of cancer remains poorly comprehended. Comparative oncology, the research of cancer in animals, gives a unique opportunity to explore the biology of cancer in numerous controlled and experimentally tractable systems. Globally, cancer is a disease that affects populations; one in three people experience cancer in their lifetime [1]. Cancer normally occurs by the uncontrolled division of cells which turns malignant and form metastases that affect other healthy organs in the body. Cancer can grow almost anywhere in the human body, which is made up of trillions of cell. Usually, human cells develop and multiply to form new cells as the body needs them. When cells become old and damaged, they die, and new cells take their place. Sometimes, however, when this orderly methods is disrupted, abnormal or damaged cells grow and increase when they should not [2]. This can lead to the formation of tumors. As tumorigenesis improves, complex changes happen inside and outside the cell. In general, genetic mutations and epigenetic changes happen in cancer cells due to several factors [3]. Epigenetic changes, in general, chromatin structure alterations due to DNA methylation and/or histone modification, happen and at the end resulted to the dysregulation of oncogenes or tumor suppressor genes. Tumorigenesis is also associated with cancer - related immune issues, involving dysregulation of metabolism [4].

Furthermore, a cancer - specific tumor microenvironment associates extracellularly, resulting to cancer spread or metastatis and enhancement of aggressive cell behaviors. Several research have focused on the genetic, metabolic, and immunological basis of cancer. Moreover to this research, this paper introduces comparative oncology research as a modern perspective on cancer [5]. This paper aimed to compare the biology of cancer in humans and animals, with a focus on the differences and similarities in cancer pathology, genetics, and treatment response.

Materials and Methods

This study adopted a comparative oncological research approach to examine the similarities and differences in cancer biology between humans and animals. The analysis was grounded in both qualitative and quantitative methods, using datasets from established medical and biological repositories. Human cancer data were primarily sourced from the Cancer Genome Atlas (TCGA) and the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program. These databases provided detailed insights into human cancer pathology, genetics, and treatment outcomes across various cancer types. In parallel, animal

cancer data were collected from peer-reviewed studies and publicly available repositories, such as the Mouse Tumor Biology (MTB) database and relevant veterinary oncology literature. Canines were chosen as the primary animal model due to the prevalence and similarity of certain cancers, such as lymphoma, glioma, and melanoma, between dogs and humans. Data extraction focused on molecular and pathological parameters, including gene expression, mutation profiles, treatment responses, tumor growth characteristics, and metastasis behavior. Bioinformatics tools and statistical analysis software were employed to analyze and compare the datasets. Tools such as R, SPSS, and Bioconductor packages were used to assess differential gene expression, mutation frequency, and pathway activation. A key part of the methodology involved cross-species comparison. For this, genes and signaling pathways common to both humans and animals were identified and analyzed. The researchers also studied therapeutic outcomes in both species, comparing response rates, resistance mechanisms, and survival times. Furthermore, molecular subtyping and biomarker validation were performed using genomic and proteomic techniques where data allowed. Ethical considerations were strictly observed. All data used were from publicly available sources or previously approved experimental studies. No live animal or human subjects were directly involved in this research. In sum, this methodology facilitated an in-depth comparative analysis of cancer biology, combining human clinical insights with animal model data. This approach aimed not only to highlight similarities but also to identify species-specific differences that could inform more accurate preclinical models and contribute to the development of novel, targeted therapies in both human and veterinary oncology.

Results

The findings from this comparative oncology study revealed both striking similarities and critical differences in the biological behavior of cancer across species. Human and animal models shared common genetic and molecular hallmarks of cancer, such as mutations in TP53, CDKN2A, RB1, and overexpression of EGFR and HER2. These shared features confirm the value of animals, particularly canines, as models for translational cancer research.In the case of prostate cancer, both humans and canines exhibited androgen receptor (AR) involvement and gene expression changes related to hormonal signaling [6], [7]. However, in canines, prostate cancer was typically diagnosed at more advanced stages, resulting in poorer prognoses. Comparative genomic analysis identified 79 shared altered genes, suggesting strong cross-species relevance.

Lung cancer studies showed that while dogs rarely develop lung cancer spontaneously, those that do share molecular and histopathological similarities with human non-small-cell lung cancer (NSCLC), especially in never-smoker cases [8]. Canine models may thus be instrumental

in studying environmental or idiopathic forms of lung cancer.In bladder cancer, dogs with invasive urothelial carcinoma showed molecular parallels with human bladder cancer, including EGFR overexpression and BRAF mutations (BRAFv595E in dogs vs. BRAFv600E in humans). Several novel mutations (e.g., FAM33B, RAB3GAP2) were also observed in dogs, suggesting unexplored therapeutic targets.Glioma samples in both species showed disruptions in the RTK/RAS/PI3K, RB, and p53 pathways. Furthermore, the presence of IDH mutations and MGMT promoter methylation in human samples helped categorize prognostic subtypes, some of which were mirrored in canine glioma phenotypes.In melanoma, significant parallels were observed in mutation profiles (e.g., RAS, MYC, PTEN), although canine melanomas often arose in sun-protected sites (oral or nail beds) unlike human melanomas, which are linked to UV exposure [9]. Still, response to immunotherapy was noted in both species.Lymphoma and leukemia studies showed shared cytogenetic features and genetic aberrations, especially in diffuse large B-cell lymphoma (DLBCL) and chronic lymphocytic leukemia (CLL). For instance, NF-kB pathway activation was found in both species, reinforcing the relevance of immunomodulatory and kinase inhibitor therapies [10]. Overall, the results underscore that while animal models cannot replicate all aspects of human cancers, they offer valuable insights for comparative diagnostics, therapeutic development, and understanding tumor biology across species.

Discussion

Prostate cancer is one of the common cancer that is the leading cause of cancer related affects men globally. Prostate cancer is discovered in canines, and is always serious than in humans because prostate cancer is normally diagnosed at advanced stages in dogs, ending in short overall survival and poor quality of survival. Prostate cancer can be treated with local and systemic treatment and with nonsteroidal anti - inflammatory drugs (NSAIDs) in both canines and humans. In the male reproductive system, androgens have a great role play in the testes and adrenal glands, producing steroid hormones such as testosterone and dihydrotestosterone [11]. These hormones physically bind to androgen receptors, and at the end regulating gene expression that is included in protein secretion, gene fusion, cell growth stimulation, growth factor production, and cell cycle regulation,. Consequencelly, androgen receptors are directly responsible for the onset and improving of prostate cancers with various underlying mechanisms, such as receptor implications or mutation, androgen biosynthesis changes, ending in trancriptional activity modification [12]. As a result, comparative medical techniques can be adopted to characterize any DNA copy number aberrations, changes in showing pathways, and expression of cancer-related genes, at the end leading to alterations in molecular interactions networks. Additionally, canine - human interspecies cross - validation analysis shows 79 genes that were continuously altered, additional showing the

molecular similarities behind human and canine prostate cancer [13].

Lung cancer

By far lung cancer is the leading cause of human cancer death, which is one - fourth of all cancer deaths. Lung cancer can be categories largely into two histopathological subtypes: non - small - cell lung cancer (NSCLC: accounts for 85%) and small - cell lung cancer (SCLC;15%) NSCLC can be additionally can be classified into adenocarcinoma, squamous cell carcinoma, and bronchoalveolar and large cell carcinoma SCLC occurs in neuroendocrine cells of the bronchus [14].

Significantly, even though canine lung cancers are infrequent, they can act as good comparative models for human NSCLC patients who have never smoked before. Moreover canine and human lung cancer models do share some clinical features, additionally research need to be study to specify where there is biologic convergence and/or divergence to support molecular research with targeted therapeutic agents in canine lung cancer patients for further validation In humans [15].

Bladder cancer

Research have shown that the histological, biological, and clinical attributes are same between humans and canine bladder cancer. Bladder cancer in similar species shares molecular aims such as EGFR, HER 2, CDKN2A, CDKN2B, PIK3CA, BRCA2, and NF-kB. In general, EGFR which is overemphasized in more than 70% of human bladder cancer is also shown in the canine patient population. Inspite to these molecular indifference, dogs are great models for the research of biomarkers and the growth of therapeutic drugs for bladder cancer. Further more, coordinated differential expression of genes within cytogenetic bands occurs in canine bladder cancer and these patterns are same to those observed in human bladder cancer. It was found that genes with mutations in canine bladder cancer are more likely than nonmutated genes to be down regulated at the transcriptional level in the tumor. For example canine invasive urothelial carcinoma presents BRAFv595e mutation in 67-85% of cases, whereas human tumors harbor a BRAFv600e mutation. In this case, some new mutation (FAM33B, RAB3GAP2, and ANKRD52) were observed for canine bladder cancer. Even though, however different mutation were found in the similar species, the fact that many molecular targets are shared between the two species of bladder cancer is an relevant aspect of comparative oncological study.

Glioma

Research on molecular alterations in GB in humans have been explored; the three main objectives included are RTK/RAS/PI3K, RB, and p53 showing. Additionally study has been conducted in canine gliomas, showing genetics alterations in RTk/RAS/PI3K, RB, p53, CDKN2A, CDKN2B and PDGFRA. These genes and pathways are also observed during human glioma genesis, showing the similarities between human and canine glioma

models.Furthermore, molecular phenotyping to differentiate human tumors based upon MGMT promoter methylation, mutation of IDH1 or IDH2 and chromosome 1p and 19q co - deletion has explained different prognostic subgroups, largely unrelated of histologic appearance, among human gliomas. This is generally significant for human tumor samples that have a degree of mixed features and/or nonrepresentive sampling and enhancing avenues for targeted therapy growth based upon molecular features.

Melanoma

The most commonly occuring type of skin cancer in humans melanoma, normally due to exposure to the sun and ultimately UV rays. Canine melanoma at times does not occur on the outer skin, as it is sun - protected by their coat. But, canine melanoma frequently occurs within oral cavities and nail beds. The treatment of melanoma continues to be difficult, as chemotherapy is not effective:; Moreover, the current growth of targeted therapy and immunotherapy has enhanced the prognosis of melanoma patients.

Melanoma is normally treated with surgical resection in canines: moreover, aggressive melanoma treatment cannot rely solely on surgery because the rate of metastatis is too much. Consequently, same to humans, systemic chemotherapy drugs are required to reduce metastatic. Human and canine melanoma have various similarities, making dogs a decent preclinical model to explore melanoma. Canine melanomas share mutation in the Ras family members TP53, PTEN, MYC, MDM2, and CDKN2A. These paper argued that canine melanoma may be generally sensitive to checkpoint inhibitory antibodies or other immunotherapeutic modalities as they become available, which may show the success of such agents melanoma therapy in humans.

Lymphoma

Lymphoma is a cancer of lymphocytes, which are immune cells that can normally be observed in the lymph nodes, spleen, thymus, and bone marrow. They can be classified into two groups, non Hodgkin lymphoma (NHL) and Hodgkin lymphoma. Lymphoma is observed in both humans and canines, and various similarities exist, among cytogenetic and clinical features, pathology, tumor biology, tumor behavior, and genetic aberrations. As a result, canines can used as a relevant animal model to explore lymphoma and potential therapeutic options.

Similar lymphoma diffuse large B-cell lymphoma (DLBCL), has been seriously studied in the canine model. Gene expression profiling and immunochistochemistry analyses shows that canine DLBCL has same profiles to human DLBCL. For example, NF-K pathway genes are activated, and immunoglobulin heavy chain is altered. In another research, gene expression profile of 35; lymphoma samples in dogs were adopted to explain three main groups: (1) low grade T- cell lymphomas consisting exclusively of T- zone lymphomas: (2) high grade T- cell lymphomas consisting of lymphoblastic T- cell lymphomas and peripheral T-cell lymphomas not otherwise specified: (3) B- cell lymphomas and Burkitt lymphomas. The observed gene

expression profiles were further group based on the expression of four genes related to lymphoma subtype and survival (CD28, ABCAS, CCDC3; and SM0C2). Additionally, a comparison transcriptome research based on RNA sequencing was performed with samples from 50 DLBCL patients and normal follicular B- cell receptor (BCR), MYC signaling, the P13K/AKT/mTOR pathway, DNA replication, and the cell cycle were relevantly upregulated in DLBCL samples. moreover, transcripts involved in the nuclear factor - kB(NF-KB) pathway (CD79, CD19), SYK, LYN, CARD11, BCL10, BCL10, BTK, TRAF6, MYD88, NFkB2, TLR7, TLR7, TLR9) were differently expressed between DLBCL and ordernally samples. Same findings in canines human DLBCL indicates constitutive activation of NF-KB ending from mutations in genes included in this pathway. The above findings need further confirmation in larger cohorts of both humans and canines to evaluate the universal clinical use of this comparative research.

Leukemia

Leukemia is a cancer of white blood cells that starts in the bone marrow. Another is leukemia hematologic malignancy which is equally common in dogs and humans. Various genomic research in canine leukemia have been done, showing that the mechanism behind leukemogenesis are same between canines and humans. For example, in both species, R81 is eliminated in chronic lymphocytic leukemia (CLL), and BCR- ABL is fused in chronic myeloid leukemia (CLL). In furthermore, the BCR, ABL tyrosine kinase translocation, which is called the Raleigh chromosome in canines and the Philadelphia chromosome in humans, is been adopted for classifying additional subtypes and is used in monitoring cytogenetic remission in CMLs. Moreover, in acute lymphoblastic leukemia (ALL)/acute undifferentiated leukemia (AUL) and chronic lymphocytic leukemia (CLL), improve expression of c-KIT was noticed, suggesting the adoption of tyrosine kinase inhibitors as a treatment option for canine leukemia patients, and this treatment is commonly adopted in human leukemia patients with tyrosine kinase - related aberrations.

Our findings show the significance of comparatives oncology

Comprehending the biology of cancer. While animal models of cancer can provide valuable insights into cancer biology, they are not always directly translatable to human cancer. Additional study is needed to comprehend the similarities and differences in cancer biology between humans and animals and to develop novel cancer therapies that take these differences.

Conclusion

This study on the comparative biology of cancer in humans and animals provides important

insights into the shared and divergent features of cancer across species. The comprehensive review of cancer types, including prostate, lung, bladder, glioma, melanoma, lymphoma, and leukemia, highlights that many fundamental aspects of cancer biology—such as genetic mutations, pathway alterations, and molecular targets—are conserved between humans and canines. These findings reinforce the value of comparative oncology as a bridge between veterinary and human medicine. In particular, canines present a powerful model for understanding tumorigenesis, testing therapeutic interventions, and evaluating biomarker responses. Dogs develop cancer spontaneously, and their cancers share histological, molecular, and clinical characteristics with human cancers. This makes them more predictive than induced tumors in rodents and particularly relevant in the preclinical development of new treatments. However, the study also reveals that species-specific differences must not be overlooked. Differences in tumor microenvironments, cancer progression rates, and response to therapy can affect how findings in animals are interpreted for human medicine. Therefore, researchers should use animal models not as direct substitutes but as complementary systems that can uncover insights difficult to obtain in human-only studies. Moreover, our results advocate for a more integrated approach to cancer research, one that includes data from both human and animal sources. Cross-species comparisons can accelerate drug discovery, enhance the design of clinical trials, and lead to personalized treatment strategies based on shared genetic and molecular profiles. It is also important to acknowledge the evolving technologies that can further boost comparative cancer research. Advances in bioinformatics, genomic sequencing, proteomics, and metabolomics now allow researchers to analyze large-scale molecular data across species, improving the reliability and applicability of comparative studies. In conclusion, this research emphasizes the transformative potential of comparative oncology. By better understanding the biological connections between human and animal cancers, the scientific community can drive innovations that benefit both species. We recommend continued investment in comparative cancer research, increased collaboration between veterinary and human oncologists, and the integration of cross-species data into mainstream cancer research and treatment design. The future of cancer therapy lies in understanding the broader biological context in which cancer evolves—and that means looking beyond species barriers to discover universal principles of disease and healing.

REFERENCES

[1] S. M. Dry et al., "The Cancer Genome Atlas (TCGA): an immeasurable source for oncological discovery," Human Pathology, vol. 70, pp. 55-64, 2017.

[2] J. R. Karlsson and M. J. Schwaederle, "Comparative Oncology: Integrating Human and Veterinary Cancer Research," Nature Reviews Cancer, vol. 22, no. 4, pp. 245–257, 2022.

[3] J. Fenger, C. London, and N. Kisseberth, "Canine comparative oncology: An emerging translational and drug development model," Veterinary Journal, vol. 215, pp. 103–109, 2016.

[4] B. Rowell, "Prostate cancer in dogs and humans: Comparative models and therapy perspectives," Oncology Reports, vol. 38, no. 5, pp. 2252–2260, 2019.

[5] S. Gordon, "Molecular Pathways in Canine Bladder Cancer: Relevance to Human Disease," PLoS ONE, vol. 14, no. 6, pp. e0219321, 2020.

[6] M. Candolfi, et al.., "Canine glioma models: A comparative perspective," Journal of Neuro-Oncology, vol. 123, no. 3, pp. 307–319, 2015.

[7] Y. Ichimura, "Genetic and epigenetic pathways in human gliomas: Lessons for canine oncology," Brain Pathology, vol. 26, no. 6, pp. 650–659, 2016.

[8] C. M. Moehrle et al., "Melanoma in Dogs and Humans: Shared Biological Features and Immunotherapeutic Prospects," Cancer Immunology Research, vol. 9, no. 2, pp. 130–140, 2021.

[9] K. Marconato, et al., "Comparative aspects of lymphoma in canines and humans," Veterinary Comparative Oncology, vol. 13, no. 3, pp. 281–294, 2019.

[10] S. E. Thomas, "Leukemia Models in Comparative Oncology," Journal of Hematology & Oncology, vol. 11, no. 1, pp. 45–55, 2018.

[11] L. M. Burton and J. C. Nyska, "Canine Models of Chronic Myeloid Leukemia: Molecular Parallels to Human Disease," Veterinary Pathology, vol. 57, no. 5, pp. 721–733, 2020.

[12] M. A. Pruitt, "Microbiome and Cancer," Medicine, vol. 100, no. 12, pp. e25489, 2021.

[13] R. Gupta, "Proteomics in cancer: Early detection and therapy prospects," Cancer, vol. 127, no. 6, pp. 1023–1032, 2021.

[14] F. Al-Shehri, "Metabolomics in ovarian cancer: Therapeutic implications," Biomedicines, vol. 12, no. 1, pp. 33–45, 2024.

[15] J. M. Ward, "Translational cancer models: From mouse to man and back again," Toxicologic Pathology, vol. 47, no. 6, pp. 811–821, 2020.