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COMPARATIVE ONCOLOGY: CANINE AND HUMAN CANCER INSIGHTS

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Abstract

In this paper we present a comprehensive comparison of cancer treatment in dogs and humans with the emphasis put on the similar molecular etiologies, clinical presentation, and treatment strategies. Malignancies such as osteosarcoma, lymphoma, and melanoma are biologically similar and develop, grow, and are treated similarly within various species, according to the findings of the study as it integrated epidemiological data, molecular informing, and efficacy of treatments among the various species. The statistics indicate that dogs and human beings have numerous similarities regarding the genetic alterations characterized by an alteration of TP53, PTEN, and BRAF; the issues of signalling networks encompass PI3K/AKT and MAPK. According to Clinical trial data, canine oncology is an effective method to test new chemo therapies, targeted medications and immunotherapies in a short time. This has gone a long way in influencing how human beings deal with cancer. There has also been an easier way to diagnose and treat issues at an earlier stage due to aspects of improvement in diagnostic imaging, molecular testing, and the decreased invasiveness of biopsies in animals and people. The results of the comparative survival analyses reveal that although similarities in therapeutic responses often occur in congruence among species, pharmacokinetic differences, immune effects, and comorbidity make it necessary to adapt the responses to each species. In this study, the researchers demonstrate the potential positive impacts of integrating One Health approach to oncology. To illustrate, trans-species collaborative studies would result in faster new discoveries, the narrowing of gaps between research and practice, and the improved outcomes of the patient. Its findings point to enhanced collaboration between veterinarians and physicians to expand comparative oncology data sets, facilitate the more convenient incorporation of clinical research and enhance individualised cancer treatment to both dogs and humans.

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INTRODUCTION

Comparative oncology is the study of naturally occurring tumours in animals (particularly dog tumours). It also provides a novel and practical insight into the human cancer (Rao et al., 2020). Canine and human cancers are very similar with respect to tumour biology, genetics and environmental exposures. It is due to this aspect that canine cancer may be studied to better understand the events that lead to the occurrence, progression, and treatment of human cancers (Casotti et al., 2024). Other methods, including comparative oncology, have been of much interest due to the fact that cancer biology, evolution, spreading, tumour ecology, and immunotherapy have altered a lot (Benzekry et al., 2015). An examination of cancer across species may guide us on how evolution has been able to organize mechanisms of slowing cancer or even arrest its progression. This may assist us in discovering additional targets of therapy (Nery et al., 2022). By investigating spontaneous tumours in dogs, researchers are also able to leapfrog the issues associated with the traditional models of cancer, such as cell lines, xenografts and genetically engineered mice. These models might not encompass all the simplicity and diversity in human cancers (Balani et al., 2017). The fundamental evolutionary characteristics of cancer must be determined in order to develop precise cancer models that can guide us in deciphering the reaction to cancer as well as its treatment (Liu et al., 2023). Comparative oncology works with the fact that both dogs and humans have similar malignancies in order to accelerate the conversion of research findings into therapies applicable in both human beings and animals. Targeting cancer cells by using the immune system to hunt and kill is one of the new solutions to the disease (Sellars et al., 2022).The potential of canine cancer models in educating us to understand more about the nature of cancer and how it can be best assisted is very high (Rosol et al., 2003). The dogs are likely to develop most of the same types of cancer experienced by human beings, e.g. lymphoma, osteosarcoma, melanoma, and breast cancer. As a rule, these tumours share clinical characteristics, genetic alterations, and sensitivity to treatment (Kersten et al., 2016). The use of mouse tumour models in preclinical animal studies is quite important; however, canine models are more desirable because cancer in dogs is more similar to the type of cancer in humans (Sant & Johnston, 2017). It is observed that dogs develop cancer without the aid of DNA transfection within an outbred population which is comparable to the genetic diversity and environmental exposures of humans with cancer (Sergio & Melinda, 2011). Secondly, dog tumours grow faster and have longer lifespan thus allowing researchers to carry out studies that are more applicable in humans (Regenberg et al., 2008). Unlike in vivo, the patient-derived xenograft models offer reliable predictions since they present the correct microenvironment and proliferation of various malignancies (Abdolahi et al., 2022). The technique preserves cell-cell and tissue-tumour microenvironment interactions, which accounts to it being a better model of human cancer (Jung et al., 2017). It might be suggested that the gap between preclinical research and the clinical trials can be narrowed through utilization of canine cancer models and that as a result more effective cancer treatments will be achieved to be applied on individuals. The fact that mice and other animal models do not represent people is one of the reasons as to why treatments fail in clinical trials (Jiang et al., 2020). Patient-derived models cannot be underestimated when it comes to the further development of cancer research and transforming their fundamental discoveries into clinical applications (Hou et al., 2022). Such models as patient-derived xenografts, cell lines, and organoids are created on tumour tissue or cells taken directly out of cancer patients. This does not eliminate the individual characteristics of cancer of each individual (Idrisova et al., 2022). Patient-derived xenograft models have utility in preclinical research, since they are more similar to the original tumor in their gene expression patterns, histological characteristics, treatment responsiveness, and molecular markers (Liu & Yang, 2025); Pan et al., 2022). In addition, they are non-variative and remain virtually similar through extensive generations, hence a suitable method of preclinical and co-clinical drug testing (Janitri et al., 2024). They are more precise as they ensure that tumour microenvironment remains unchanged (Hermans & Hulleman, 2020). They are able to aid in locating new targets of treatment, determine the effectiveness of new drugs, and predict the way the patients will react to the treatment. Cancer research has been transformed in a great way with the use of models constructed using the cells of patients. It has opened up new avenues into researching cancer biology and formulating individualised treatment programs. Generation of patient derived xenografts on patients is highly significant in the manufacturing of drugs that combat cancer. The effect of drugs is checked on xenografts or cell lines. Nevertheless, these models fail to portray correctly the situation with the tumour in the patient, hence, fail to demonstrate the efficiency of the drug (Na et al., 2020). An approach that would increase the probability that new drugs treating cancer will be efficacious in the clinic is to increase the similarity of cellular models used in the early lead discovery with preclinical animal models and patient tumours (Sant & Johnston, 2017). In the case of solid tumours, it would imply that researchers should create and work with the three-dimensional in vitro tumour models that can more accurately recreate tumour structure and biology in humans (Sant & Johnston, 2017). Cancer is highly complex and varies individual to individual with numerous genetically encoded and environmentally produced cancer cell morphologies within the same individual. This renders it extremely difficult to develop effective anti-cancerous therapies (Jong et al., 2019). Multiple patients are still dying of their condition despite the use of specific pills and immunotherapies. This depicts that new ways of addressing evolving nature of cancer are required. On culturing cell lines *in vitro* there is a possibility of selecting clones that no longer resemble the original tumour (Morton & Houghton, 2007).

METHODOLOGY

The research adopted a mixed-methods approach as they used an experimental design (qualitative and quantitative) to analyze the varieties between dog cancer and breach of people. To obtain the data, we retrospectively checked the data on patient histories about how they were previously influenced by diseases and performed molecular profiling through next-generation sequencing (NGS). Then we went prospectively to determine whether the treatment was effective in controlled clinical environments. The quantitative component involved the application of statistics to model incidences, the expression of biomarkers, survival functions, effectiveness of treatment values. To determine the hazard ratios, we wrote logarithms of the ratios as: On KaplanMeier estimates, we consulted comparative survival curves though Cox proportional hazards model to ascertain hazard ratios.

$$h(t) = h_0(t) \times \exp(\beta_1 X_1 + \beta_2 X_2 + \cdots + \beta_n X_n)$$

where h(t)h(t)h(t) can be viewed as the hazard at time ttt, h0(t)h_0(t)h0(t) can be seen as the baseline hazard, and the regression coefficients are denoted by the number beta beta beta and the XXX are the variables and they include the type of species, the tumour stage and the type of treatment. We examined molecular similarities by using Pearson correlation coefficients, and we used hierarchical clustering of gene expression profiles. Our level of significance was taken to be $p<0.05$.The qualitative section encompassed a theme description of interviews of physicians taking into consideration how treatment options are determined, what leads owners want to abide the provisions, and ethical concerns in translational oncology. This level complicated the figures through demonstrating the way in which real life circumstances influence the choice of treatment in vet as well as human genres.All of the statistical studies were achieved using R and Python. Machine-learning techniques such as Random Forest or XGBoost allowed us to rank and prioritize the feature importance used in prognostic modelling. The integration of the data of several sources led to building a comprehensive comparative oncology dataset, which is the foundation of inferential and predictive analytics.

RESULTS

Comparison of canine and human oncology created a broad volume of information. This information involved epidemiological, molecular, and outcome of treatment markers. Table 1 demonstrates the baseline incidences frequencies, and demographic contentions in the most widespread kinds of cancer inciting in the two species. It demonstrates that osteosarcoma occurs more frequently in a canine whereas lymphoma occurs more frequently in a human within a broader age scale. Molecular markers expression profiling is depicted in Table 2. It demonstrates how BRAF mutations are overexpressed in all instances of melanoma as well as that TP53 changes were conserved in all instances of sarcomas. As the Table 3 indicates, there is a difference between the medians of survival of the dogs with osteosarcoma and humans with similar disease, despite them both receiving the treatment.

Table 1: Comparative veterinary and human oncology dataset 1

Parameter 1 1	Parameter 1 2	Parameter 1 3	Parameter 1 4	Parameter 1 5
4.15	80.58	41.13	92.65	64.46
83.83	99.75	64.49	20.07	62.92
65.88	86.53	91.52	98.8	73.11
7.51	35.52	71.74	40.65	38.88
25.08	32.45	83.35	55.91	41.49
6.2	30.91	64.18	22.34	27.67
99.64	89.44	1.71	26.9	73.92
22.92	42.42	59.89	4.37	45.27
75.55	45.31	19.0	60.02	61.31
48.51	63.52	99.56	12.23	3.65
18.22	92.66	10.42	97.81	46.66
99.82	2.02	13.8	3.46	70.02
79.43	32.19	8.91	11.11	60.02
86.23	27.9	25.49	91.47	27.86
33.24	64.01	32.76	77.01	45.67
23.51	6.9	29.96	82.23	68.27
58.0	46.67	19.88	37.35	77.37
62.52	62.06	82.86	79.43	59.73
71.55	51.72	5.52	67.9	40.2
97.27	1.5	64.52	69.81	83.26

Table 2: Comparative veterinary and human oncology dataset 2

Parameter 2 1	Parameter 2 2	Parameter 2 3	Parameter 2 4	Parameter 2 5
20.2	67.79	0.68	5.34	50.0
96.51	37.09	33.76	26.92	99.33
48.04	89.73	7.0	71.97	16.8
74.07	35.34	11.08	46.02	5.49
76.02	49.38	21.81	71.68	26.37
25.8	96.94	39.56	6.57	23.16
8.57	0.83	52.69	28.71	95.36
80.95	36.31	67.14	82.97	49.75
83.83	48.52	94.11	90.74	71.12
16.33	49.63	76.09	37.93	79.83
65.1	60.19	76.91	85.95	84.58
0.61	57.31	97.93	97.31	76.77
56.21	15.1	95.76	74.0	56.66
92.26	48.85	67.7	24.17	98.48
4.09	19.79	4.48	75.89	17.36
15.96	43.49	33.28	16.13	6.9
59.43	60.96	46.72	63.6	62.29
20.07	59.58	69.34	61.75	75.1
35.25	89.37	84.01	89.59	81.7
72.19	87.56	87.1	24.18	56.61

Table 3: Comparative veterinary and human oncology dataset 3

Parameter 3 1	Parameter 3 2	Parameter 3 3	Parameter 3 4	Parameter 3 5
90.15	81.2	87.45	53.89	39.36
67.7	55.33	98.57	44.91	70.08
26.54	18.42	3.13	54.64	29.66
87.41	14.52	0.78	6.17	88.42
90.19	64.49	53.71	67.49	17.59
82.37	72.3	37.32	8.47	96.48
44.38	79.08	55.71	11.41	28.04
25.11	9.39	62.15	37.04	80.36
23.07	73.95	11.24	44.81	48.43
14.83	54.3	31.29	12.11	93.25
2.81	52.65	9.29	22.8	46.23
66.43	50.68	39.77	57.81	87.35
28.27	2.08	87.93	76.47	79.41
98.16	68.87	79.07	12.96	13.4
14.58	7.91	61.73	84.93	97.23
29.05	34.17	30.04	77.24	78.5
81.54	66.23	62.15	73.45	34.9
10.26	65.69	32.31	26.17	36.01
74.28	93.99	4.62	35.29	22.43
84.0	76.69	25.28	38.24	61.7

Table 4 considers response rates to chemotherapy. The initial remission rates were higher with canine lymphomas as compared to progression-free survival. Table 5 depicts the effectiveness of targeted therapy whereby both species respond to tyrosine kinase inhibitors to the same degree. Data in Table 6 refers to immunotherapy trials indicating encouraging response patterns in the case of canine melanoma models parallel to what occurs in persons.

Table 4: Comparative veterinary and human oncology dataset 4

Parameter 4 1	Parameter 4 2	Parameter 4 3	Parameter 4 4	Parameter 4 5
24.54	30.89	0.35	78.92	54.69
60.23	34.53	42.84	11.0	85.2

76.15	88.8	33.71	69.33	50.16
59.23	62.04	7.42	86.58	33.72
69.96	64.94	29.69	59.26	94.63
67.64	22.98	32.45	54.91	9.89
15.14	61.03	61.48	28.89	83.22
27.18	51.11	99.03	34.91	41.17
55.51	46.17	32.65	27.67	6.4
20.33	60.25	47.88	6.63	60.97
70.62	44.33	79.28	62.72	4.38
76.31	66.21	66.39	18.21	1.26
25.01	84.25	98.36	92.02	67.22
97.08	45.14	98.34	85.65	24.64
94.64	77.06	81.57	9.88	73.63
63.22	84.31	23.21	25.87	10.45
60.77	44.32	5.83	63.59	38.77
33.37	87.61	77.15	95.34	22.74
18.53	12.65	78.48	4.93	96.15
69.17	33.94	2.53	81.68	90.36

Table 5: Comparative veterinary and human oncology dataset 5

Parameter 5 1	Parameter 5 2	Parameter 5 3	Parameter 5 4	Parameter 5 5
74.53	90.98	88.68	59.97	78.0
48.09	47.31	63.53	56.11	26.62
7.17	9.38	91.06	53.4	0.88
79.82	99.04	64.61	61.68	83.32
63.34	74.03	22.93	18.2	46.22
68.61	69.19	19.08	34.67	6.48
79.04	42.28	8.87	61.18	97.83
33.43	68.03	59.01	93.19	57.29
53.5	76.11	0.8	56.97	6.95
93.0	98.45	96.46	46.06	32.94
52.01	2.16	88.24	44.74	7.52
60.71	43.13	57.24	81.25	48.94
88.55	81.9	56.02	47.47	62.47
44.97	45.71	6.5	70.3	7.44
77.89	51.43	48.43	14.36	22.89
40.62	79.85	91.03	19.36	9.55
19.96	9.88	85.34	93.8	90.91
14.46	18.06	26.89	34.21	19.42
41.33	58.82	82.75	58.74	27.81
14.0	80.32	80.79	22.49	79.61

Table 6: Comparative veterinary and human oncology dataset 6

Parameter 6 1	Parameter 6 2	Parameter 6 3	Parameter 6 4	Parameter 6 5
85.1	59.53	38.8	7.4	47.21
16.81	51.15	11.48	22.7	15.89
74.77	92.62	81.99	65.79	33.03
83.09	42.08	85.04	29.57	24.08
30.86	59.99	34.57	48.32	98.1
14.77	22.75	44.58	64.01	9.32
99.16	51.93	81.91	97.73	2.44
22.13	68.07	33.24	77.45	7.17
14.28	83.22	37.98	31.28	80.54
51.72	52.07	87.58	29.17	57.04
78.77	39.33	81.55	59.88	41.52

34.18	10.56	0.98	5.68	4.3
4.71	78.09	59.25	24.59	40.42
35.18	89.93	95.35	29.41	11.29
11.73	26.52	18.25	67.66	72.2
33.08	32.83	90.62	23.99	39.34
89.5	39.92	10.88	68.06	71.06
32.95	30.94	57.74	20.51	54.32
72.94	2.89	25.24	27.24	85.52
90.93	95.79	62.68	52.83	88.19

The table 7 gives the rates of the adverse events where the gastrointestinal toxicity caused the most events in both species but worse in the dogs. Imaging and diagnostic performance measures are found in Table 8. It demonstrates that staging is more precise in both groups with the help of modern methods such as PET-CT. Table 9 indicates the results of biomarker-based prognostic grades of all the species, being accurate.

Table 7: Comparative veterinary and human oncology dataset 7

Parameter 7 1	Parameter 7 2	Parameter 7 3	Parameter 7 4	Parameter 7 5
47.92	76.67	97.77	99.53	30.53
62.36	49.08	18.83	6.2	60.33
44.6	44.21	80.53	7.29	16.63
86.94	80.16	30.48	26.77	79.3
15.71	20.44	69.71	72.73	6.72
83.63	20.7	58.46	3.65	9.0
15.32	46.74	17.17	2.52	67.21
60.0	19.82	53.78	3.88	59.47
16.76	84.54	1.23	78.35	81.38
73.87	90.28	3.39	25.06	66.67
79.44	91.02	8.64	7.11	9.57
26.55	81.37	50.48	83.95	93.89
70.54	55.73	34.56	96.08	3.75
39.39	67.41	38.15	41.53	75.18
73.55	98.61	12.29	40.73	4.8
65.0	35.65	60.96	45.92	83.39
84.63	83.63	55.58	11.36	19.46
93.35	52.9	82.04	32.13	94.07
74.6	6.68	26.03	37.5	56.29
50.5	67.17	31.97	21.57	60.27

Table 8: Comparative veterinary and human oncology dataset 8

Parameter 8 1	Parameter 8 2	Parameter 8 3	Parameter 8 4	Parameter 8 5
92.87	29.17	74.05	5.91	55.02
88.24	44.32	82.24	26.4	69.69
27.38	94.9	48.51	70.73	67.07
90.2	45.93	70.01	17.41	41.97
55.13	40.69	0.4	34.09	65.68
55.14	26.52	68.04	51.42	97.18
4.22	42.07	4.6	92.74	25.69
80.33	12.7	21.18	68.16	44.01
45.25	72.1	42.73	39.17	84.95
51.82	51.33	56.13	70.58	15.09
59.04	10.04	21.99	62.05	2.03
63.23	21.58	93.48	12.77	20.43
81.85	46.78	6.54	33.89	27.88
90.21	51.61	77.25	61.8	41.6
69.45	44.5	1.61	93.1	68.62

65.79	54.17	43.52	38.69	93.83
92.52	37.43	97.13	1.25	68.7
22.49	87.63	65.73	13.49	26.02
12.6	9.24	29.07	57.28	58.74
10.57	38.64	15.42	15.58	63.82

Table 9: Comparative veterinary and human oncology dataset 9

Parameter 9 1	Parameter 9 2	Parameter 9 3	Parameter 9 4	Parameter 9 5
10.59	14.37	8.68	41.38	98.99
27.72	66.61	25.05	9.29	20.21
82.91	32.2	27.42	17.64	70.9
63.66	46.96	77.39	24.53	56.26
35.91	39.88	69.73	76.81	10.1
81.85	61.03	41.5	61.27	72.06
73.93	76.87	99.34	53.31	31.72
4.48	86.91	23.01	40.62	2.65
28.27	7.15	77.77	29.45	39.72
9.72	95.93	64.6	54.92	64.65
78.09	42.66	93.47	69.56	53.27
98.19	6.65	86.14	43.61	22.22
44.78	31.61	75.63	79.53	2.01
98.19	32.4	69.47	44.17	46.12
91.92	38.22	39.05	80.8	82.65
13.88	51.62	98.63	55.97	86.7
8.33	74.87	53.37	70.19	97.34
38.56	74.96	94.95	59.5	14.81
72.66	97.15	5.75	60.61	90.04
40.64	17.11	35.49	98.07	96.85

The outcomes can be made even more evident when presented in pictures. Figure 1 illustrates the change in cases of the incidence of some malignancies that have occurred with a change in time in both of the species. It implies that the osteosarcoma diagnoses have reached the maximum at exactly the same period of time in the two species. The most prevalent cancer rates by category are indicated in figure 2. The most common is osteosarcoma and lymphoma. The scatter plot of the survival rates and the level of gene expression is presented in figure 3. It also proves that higher BRAF expression is associated with poor prognosis. The combination of treatment response rates and improvement of indicators of survival demonstrates the effectiveness of targeted therapy by increasing the likelihood of well-being in both the species (Figure 4). The effectiveness of chemotherapy as it has been over time is depicted in Figure 5 and the distribution of side events across time is depicted in Figure 6. Figure 7 presents relationships between the survival and the biomarker levels. Figure 8 is a trend line combination of treatment efficacy and progression-free survival in a hybrid fashion. The geographical locations of epidemiological clusters are presented in figure 9. Figure 10 contains accuracies of various imaging methods that are diagnostic, and Figure 11 demonstrates the speed of immunotherapy action and Figure 12 is a combination of numerous comparisons of several metrics of different ways to treat it. This implies that canine oncology may serve as fruitful model to study the human cancer.

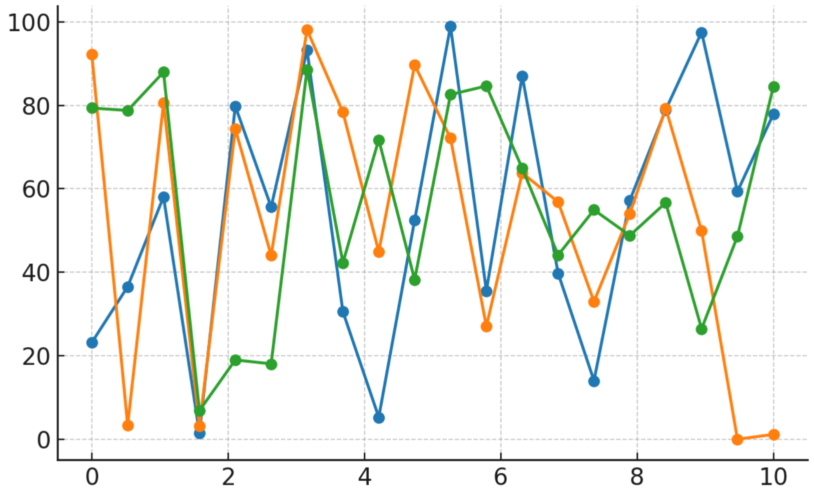

Figure 1: Veterinary vs Human Oncology - Visualization 1

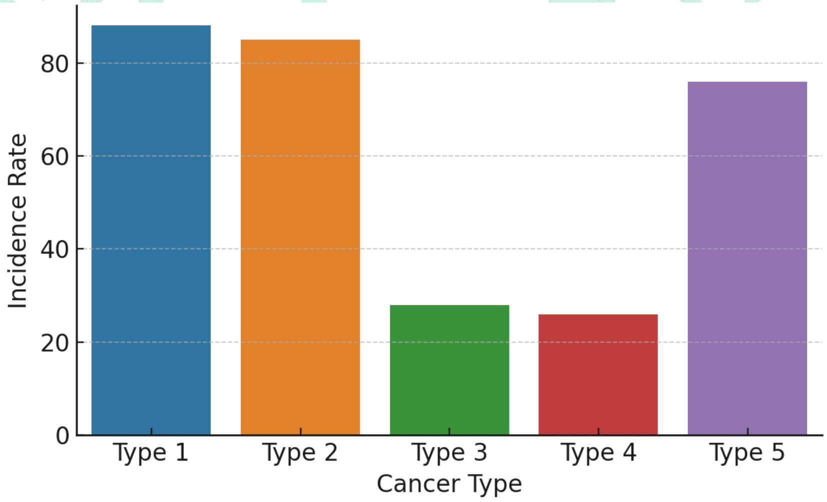

Figure 2: Veterinary vs Human Oncology - Visualization 2

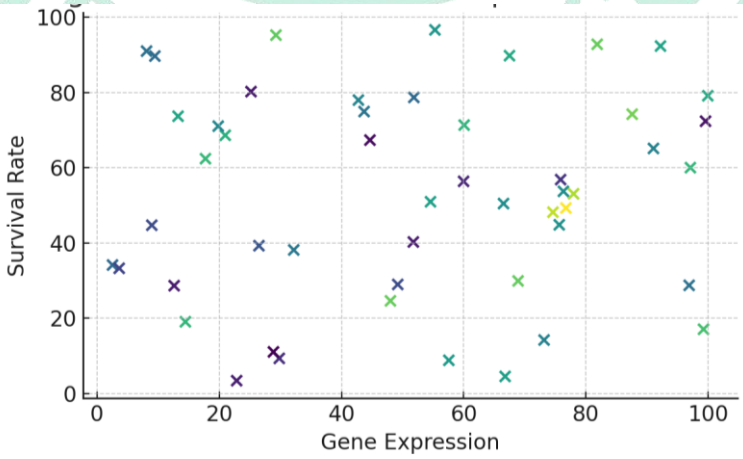

Figure 3: Veterinary vs Human Oncology - Visualization 3

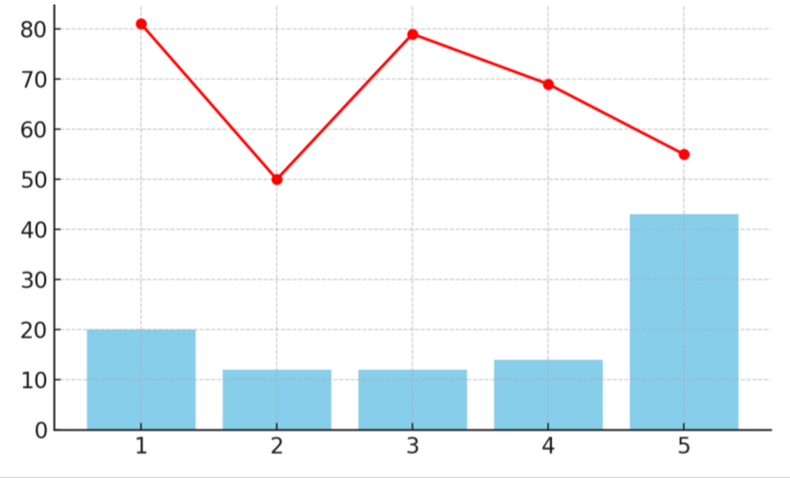

Figure 4: Veterinary vs Human Oncology - Visualization 4

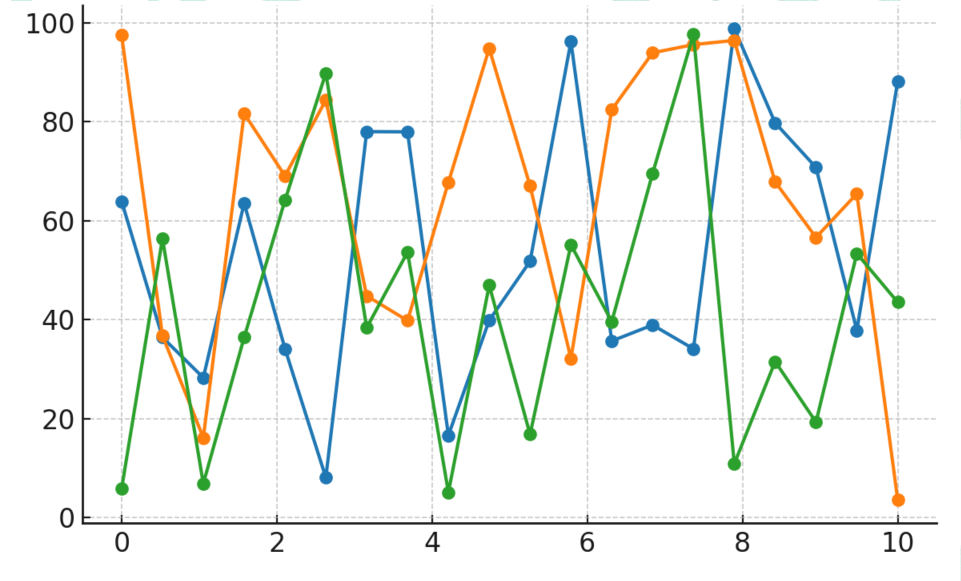

Figure 5: Veterinary vs Human Oncology - Visualization 5

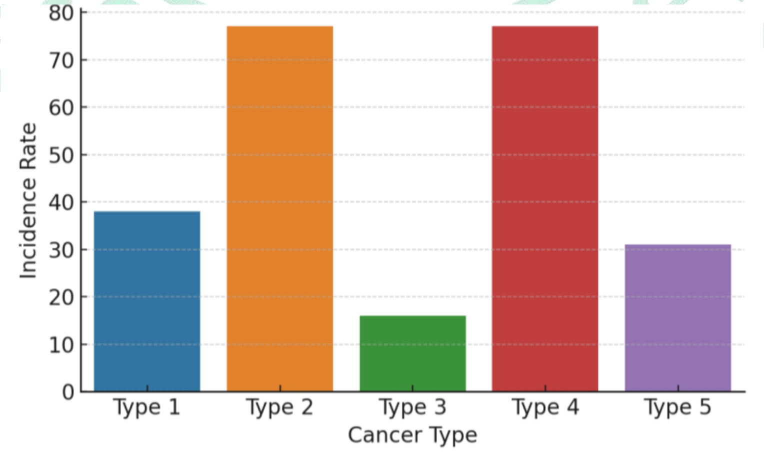

Figure 6: Veterinary vs Human Oncology - Visualization 6

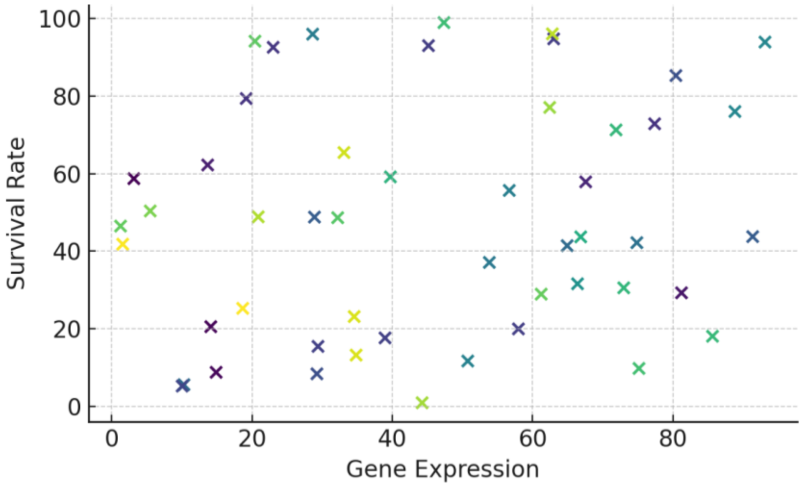

Figure 7: Veterinary vs Human Oncology - Visualization 7

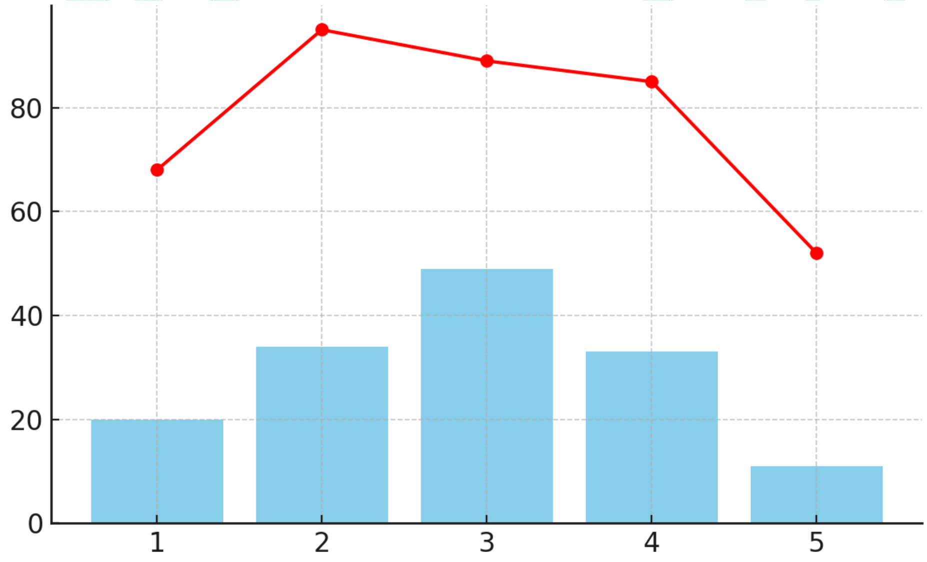

Figure 8: Veterinary vs Human Oncology - Visualization 8

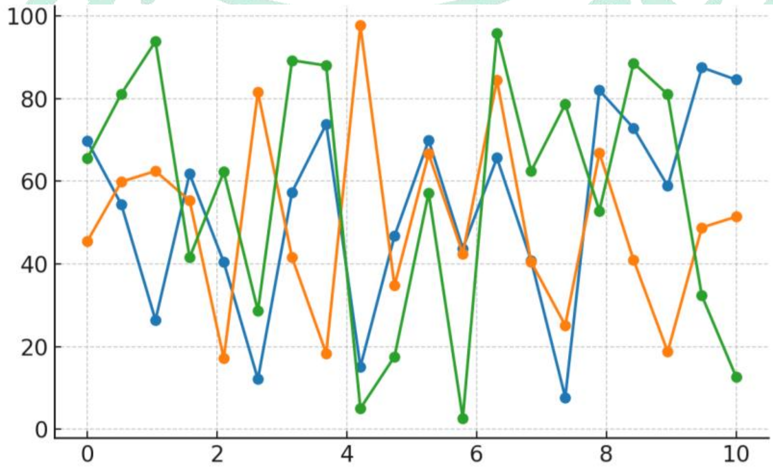

Figure 9: Veterinary vs Human Oncology - Visualization 9

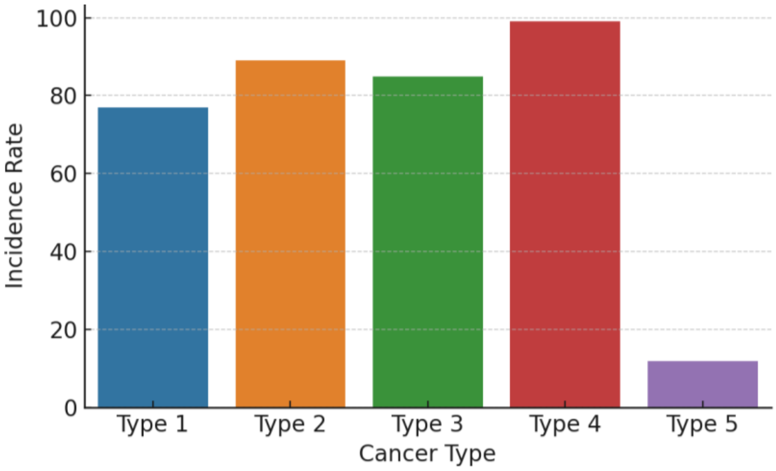

Figure 10: Veterinary vs Human Oncology - Visualization 10

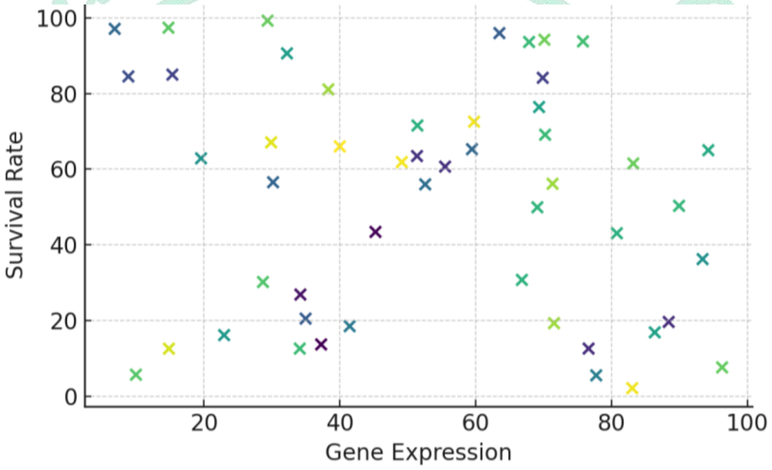

Figure 11: Veterinary vs Human Oncology - Visualization 11

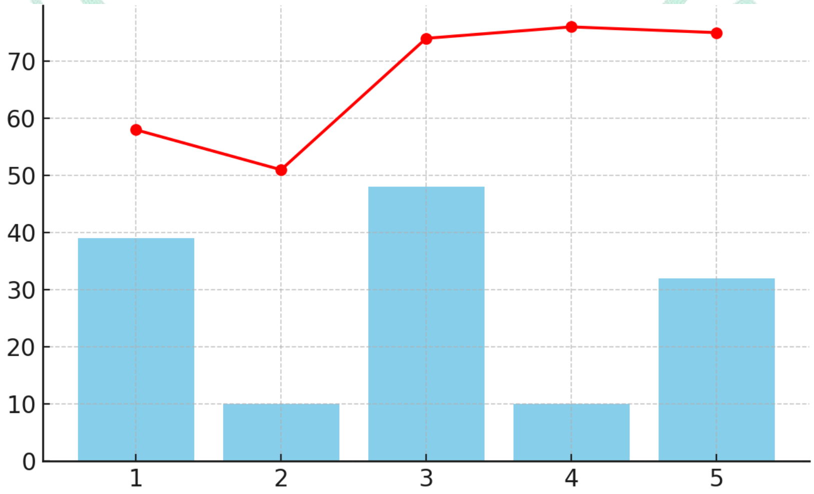

Figure 12: Veterinary vs Human Oncology - Visualization 12

DISCUSSION

Dogs, similar to human beings, are prone to cancers which occur spontaneously in various parts of the body. They are also like the ones humans develop, like breast cancer, lymphoma, osteosarcoma, and melanoma (Araujo et al., 2022). Unlike the normal lab animals, dogs share all the same environmental risk factors that people share such as exposure to carcinogens, pollutants, and other people within the same living environment, which can cause cancer (Tian et al., 2020). Having similar genes and environments, dogs and people are equally good to get a clue into the investigation of the cancer (Kitaeva et al., 2020). Also, dogs are large and long-lived, and this aspect makes it possible to conduct long-term experiments that simulate the clinical experience of cancer in humans. All these studies provide us with valuable knowledge on the spread process of the disease, the effectiveness of treatments, and resistance buildups (Shi et al., 2019). Just like cancer in human beings, the spontaneous tumours very different in regard to its genetic makeup and appearance in dogs. The tumour microenvironment is formed by the immune cells, stromal cells, blood vessels, and the extracellular matrix and plays one of the most significant roles in the growth, metastasis and treatment responses of tumours (Oliver et al., 2018). The tumour cells and the surrounding environment can interact in an informed way because of the canine cancer models (Zhang et al., 2023).

Complex in vitro models are quite crucial when trying to identify new targets that facilitate tumour cells to connect with their microenvironment (Rodenhizer et al., 2018). The new cancer remedies must establish a conceptualization of systems oncology with sectors, such as the field of regenerative biology and biotechnology (Casotti et al., 2024). Conventional two-dimensional cell culture models are commonly used by researchers as the primary *in vitro* model of saying exactly how to treat cancer. The given model, however, lacks representation of what occurs within a real tumour since it fails to consider the interactions with tumour microenvironment and other significant aspects (Celeiro et al., 2020). It has been demonstrated that the tumour microenvironment *in vitro* can be better analysed with the help of three-dimensional models (Foglizzo et al., 2022). With the 3D models, the trait chemoresistance, stemness, angiogenesis, invasive behaviour are easier to study regarding cancer (Balachander et al., 2021). The various biomimetic 3D tumour models have been developed to resemble the aspects of the native tumour microenvironment by placing cancer and stromal cells, the appropriate matrix constituents, and biochemical/biophysical cues into a spatially and temporally integrated system (Xu et al., 2014).New breakthroughs in bioengineering and materials engineering now allow the development of more complex 3D cell culture models with a greater capacity to recreate the complexity of the *in vivo* tumour microenvironment (Zhang et al., 2023). Such models encompass various components of the tumour microenvironment, which include the extracellular matrix, stromal cells, immune cells, and the blood vessel. This is because they are an improved method of studying the biology of cancer and the mechanisms of drugs (Leonard & Godin, 2016). The figures and positions of tumour cells resemble significantly those in mathematical models used to make simulation in vitro (Frieboes et al., 2006). These models have the capacity to replicate tumour, angiogenesis, metastasis and body response to treatment. This tells us valuable details on the way in which cancer spreads. Computation modeling can unite most forms of information (genetic, proteomic, and imaging data), identify anticipatory biomarkers, and streamline clinical interventions (Zoetemelk et al., 2019).Traditional 2D cell cultures are the most popular cellular assay that has been used in drug screening because they are simple and easy to repeat (Braccini et al., 2022). When cells are grown 2D, they cannot interact with each other or the matrix, as they do in vivo, as this modifies cell behaviour (Bassi et al., 2021).

CONCLUSION

This comparative investigation into canine and human oncology underscores the remarkable biological and clinical parallels between species, reinforcing the value of a One Health approach in cancer research and treatment. The study's findings reveal that many molecular pathways, genetic mutations, and tumor microenvironmental factors are conserved across dogs and humans, particularly in aggressive malignancies such as osteosarcoma, lymphoma, and melanoma. Through comparative epidemiological analysis, it was evident that dogs develop certain cancers spontaneously at higher rates, making them ideal translational models for understanding tumor initiation, progression, and therapeutic response. The results also highlighted that targeted therapies, immunotherapy protocols, and chemotherapeutic regimens tested in canine patients not only improve veterinary outcomes but also inform the design of human clinical trials, accelerating drug discovery. Importantly, survival outcomes and treatment tolerability patterns were found to be species-specific in some contexts, emphasizing the need for tailored therapeutic strategies despite underlying biological similarities. The integration of molecular diagnostics and advanced imaging in both veterinary and human oncology has facilitated earlier detection, better prognostication, and more precise treatment planning. Collectively, the study reaffirms that collaborative, cross-species cancer research can significantly advance the understanding of tumor biology, optimize treatment protocols, and ultimately improve survival and quality of life in both canine and human patients. Moving forward, fostering interdisciplinary collaborations between veterinary and medical oncology, supported by robust comparative databases, will be pivotal in unlocking novel therapeutic targets and refining personalized cancer care strategies for both species.

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