

Spring 2026 Research Competition Studies Funded by Smiling Blue Skies

Understanding aggressive behaviour of dog bone cancer

Dr Geoff Wood

Bone tumours are common and highly lethal cancers in dogs. Standard treatment consists of surgery (limb amputation) followed by chemotherapy but expected survival times are still only a year on average. The eventual cause of death in these dogs is spread of the cancer to other tissues, mostly lung. Currently, there are no reliable predictors of how well dogs with bone cancer will fare after treatment. However, our recent publications show that levels of small molecules called microRNAs are different in dogs with long versus short survival times after treatment. MicroRNAs are not just makers of disease outcome, they are also functional molecules. Each type of microRNA can change the expression of dozens of genes. In this research proposal we aim to measure microRNAs in dog bone cancer cell lines in culture. We have already found that levels of some microRNAs correlate with aggressive behavior of cells. We aim to manipulate the levels of specific microRNAs in cells and test whether this effects their behavior and also study the changes in genes that underly this effect. Novel treatments to prevent spread of cancers using drugs that target microRNAs are currently being tested in human cancer patients and out hope is that these can also be applied to dogs once we know which microRNA are the best targets.

CDK expression and inhibition in canine osteosarcoma with MYC amplification

Dr Courtney Schott

Osteosarcoma is the most common malignant bone cancer of dogs and people. It often spreads from its original site within a limb bone to distant sites in the body, like the lungs, causing patient mortality. The treatment, involving surgery and chemotherapy, has remained relatively unchanged for decades in both dogs and people; therefore, patient life expectancy has not significantly improved in a very long time, and new treatment strategies are desperately needed.

Genes that control normal cell growth are sometimes abnormal in cancer cells, which can explain how some cancer cells are able to grow uncontrollably. MYC is one of these genes and sometimes cancer cells have extra copies of this gene. In humans and dogs, when a patient's bone cancer has extra copies of MYC, it is associated with shorter patient life expectancy. It has also been shown that knowing the number of MYC copies possessed by human bone cancer cells can predict how sensitive the cells will be to treatment with certain drugs in experimental cancer models. Performing tests in cancer models (e.g., experiments with cancer cells in Petri dishes or mice) must occur before drugs can be tested in human or canine patients. Human bone cancer cells with extra copies of MYC are more sensitive to a drug called dinaciclib. This drug targets several cyclin-dependent kinases (CDKs) which are a family of enzymes involved in cell replication, and the MYC protein interacts with these CDKs to drive cell replication, promoting tumour growth and negatively affecting the patient.

Knowing that MYC copy number is relevant to dogs diagnosed with bone cancer, we are going to explore whether measuring MYC protein level could serve as test to predict outcome in dogs and whether dinaciclib has a similar effect on canine cells to that observed in human cells. First, we will determine how much MYC protein is found in canine bone tumours and check whether this is associated with how long dogs live after diagnosis; if MYC protein level predicts outcome, this test could eventually be used in the clinical management of canine bone cancer. We will compare how canine bone cancer cells with or without extra copies of MYC respond to dinaciclib. Uncovering whether canine bone cancer cells with extra copies of MYC respond differently to dinaciclib treatment in our model is a step in the direction of developing a new treatment strategy for patients with osteosarcoma.

Battling Dog Cancers: Investigating the Ability of Dendritic Cells, a Subset of Immune Cells, to Modulate Type 2 Innate Lymphoid Cells, Another Immune Cell Subset, to Attack Canine Osteosarcoma Cell Lines.

Dr Khalil Karimi

Innate lymphoid cells (ILCs) are white blood cells that regulate immune responses in cancer and are classified into ILC1s, ILC2s, and ILC3s, with documented roles in cancer. In dogs, ILCs are poorly understood and need more research. A study shows that human ILC2s expanded ex vivo can kill cancer cells when transplanted into mice with tumors. Human cancer immunotherapy offers targeted, personalized treatments to boost immune response. However, dendritic cell (DC)-based vaccines still face challenges due to immunosuppression. Autologous DC vaccines, made from patients' own DCs loaded with tumor antigens, are personalized treatments that have been tested across cancers such as glioblastoma, melanoma, and multiple myeloma. These vaccines have demonstrated feasibility, safety, and potential for tumor-specific immunity and long-term control, especially when combined with other therapies. Because dogs can develop cancers similar to those in humans, the current proposal aims to advance strategies for using autologous DC vaccines in dogs by i) optimizing the culture of autologous DCs derived from canine blood monocytes, ii) establishing an in vitro co-culture system between canine autologous DCs and ILC2s, and iii) assessing the potential to enhance ILC2s' anti-tumor functionality. We have primarily cultured DCs from canine peripheral blood mononuclear cells (PBMCs) in vitro and, through experiments funded by the OVC Pet Trust, have effectively enhanced the expansion of canine ILC2s derived from healthy dogs' blood. Remarkably, overnight co-culture of these expanded ILC2s with osteosarcoma cells increased granzyme B, possibly indicating an anti-cancer mechanism. Based on this observation and what we have learned from studies of human ILC2s, the current proposal will examine whether expanded canine ILC2s can be educated by in vitro-differentiated autologous DCs and effectively kill osteosarcoma cells and inhibit tumor growth. We will employ multiple techniques to analyze the phenotype and function of ILC2s when co-cultured with the DC vaccine. Furthermore, we will use several immunological assays to determine what ILC2s produce after co-culture with tumor cells and whether ILC2s' anti-tumor efficacy is enhanced by an autologous DC vaccine. Understanding how autologous DC vaccines interact with ILC2s to influence tumors is essential for developing new treatments. These treatments involve autologous DC vaccines and ILC2 cell transfers into dogs with osteosarcoma, aiming to boost anti-tumor immunity and improve survival and recovery.

Exploring new molecular biomarkers for a subtype of canine lymphoma

Dr Katy Morrison

T-zone lymphoma (TZL) is a common type of slowly progressive or “indolent” lymphoma that affects older dogs. Many dogs with TZL live for years with few signs of illness, but diagnosing this disease accurately can be challenging. Dogs with TZL can develop enlarged lymph nodes and have increased numbers of lymphocytes (a type of white blood cell) in the blood, or both, and these findings can look similar to other types of lymphoma or even non-cancerous immune responses. Recognizing TZL is important as its treatment and prognosis differ considerably from other lymphoma subtypes. Current diagnostic tests rely mainly on the appearance of cells and the detection of specific proteins on their surface, but these tests cannot fully explain why TZL behaves differently from more aggressive lymphomas or why some dogs develop more severe disease.

This project explores the use of microRNAs—small molecules that help regulate how genes are turned on and off—as new biomarkers for TZL. Changes in microRNAs reflect the underlying biology of cancer cells and can provide information that traditional tests cannot. Furthermore, microRNAs can be measured from fine needle aspirate (FNA) samples, which are already routinely collected during diagnosis, meaning no surgery or additional procedures are needed.

By comparing microRNA expression patterns in dogs with TZL to those in dogs with other lymphomas, this study aims to identify molecular features that can distinguish TZL from other lymphoma subtypes while also providing insight into the biological basis of its typically indolent behaviour. Understanding these differences will help veterinarians make more specific diagnoses, avoid unnecessary chemotherapy in dogs with slowly progressing disease, and better identify dogs who may need closer monitoring or treatment.