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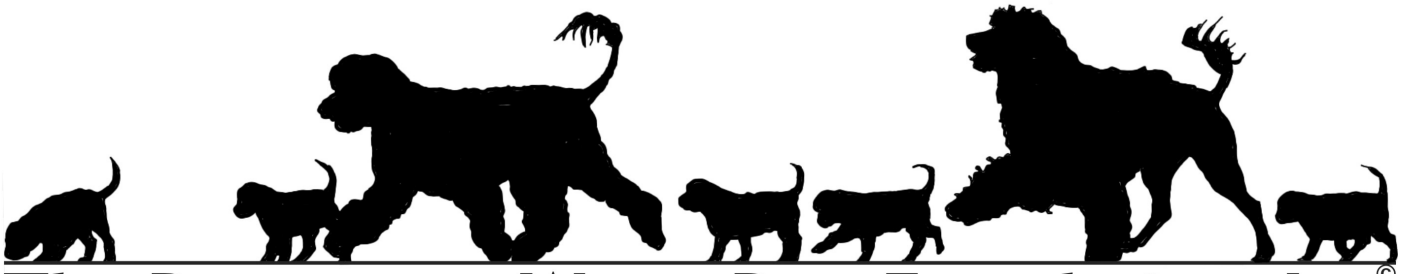

# The Portuguese Water Dog Foundation, Inc.©

**It's the Foundation's 20th Anniversary 1998 — 2018**

*We are dedicated to funding canine medical research focused on issues that affect the health and well-being of Portuguese Water Dogs everywhere.*

**Inside this issue**



## Microphthalmia and Delayed Growth Syndrome in the PWD

**Margret Casal, DVM, PhD, University of Pennsylvania**

At press time, we received the first progress report on PES research. The project is on target. More detail is available on our website. To date, the study has been able to collect DNA from 19 affected dogs and from 4 known producers. Males and females can be affected, although researchers have seen more affected females (about 70%). The frequency of this syndrome is as high as 25% in certain lines. Preliminary pedigree studies suggest an autosomal recessive inheritance. Dr. Casal has been been busy getting enough blood samples so she can run a Genome-wide association study (GWAS) scan, and is putting out a call for more DNA.

## The Foundation's 20th Anniversary

In 1997, the Portuguese Water Dog Foundation was formed for the purpose of supporting research to improve the health of the breed in breed specific diseases but also diseases that affect the general canine population. In 1998, the Foundation was incorporated as a non-profit corporation and in 2000, the Foundation was granted status to act as a 501(c)(3) charitable organization. Thank you for your support!

## A Novel Approach for Prevention of Canine Hemangiosarcoma

**Jaime Modiano, VMD, PhD, University of Minnesota**
**Mid-Year 3 Progress Report**

Hemangiosarcoma, an aggressive form of cancer in dogs, is the cause of death for one out of every five Golden Retrievers in the United States. Portuguese Water Dogs and Boxers also have an especially high risk for this disease which is devastating for all dogs. Hemangiosarcoma is incurable partly because the cancer is not detected until a very advanced stage when it is resistant to conventional therapies. Thus, an unconventional approach to improve outcomes for hemangiosarcoma patients will involve effective methods for early detection and for disease prevention. This project pairs two novel technologies consisting of a patented test to detect hemangiosarcoma cells in blood samples, and a treatment that attacks the cells that establish and maintain the disease.

During the 30 months that the project has been active, we have made substantial progress toward our objectives, and from the experience we have gained, we have made some adjustments to the experimental set-up.

1. As of August 20, 2018, we had tested the parameters of the detection test on 74 dogs that had diagnoses of hemangiosarcoma, non-malignant spleen masses, other tumors, or no apparent illness.

2. We updated and refined the criteria and the algorithms to identify hemangiosarcoma cells in circulation, and to use this as a test for early detection of hemangiosarcoma for dogs at risk. We have observed cells that have the predicted "hemangiosarcoma progenitor" markers in dogs with other conditions (other tumors such as melanoma, osteosarcoma, and possibly dogs with benign splenic hematomas). So, we have included additional markers to increase confidence in in the results that the cells we can identify in the blood represent hemangiosarcoma progenitors. Even if they do not, their presence seems to be

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infrequent in healthy dogs from a low-risk population, so we anticipate positive tests will be informative. If the cells of interest are associated with other tumors, it would mean that the test could be deployed more widely, and particularly for other tumors that we expect would be equally responsive to eBAT prevention.

3. We opened Shine On Phase-3 on January 2, 2018. As of August 20, 2018, we had enrolled 78 healthy dogs (Golden Retrievers, Boxers, and Portuguese Water Dogs). Even though we have not completed recruitment to establish sensitivity and specificity for the blood test, we have concluded from our data that the best way to determine if we can use this method to identify dogs with a high probability to eventually develop hemangiosarcoma, is to examine samples from dogs that are presumed to have "moderate risk" (based on age and breed) prospectively, and to follow these dogs through an extended period of time to determine if they eventually develop the disease. To our knowledge, this is the first time this type of bold experiment will have been done in dogs, and we believe the results will be more definitive than what we would obtain by further "tweaking" the assay in the phase-1 and phase-2 design. By implementing secondary safeguards (additional markers to define "circulating hemangiosarcoma progenitor cells"), we believe that we will be able to protect against false positive results. As part of the modifications to the study, we have been more emphatic in our communication with potential enrollees that, like every clinical trial, Shine On phase-3 is an experiment. Our institutional and programmatic philosophy is to make sure pet owners understand that clinical trials, regardless of their nature, do not have guaranteed benefits, and so, specifically for Shine On, the study website, the enrollment form, the FAQs, and the study reports all include the following statement: "The Shine On study is not intended as a diagnostic for disease. It is an experiment to determine whether the blood test can be used as a tool for early detection of hemangiosarcoma. At this point, we do not know if a negative test result means the dog does not have, or will never get, hemangiosarcoma. We also do not know if a positive test result means that a dog will definitely get hemangiosarcoma. The study is designed to answer some of these questions.

We have added a new parameter to the assay, using a human adenovirus that is "crippled" so it cannot replicate and cause disease, but it can still infect the putative hemangiosarcoma progenitor cells in the blood, but not normal while blood cells. We will continue developing this new parameter to further enhance the potential sensitivity and specificity of the early detection test. Managing Shine On holistically, and evaluating all the data in aggregate, will provide more robust data than if we were to parcel out the results by phase. This was not something we could have easily predicted in foresight, but it is clear in hindsight. In other words, since interim analyses are fraught with risk, we have decided to avoid strong conclusions from incomplete data. We will, of course, conduct a complete and thorough analysis of all the data once all the samples are collected and processed through the assays, and the results, whether positive or negative, will be reported to the AKC CHF and through them to the foundations that have supported the study. We also intend to publish the data in the peer reviewed literature. Regardless of the final result, there is significant innovation in this trial that will be of interest to the biomedical and translational communities.

## Broad-Range Detection of Canine Tick-Borne Disease and Improved Diagnostics Using Next-Generation Sequencing

**Pedro Diniz, DVM, PhD, Western University of Health Sciences, Final Report**

The long-term goal of our research team is to expand the current diagnostic tools to include a larger spectrum of potentially hazardous microorganisms. Our innovative approach is based on four pillars: (1) large-scale DNA sequencing to identify known and potentially new organisms present in blood of dogs naturally exposed to vector-borne diseases; (2) increase in sensitivity and specificity of large-scale sequencing by targeting major families of potentially hazardous organisms, (3) advanced bioinformatic analysis of millions of DNA sequences from a large number of dogs suspected of infection; and (4) comprehensive quality-control measures to support and validate the impact of our results. We confirmed that large-scale DNA sequencing can simultaneously detect one or more microbes in dog blood and can identify which microbe is present based on its unique DNA sequence. We confirmed this in both simulated infections as well as in samples from naturally infected dogs. We also detected potentially new organisms from sick dogs. With the crucial support from the AKC CHF, we were able to expand our expertise in using large-scale DNA sequencing technology for the detection of tick-borne pathogens in dogs and generate a large body of knowledge to support the further development of this technology. Ultimately, the results of this study will support early diagnosis and better medical care to dogs worldwide.

### Clinical Advancement of a Cancer Vaccine in Dogs

*Nicola Mason, BVetMed, PhD*
*University of Pennsylvania*

Canine lymphoma is the most common blood-based cancer in dogs with an estimated annual incidence of 30/100,000. Chemotherapy induces remission in 75-85% of patients; however, the majority of patients relapse with drug-resistant lymphoma within 8-10 months of diagnosis and most dogs die of their disease shortly thereafter. Cell-based vaccine strategies that stimulate anti-tumor immunity have shown promise in the treatment of many different cancer types including non-Hodgkin's lymphoma (NHL) in humans. In a previous study Dr. Mason developed a cell-based vaccine to induce anti-tumor immunity in dogs with NHL. Initial studies were hopeful as this early vaccine significantly prolonged second remission duration and overall survival, but ultimately the vaccine did not prevent relapse. These early findings suggest that while the lymphoma vaccine stimulated anti-tumor immunity it will require immunological boosting to achieve prolonged cancer-free survival. In the current study, Dr. Mason will optimize her cell-based vaccine approach to induce functional, long lasting tumor-specific immune responses that will prevent relapse and prolong survival in dogs with NHL.

**Final Report**

Our previous work has shown that white blood cells known as B cells found in the peripheral blood can be activated and grown outside of the body using special "feeder cells" that express a molecule known as CD40L. The stimulated B cells (known as CD40-B cells) can be loaded with genetic material (RNA) that has been extracted from the patient's tumor. When re-injected back into the patient, the CD40-B cells present the tumor material to the body's immune system and stimulate an anti-tumor immune response. We have shown in a phase I clinical trial that this approach has produced promising results with respect to prolonging overall survival in dogs with lymphoma. Since then we have been working to further improve this vaccine in 2 ways. Firstly, we aimed to generate a more robust system that induces greater B cell proliferation and produces B cells that have improved capacity to stimulate the patient's T cells against the cancer; and secondly to generate a more user-friendly system of B cell activation and expansion that would only require basic laboratory equipment to make these vaccines for canine patients. We saw this as an important step towards potential commercialization of the product enabling its use for many more dogs.

Current methods of generating the CD40-B cell vaccines from lymphoma patients are labor-intensive and require specialized laboratory equipment that is not available in most facilities. Therefore, we made second-generation feeder cells that stably express the human or canine form of CD40L (we previously used feeder cells that transiently express human CD40L). We found that our second-generation canine or human CD40L expressing feeder cells were simpler to maintain than the previously used transfected cells expressing human CD40L. We also performed several experiments to determine whether these cells could be irradiated prior to cryopreservation and then thawed to stimulate PBMCs. This would enable a master cell bank to be created. However, we found that B cell expansion using thawed, previously irradiated KTcCD40L feeder cells while possible is sub-optimal when compared to freshly irradiated feeder cells. We did experience technical difficulties in growing canine B cells with transduced feeder cells midway through our work and so we opted to use freshly irradiated KthuCD40L cells to generate CD40-B cell vaccines as previously described in our first pilot study for the current clinical trial.

The overall goal of the clinical trial was to determine whether repeat vaccinations with tumor RNAloaded CD40-B cells administered to dogs with B cell lymphoma after successful induction chemotherapy would prolong remission time and overall survival. We recruited 20 dogs. 7 dogs failed screening and 3 dogs failed CHOP based chemotherapy. Ten dogs were in clinical remission at restaging, and received their autologous CD40-B cell vaccines. The vaccine series consisted of an initial series of three vaccines, given three weeks apart, followed by a maintenance phase of booster vaccines, given once every 2 months.

Of the 10 dogs that received CD40-B cell vaccines, 7 completed their initial series and 3 dogs relapsed with clinical disease prior to completing the initial series. Of the 7 dogs that completed the initial series, 5 received booster vaccines. The other 2 dogs relapsed at re-staging and were not eligible to receive further vaccinations. Of the 5 dogs that received booster vaccinations, 4 dogs received only one booster before relapse and 1 dog received 4 boosters before clinical relapse. All dogs participating in the clinical trial received rescue chemotherapy although different rescue protocols were used depending on clinician preference. We found a significant correlation between the number of vaccines a patient received and the time between first relapse and death. This finding supports the hypothesis generated from our original study that vaccine-induced anti-tumor immunity synergizes with rescue chemotherapy to prolong duration of second remission in dogs with B cell lymphoma. An alternative hypothesis is that long term survivors in this group had less aggressive disease which enabled prolonged time to progression and overall survival unrelated to vaccination. We are currently seeking to evaluate the immune response of these dogs and the genetics of the dogs' tumors to try to identify whether anti-tumor immunity or intrinsic differences in tumor behavior are responsible for these findings. We also found that the TTP and OS times of dogs in this study were considerably shorter than reported in our original publication on this topic. The reason for this difference is unknown although the small cohort sizes in each study are likely to contribute to the discrepancy.

### The Role of Complex Translocations Associated with TP53 Somatic Mutations for Aiding Prognosis of Canine Diffuse Large B cell Lymphoma

*Matthew Breen, PhD*
*North Carolina State University*

Lymphoma accounts for up to 24% of all cancers diagnosed in pet dogs. Among these cases diffuse large B-cell lymphoma (DLBCL) is the most common subtype. Despite continued advances in veterinary medicine, the response to treatment for canine lymphoma remains highly variable with no reliable means to predict response. Studies of lymphoma in people have identified characteristic genome changes that have both diagnostic and prognostic significance. In human DLBCL, mutations in the TP53 gene, and genome rearrangements involving the MYC, BCL2 and BCL6 genes have been shown to confer particularly poor prognosis in cases treated with standard of care multi-agent (CHOP-based) chemotherapy. The investigator's previous CHF-funded studies have shown that canine cancers, including lymphoma, exhibit genomic changes that are conserved with those observed in the corresponding

human cancers, and have identified MYC and BCL2 rearrangements and a high frequency of TP53 mutation in canine DLBCL. This research will screen a well-defined collection of over 450 pre-treatment, canine DLBCL samples to determine accurate frequencies of these genome changes. The researchers will investigate the correlation of these target aberrations with duration of first remission, and identify key genomic signatures that may aid prognosis of prospective canine lymphoma cases. The data generated should assist owners and veterinarians with decisions regarding treatment with CHOP. Patients with signatures predictive of poor response to conventional CHOP chemotherapy may benefit from more aggressive treatment at the outset to improve outcome.

**Progress Report**

This study involves the evaluation of a cohort of canine lymphoma specimens for the presence of tumor-associated aberrations associated with four key cancer-associated genes (MYC, BCL2, BCL6 and TP53). The presence of these abnormalities, alone and in combination, has been shown to be predictive of the response to standard treatment modalities in human lymphoma patients, and provides powerful opportunities to predict prognosis in newly diagnosed patients. We hypothesize that the same may apply in dogs. Our hypothesis will be tested through analysis of the genomic status of each of these genes within a retrospective cohort of canine lymphoma cases treated with a common chemotherapy regimen.

We have designed appropriate methods for analysis of each gene, and have completed data acquisition. Data for three genes (MYC, BCL2 and BCL6) are currently undergoing detailed biostatistical analysis in context with the clinical data for each case. Data for TP53 are being evaluated for each individual case, and will then be compiled in readiness for integration with the full dataset.

Our findings show that structural aberrations of MYC and BCL6 occur in canine lymphoma at a broadly similar frequency to the human counterpart. We identified a small subset of cases bearing aberrations of both of these genes. This is potentially significant since the presence of both aberrations within the same tumor has shown to be associated with a particularly aggressive form of disease in human patients. We did not identify significant evidence for aberrations of BCL2 in our canine cohort. Abnormalities of the TP53 gene are widely variable in nature when compared between cases; however, we have thus far identified one distinct abnormality that is recurrent within the cohort, and which has been reported in several human cancers.

On completion of this analysis, all findings will be evaluated as a single comprehensive dataset and compared with the clinical outcome of each canine lymphoma case. This will indicate whether our data support a significant relationship between abnormalities of these four genes (whether individually or in combination) and the patient response to chemotherapy.

### A Novel Mechanism to Regulate the Growth of Canine Hemangiosarcoma

*Erin Dickerson, PhD*
*University of Minnesota*

Hemangiosarcoma is an extremely aggressive cancer that is rapidly fatal in dogs. While the lifetime risk is alarmingly high for some breeds such as Golden Retrievers and German Shepherd Dogs, the disease does not discriminate, and it can strike any dog at any time. Despite considerable efforts by veterinarians and scientists to find effective treatments, the outcome for dogs with hemangiosarcoma has changed very little over the past few decades. Recent evidence provides essential clues into how these tumors grow and progress, generating new ideas for treatment approaches. Such new evidence suggests that hemangiosarcoma cells rely on the metabolism of lipids or fatty acids to supply energy for tissue invasion or continued tumor growth. To obtain these lipids, hemangiosarcomas may take over the metabolic machinery of neighboring cells, forcing them to produce nutrients for the tumor cells to help them proliferate and grow. This study will verify that tumor cells rely on lipid metabolism for growth, and determine if tumor cells alter the metabolism of fat cells to obtain cellular nutrients and accelerate tumor cell lipid metabolism. Identifying and exploiting a novel mechanism that may disrupt this process by inhibiting the interactions between tumor cells and cells in the tumor environment will speed clinical investigations, and ultimately lead to improved outcomes for dogs with this devastating disease.

**Progress Report**

Hemangiosarcoma is an incurable cancer that is almost uniformly fatal. The tumors often grow quickly and spread rapidly, with half of all dogs dying within six months of diagnosis, even with treatment. Because the prognosis has not changed over several decades, a better understanding of the disease is needed to develop new treatment approaches. We have found that hemangiosarcoma cells appear to rely on the metabolism of lipids to supply some of the energy and essential building blocks needed for tumor growth. We also found that propranolol, a common drug used to treat heart disease in both dogs and people, limits the uptake of lipids into cells and blocks the cell's ability to process these compounds. Cancer cells have been shown to impose a self-serving metabolic program on normal cells by forcing normal cells to supply nutrients, such as sugars and lipids, to the tumor. Recent studies have shown that cells like adipocytes (fat cells) can be remodeled by tumor cells to help create a niche favoring tumor growth. Because propranolol can block the use of lipids by tumor cells, propranolol may be able to reverse the cancer-imposed metabolic reprogramming on adipocytes or other normal cells, limiting tumor growth. For this study, we sought to: 1) characterize the lipid metabolic program(s) in hemangiosarcoma cells and determine if the use of lipids by these cells could be blocked by propranolol; 2) determine if hemangiosarcoma cells alter the metabolic program(s) of adipocytes; and 3) whether these changes in adipocytes enhanced the tumor cell growth programs and the invasive nature of hemangiosarcomas. We found that propranolol inhibited key metabolic processes in hemangiosarcoma cells, including the uptake and processing of lipids. We also found that hemangiosarcoma cells reprogrammed normal adipocytes in a way that may force the adipocytes to produce nutrients for hemangiosarcoma cells to help them proliferate and grow. Parallel studies supported this idea by showing that adipocytes accelerated metabolic growth programs in hemangiosarcoma cells and enhanced programs favoring more aggressive disease. Future studies will be directed toward further assessing the metabolic programs of hemangiosarcomas and determining whether drugs like propranolol can be used to prevent the manipulation of adipocytes by tumor cells and reduce tumor growth and invasion.

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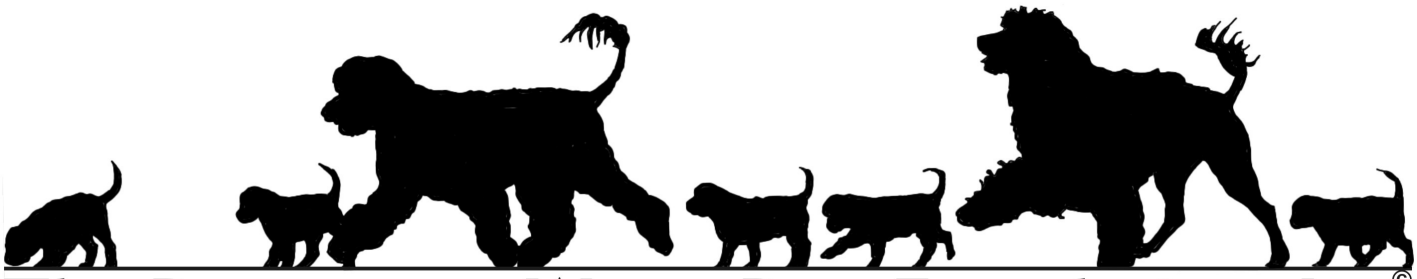

# The Portuguese Water Dog Foundation, Inc.©

**The Portuguese Water Dog Foundation, Inc.**
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Madelaine McCrorie & Rick Johnson in memory of our beloved Sunny McCrorie and Sonny Johnson – The Sunnys. We love you!

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Anonymous in memory of Boann

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Deb Carpenter & Regala's Kai Monster Ballou in memory of Eleanor Pierce

Julie Conger in memory of Nino

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Robert & Susan Dick in memory of PWDs "Chukker", "Polo", "Lisbon" and "Lolita; and in honor of PWD "Adele"

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Maxine Brainer in memory of Harley and Hanna – best two dogs in the world

Elle Brunner

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Linda Carey, Rockmere PWDs in loving memory of "Tomba" Rockmere's Tomba LaBomba. Loved and missed by Kim, Jeff & Bowen Barrows.

Christine Chandler in honor of the PWD PSG

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Bill & Judy DeSana in memory of Murphy (Our first PWD – passed in March 2017 at 15 years. Made 600 visits as a Therapy Dog.)

Gail DesRoches

Steve & Christine Dostie in memory of Eleanor Pierce

Roslyn Eskind in honor of the PWD PSG

Paul & Fran Fehling in honor of Daisy & Calvin

Ordean & Dorothy Finkelson in memory of Neptune and Osha

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Linda K. & Krista K. Hunt, Kalista in congratulations on your new titles to "Titan" CH Sun Joy's Back In Time At Kalista AX AXJ XF for his AKC Championship and AX titles!!

Morgan Jennings in memory of Marlene's Lucy

Beth Keicher in memory of Dr. Ken Sears and his dog, "Lucy"

Judy Machens Knorr in memory of "Carly" DreamStar Carly of DelCardo CD RA TD NA NAJ NF VCD1 CWDX GROM, and "Gypsy" Cortereal Gypsy

Sandy Kott in memory of "Cruz" CH Bayshore Cruz of Hope AX AXJ WWD SROM owned and loved by Lori Decheine

Sandra Kott in memory of MACH6 Mr. Darcy of Pennberlee owned and loved by Rob Moore and Paula Henthorn

Michael and Dona Lee in honor of Homeport Titan

Jenny & Mike Mobley in honor of the PWD PSG

Barb & Don Niemann in memory of "Indy" CH MACH PACH Finisterra Tangled Up In Blue CD RAE MXS MJG MXP4 MXPB MJP4 MJPB PAX OF WWD GROM

Pacific Northwest PWD Club

Nancy & Jack Perkins in memory of Falcor-Hytide's Madeira

Nancy Perry

Robert & Lois Pierce in memory of Eleanor Pierce

Carole Prangley-McIvor

Karen Pratt in memory of Elaine & Chris Rasmussen's "Maquah"

Karen Pratt in memory of John & Susan Cucura's beloved "Indy"

Karen Pratt – Thank you to the PNWPWDC for inviting me to judge your water trial in 2017!

Karen Pratt – Thank you to the USSPWDC for inviting me to judge your water trial in 2017!

Karen Pratt – Thank you to the Colorado PWDC for inviting me to judge your water trial!

Denise Ratner

Janice F. Reilly in honor of the PWD PSG

Barbara Rossi in memory of Farley, Jamie and Maggie

Martha Ruskai in honor of the PWD PSG

Mary Salvary in honor of the PWD PSG

Jeanne Sandeno

Sandra Saybolt in honor of the PWD PSG

Marion Schaufler in honor of the PWD PSG

Nan Sharp & Robert Hassler in loving memory of Leilani and Keoki

Peter & Geralyn Shea

Kersten & James Terry in memory of Kika & Padi

Philip & Marilyn Tierney in honor of Kammy, Lucy & Whitey

Merlin Tretter & Janet Venne in thanks to Linda Hunt for Moby

Debbie Tubbs in memory of "Zoe" CH Cortereal Electra VCD1 BN RE TDX AX AXJ NAP NJP NFP CWDX CGX MAC3 GROM

Jenn Urban in thanks to Olivia Stanwood from Village Taphouse in West Van. Thank you for your support of Whistler Brewing Company Rescue Ale and for supporting animals in need! Great job on winning our promotion.

Nancy Vener in memory of "Cassie" devoted companion of Donna Buckenmaier

Sian Wilstrup in honor of the PWD PSG

Elana Winsberg & Mike Barber in honor of Marisol

Jerry & Kim Wolcoveick in memory of Alice Vicha and all the Norvic PWDs

Kendra & Matt Yociss in honor of our dogs, Taylor Belle and Sasha Rayne Yociss – we love you both so much!

**Deck Hand up to $49**

Susan & Michael Burke

Carol Clark in memory of "Tide", beloved companion of Jean Hassebroek

Carol Cooke in remembrance of the one year anniversary of Randy Latham's passing

Deb & Tom Fenstermacher in memory of Eleanor Pierce

Miriam & Kal Goren in memory of "Tug" missed and loved by Ben and the "girls"

Linda K. & Krista K. Hunt, Kalista in congratulations on your new title to "Jagger" CH Kalista's Ready To Rock UD BN GN RA WWD CGC TKA for his RA title!

Linda K. & Krista K. Hunt, Kalista in congratulations on your new title to "Titan" Sun Joy's Back In Time At Kalista OA OAJ OF for his OA!

Linda K. & Krista K. Hunt, Kalista in congratulations on your new title to "Y" MACH3 Kalista's Now What BN CD RA MXP MXF T2B TKI CAX CWDX for his MACH3!

Linda K. & Krista K. Hunt, Kalista in congratulations on your new title to "Lyra" Kalista's Vega A Lyrae RN TKN JWD for her RN!

Linda K. & Krista K. Hunt, Kalista in memory of "Mimi" Kalista's Jewel of The Sea, loved and missed by the Mesna Family

Linda K. & Krista K. Hunt, Kalista in memory of "Tandy" Kalista's Irresistibly Tandy BN RE OA NAJ AWD, loved and missed by Ellen Gray

Linda K. & Krista K. Hunt, Kalista in memory of "Loki" Kalista's Loki The Shadow, loved and missed by Rod & Janine Dahlquist

Linda K. & Krista K. Hunt, Kalista in congratulations on your new title to "Tally" CH Kalista's What A Catch TKI AWD on her AKC Championship!

Linda K. & Krista K. Hunt, Kalista in memory of "Ginger" Therapy Dog loved and missed by Sandy & Gary Grundman & Rudder

Jackie Meltzer in memory of Eleanor Pierce

Miles & Gerry Nogelo

Sandy & Paul Novicki in memory of "Sailor"

Karen & Walter Paulick

Carol Prangley-McIvor in memory of Dick Kraus

Carol Prangley-McIvor in memory of Eleanor Pierce

Southern California PWD Club in memory of "Indy" CH MACH PACH Finisterra Tangled Up In Blue CD RAE MXS MJG MXP4 MXPB MJP4 MJPB PAX OF WWD GROM

Southern California PWD Club in memory of "Keel" CH Seadream Even Keel CDX TDX CWD HIT GROM

Janis Watts in memory of "Lucy" Legado Querida Lucia

Janis Watts in memory of "Nabia" Legado Queda D'Agua

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Missy McIvor
Helene Rothman
Heather Rideout
Art Prangley
Nina Grossman
Ceil McIvor
Verne Foster
Marie Genteal
Deyanne Miller
Christopher McIvor
John McIvor
Mark Miller
Beth Cramer
Carl Marinaccio
Russ (Skip) Pringle
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Judy Miller
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