



AMSCOPE

Newsletter of the AMERICAN MINIATURE SCHNAUZER CLUB
Member of the American Kennel Club

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SPECIALTY WINS

Greater Columbus Miniature Schnauzer Club
November 2010

Puppy Sweeps

Judge: Ms. Trish Clute

Best in Sweeps...Wards Creek's Straight Talk/
Schnetzer

BOS in Sweeps...Jacqueminot Jokes on You/C
Coffman/M Coffman

Regular Classes

Judge: Mrs. Lydia Coleman Hutchinson

WD...Wards Creek's Straight Talk/Schnetzer

RWD...Tee Jay's National Treasure/Young

WB/BW/BOS...Loneacre's Tinkering With
Chance/Harmon

RWB...Char N Co She's Electric/Stukey

BOB...CH Briar Hill Full Throttle/Lyons/Stukey

SEL...CH Rampage's Triple Crown/Ramel/
Hudziak

**DON'T GET STUCK....
GET YOUR DUES
IN ON TIME!**



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Cancer, Immune Problems And Vaccinations

By Dr. Susan Thorpe-Vargas

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According to the AKC figures, the incidence of cancer in the purebred dog is epidemic. Why is this so? Due to advances in veterinary healthcare, many dogs are living to an age where cancer is more likely to appear. We are also living in a polluted environment. Our canine companions are at an even higher risk for exposure to environmental toxins. Not only do some of us load our dogs up with flea and tick collars and dips, but their grooming habits make it much more likely they will ingest pesticides and other chemical carcinogens. One ubiquitous carcinogen is found in the outgassing of asphalt on hot days. It is also seen in meat that has been charcoal broiled. The chemical name for this substance is benzo [o] pyrene, and dogs simply crossing the street can get it on their paws and later lick it off. This chemical does its dirty work by causing missense mutations, a type of mutation that causes the replacement of a different amino acid in a protein and that can result in cancer.

At a recent conference, hosted in part by the AKC, it was revealed that cancer is the leading cause of death in dogs, after euthanasia. Lymphomas are the most common cancer found in canines, comprising about 20% of all malignancies. In humans, this type of cancer has been associated with chromosomal anomalies, it is most likely that this will prove to be true for dogs as well. What must be emphasized is that all cancers have a genetic component. We know that there are familial and breed related cancers and that only emphasizes the genetic aspects of the disease. Identification of affected families within the canine population may lead to the discovery of cancer susceptibility genes. It is no surprise to learn that those breeds with very small foundation numbers and those breeds with an overabundance of popular sires are those most valuable for this study.

The Genetics of Cancer

Two classes of genes are suspected of being involved in the occurrence of cancer when they are mutated: Tumor-suppressing

genes and proto-oncogenes, genes that function to encourage and promote normal growth and division of cells. The progression of tumor growth correlates with mutations that activate oncogenes (mutated proto-oncogenes) and render tumor-suppressor genes inactive. These mutations somehow "uncouple" the same mechanisms that allow normal cell division. What is so frustrating for both researchers and clinicians alike is that different combinations of mutations are found in different types of cancer and even in cancers of supposedly the same type in different patients. This reflects the random nature of these mutations.

Cancers caused by these loss-of-function mutations are more likely to be inherited. Parents that pass on a mutation in one copy of the gene produce offspring with a predisposition for cancer in that the disease requires only one mutation in the remaining "good" copy of the gene to be expressed.

Another familial type of cancer predisposition would be those that involve DNA repair. The body has mechanisms in place to detect errors in duplicated DNA. If mutations occur in the genes that code for the proteins responsible for this repair process, bad copies of the cellular DNA will accumulate. The initiation of cancer requires multiple events, which is perhaps one of the reasons cancer is seen more often as we age. The first mutation that is not repaired is thus inherited by any subsequent daughter cells. Cells thus affected do not undergo **apoptosis**—cellular suicide—and are rendered immortal. (both definitions are fine) Even though immortalization is not the same as carcinogenesis, which is the generation of cancer from normal cells, most transformed cell lines do not die after their normal number of cell divisions. This is a requirement for the further development of malignancies.

There appear to be several stages in the development of tumors. First, there is an initiation phase, in which an optimum or threshold level of mutations occurs and "tips

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LOOK

Please let me know if you make a change.

***DECALS & PINS.** You can get AMSC decals (\$1.00) and Replacement pins (\$7.50) from:

Mary Ann Shandor
2302 Cumberland Court, SW
Decatur, AL 35602
256-351-6942
tuckarry@aol.com

***LOCAL CLUBS...** please be sure to include *all* information when sending Specialty tear sheets for inclusion in AMSCOPE e.g. Judges, dates, entry, obedience.

*** LOCAL CLUBS...PLEASE send a copy of your newsletter to the following members of the Local Club Bulletin Committee:**

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***E-MAIL...** If you would like to send your Bouquets and Biscuits to AMSCOPE via E-MAIL, use the following address and include it in the body of the email.
CBORR@AOL.COM

**The deadline
for the
February issue
is January 20.**

APPLICANTS FOR SPRING 2011 Board Meeting

Calvo, Oscar Enrique
14 Columbine Ave North
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516-670-4933
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Sponsors: Harriet Catalina Meschi
Margo Klingler

Oscar Enrique Calvo owns 1 Miniature Schnauzer which he co-owns with one of his sponsors and with his mentor. He is not a breeder. He has attended 11 shows in 2009 and 2010 thus far. He plans to apply for membership in the Riverhead Kennel Club. He is a Landscape Architect and is interested in all listed AMSC activities.

Effland, Deborah L.
59 N Westen Avenue
Elgin, IL 60123
847-742-4006
Email: mystique@mac.com

Sponsors: : Marcia Feld
Michele Smith

Deborah Effland is a past member of AMSC whose membership accidentally lapsed. She has had Miniature Schnauzers since childhood. She does breed her dogs, producing 2 litters in the past 5 years. She is active in Conformation, Agility, Obedience and has trained for Tracking but has not competed. She attends 10-20 dog events yearly and has done pet grooming for 31 years. She is a member of the Chicago Miniature Schnauzer Club having served as President, Board, Secretary and Rescue. Her occupation is a Production Specialist.

Kolca, Francine M. Mieszala, Patricia
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Burbank, CA 91504
213-220-0500
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Sponsors: Kathy Colby
Vera Potiker

Has owned Miniature Schnauzers for over 30 years and has decided to become more dedicated to the breed and is learning to owner handle her own dog. She does not list herself as a breeder. They are members of the Miniature Schnauzer Club of Southern California. Ms Kolca has an MBA/ Business Management and Ms Mieszala is an RN – Consultant/Educator.

Wang, Sonny
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Sponsors: Vera Potiker
Kathy Colby

Sonny Wang has had both Miniature Schnauzers and Doberman Pinschers for 15 years. He is a breeder having bred 15 litters in the past 5 years. He is involved in breeding and in conformation attending approximately 20 dog events yearly. He is a member of the CNKC (China National Kennel Club) in Beijing China, serving as Vice President

Zemany, Susan M. Zemany, Gregory W.
6881 Thomas Dr.
Liverpool, NY 13088
315-453-7263
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Sponsors: Harriet Catalina Meschi
Margo Klingler

The Zemany's have owned Am Staffordshire Terriers, a Beagle, Welsh Terrier, and currently, a Miniature Schnauzer. They state they do not breed their dogs. They are active in conformation and obedience events, attending 10 shows in 2010. They do not belong to any other dog clubs but will be applying to the Onondaga Kennel Club. Susan is a Human Resources Director and Gregory is retired.

2011 Membership Dues

Online payment is made by secure credit card (VISA, MasterCard, Discover, or American Express) or PayPal transaction by using the following link. Note this link is only available directly, and cannot be accessed through the AMSC web site or through PayPal. You must have a PayPal account in order to pay via PayPal. Foreign members can pay with currency conversion to US dollars from most countries. Online dues payment: <http://amsc.us/dues.html>

Payment by check may be made by sending a check payable to the AMSC to the address below. Foreign payments must be a bank draft or certified check in US dollars. Sharon Edwards, 21301 Golf Estates Drive, Laytonsville, MD 20882
phone 301 947-8811
email: Treasurer@amsc.us

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the scales" toward tumor genesis. Once the cell has been transformed, there is a latent stage, in which mutations that have a selection advantage start to proliferate. During the clinical phase, the tumor becomes large enough to induce symptoms. These symptoms are caused by tissue destruction, or the production of soluble factors that can be detected in the blood or the tumor can depress vital functions and act as a space-occupying lesion in a confined anatomical space.

Genetic mistakes initiate cancer

Since apoptosis is also under genetic control, it is not surprising that many of the proto-oncogenes and tumor-suppressor genes altered during apoptosis are those genes involved with cell death. Many proto-oncogenes code for proteins involved in mechanisms that regulate the social behavior of cells. Signals from those cells in the immediate environment induce their neighbors to divide, differentiate and even undergo apoptosis. It also appears that both types of genes are involved with or expressed during the control points of the cell cycle. Human cancer studies show that mutations in the tumor suppressor gene called p53 account for many tumors. One of the functions of this gene is that it normally prevents cells with damaged DNA from proceeding through the cell cycle. The presence of the protein product encoded by p53 induces the expression of the waf-1 gene. The waf-1 gene produces a protein that normally inhibits the activity of several similar cellular proteins called **kinases** (enzymes that catalyze the conversion of proenzymes to active enzymes) that are involved in stopping cell cycle progression. A mutation in either the p53 or waf-1 gene sometimes can cause the loss of that "emergency brake" function and allow uncontrolled growth. One recent study has linked a case of benign canine melanoma to loss of this function. However, loss of apoptosis isn't the only culprit that causes cancer.

Many types of genetic mishaps can occur and can lead to disease. The basic types of genetic accidents include point mutations, deletions and chromosomal translocations mentioned earlier. The insertion of mobile genetic elements such as transposons—segments of DNA that are capable of moving to a new position within the same or another chromosome—or retroviral DNA—retroviruses are potent disease agents with the capability of incorporating their DNA into the host cell's DNA—into the cell's genetic material are

two other types of mutations.

Malignant transformations occur for a variety of reasons. Oncogenes, exposure to chemical carcinogens and ionizing radiation such as X-rays all play a role in inducing neoplasias. We even can "catch" cancer. In a number of species, although not yet demonstrated in dogs, **retroviruses** have been proved to be the cause of a variety of different diseases, including cancer.

A virus does not have the ability to reproduce itself but instead hijacks the host cell's reproductive capability by inserting its own DNA into the genome of the cell it has infected. It then forces that cell to produce the proteins it needs. This can cause something called an insertional mutation. Depending on where it inserts its viral DNA, the mutation can wreak havoc in a variety of ways. The result of these genetic accidents can alter the gene sequence so that it produces a protein with abnormal activity or even no activity at all.

Free radicals can attack DNA

Outside influences also can lead to mutations and changes in cellular genetics. Because we breathe an atmosphere that contains oxygen and we digest food, our bodies are constantly producing **free radicals**—highly reactive oxygen molecules that occur naturally in the body because of metabolic processes. Environmental factors such as air pollution, radiation, pesticides, herbicides, many drugs and cigarette smoke react within the body to cause free radical production. These molecules can damage DNA, affect the structure and function of cell membranes and damage certain regions of proteins that have enzymatic functions. Older humans and animals are more at risk due in part to increased levels of free radicals as well as an impaired ability of the immune system to eliminate altered cells. Very inbred dogs also have weakened immune function. (<http://cc.yosu.edu/~helorime/inbrimmune.html>)

Autoimmunity & Vaccinations

Autoimmune disease is genetic but like many other polygenic diseases, there is an environmental component. In the case of thyroiditis and diabetes, there is an established link to environmental *triggers*. Why are we seeing a rise in such diseases in the purebred dog? One could suggest it might be poor and outmoded breeding practices, i.e., inbreeding referred to as line breeding by many dog breeders. The portion of the genome that codes for the genes that

help us recognize "self" is called the MHC—the Major Histocompatibility Complex. These genes are located very close to each other and therefore it is very rare for recombination to occur. This in effect means that the genes from each parent are inherited intact as haplotypes. If the parents are closely related, then the possibility exists that they share the same genes at that site, i.e., they are homozygous by descent. This essentially cuts the functionality of the immune response in half— not a good thing. Normally, autoimmune diseases can be separated into diseases that are "organ-specific" and "systemic" categories. For instance, an organ specific example would be Graves' disease that is characterized by the production of antibodies to the thyroid-stimulating hormone (TSH) receptor in the thyroid gland. In the case of Hashimoto's, thyroiditis antibodies are formed against thyroid peroxidase; and in type I diabetes (the type most often seen in dogs) by anti-insulin antibodies. An example of a systemic autoimmune disease would be SLE (systemic lupus erythematosus). It also appears that some individuals are more at risk than others of developing particular diseases. As mentioned before, susceptibility to autoimmune disease is controlled by environmental and genetic factors, especially MHC genes. Results from both twin and family studies show an important role for both inherited and environmental factors in the induction of autoimmune disease. In addition to this evidence from humans, certain inbred mouse strains have an almost uniform susceptibility to particular spontaneous or experimentally induced autoimmune diseases, whereas other strains do not. These findings have led to an extensive search for genes that determine susceptibility to autoimmune disease.

One way of determining this in humans is to study the families of affected patients; it has been shown that two siblings affected with the same autoimmune disease are far more likely than expected to share the same MHC haplotypes. The more closely related two individuals are, the more likely that they share the same haplotype. The association of MHC genotype with autoimmune disease is not surprising, because autoimmune responses involve T cells, and the ability of T cells to respond to a particular antigen depends on MHC genotype. It appears that susceptibility to an autoimmune disease is determined by differences in the ability of variations of the MHC haplotypes to present various proteins that mimic "self" to those T cells that react to them. Inbred animals have fewer allelic variants. An alternative

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hypothesis for the association between MHC genotype and susceptibility to autoimmune diseases emphasizes the role of MHC alleles in controlling the variety of T-cell receptors. This lack of diversity means that developing and immature immune cells that are specific for particular self-antigens are not selected against and so are allowed to reproduce themselves.

However, MHC genotype alone does not determine genetic susceptibility to disease. Identical twins, sharing all of their genes, are far more likely to develop the same autoimmune disease than MHC-identical siblings, demonstrating that genetic factors, other than the MHC also affects whether an individual develops disease. One of these genetic factors would be B or T cell immunodeficiencies. Symptoms of this condition include eczema, dermatitis, heart disease, inhalant and food allergies and neurological disease. These conditions are often seen in the purebred dog.

This however begs the question as to why we are seeing a rise in autoimmunity associated with vaccinations. JAutoimmun. 2000 Feb;14(1):1-10. Vaccination and autoimmunity-'vaccinosis': a dangerous liaison? Shoenfeld Y, Aron-Maor A.

The whole duration of immunity and the timing and necessity for various vaccinations are being questioned. Many veterinary training schools are changing their recommended vaccination protocols. The practice of annual vaccinations lacks scientific validity or verification. There is no immunological requirement for annual vaccinations. The practice of annual vaccinations should be considered of questionable efficacy. Instead, clinicians, in the absence of legal requirements, should educate their clients that an annual physical examination is the better option.

Alternatives to vaccinations Monitoring Serum Antibody Titers

As mentioned before, one of the "unknowns" with animal vaccinations is the



duration of immunity. What this means is that the pharmaceutical companies have not determined how long a vaccination will protect against infection. One way to find out if an animal is still protected is to measure the amount or level of antibodies to a particular antigen is still present in the blood serum. This is called titering. Once an animal has been exposed to a particular disease pathogen the body makes antibodies against that organism. After they have done their "job", some of those antibodies change and become dormant so that the next time the animal is exposed to that same pathogen there is a stockpile of clones that can jump in and fight off that same infection. It is these antibodies that are being measured when an animal is titered. One point to consider is that titer levels do not really reflect the ability of an animal to fight of an infection, but rather how recently they have been "challenged" or exposed to that infectious agent. What this means is that the dog that stays at home and is never around other dogs is more at risk than those that have an active social life.

New Vaccination Protocols

Colorado State University

<http://www.vth.colostate.edu/vth/savp2.html>

UC Davis

<http://www.vmeth.ucdavis.edu/vmeth/clientinfo/info/vaccinproto.html>

University of Pennsylvania

<http://www.vet.upenn.edu/comm/publications/bellwether/48/vaccination.html>

University of Florida

<http://www.vetmed.ufl.edu/sacs/Misc/2001vacprot.htm>

Washington State University

<http://www.vetmed.wsu.edu/rdvm/vaccine.html>

Recent research may have uncovered the link between vaccinations and autoimmunity. Most autoimmune disorders appear to be triggered by some type of toxic assault or a viral or bacterial exposure. Why is this important? Preliminary studies have shown that something called molecular or antigenic mimicry may be involved. This intriguing model argues that the body is reacting to small protein-like fragments of the pathogen that are homologous to normal cellular components. If true, this would be a form of antigen or molecular mimicry in which antibodies formed against one molecule react with another similar looking molecule. Another factor to consider

is illustrated in a recent study that showed that contaminants (specifically bovine thyroglobulin in rabies vaccine) that remain from growth of the cells in culture during vaccine production cause the dog to make antibodies against these contaminants. Since the bovine thyroglobulin molecule is very similar to the dog's own thyroglobulin, the antibodies produced against the bovine thyroglobulin cross-react with the dog's thyroglobulin. This could explain why so many dogs have thyroiditis characterized by high levels of antithyroglobulin antibody in their serum. This may in turn, explain why we currently have an epidemic of hypothyroidism in dogs in the United States.¹³

Vaccination Take Home Message

1. Vaccination should only be given at age appropriate times - the most common reason for vaccine failure is maternal antibody interactions.
2. Never vaccinate a dog that is ill or malnourished (the second most common reason for vaccine failure is nutritional deficits).
3. Only vaccinate for the: (a) "core" diseases like distemper and parvo, (b) those diseases appropriate for your dog's environment and (c) those mandated by law.
4. Follow the new guidelines for frequency of vaccinations and suggested combinations of vaccines.

* MOVING?

**Be sure to send your
change of address
and new phone number
and new email
address to us so that
AMSCOPE can follow
you...you wouldn't
want to miss one
issue!**

TROPHY DRIVE...

**please don't forget
the trophy drive.**

Terrie Houck

**105 Fite's Creek Road
Mount Holly, NC 28120-1149**

In Memoriam

Mildred Schultz



Long time club member Millie Schultz passed away on Sunday, December 5. Millie was made a life member of AMSC this year.

The family has asked that Instead of flowers, they would appreciate donations to Miniature Schnauzer rescue. Over many years Millie has rendered valuable service to our club and our mission. Most notably Millie ran our special projects committee for many years. Due to her determination and enterprising spirit, she helped to keep the club in black when otherwise we would had been in a deficit. Before the special projects committee, Millie did Rescue for the AMSC.

Bouquets & Biscuits

* **MACH3 Southcross Speed Trap OF** (Ch Wards Creek's Extreme Commitment x Ch Classic Southcross Eclipse CD RN) earned his third Master Agility Championship title on November 12, 2010 in Lubbock, Texas at the South Plains Obedience Training Club's fall agility trial. Radar was bred by Sonny Lelle and is owned, trained, and adored by Teresa Janssen.

Canine Body Language: What Does It Mean?

Dogs can communicate with other dogs through a series of signals, including a variety of facial expressions, body postures, noises, and scents. Your dog will use his mouth, eyes, ears, and even his tail to express his emotions. By reading the combination of body signals, you should be able to work out who is top dog in any confrontation or situation.

Big=Aggressive

A dog that feels brave or aggressive will try to give the impression of being a larger, more powerful animal. He will stand tall with his ears and tail erect and thrust his chest forward. He may also raise the hairs around his neck and along his back (his hackles). Aggressive dogs may also wave their tail slowly and growl.

Small=Submissive

A submissive dog, on the other hand, will try to appear small and puppy-like. Adult dogs will chastise puppies, but they don't attack them. A submissive dog's approach to a more dominant canine or human is likely to be from the side, crouching near to the ground with his tail held low and wagging enthusiastically. He may also try to lick the hands or paws and face of the dominant dog or person. If this isn't appeasing enough, he might then roll onto his back to expose his stomach, or even involuntarily pass a small amount of urine.

The many meanings of tail wagging

One pattern of behavior that's characteristic of dogs and familiar to almost everyone is tail wagging. Most people recognize that loose, free tail wagging indicates pleasure or friendliness. Exaggerated tail wagging, which extends to the entire rump, is sometimes seen in subordinate dogs, as well as dogs with very short tails.

The tail, however, is also an indicator of other emotions. A tail waved slowly and stiffly, in line with the back, expresses anger. Clamped low over the dog's hindquarters, it's a sign that the dog is afraid. Anxious or nervous dogs may stiffly wag their drooping tails as a sign of appeasement.

Facial expressions—and more

The facial expressions of your dog will tell you a lot about his mood—whether he's anxious or excited, frightened or playful, or any one of a vast range of emotions he may express. His ears prick up when he's alert or listening intently, but are held back or

flattened onto the head when expressing pleasure, submission, or fear.

To successfully read his mood correctly, you must watch for other body signals at the same time. The narrowing or half-closure of the dog's eyes indicates either pleasure or submission, but when his eyes are wide open, he's signaling aggression.

Eye contact: Who blinks first

In the wild, the pack leader can maintain control simply by staring at a subordinate dog. The two animals will continue to stare at each other until one challenges the other, or until one lowers his head and turns away. If the staring continues after the submissive dog has looked away, he will feel confused and may bite out of fear. If eye contact is not broken, the dominant dog will reinforce his threat by snarling, growling, or even attacking.

You should not try to outstare your dog if he has aggressive or nervous tendencies because this could provoke an attack. Nevertheless, regular, gentle eye contact reassures the dog and reinforces your relationship.

Smile, we're communicating

Submissive dogs may appear to be "smiling" when they open their mouth to show their teeth in a lop-sided grin of friendliness. In the snarl of aggression, however, both lips are drawn right back to expose most of the teeth, and may be accompanied by a growl.

A dog will indicate his desire to play by raising a front paw, or by performing the play bow, which is often accompanied by barking to attract attention. Other gestures include offering a play object or bounding up to another dog to invite a fun and friendly chase.



Battling Canine Cancer

From the AKC Q4 2010 Edition

Humans often refer to it as "the dreaded 'C' word." In the canine world, cancer is just as disturbing and pervasive. And at PetPartners, the exclusive provider of the AKC Pet Healthcare Plan, we are seeing an increase in canine cancer claims.

Better Prevention, Earlier Diagnosis and Advanced Treatments

The good news is that, with more emphasis on prevention, our pets stand a better chance against falling victim to cancer in the first place. But if they do, early detection can help ensure the best possible outcome.

Good cancer prevention starts by researching dog breeds and make certain you adopt your dog from a reputable breeder. Understand your dog's inherited risks but keep in mind that even the best-bred dog from the best breeder can develop cancer. While any breed can be affected, some studies suggest that certain breeds (see the AKC Gazette article below) are more susceptible to cancer than others. A recent edition of AKC Breeder contains helpful links to a series of articles, videos and podcasts on canine cancer issues, risks and therapies.

Dogs need a healthy immune system to best avoid illnesses. Frequent exercise, along with regular medical and dental check-ups is necessary to maintain good health. As many veterinarians agree, lots of love and attention are also critical for a dog's physical and emotional well being. A fit, happy and healthy dog is less likely to develop cancer.

Quality food and clean water will also go a long way toward keeping your dog healthy. Discuss with your veterinarian the best type of food for your dog, as well as appropriate meal portions to avoid obesity (which can also contribute to cancer). Ask your veterinarian about adding omega-3 fatty acids or supplements to your dog's diet, another way to potentially reduce the risk of cancer. Avoid foods that contain known carcinogens such as growth hormones, insecticides, preservatives and artificial colors.

Research has shown that spaying and neutering can be an effective method of preventing canine cancer and other illnesses. As a side benefit, some studies suggest that spaying and neutering helps prevent aggression and roaming. Ask your breeder and your veterinarian about the pros and cons involved in spaying and neutering.

Maintain a Safe Home Environment

Healthy dogs should avoid exposure to pes-

ticides and harmful substances in their homes and yards. Our pets will be at their healthiest when their environment is carcinogen-free.

Be aware that a harmless-looking puddle in the yard might contain certain cancer-causing substances such as asbestos dust from brakes. Also, many cans and bottles in the garage, under the sink and by the pool might look like chewable fun to your dog but can cause cancer down the road. Contaminants can cause immediate illness or symptoms that develop later on after initial exposure. Some other known carcinogen sources include coal, kerosene heaters and excessive diagnostic x-ray work.

Despite your best efforts to keep harmful contaminants away from your dog, sometimes accidents do occur. Dogs are curious by nature and the unthinkable can happen when they get into things they should not. This is where pet insurance can help cover the costs for unplanned emergency clinic visits. Some recent claims we have seen include \$2,423 for rodent poison toxicity, \$1,500 for fertilizer toxicity, \$1,042 for cocoa mulch poisoning and \$3,464 for household cleaner poisoning.

Connect the Dots of Warning Signs for early Diagnosis

The best weapon for fighting canine cancer is early detection. Be aware of these visible symptoms while cancer is still optimally treatable:

- Sudden weight loss
- Abnormal swellings that grow
- Loss of appetite
- Sores that do not heal
- Bleeding or discharge
- Problems eating or swallowing
- Offensive odor
- Loss of stamina or desire for exercise
- Lameness or stiffness
- Difficulty breathing, urinating or defecating

Although you might do all of the "right" things, there is no guarantee that your dog won't develop cancer. However, thanks to advances in veterinary diagnostics and therapies, canine cancer can be caught and treated earlier. This helps increase the chance for survival and a high quality of life. While new anti-cancer treatment therapies become available almost daily, early diagnosis remains critical -- the smaller the tumor the better the outcome.

The Future Holds Even Better Treatments

Today, our dogs are lucky that they can receive and benefit from many of the same

cancer treatments used by humans. In fact, the most commonly seen cancer in dogs - lymphoma - is generally treated using protocols adapted from interdisciplinary human and veterinary studies.

Supporting efforts to fight canine cancer is the AKC Canine Health Foundation. In recent years they have allocated new grants of almost \$2 million toward 27 clinical research projects, with the common goal of:

Discovering new treatments to improve to existing therapies to eradicate cancer or slow down tumor growth

Finding methodologies for earlier and more accurate cancer detection

Increasing understanding of the genetics of lymphoma in order to develop alternative canine cancer treatments

The AKC Pet Healthcare Plan includes cancer coverage and other serious conditions in all of our illness plans. Many other pet healthcare plans exclude cancer coverage or provide a cancer 'rider' for an additional fee. Others consider cancer a genetic condition and exclude it from coverage on that basis. All of our illness plans include cancer diagnosis and treatment costs. For more information on our plans and a no-obligation quote, contact us at 866-725-2747 or visit www.akcpethealthcare.com.

Back to the Drawing Board: Hemangiosarcoma

While researchers across the country are working hard to learn more about the causes and potential treatments of cancer, hemangiosarcoma remains one of the more frustrating cancers in dogs. Experts note hemangiosarcoma tumors account for an estimated 5 to 7 percent of all tumors seen in dogs. Its aggressive and highly malignant nature remains particularly challenging to researchers and veterinarians because it has usually metastasized by the time it's diagnosed. Equally frustrating, the symptoms are often subtle or nonexistent until the final stages, with some dogs succumbing to the disease within days of the initial diagnosis.

What seemed to be a simple, albeit aggressive disease is much more complex in both origin and biology than what researchers originally thought. What they have learned in 40 years of assumptions about hemangiosarcoma may not be entirely correct, and a "back to the drawing board" approach is likely to be the most efficient way to find this cancer's Achilles' heel, according to Jaime Modiano, VMD, Ph.D., Perlman professor of

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oncology/comparative medicine, College of Veterinary Medicine and Masonic Cancer Center, University of Minnesota.

"We believe that hemangiosarcoma arises from a specialized cell that originates in the bone marrow and may have multipotential-meaning it can give rise to more than one type of cell, i.e., has stem-cell properties—or perhaps one that contributes to blood-vessel formation but is not, strictly speaking, the 'endothelial' cell we recognize in normal vessels," Modiano says. "While we remain fairly certain the cells that give rise to hemangiosarcoma come from the bone marrow, we do not know if the transforming event(s) take place there or in the target origins."

Any breed can be affected, but some breeds appear more susceptible, including Boxers, German Shepherd Dogs, Portuguese Water Dogs, English Setters, Great Danes, and Pointers, among others. According to an Australian Shepherd breed-club health survey, it's the number-one cancer in Aussies. A similar Golden Retriever health study published in 2002 estimated the lifetime risk for hemangiosarcoma is one in five. While dogs of any age are susceptible, it occurs more commonly in middle-aged dogs with the mean age between 8 and 13.

The good news is that it's not all bad news. In addition to working to define the genesis of the tumor and to explore the extent of breed-specific variation in the biology and behavior of hemangiosarcoma, researchers at universities across the country are conducting various hemangiosarcoma research projects, including working to discover pathways or molecules that are essential for hemangiosarcoma proliferation and survival, and will be amenable to targeting using pharmacologic or immune-based strategies.

These excerpts first appeared in an article by Tracey Libby in the November 2009 AKC Gazette and are reprinted with permission. To subscribe to the AKC Gazette visit: www.akc.org/pubs/index.cfm.

DUES....DUES....DUES...

**DO SENT IN YOUR
DUES**

**with correct address
and phone and email!**

BLACK RIBBONS

*** Dynasty's Classified Page (Classy)** followed her breeder, Millie Shultz, to the Rainbow Bridge on December 12, 2010 at the age of 14 ¾ years old while laying on my husband's lap. Classy came to live with Imre and I and my first show dog, Dynasty's Beyond the Abacus (Abby) in February 1999 at the age of 3 years old. Classy was coming as a companion to Abby, but I immediately recognized how nice Classy was and asked Carma Ewer, her owner, if I could keep her intact and show her. Carma was generous and let Classy live with me and let me show her. Carma warned me that Classy hated the show ring and true to form, every time I would get her ready to show she would chew her furnishings, so she never finished her championship. I talked Carma into selling Classy to me in December 1999. Classy was already a top producing bitch with three champions from her first two litters with Carmel's Special Dark (Hershey) when I purchased her from Carma and another champion finished from the second litter before my litter with Classy was old enough to show. [Classy's babies from her first two litters were very successful - Ch. Dynasty's New Image (black dam of 2 champions), Ch. Carmel Kokopelli (black dam of 7 champions), Ch. Carmel Hershey's Hugs n Kisses (black dam of 3 champions) and Ch. Dynasty's Media Blitz, also a black bitch.] The litter from Classy and Hershey was the beginning of Abacus Miniature

Schnauzers as my first champion came from that litter – Ch. Abacus Consider The Ethics, a salt and pepper male with a champion son and daughter and champion grandson to his credit. Classy will be missed at my house, but her legacy lives on in her descendents – she is in the pedigree of every good dog I have.

Linda Wahlquist-Soos

*** Buggy..** It is with great sadness that I am writing to you. At age 15 1/2, I had to put Buggy down on Friday, November 12, 2010. He lived for 4 1/2 years longer than all of his doctors expected. He had severe heart disease which ultimately led to kidney disease. Dogs aren't supposed to be on Enalapril as long as he was! He lived a normal, high quality of life any prince would live. My 2 children adored him and we can't believe life still goes on w/o him. Zoey is still searching our house and dog yard.

I know there is a section in the AMScope and I would like to let the Schnauzer world know just how much he will be missed. As my Grandmother once told me, "You never forget your first true love."

Ellen Levy-Gray

Avatar Miniature Schnauzers

2011 Membership Dues

*Members receiving AMSCOPE by **Email** - **\$30 individual** - **\$52.50 joint**
*Members receiving AMSCOPE by **regular mail (hard copy)** - **\$55 individual,**
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new logo on floor mats and vests

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This beautiful colorful woven 100% cotton afghan of Miniature Schnauzers in a garden setting will be a keepsake. Black, Salt/Pepper and Black/Silver Minis are at home on green grass with colorful shrubs, trees and flowers in the background. Lori Bush and Mildred Shultz collaborated with the artist at We Love Country on this very unique design. \$75. Includes Shipping

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FRONT



BACK



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AMSCOPE

Carla M. Borrelli, Editor
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UPCOMING SPECIALTIES

Portland MSC January 19, 2011
Regular Classes: Ms Gay Dunlap
Sweepstakes: Julie Cooper

Chicago MSC/IKC Specialty February 26, 2011
Regular Classes: Lorraine Boutwell
Sweepstakes: Diana Francois

Cincinnati MSC (Louisville) March 19, 2011
Regular Classes: John Constantine
Sweepstakes: David Owen Williams

Cincinnati MSC May 26, 2011
Regular Classes: Penny Hirstein
Sweepstakes: Arlene Smith

MSC of Atlanta April 17, 2011
Regular Classes: Carole Luke Weinberger
Sweepstakes: Linda Drost

MSC of Southern California June 24, 2011
Regular Classes: Clay Coady
Sweepstakes: Carma Ewer

MSC of Southern California June 25, 2011
Regular Classe Ken McDermott
Sweepstakes: Shirley Cole

AMSC SPECIALTIES

Louisville Ky. Kennel Club March 20, 2011
Regular Classes: Dale Miller
Sweepstakes: Lisa Sarvas
AMSC Obedience: Marilyn Gromley March 16

GWTA June 26, 2011
Regular Classes: Geri Kelly
Sweepstakes: Gale Schnetzer

Montgomery County KC October 9, 2011
Regular Classes: Wyoma Clouss
Sweepstakes: Susie Atherton

Fort Worth March 25, 2012
Regular Classes : Carole L. Weinberger
Sweepstakes: Jan Taylor

Great Western June 24, 2012
Regular Classes: Lynda Berar
Sweepstakes: Carla Nickerson

Montgomery Co. October 7, 2012
Regular Classes: Ken McDermott
Sweepstakes : Linda Drost



**Have you been
a member of
AMSC for 50
years?**

**If the answer is YES,
please contact
Susie Atherton at
lakiel@aol.com**

FOR YOUR CONVENIENCE: The following information is given to help conduct AMSC business more efficiently. Please remember that the Secretary and the AMSCOPE editor should **BOTH** be notified of address changes, club officers and specialty results.

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