THEORETICAL EXPLANATION OF NEOPLASENE EFFECTS ON CANCEROUS AND OTHER ABNORMAL TISSUE

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Abstract

A theoretical basis is presented that explains the effect that neoplasene and neoplasenex have on neoplastic tissue. Neoplasene compounds attack neoplasm by preferentially triggering apoptosis and sparing healthy tissue. Squamous cell, mast cell, spindle cell, sarcoid, transitional cell, melanoma, osteosarcoma, nerve sheath, hemangiosarcoma, lymphoma and many more classifications of neoplasm, bacteria, including MRSA and viruses have been eliminated with neoplasene and neoplasenex. Treatment protocols have been generally the same with variations specific to the situation presented. Treatment has been largely successful with less than satisfactory outcome associated most often with noncompliance to protocol and situations where the neoplasm has been greater in size and growing more rapidly than can be handled by even the most aggressive treatment efforts. Many representative case histories are presented including squamous cell, osteosarcoma, mast cell, sarcoma, spindle cell, neurofibroma, and transitional cell carcinoma of the bladder.

Introduction

Neoplasene and neoplasenex are names coined by the author to apply to the subject medicines. Confusion and misinformation abounds in the lay and trade literature regarding the nature of neoplasene compounds. They have alternatively been inappropriately referred to as “bloodroot”, “black salve” and as “escharotic”. Neoplasene is made up, in part, of plant alkaloids extracted from the botanical known as bloodroot and other plant species. These alkaloids are a proportionately very small constituent of the materials used to produce neoplasene compounds. Certainly neoplasene compounds cannot be accurately described as “bloodroot” or even bloodroot preparations. The action of neoplasene is apoptotic not escharotic and lay preparations called black salves vary widely and by the author’s observation may, or may not, be escharotic, apoptotic or both in their effect. The menstruum used to extract the active chemicals from the plant material used to formulate the medicines is a collection of halogen species. The isoquinoline alkaloids sanguinarine, sanguidimerine, chelerythrine, protopine and others are, in addition to being

extracted, also chemically modified and this, it is theorized, contributes to the efficacy of neoplasene. The compounds are acidic not basic and therefore are not caustic and do not create an eschar. That the compounds are preferential in their attack on neoplastic cells is telling in that a caustic escharotic would not differentiate between normal and neoplastic tissue.

Neoplasene compounds attack fast growing cells of broad type including: mast, basal, squamous, spindle and transitional cell; sarcoid, hemangiosarcoma, osteosarcoma, melanoma and lymphoma; in fact there has not been any cancer type that has demonstrated immortality against attack by neoplasene compounds. In addition viruses and bacteria of all types observed also succumb to neoplasene compounds. Neoplasene compounds are potently active against methicillin resistant staphylococci aureus.

Neoplasene is administered topically, orally, by intralesion and intravenous injection and it also is infused into the bladder, prostate, udder, nares, etc. The notable side effects are that systemically neoplasene is an emetic and intravenous injection may result in anaphylaxis. Antiinflammatories of any kind inhibit the action of neoplasene compounds and are to be avoided during their use. The mechanism of this inhibition is not known, however it has been additionally observed that a suppressed immune system also inhibits the action of neoplasene compounds. Therefore it may be that the suppression of the immune system with anti-inflammatory drugs plays the predominant role in shutting down neoplasene compounds. Neoplasene compounds are preferential in attack and will respond specifically to residual neoplasm. The mechanism of preferential attack is theoretically connected to the character of the neoplastic cell membrane.

Many thousands of clinical applications have been accomplished as of June 2008. The several case histories presented herein are representative of these clinical cases. The theory set forth herein is based upon and consistent with the results obtained with these numerous clinical efforts.

Theory

By the author’s observation, neoplasm is widely viewed, by lay and professional persons alike, as a visible and palpable mass with structural organization and defined boundaries (i.e. a tumor) and there are but two broadly recognized conditions, namely a tumor is benign, therefore great relief is felt, or it is malignant and the death knell is sounded. Widespread occurrence of the disease throughout the patient is routinely attributed by the medical practitioner to metastasis, a spreading of the disease from the tumor by the circulatory system to distant locations in the body of the patient. This thought model is in general incomplete. Neoplasm may present as a tumor, a field of diseased cells, small

sets of cells or even isolated cells that escape notice; more often, all of these present simultaneously. It is theorized that there is a continuous process by which healthy normal tissue is invaded with unnoticed diseased cells. This neoplastic tissue may progress to become more and more diseased, aggressive and noticeable. Thus there are an infinite number of states of diseased neoplastic tissue. A benign tumor is diseased but just not as diseased and hostile as it may, and often does, become. Metastasis while a part of the progression of the disease is secondary to the independent development of neoplasm at many anatomical locations as a result of the ongoing debilitation of the normal apoptotic process. The apoptotic process is a major physiological function; gone awry much damage is accomplished.

In order to gain an appreciation of the scale of the apoptotic function it is assumed there are about 40 billion cells per pound of flesh each of which will divide by mitosis about fifty times, creating daughter cells numbering about 1,100 million, million cells before it dies itself. This being the case without an ongoing process of cell suicide by genetically controlled apoptosis, all creatures would, if enough food and space were available, become behemoth. This exponential growth is prevented by the rapid process of apoptosis which rids the individual of cells created that are not needed for growth and replacement, thereby balancing apoptosis and mitosis. Slowly, usually with advancing age, hormonal changes, exposure to external carcinogens, exposure to radiation, etc., widespread variable degeneration occurs in a small percentage of cells that modifies or in the limit eliminates the apoptotic gene giving rise to the build up of tissue that does not self destruct as it should. It may self destruct after a fashion or it may not, or it may exhibit a spectrum of abnormal apoptotic behavior throughout the individual. The debilitated tissue is disrespectful of cell type and includes cells of various anatomical origination. Lesions may ultimately develop as the first noticed evidence that neoplasm is present. Left untended the disease is progressively more debilitating and is commonly fatal.

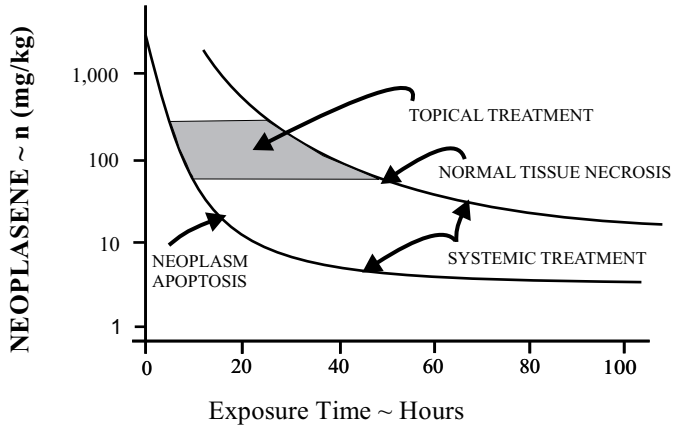
Therefore, a tumor that exhibits structure, with a discernable shape and boundaries is, by observation, in fact, usually surrounded by tissue that appears normal to inspection. This diseased tissue is sometimes judged pre-cancerous or questionable on histopathology and sometimes this diseased tissue escapes notice. The tissue frequently is diseased in that the apoptotic function is compromised to some degree, but not so far debilitated as to be readily recognized. Further multiple classifications of diseased tissue type are observed to be simultaneously present in patients with debilitated apoptosis. Therefore since the conclusions based on tissue analysis of a lesion are troublesome and there is little connection between the cell type of any particular lesion and the cell type of other present neoplasm, lesion or not, and the medical course of action is to eliminate the tumor, not study it, the value of biopsy is diminished. Biopsy, it is predicted, will ultimately become but an academic curiosity and a vestigial remain of the ongoing research into the character of cancer on the cellular level.

If multiple lesions present, it is generally assumed by client and practitioner that the disease has metastasized when the many lesions more likely developed independently due to degeneration of the apoptotic process. Further, the reappearance of a tumor after resection is frequently misthought of as regrowth when probably a new tumor has simply developed in proximity to the resection from diseased tissue left behind that at the time of surgery escaped detection. The practice of removing a margin around an excised tumor blindly removes a portion of this field of diseased tissue. The closer to the structured tumor the more actively diseased this field is typically observed to be. Resection followed by irradiation and chemical therapy is less than preferential and blind to residual neoplasm and collateral damage is done without elimination of all the diseased cells.

Whether tumors arise from connective tissue, melanocyte, fibroblast, basal cell, squamous, epithelial, mast cell, or any other anatomical origination site they do have in common that they are abnormal. They are, it is theorized, uniquely characterized by a cell membrane that is different from the normal cell membrane or the neoplastic cell membrane is different just because it is not fully developed. It is theorized that a single diseased cell or cells in a field of diseased tissue, just as the cells in a flagrantly malignant structured tumor, exhibits also this cell membrane difference. It is believed that at least the mix of polysaccharides in the diseased cell membrane is different than that in normal healthy cells which characteristic lends them vulnerable to preferential attack by neoplasene compounds because of this characteristic alone. This predicted universal characteristic of diseased cells is, in theory, the key to the highly preferential attack of neoplasene on neoplastic cells.

There are some chemicals other than the alkaloid(s) and their salts in neoplasene compounds that do attack neoplasm preferentially. The viscotoxins in mistletoe are observed to be feebly effective, curcumin and epigallocatechin are much better and similar to sanguinarine [reference 1 and the references therein]. Benzyl isoquinoline alkaloids and their salts (i.e. neoplasene compounds) are observed to be however very preferential. These alkaloids, including sanguinarine, [reference 1 and the references therein], clearly attack neoplasm preferentially. The cell membrane of neoplasm is permeable to neoplasene compounds. Healthy cells possess a cell membrane that is resistant to trespass and subsequent attack unless dosage or exposure times are high, then attack results in necrosis not apoptosis. Theoretically the active principles interact with DNA, trigger apoptosis, inhibit adenosine triphosphatase and inhibit nuclear transcription factor NF-κB. All this is similar to that which occurs with sanguinarine alone [reference 1 and the references therein]. These are theorized to be the key mechanisms of the affinity of neoplasene to trigger apoptosis. It is unknown if neoplasene's constituent alkaloid salts are linear or nonlinear in their apoptotic effect. It may be that the predominate neoplasene constituent is a single compound or a linear or non-linear mixture of compounds. Conclusive chemical analysis has not yet been accomplished. Much evaluation and research opportunity exists.

There is a functional relationship between the death of neoplastic cells and exposure to neoplasene. It is observed that the apoptosis and necrosis resulting in the death of both diseased and normal cells is directly proportional to the concentration of the active principles and the concentration required for mortality monotonically decreases as a function of exposure time to neoplasene, figure 1. By adjusting concentration and exposure time neoplasm is condemned and normal cells are spared.



APPROXIMATE RELATIONSHIP FOR APOPTOSIS AND NECROSIS

figure 1

General Theory of Treatment

The strategy is to eliminate, by preferentially stimulating apoptosis, the diseased tissue. The task in treatment is to simultaneously get enough of the neoplasene compounds in contact with the neoplasm to cause apoptosis faster than it is growing by mitosis and restrict the concentration and exposure time so that healthy tissue is not attacked. Further the dose needs to be low enough to allow the body to rid itself of the dead tissue as fast as it is produced and also to avoid anorexia.

There are situations where this is easy, others where it is difficult and still others where it is impossible. On the easy extreme are those situations where the tumor is sloughed or excised and the residual neoplastic tissue is eliminated with injection and/or long term low dose oral treatment. The difficult situations include aggressive inoperable growths where oral treatment is the only option. Anorexia may be avoided with careful adherence to oral protocol and large doses of metoclopramide or other antiemetics. Impossible situations present

when the diseased tissue is being created at such a rate that the required dose of neoplasene ensuring diseased cell destruction faster than diseased cell creation cannot be accomplished without anorexia and/or overloading the body's ability to eliminate necrotic neoplastic cells.

It is the unusual situation wherein the disease is limited to an observed lesion. The common situation encountered is that diseased cells with compromised ability to self destruct are widespread in the patient. They often involve multiple cell types (e.g. squamous cell, melanocyte, mast cell, etc.). Usually the presence of the disease is not apparent until a debilitating limp, lump, lesion or other signal of compromised function presents. Elimination of the lesion by resection or use of topical neoplasene or injectable neoplasenex does not necessarily resolve the problem. Diseased tissue usually remains and is continually produced because of a widespread compromised apoptotic cell destruction mechanism that gave rise to the diseased tissue that originally drew notice.

Smearing or injecting the margins after resection followed by long term low dose oral treatment is an effective treatment protocol. Oral treatment may continue for a year or more. After about a year of treatment the oral dose may be reduced or stopped on older patients. Once oral treatment stops it takes some significant measure of time for the redevelopment of widespread tissue with debilitated apoptotic genetics. Hopefully the patient, thusly treated, will succumb to old age by a more normal organic shut down prior to the resurgence of cancer. On younger animals the prophylactic use of neoplasene continually or at least periodically, for the remainder of the patient's life is predicted to be an effective protocol. On animals prone to develop cancers the prophylactic use of neoplasene orally is predicted to be a practical way to prevent the serious consequences of late life development of cancer.

Many cancers can be treated topically with adjunct long term oral treatment. Osteosarcomas, bladder carcinomas, lymphomas and other internal or widespread neoplasm may be treated orally, by injection, by infusion or by a combination of administration techniques. When neoplasm is attacked there is localized inflammation and soreness because the immune system which does not readily recognize neoplasm does recognize dead neoplastic cells and the body begins their elimination. Normal bodily processes that eliminate necrotic cells usually do not have trouble ridding the system of the dead cancer cells because the increase in load over normal bodily processing is comparably small due to the dose controlled low rate of add on apoptosis. If the cancer is large and aggressive and the rate of apoptosis high enough to eliminate neoplasm as fast as it is produced it may overwhelm the ability of macrophages to eliminate the necrotic tissue, in this situation debulking is indicated to allow a lower dose and resultant required detoxification at a realizable rate.

The oral dose is initially high and is followed by long term low level oral protocol. The active principles are emetic and have been observed to induce gastrointestinal distress leading to anorexia if the concentration is sufficiently high or the exposure time is too great. It has been found that, if the medicine is administered by mixing thoroughly with a wet cooked meal, dosages below eleven milligrams per kilogram are usually beneath the threshold of nausea leading to anorexia. General treatment protocols for a wide variety of situations have been developed and are available [reference 2].

Several case histories are presented to demonstrate this theory, the use of neoplasene and the developing protocols. Neoplasene salve has been in use topically since mid-2003 and oral, injectable and infusible forms have been used widely since early 2006. The methods employed to accomplish effective treatment are varied and limited only by practical realities and the creativity of the practitioner. The presence of neoplasm, while serious, is now not as grim a situation as it has been previous to the advent of neoplasene compounds.

References:

[1] Ahmad et al., Differential Antiproliferative and Apoptotic Response of Sanguinarine for Cancer Cells versus Normal Cells, Clinical Cancer Research, Vol. 6, pgs. 1524-1528, April 2000.

[2] Fox, T.S. et al., The Treatment of Neoplasm, Proud Flesh and Warts with Sanguinarine and Related Isoquinoline Alkaloids, accessed May 2008 at www.buckmountainbotanicals.net.

CASE HISTORIES

James H. Bailey, DVM: Associated Veterinary Service: Great Falls, MT

Patches a neutered feline, fourteen pounds, ten years old was referred on April 13, 2004, for non-healing lesion on and in the right nostril one centimeter in diameter. The lesion involved the right nares extending posterior into the nasal cavity with apparent bone involvement. The tissue was identified as a squamous cell carcinoma

On April 26, 2004, Patches was anesthetized and the area was clipped and cleansed. A layer of Neoplasene was applied to the entire area. Patches was maintained on Isoflorane for thirty minutes and then the Neoplasene was cleaned off. The tissue was gray and necrotic after this treatment. The lesion was reevaluated on April 27th. Wound Balm was applied to keep the lesion soft. One week later the lesion was retreated with Neoplasene under Isoflorane

anesthesia. On May 6, 2004, fluid drainage from the nostril with frequent sneezing occurred. Clinical exam revealed heavy scab and tissue separation in the middle of the mass. On May 13, 2004, the lesion was retreated. Neoplasene was applied to an area of normal looking healing tissue. On May 25, 2004, a normal healing lesion presented. Three weeks later there were two small areas of raw tissue in the depth of the lesion. It was covered with Neoplasene under Isoflorane anesthesia. Excessive salivation after recovery resolved in thirty minutes without treatment. On July 2, 2004 the raw areas were retreated with the Neoplasene without anesthesia.

In early August there was a fine line of raw tissue deep into the nares. It was retreated with Neoplasene under Isoflorane anesthesia. On September 13, 2004 no active lesion could be seen and Patches was acting normal. On October 10, 2004 some nasal discharge and excess sneezing occurred. No areas of visible tumor presented but a thin red line deep in the nares was treated with Neoplasene. After three hours the medication was wiped away. No area of reactivity could be seen. The Neoplasene acts as its own confirmation or denial of the presence of diseased tissue.

On November 18, 2004; March 10, 2005 and July 27, 2005 the tumor bed area appeared normal and no treatment was warranted. On August 23, 2005 bloody mucous discharge was reported. There was no active lesion. One week later Patches was normal. The cause for the bloody nose was not identified. On September 25, 2005, Patches was a normal, happy cat and the owner was very pleased.

Treatment was a protocol dictated by response to the treatment. General anesthesia was used because of the location of the lesion and the patient's ability to do damage with its front claws. At no time during or after the treatment did Patches appear to be uncomfortable or in pain. The response of the tumor to the Neoplasene was dramatic in that minimal tissue was destroyed which would have been impossible with a surgical approach. Normal tissue does not appear to be affected by the Neoplasene. This ability to spare normal tissue is a great asset.

4/26/04 10/11/04 3/10/05

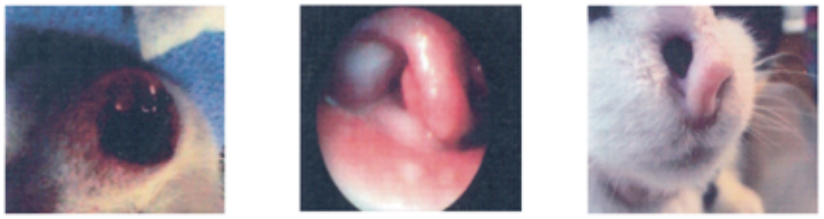


figure 2 Patches - Squamous Cell Carcinoma Photo Chronology

On January 25, 2007 Gingko, a seven year old, ninety-two pound neutered Rottweiler mix was diagnosed with a mast cell tumor involving the fifth digit of the right hind paw.

The lesion appeared to be confined to a red, raised dermal growth, approximately one centimeter in diameter. On January 26, 2007, Gingko was pre-anesthetized and maintained on Isoflourane anesthesia. The tumor was found to be more invasive than anticipated, encircling the bone and involving the majority of the tissue of the fifth digit. Amputation of the digit to the level of the distal third of the fifth metatarsal was performed.

Histopathology supported a morphological diagnosis of grade II mast cell tumor with the appearance of complete excision at the amputation border. After consultation with the School of Veterinary Medicine at Madison, Wisconsin (SVMM), Gingko was started on the Vinblastine/Prednisolone chemotherapy program.

On June 2, 2007, approximately six weeks after the last Vinblastine treatment, a small growth was found involving the same location as the surgery. A referral for cancer staging and radiation therapy was declined. On June 5th an aggressive debridement of the affected region was executed. Histopathology identified perivascular mast cell infiltrates consistent with a grade III Mast cell tumor.

CCNU therapy was started on the 19th of June. Despite two doses the growth was returning, prompting investigation of alternative treatments, which led to Neoplasene. Neoplasene was elected and on the 20th of August Gingko was sedated for NeoplaseneX infiltration. Five cubic centimeters of NeoplaseneX was diluted to ten cubic centimeters with lactated ringers and the entire dorsal and lateral aspect of the foot was injected with three-tenths cubic centimeters volumes of the diluted solution. Following recovery, Gingko was started on Tramadol for pain control. The following day, redness was noted throughout the treated area. Over the next two days, the tissue started to swell and ooze a serous fluid and twice daily applications of Wound Balm was started. Neoplasene 300 at one and six-tenths cubic centimeters twice daily was initiated on August 23rd. Two days later, multiple areas of the affected tissue began to slough and on August 31st, the tissue sloughing continued to the point of concern that the distal aspect of the foot may become devitalized. Cephalexin 1000 milligrams by mouth three times daily and foot soakings with Chlorhexidine was started due to evidence of a gram positive bacterial infection secondary to the tissue damage. Treatment continued throughout the next two weeks and on September 13, the wound was almost completely filled in with granulated tissue.

On March 27, 2008, Gingko was re-examined and there was no evidence of cancer recurrence at the surgical site. The foot healed nicely with excellent re-growth of the hair coat. Gingko is bright, alert and happy. There is no lameness or pain noted and Ginkgo continues to receive the oral Neoplasene at the normal after care dose.

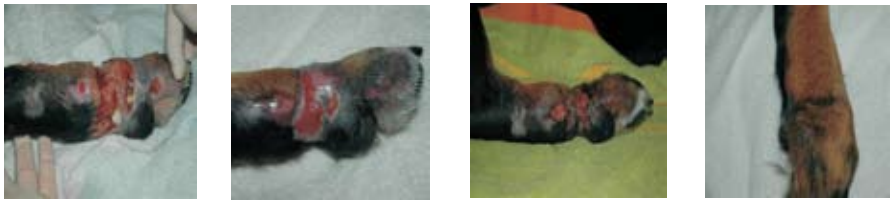


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figure 3 Gingko - Mast Cell Treatment Photo Chronology

Brandie, a ten year old, thirty-three pound, spayed Cocker Spaniel presented with a seven and one-half centimeter diameter raised, fluctuant, fluid filled mass involving the right side of the anterior chest. The owner noticed it two months prior to presentation but thought that it had the feel and appearance of a fatty tumor. Fine needle aspirate resulted in a determination of a mast cell tumor on December 4, 2006.

On December 15th, a wide excision of the mass and surrounding tissue was completed and histopathology report indicated a Grade II Mast cell tumor with the appearance of clean margins. After consultation with SVMM, Vinblastine/ Prednisolone chemotherapy program was started.

On June 15, 2007, approximately ten weeks after the last Vinblastine treatment, a mass similar to the previously removed tumor involving the same location was found. Surgical removal was elected. Follow-up Histopathology reported the presence of a possible Grade III Mast cell tumor. Two weeks after surgery, CCNU therapy was initiated, but after two doses, three weeks apart, the mass recurred with palpable soft tissue densities and fluid accumulation. Investigation into alternative therapies identified Neoplasene as an option and the owner elected to proceed.

On August 22, 2007, Brandie was pre-anesthetized with Hydromorphone and sedated with IV Telazol and the entire anterior chest region was infiltrated with three-tenths cubic centimeters volumes of a diluted solution of NeoplaseneX. Tramadol was started for post-treatment pain once Brandie was recovered from the anesthesia.

On August 25th, Brandie was started on six-tenths cubic centimeters Neoplasene 300 given twice daily. On August 31st, the infiltrated chest region started to drain a malodorous, cloudy fluid through several small necrotic areas and Brandie was started on 250 milligrams Cephalexin by mouth three times daily. The owner was instructed to keep the area clean with frequent warm soaks with an antiseptic shampoo.

On September 12th, Brandie was re-examined and the area was dry and clean with no evidence of drainage. On October 5th, Brandie was found to be completely healed with no evidence of recurrence of the cancer, although there were a few small areas of thickened skin associated with the drainage sites. On March 24, 2008, Brandie was re-examined and although still overweight, appeared to be alert and healthy. She has no evidence of recurrence of the cancer and the focal areas of thickened skin were resolved. Her owner continues to administer twice daily doses of Neoplasene oral aftercare.

Sarah L. Green, DVM: Veterinary Housecalls: Arcata, CA

Cedar, an eight year old neutered pit bull terrier mix presented on April 4, 2005 to evaluate a swelling on the dorsal aspect of his right metatarsals. A biopsy revealed soft tissue sarcoma three by four centimeters of fibroblastic origin. Treatment options include resection with wide margins followed by irradiation. Large tumors on distal limbs pose problems of closure. Where the tumor is locally invasive amputation may be offered as an alternative. The clients did not wish to pursue surgical approach or irradiation. Treatment with Neoplasene was offered.

A layer of Neoplasene approximately five millimeters thick was applied to the tumor surface. The treatment site was bandaged for ten hours. The bandages were removed and the area rinsed with hydrogen peroxide. Several areas of tissue necrosis were evident primarily surrounding the biopsy sites. The site was coated with Wound Balm and bandaged. The following morning an additional treatment was performed. By that evening a larger area of discoloration and necrosis was evident. The paw was mildly swollen. Three days later the dorsal and superficial layers of the tumor began to separate and large areas of tumor necrosis were evident.

Four days later the skin overlying the tumor mass had completely separated from the surrounding skin and an extensive area of necrosis was present at the center of the tumor. The tissue at the periphery of the mass appeared viable so an additional application was performed. Three days later the entire tumor mass appeared necrotic and partially separated. The following day the mass sloughed. The client cleansed the area daily with hydrogen peroxide, applied Wound Balm or a triple antibiotic ointment topically and bandaged the area.

Four days later the tissue appeared healthy. There was no evidence of pain or inflammation. Normal wound contracture was observed. At a six month follow up there was no evidence of tumor recurrence and hair re-growth had occurred over approximately three fourths of the original treatment area.

A follow up biopsy was not performed. The tumor was successfully removed without surgery or irradiation. This author (SLG) has subsequently used Neoplasene in the treatment of hemangiopericytoma, nerve sheath, squamous cell carcinomas, oral melanoma, and an apocrine cell carcinoma, as either the sole method of therapy or following surgical debulking of the tumor mass. These experiences and those of other practitioners indicate that Neoplasene differentiates between neoplastic and non-neoplastic tissue, causing rapid necrosis of neoplasm without harming the adjacent healthy tissue. The use of this compound is an effective alternative to surgery and irradiation in the removal of small to medium size cutaneous and subcutaneous neoplasms and a useful adjunct to surgery in the removal of larger masses. The potential benefits of a therapeutic agent that differentiates neoplastic from non-neoplastic tissue at the cellular level are enormous. These would include not only the successful eradication of neoplastic cells beyond the visible margins of a mass, but also elimination of the need to remove large amounts of healthy tissue to ensure 'clean margins'. In situations where this treatment is used as the sole therapeutic modality, anesthesia is generally not needed, with the possible exception of intraoral neoplasms, or those in very close proximity to the eye.

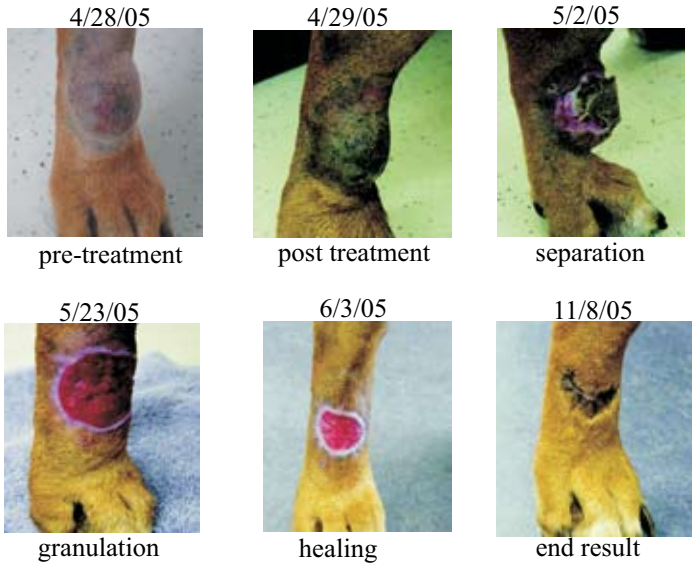


figure 4 Cedar - Soft Tissue Sarcoma Treatment Photo Chronology

This author (ABL) has treated many patients with Neoplasene. In all the cases the Neoplasene has been applied in a thin layer. In a few cases the salve has been reapplied in twenty-four to forty-eight hours.

Ali a seven year old spayed female yellow Labrador Retriever, presented on December 9, 2004 with a six centimeter spherical mass on her front left leg. The skin over the lump appeared normal. Twenty cubic centimeters of blood were removed with a syringe. A hard mass palpated in the lesion. Surgical removal was accomplished. Histopathology identified that the mass was a spindle cell tumor. The tumor returned aggressively and within seven days had regrown to nearly the size as it was at the time of surgical removal, figure 5.

Neoplasene was applied and the leg bandaged. At eighteen, twenty-five and thirty-seven hours the Neoplasene was reapplied. At sixty-four hours a large plug of necrotic tissue fell out of the treated area. The area in and around the tumor bed was inflamed and showed signs of healing at the end of seven days. A ridge of granulation tissue looked suspicious and was retreated. The ridge sloughed and the area continued to heal. After initial treatments which were bandaged for about twenty-four hours each, the patient was allowed to remove devitalized tissue with lingual abrasion. After fourteen months, the area that had tumor tissue appeared normal and was hair covered. As of January 2008 the patient was doing well.

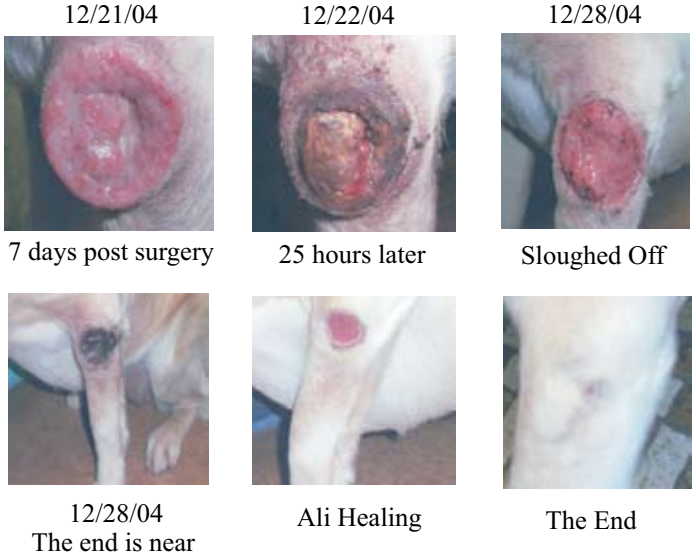
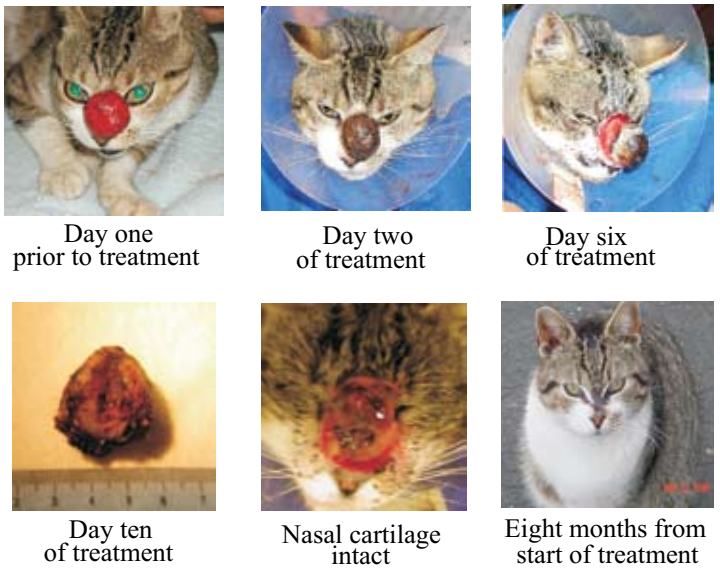


figure 5 Ali - Spindle Cell Carcinoma Treatment Photo Chronology

A feral cat with a tumor covering the bridge of its nose was presented. The duration of the condition was unknown. Histopath was not performed on this tumor however neurofibroma is presumed.

Neoplasene was applied on days one, two and four for twelve hours. After the twelve hours the salve was cleaned off with hydrogen peroxide and Wound Balm was applied. Because the cat was fractious, sedation was required for each treatment. The tumor was kept protected with Wound Balm. The tumor began to separate from the normal adjacent tissue by day six and fell off on day ten.

Once the tumor sloughed the wound was treated daily with Wound Balm without sedation for almost a week until the patient was too fractious to treat further and was discharged. Skin margins were migrating in at that time. Treatment at home did not continue because of his fractiousness, figure 6.



Rudolph - The Fractious Feral Feline Treatment Photo Chronology

figure 6

Beth D. Wittenberg, DVM: El Dorado Animal Clinic: El Dorado, KS

Maggie an eight year old, spayed female, Scottish terrier was diagnosed with transitional cell carcinoma of the bladder at Kansas State University on September 10, 2007. The patient was ultrasounded then and again on October 8, 2007 to find that the tumor was growing and also causing hydronephrosis of the left kidney. Treatment with Neoplasene 300 orally and NeoplaseneX with sterile saline bladder infusions were elected. The patient was placed on Neoplasene 300 orally at a dose of 125 milligrams twice daily and Astragalus extract orally at one-quarter milliliter twice daily. Maggie was also placed on an all natural, preservative and additive free food, and supplemented with fruits and vegetables. The patient was anesthetized each time for bladder infusion.

On October 9, 2007. Maggie was anesthetized and infused with fifty milliliters saline/one milliliter NeoplaseneX intrabladder.

On October 12, 2007. Maggie was anesthetized and infused with thirty-five milliliters saline/two milliliters NeoplaseneX intrabladder.

On October 15, 2007. Maggie was anesthetized and infused with thirty-five milliliters saline/five milliliters NeoplaseneX intrabladder.

On October 19, 2007. Maggie was anesthetized and infused with thirty-five milliliters saline/seven milliliters NeoplaseneX intrabladder.

On October 26, 2007. Maggie was anesthetized and infused with ten milliliters saline/three milliliters NeoplaseneX intrabladder.

Ultrasound on October 31, 2007 showed reduced transitional cell carcinoma mass. Ultrasound on November 7, 2007 showed further reduction of transitional cell carcinoma, narrow band of tumor tissue and no fingerlike projections had been previously noted.

On November 14, 2007. Maggie was anesthetized and infused with six milliliters saline/two milliliters NeoplaseneX intrabladder.

On November 21, 2007. Maggie was anesthetized and infused with six milliliters saline/two milliliters NeoplaseneX intrabladder.

An ultrasound on December 19, 2007 showed no evidence of a bladder tumor. Ultrasound on January 16, 2008 showed no evidence of a bladder tumor. Ultrasound was repeated on 4/1/08 and no evidence of a tumor was found.

Maggie will continue to be monitored using ultrasound and urinalysis. So far she is doing well, has a good energy level, and urine output. Seven months has elapsed since treatment began. The plan is to do bladder infusions as needed and to maintain her on Neoplasene 300 and Astragalus by mouth daily.

Laurinda K. Morris, DVM: Danville Veterinary Clinic: Danville, OH

This author (LKM) has been using the Neoplasene drugs for about two years.

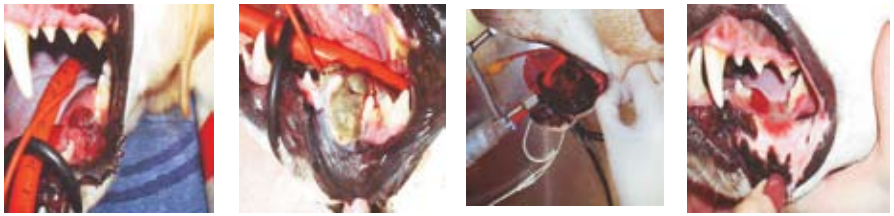
Zoey a rescued spayed Labrador retriever approximately seven years old presented 10/30/06 at seventy-nine pounds for what was assumed an abscessed tooth at the buccal aspect of #307. There was a two centimeter, ulcerated, firm, erythematous mass that was painful and bleeding. Tooth #307 had been displaced by the mass. A firm, bony mass extended lingually from #305-309. Radiographs were performed and lysis noted in the left mandible from the apex of the lower left canine #304 to #308. Histopath results were osteosarcoma.

Referral consultation resulted in recommendation of hemimandibulectomy, radiation and chemotherapy. Neoplasene treatment was elected. On 11/14/06 Zoey was presented and general anesthesia induced with Isoflurane, figure 7.a. Neoplasene salve was applied to the buccal mass for thirty minutes and then rinsed away with hydrogen peroxide and water, figure 7.b. Zoey was released with Tramadol and Chlorhexidine rinse. Neoplasene Oral 300 at a dose of six and one-half milligrams per kilogram by mouth three times daily was started. Some salivation and oral hemorrhage was observed. One week later a second topical treatment was administered. Indicators were normal. General anesthesia was induced and the mass evaluated, figure 7.c. It fell apart exposing the alveolar socket for #307. The displaced #307 was extracted. Salve was again applied for thirty minutes and then rinsed away. Zoey was sent home with astragalus extract at a dose of one and one-quarter milliliters three times daily.

Zoey presented 11/28/06 for a third treatment. Health indicators were normal and general anesthesia was induced. There was no buccal mass present. Neoplasene was applied to the lingual mass for thirty minutes. Prior to this third treatment there had been substantial hemorrhage, which resolved.

A fourth treatment was performed on 12/5/06. The oral Neoplasene dose was increased to eight milligrams per kilogram by mouth three times daily. She was rechecked on 12/12/06, figure 7. d., and appeared stable. Another recheck on 12/18/06 demonstrated no new visible tumor growth. Zoey's owner reported she was back to eating hard food. The Neoplasene Oral 300 dose increased to ten and one-half milligrams per kilogram by mouth three times daily. Famotidine forty milligrams was also dispensed at one-half tablet with each Neoplasene dose. A fifth Neoplasene salve treatment was performed on 1/22/07 under general anesthesia.

Zoey was rechecked on 2/5/07, 5/10/07 and 10/15/07. She is at this writing over a year and one-half post diagnosis, has a normal life, catches a Frisbee and is doing very well. She continues on the Neoplasene Oral 300 three times daily and astragalus two times daily. Her disease is controlled and she has an excellent quality of life.



a b c d

figure 7 Zoey - Osteosarcoma Photo Chronology

Jesse, a female brindle boxer, birth date 5/14/98 originally presented 7/6/05 for vaccinations and evaluation of a pink, depigmented area medial to the nares opening on the left side of her nose of approximately three month's duration. Biopsy revealed a well-differentiated mast cell tumor.

On 9/23/05 Jesse was accepted into a double blind study to evaluate a chemotherapy drug for mast cell tumors. She had a second tumor on her right shoulder. She began the program on 10/18/05. At the conclusion of the study it was learned that she received the drug, not the placebo. She now had four tumors, was inappetent and her weight had dropped from near eighty pounds to seventy pounds. The study ended in December, 2006.

She began Neoplasene therapy on 1/15/07. Two milliliters of NeoplaseneX injectable was diluted in two milliliters of lactated ringers solution and one-half milliliter was injected into eight sites around a two centimeter mast cell tumor on her left lateral thorax. This procedure was repeated in a mass on her left hind leg as well. She had immediate swelling and appeared uncomfortable. She was sent home to start Neoplasene Oral 300 three days later at a dose of nine and four-tenths milligrams per kilograms by mouth two times daily and one and one-quarter milliliters of astragalus by mouth two times daily on her food. If there were no signs of vomiting or anorexia, the owner was to increase the Neoplasene Oral dose to three times daily.

Jesse was rechecked on 1/24/07. The mass on her left side had ruptured and was draining serosanguinous fluid. A wrap was devised to absorb the exudates. Her weight had increased to seventy-one pounds. Her left hind leg was markedly

swollen. Her Neoplasene dose was increased to eleven and three-tenths milligrams per kilogram by mouth three times daily. Metoclopramide was also dispensed to give with each Neoplasene dose. She returned on 1/26/07 with further swelling of her left hind leg the leg was hot packed and drained twice. Copious amounts of serosanguinous fluid drained from the tumor area. She was sent home with a mild pressure bandage and instructions to continue hot packs. The leg returned to normal size and function within twenty-four hours.

Jesse has continued on the oral protocol ever since. Her weight as of 8/31/07 was eighty-seven and two-tenths pounds. She is active, happy and has excellent quality of life. She does not act her ten years.

2/23/08

Lepsilyte

Seizures of unknown origin are collectively referred to as epilepsy. Seizures are an all too common malady affecting animals. Phenobarbital and/or potassium bromide are routinely used. They are both toxic in therapeutic dose concentrations. Specifically they are hard on the liver.

Of special note is that the inhibition of gamma amino butric acid (GABA) induces seizure. The logic is that enhancement of GABA should inhibit the frequency and severity, of epileptic fits. It turns out to be so!

Those things that enhance GABA include (per 40 pounds):

- Lepsilyte (valerian & skullcap) 1 ml. s.i.d. to q.i.d. depending on severity
- Vitamin B_6 15 mg. t.i.d.
- Vitamin E_1 100 iu. s.i.d.
- Taurine 100 mg. t.i.d.
- Mg 75 mg. t.i.d.
- Mn 3 mg. t.i.d.
- Selenium 25 ug. s.i.d.
- Zn 10 mg. s.i.d.

It is recommend the Lepsilyte and the vitamins be administered. If seizures are controlled that is all that's needed. If seizures continue, at any level, add the remainder of the mineral supplements.

6/16/08

Hyperthyroid

Bugleweed extract effectively binds thyrotropin and Graves thyroid stimulating immunoglobulin (TSI) and precludes receptor activation. Lemon balm extract also does so to a lesser extent. The mechanism of action of the extracts of Lycopus (i.e. bugleweed) and Melissa (i.e. lemon balm) is debated. However that it is effective is well documented.

Levo-carnitine (L. carnitine) is a thyroid inhibitor.

Bugleweed used alone is usually effective on mild cases of hyperthyroid. The appropriate dose is 0.1 to 0.2 cc per 10 pounds weight, bid. For more severe conditions, we supply a product called Thyrolyte. It is Lycopus and Melissa extract with 350 mg/ml of L-carnitine per cc.

Results obtained since these products were introduced in mid-2007 indicate that a high level of success can be expected. For patients already on other drugs (e.g. Tapazole) we recommend phasing out present drugs and phasing in Bugleweed a/k/a Hyperthyroid Support, or Thyrolyte depending on the severity of the problem. Neither of these medications is hard on the liver or kidneys.

5/2/07

Parasite Dust for Animals

Nearly all insects are immediately disabled and then die after contact with Buck Mountain Parasite Dust (e.g. fleas, ticks, lice, flies, etc.). Fleas and their eggs, larvae and pupae, die quickly when they come in contact with this product. However, there have been instances where pet owners have expressed difficultly in controlling fleas.

If your pet has fleas you can expect that the pet's surroundings are also infested with fleas, flea eggs, larvae and pupae. Therefore a steady supply of fleas will reinfest your pet about as fast as you eliminate the pests on your dog or cat.

Fleas and their eggs, larvae and pupae are in the carpet, upholstered furniture, pet bedding, cracks and crevices of baseboards, the lawn and near everywhere. The best procedure to eliminate the fleas on your pets and reduce the flea supply is:

Procedure for Fleas

1. Apply the dust by running one hand against the fur and holding the parasite dust container in the other hand sprinkle lightly a little dust on the skin and hair. Then rub it in briskly and lightly. A little parasite dust goes a long way. One teaspoon full is plenty for a dog and way more than is needed for a cat. If you can see the dust when it's brushed in, you are using too much.

2. Dust from the tail to the head. Dust around the neck and around the rear of the pet as fleas will head either direction for moisture and will come in contact with the dust, become disabled and die. Dust all pets in your household.

3. Vacuum or launder the pets bedding and sprinkle lightly with Parasite Dust. Rub the dust in with your hand or brush. If you can see the dust when you are done you are using more than is necessary.

4. Vacuum any rug, carpet or upholstered furniture frequented by your pet. Sprinkle it with and brush in the Parasite Dust.

5. You should not need to repeat this procedure for a month or more. It is not necessary to repeat treatment unless you see further evidence of live fleas.

Other parasites such as lice or ticks are much easier to treat because they don't present a steady supply of replacement critters.

General Procedure

For animals infested with fleas, ticks, lice (arthropods) sprinkle a small amount from tail to head along the spine. Run the hand from rear to front, against the way the hair lies sprinkling a small amount on the skin as it becomes visible.

On horses do the same thing ¼ lateral on each side. Three tablespoons, one for each front to rear application is about right. If dust can be seen brush with the hair to work it down to the skin. If it can't be worked down to the skin too much dust has been used.

For flies the underside and legs are also treated by use of the cupped hand as an applicator. Horn flies (Siphona irritans) are continually drawn to the treated horse and a whole hatch will be wiped out in about three days. Even engorged ticks will fall away after 24 to 36 hours. The dust will decrease by 1,000 times the number of flies in the stable by sprinkling it here and there around windows, stalls, etc. For lice also spread the dust on all infested areas with your hand.

In work and living buildings a teaspoon full laid in a line the height, and width of a pencil on a sash or two and along a baseboard will eliminate flies, ants and beetles of all kinds. The only bug you will see post treatment will be dead, dying or it just came in. Azadirachtin degenerates in light. Thus in windows and on hairless skin it has to be reapplied every five to seven days or so. In the vegetable or rose garden, aphides, leaf hoppers, etc. are history as well.

It lasts a very long time unless it is washed away. In dryer climates treatment once each year for horses when they are louse and tick infested is enough for the season.

There are no known side effects but avoid a cloud that would be inhaled and keep it out of the eyes, nose and mouth. There is no reason to be overly concerned it just seems like good sense.

Active ingredients

Neem, Azadirachta indica, is very healing and is a vermifuge and pesticide. It is claimed also to be an insect repellent. We have not found it to be an effective repellent.

Chemistry

Neem contains a fixed oil (10%) of glycerides. The oil is bitter. It smells like garlic and is yellow. It also had about 2% principles, nimbidin, nimbin, and a lot of related triterpenes.

Azadirachtin is the most active constituent insecticide, however related chemicals also have insecticide and claimed repellent properties. Other insecticidal constituents include deactyl-azadirachtinol and salannin.

Pharmacology

Neem is used as a contraceptive, antidiabetic, anti-ulcer drug. A primary importance in veterinary medicine is as an insecticide. Azadirachtin is, the most potent insect antifeedant and ecdysis-inhibitory botanical compound known. Azadirachtin is also a potent inhibitor of insect cell replication. It works.

Azadirachtin has not, as yet, been synthesized. Therefore only natural products are on the market. It is effective in concentrations of only one part in 10 million, so it is claimed in the literature. Buck Mountain parasiticide is about 7,500 parts in 10 million azadirachtin. It is nonmutagenic and nontoxic to warm blooded animals, fish and birds.

Clinic use

The oil of neem is tough to work with because it is messy. Powdered neem herb is very effective if insects contact it. Therefore, a very fine powder is a building block of our ecto-parasiticide.

Over the years treating wounds on horses, it was observed that yarrow when applied as a vulnerary to fly covered wounds, resulted in the flies vacating the horse. Buck Mountain was unsuccessful in making a yarrow extract that repelled insects – only the powdered whole herb acts as a repellent and is an active ingredient in our product.

Lice, mites, ticks, etc. infestations result in dermatitis of one degree or another. Yarrow is very healing and is a repellent, albeit a mild one, to some insects. Diatom flour cuts the exoskeleton of many bugs – e.g. ants, beetles, etc. and they dehydrate and is an active principle in our product.

A combination of neem, yarrow and diatom flour makes a fine parasiticide for use on animals in the stable, home, office, clinic, and in the garden.

As always, we stand ready to discuss recommended procedure with any interested person.

Milk thistle – Silybum marianum

Milk thistle is deserving of wonder drug status, it enjoys a rich history dating back millennia. The Roman Pliny the Elder wrote of Milk Thistle's virtues. Culpepper – England's infamous herbalist set forth Milk thistles utility for liver and spleen health as well as its efficacy for jaundice.

Four primary flavonolignans, silybin (silibinin), silychristin (silichristin) silidianin, silihectin and others make up what is known collectively as silymarin. Silybin is the most active, of these chemicals, as to hepatoprotection and antioxidant effects. Several other lignans are also present. About 20% oil made up of oleic lipoic acids and other acids.

The drug silymarin is a mistake when it is standardized for oral administration. It is poorly soluble in water or HCl and poorly available from the gastrointestinal tract. Therefore standardized 80% silymarin is an expensive ineffective medicine that could best be given parenterally.

The seed of the plant contains up to 8% silymarin. The many chemicals in the seed prove to be the best delivery vehicle for silymarin. Buck Mountain does not try to isolate known or suspected primary pharmacologic constituents. Improving on millions of years of evolution is an illusion and ill conceived and ill achieved effort.

Pharmacology

Milk thistle's most important therapeutic efficacies are: cell regeneration, antioxidant and hepatoprotection against a vast array of toxins.

The mechanism of Milk thistle's favorable influence on the liver is not well understood. There have been numerous research papers published that leave us with our continued poor understanding. What is clear is that silymarin, and related plant chemicals, do demonstrate amazing influence on the liver, and its function. Silymarin (silybin) potentiates DNA and RNA production. This results in cell growth and liver repair through protein synthesis. As an antioxidant it is many times more potent than Vitamin E! Silymarin is a prooxidant free radical scavenger and it does encourage concentration of glutathione in cells -- a big plus 35% to 50% over normal. Glutathione (GSH) is required in detoxification. Just how these desirable outcomes are accomplished is a matter of conjecture in the literature. Silymarin also inhibits peroxidizing enzymes blocking peroxidases of fatty acids and membrane damage.

Clinical Use:

The seed of the Milk thistle is hard, a bit shiny and preserves its contents indefinitely. A large proportion will, and does, pass through the G.I. tract and is wasted if it is ingested whole. The ground encapsulated seed is adequately preserved convenient and efficient to administer.

The whole ground seed:

works better than 80% standardized silymarin products,
usually corrects liver enzyme imbalances,
protects the liver from harsh drugs,
protects the liver when the animal is exposed to toxic chemicals and
it is easy to administer.

Further

it boosts milk production and is a very effective cure for jaundice. It is a preventative treatment for glaucoma and cloudy eyes as in aging dogs (i.e. lenticular sclerosis).

It is a positive treatment for: hepatitis, gallstones, psoriasis, and cirrhosis.

Visit our website at:

www.buckmountainbotanicals.net

and

www.neoplasene.net

On the home page find files for:

- The Clinical Guide for use of Neoplasene
  - 96 pages -
- Recommended Procedures for Neoplasene Compounds
  A two page protocol for topical use of Neoplasene
- Other product information

For further information call us at (406) 232-1185

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