Lymphoma connotes a solid-tissue tumor composed of neoplastic lymphocytes in visceral organs, skin, or lymph nodes throughout the body (Antinoff). To date, lymphoma is the most common malignant neoplasia reported in the domestic ferret at 10-15% of all neoplastic presentations in the US and Europe, and lymphoma is the third most common neoplasia of ferrets, behind adrenocortical neoplasia and insulinoma. It is documented as a spontaneous neoplasia (Mayer, Quesenberry), however there have been reports of horizontal transmission via cell and cell-free inoculation (Erdman), which suggests that there may a viral etiology, however an agent has never been reported. There is one report of *Helicobacter mustelidae*-associated (MALT) gastric B-cell lymphoma (Erdman), and this syndrome appears to mimic gastric B cell lymphoma caused *H.pylori* in humans.

Ferret lymphoma can occur across a number of age groups and has no specific sex predilections. In the early literature describing the disease, ferret lymphoma was classified by age of onset and assigned distinct prognosis, i.e. the aggressive and quickly fatal juvenile onset lymphoma form and the adult chronic onset form. This generalized classification scheme has been since retracted due to new clinical reports that reveal there is no specific age and cell-type trend. Most resources characterize lymphoma by cell line, i.e. large cell, lymphoblastic lymphoma (T cell) or small cell, lymphocytic lymphoma (B cell). Finally, there are several studies that report disease based on location, which include but are not limited to multicentric lymphoma (Ferreira), cutaneous lymphoma (Xi, Rosenbaum), malignant B-cell lymphoma with Mott cell differentiation (Gupta), polyostotic lymphoma (Long), epitheliotrophic gastrointestinal T-cell lymphoma (Sinclair), focal thoracolumbar spinal cord lymphoma (Ingrao), myelo-osteolytic plasmacytic lymphoma in the femur (Eshar), and gastrointestinal lymphoma (Lee).

Due to the substantial variation in lymphoma classification in ferrets, there has been a call for an adoption of the standardized classification system for ferret (Mayer). Currently, most clinicians develop diagnostic plans to (1) stage, (2) grade, and when possible, (3) phenotype lymphoma in clinical patients. Until a universal classification scheme can be established for ferret lymphoma, most pathologists and oncologists characterize lymphoma based on the World Health Organization (WHO) staging system. Staging identifies the anatomic location of the neoplasia and the measure of dissemination throughout the body. A 5 level staging scheme (Table 1) has been adapted from Antinoff and Mayer. Cell morphology characterization, or grading, is also imperative when classifying lymphoma type and qualifies prognosis (Table 2). In clinical practice, large cell versus small cell, round versus irregular, and nuclear size are used to classify cell type and tumor behavior from tissue aspirates.

<table>
<thead>
<tr>
<th>Staging</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multicentric</td>
<td>Multiple Lnn, usually on both sides of diaphragm, may also involve liver, spleen, bone marrow, or other extranodal sites</td>
</tr>
<tr>
<td>Alimentary</td>
<td>Solitary mass within GI tract or mesenteric node, multiple masses with or without regional involvement of intra-abdominal node</td>
</tr>
<tr>
<td>Mediastinal</td>
<td>Mediastinal Lnn, not involving the thymus</td>
</tr>
<tr>
<td>Extranodal</td>
<td>Renal, CNS, Ocular, Cardiac</td>
</tr>
<tr>
<td>Cutaneous</td>
<td>Severe skin ulceration with or without nodular skin masses, usually seen along the ferret, sacral area, inguinal area, extremities</td>
</tr>
</tbody>
</table>
Table 2. Grading Cell Morphology for Ferret Lymphoma

<table>
<thead>
<tr>
<th>Nuclear Size (relative to [RBC] Size)</th>
<th>Small: ≤ 1 RBC</th>
<th>Medium: &gt;1 but &lt; 3 RBC</th>
<th>Large: ≥ 3 RBC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitotic Index</td>
<td>Low: &lt; 3</td>
<td>Intermediate: 3-8</td>
<td>High: &gt; 8</td>
</tr>
<tr>
<td>Other Descriptives</td>
<td>Nuclear morphology</td>
<td>Nucleoli</td>
<td>Round</td>
</tr>
</tbody>
</table>

Phenotyping provides vital prognostic information and a definitive diagnosis. If tissue can be acquired, immunohistochemical stains help define the cell line as B or T cell. CD3 is a T cell marker, and CD 79α is a B cell marker. As in feline and canine medicine, the prognosis is based on cell line characterization, which predicts the progression of the disease process and response to treatment. As flow cytometry becomes more widely available for ferrets (Music), this may also be used to help define cell lines based on blood sample acquisition alone.

Clinical signs, physical examination and diagnostics

There is no universal clinical presentation for ferret lymphoma, and in some cases ferrets are asymptomatic. Clinical signs are nonspecific, but can include lethargy, anorexia, weight loss, ataxia, weakness, diarrhea, dyspnea, and respiratory signs (Soran, Antinoff, Mayer). Gastrointestinal signs are common, however this should not be confused with chronic gastrointestinal disease. Mesenteric and peripheral lymphadenopathies can be present with other disease processes, this clinical finding is not pathognomonic for lymphoma, however, intra-abdominal lymphadenopathy occurs with multicentric lymphoma in ferrets.

The diagnostic approach for ferrets with suspected lymphoma should include a minimum database (CBC, biochemistry screen, urinalysis), diagnostic imaging (radiographs, ultrasound, CT or MRI), and cytology obtained from aspirates, however biopsy tissue is preferred. The most consistent CBC finding in ferrets with lymphoma is a nonregenerative anemia. (Ammerbach). Lymphocytosis and thrombocytopenia are rare. If a lymphocytosis is present, with total white blood cell counts exceeding 30,000, a true lymphocytic or lymphoblastic leukemia may be present (Mayer). Hyperglobulinemia has rarely been reported in ferrets with multiple myeloma, however a serum or urine protein electrophoresis may help characterize if a monoclonal gammopathy is present (Eshar). It is important to note that the presence of a hyperglobulinemia is also a hallmark of ferrets that suffer from Aleutian’s disease, which is a viral condition that also causes a hyperglobulinemia (Hess). In some cases, biochemical analysis reveal elevation in liver and renal enzymes if disease is causing organ function compromise, but these findings are not specific to lymphoma. One report of a hypercalcemia as a paraneoplastic syndrome of lymphoma has been reported (Fisher), however it is rare.

Cytology from organ or lymph node aspirates, as seen in humans, show poor correlation to definitive histologic diagnosis based on WHO classification, however it can help provide the bases for tissue sampling based on cytological features (Antinoff, Hehn). Cytologic hallmarks for lymphoma are a monomorphic population of lymphocytes and the absence of peripheral blood elements (Antinoff). Evaluation of these samples by experienced pathologists is imperative, as false positives due to misdiagnosis in ferrets with reactive lymph nodes can occur. Once study revealed that the lymph node cell distribution in normal ferrets includes 50-60% lymphocytes, 2-3% lymphoblasts and promylocytes, and 0-1% macrophages, plasma cells and nondegenerate neutrophils per 200 x field (Paul-Murphy). Biopsies are strongly recommended for a definitive diagnosis. Gastric biopsies have been described. Scapular and popliteal lymph node biopsies can be easily obtained with little surgical complication in ferrets. Patients that present with bone lesions should undergo bone marrow aspiration or careful bone biopsy to further characterize aggressive ostotic disease.

Diagnostic imaging can help aid in staging and collecting samples for evaluation. In a recent study evaluating the image findings in ferrets with lymphoma, Soran et al. concluded that the most common imaging finding was intra-abdominal lymphadenopathy and mild peritoneal effusion in ferrets with multicentric lymphoma, which is the most commonly reported in ferrets greater than 3 years of age. Characterization of lymph nodes on ultrasound revealed hypeochogenicity as the single most consistent abnormality, as lymph node sizes were within reported ranges. Malignant changes seen in cat and dogs, such as increased short to long axis length ratios rations, hyperechoic perinodal fat, nodal heterogenicity, irregular nodal contour and shapes, were not appreciated in ferret lymphoma cases (Soran). Splenic infiltration was noted but correlation to splenomegaly should not be assumed, as extramedullary hematopoiesis occurs...
in ferrets and this can confound assessment for the cause of splenomegaly. Extraneal infiltration was then characterized in the liver, kidneys, lungs, and in aggressive bone lesions.

Management
There are several modified chemotherapeutic protocols, which should be chosen based on consultation with a knowledgeable oncologist. There are 3 common protocols that have been adapted for use in ferrets with lymphoma, and BSA calculations have been validated for the species, which is $9.94 \times \text{(body weight)}^{0.75}$ (Jones 2015) with weight and $m^2$ charts available. The Tufts protocol provides an intravenous-free 12 week chemotherapy protocol that employs L-asparaginase, cyclophosphamide, cytabarbine, prednisolone, leukeran, and procarbazine, and methotrexate. The Gulf Coast chemotherapy protocol utilizes a 52 week protocol that employs L’asparaginase, prednisone, vincristine, and cyclophosphamide (Antinoff). Older protocols employ vincritsine, asparaginase, prednisone, doxorubicin, cyclophosphamide, and methotrexate. All physical protocols and dosages are available in the Ferret, Rabbit, and Rodents third edition chapter for Neoplasia in ferrets, (pp112-115). Copies available upon request. For localized cutaneous lymphoma, surgical excision of lesions may help improve quality of life, however recurrence is common without concurrent chemotherapy. Radiation therapy has been employed to reduce tumor size as rescue treatment for solitary mediastinal masses to relieve respiratory distress. While the tumors are very radio-sensitive, due to the ferret’s body conformation, limiting radiation exposure to other organs can be very challenging.

Adjunctive therapies include optimizing nutrition, screening for leukopenia (< 1,000) and neutropenia and providing systemic antibiotic therapy when indicated, and co-management of additional morbidities, which often include management of Helicobacter gastritis, and other neoplastic conditions (adrenocortical neoplasia, islet cell tumors). Avoid employing homeopathic therapies without consulting a knowledgeable specialist, as some treatments can and will cause harm. One such case has been proven in a clinical report in dogs, receiving bloodroot (Sanguinaria canadensis) treatments, and the agent has been found to cause dermal necrosis (Childress).

Prognosis
Survival times strongly correlate with cell type and dissemination. Staging also heavily influences survival estimates, as disseminated T cell lymphoma may result in shorter estimates. In one study evaluating the phenotype, treatment and survival of 29 ferrets with lymphoma, Ammerbach et al. concluded that the mean survival of ferrets not immediately euthanized was 5.0 months (T-cell lymphoma) and 8.4 months (B-cell lymphoma). Ferrets treated with chemotherapy survived an average of 4.3 months (T-cell lymphoma, n = 9) or 8.8 months (B-cell lymphoma, n = 4). Ferrets in this study were diagnosed with peripheral T-cell lymphoma (n = 17), anaplastic large T-cell lymphoma (n = 5), anaplastic large B-cell lymphoma (n = 4), diffuse large B-cell lymphoma (n = 1), and Hodgkin-like lymphoma (n = 2).

Conclusion
Ferret lymphoma is one of the most common neoplasias recognized in practice. Clinical staging, grading, and phenotyping can help optimize treatment approach and qualify prognosis. Several chemotherapy protocols have been adapted for use in ferrets to help with ease of administration and improve compliance, improve quality of life, and reduce drug-induced morbidity.

References

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Exotic Animal Owners 101: How to Increase Your Clientele
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According to the 2015-2016 American Pet Products Association Annual Survey (http://www.americanpetproducts.org/press_industrytrends.asp), there were a reported, 14.3 million birds, 9.3 million pet reptiles and 12.4 million pet small mammals in US households. The demand for advanced medical care for these species has grown, but the clinical comfort of practitioners also needs to grow to match this demand. The best way to know improve care for these patients is by first knowing their owners. Ultimately, to improve their care, you will need to shape the behaviors of the human that’s attached to the beloved pet. When you understand their attachments, their strengths, and their commitment, you gain the trust of those who need your services. Let’s take a peak into different pet owner personalities and outline ways to improve compliance for your patients.

Rabbit owners
In general, most rabbit owners share one common thread; they love their bunnies. Chinchilla owners can be very similar. There are 2 types of owners, the well-meaning and the super-informed. The type one owner loves their rabbit, likely has dated husbandry practices but has a huge heart and is open to change. The type 2 owner may almost seem obsessed with minute changes in the rabbits behavior, and you should grow to trust their observations. They know their bunnies and are keen observers. They can pick up on subtle changes and that’s worth its weight in gold. For both clients, give them homework. Provide House rabbit society homework for the Type 1 and “academic” level homework for the Type 2. I often direct Type 2s to anatomy and physiology papers to read- this qualifies that you have a thorough understanding of medical physiology and that guides your treatment practices. Incorporate the owners into the treatment plan. When they feel they are contributing to the solution and are working with you, their compliance can be phenomenal.

Ferret owners
Ferret owners are not too dissimilar from some rabbit owners, except that their long-term memory and attention spans can sometimes be faulty, just like a ferret! Type 1 owners are very well-meaning, but compliance can be poor. Type 2 owners will do WHATEVER is necessary to save their ferrets and are open to advanced surgical and medical treatment options. For both owners, I recommend talking to them directly WITHOUT the pet in the room. They can become very distracted and miss specifics of your clinical findings and recommendations. Make sure you have “Ferret” disease information sheets on hand, including “Diarrhea, Insulinoma, Adrenal Gland Disease Handouts” so that they have a tangible reference. When a ferret owner is distressed, all they care about is relief for their loved pets. Do not be surprised that you will have to repeat yourself. Also send them home with treatment charts to help them stay on track. Have reminder calls/cards go out frequently for these clients to help improve compliance.

Guinea pigs
Similar to Type 1 and Type 2 rabbit owners. There is a Type 3, the super-devoted and very attached adult owner. These owners are incredibly sympathetic to their pets needs and have C&C cages that occupy entire rooms for their pigs. Avoid making the mistake of assuming monetary value for human-animal bond value. These owners often seek advanced medical care for their pigs and have excellent husbandry and compliance. Have common disease hand-out sheets ready and give them homework!

Rats
Many cat and dog owners argue that their pet rats are the most intelligent and the superior companion of their pets. Usually owners that are seeking rat care are extremely invested in their pets’ well being. Many come in desperate for care. The common diseases that afflict rats can be fatal and their lifespans are so short. It’s always a bittersweet bond. Owners are often prepared for any salvage or supportive care measures to prolong life if possible. There are many advanced care and preventative care options you can afford these owners to prolong the life of these enriching pets. By providing a compassionate approach to their care, even compassionate euthanasia options, you will undoubtedly gain you a loyal rat owner clientele.

Bird owners
There are 3 major owner types, and there are mixes of some! The Type 1 owner is the well-meaning but misinformed. They perhaps adopted the bird from a bad situation or may be first time bird owners that impulse-buy, etc. It’s clear that they care but they need guidance to maximize the bird’s physical AND mental health. Type 2 owners tend to be informed and do not trust veterinarians. They have their reasons, some may have been mistreated, their pets not treated and they require a lot of reassurance. Kill them with kindness and be honest. They are like their pets, they will either choose to accept you or you can kindly offer the contact information
of colleagues who are avian practitioners. Type 3 owners I lovingly refer to as the “ornithologists”. They have a deep ethical commitment to their feathered companions and are keen to make sure you know that. This is fine. Work to reassure them with your honesty, not with irritation. Have websites, husbandry references, bird club information and medical hand-outs ready; this will reveal to the owner that you are well informed. Human-avian bonds have a range of medical, mental and emotional spectrums and consequences involved. Be prepared to have a trusted behaviorist on stand-by and be honest about what you are qualified to treat- the bird’s MEDICAL illness.

Reptile owners
If you thought gaining the trust of a reptile owner was difficult, you’re right, it can be. Historically, many practitioners have held common negative stereotypes about reptile owners. Poor husbandry means that the owners do not care about their pets. And worse, reptile owners often do not have the financial means to pay for veterinary care. You can kick all of these beliefs to the curb! Reptile owners have doubts about practitioners. They may not know who can provide clinical care and who has expertise to provide that care? The cards have changed. We practitioners have a diverse group of animals to serve and the owners expect more from us. The author has been each of these owners and hopes that practitioners can appreciate the plight that the owners that own these very unique pets.

First, we have the Herper Kid owner type. Young naturalists are usually very intelligent and motivated about their pets. Most importantly, you can influence them to get compliance from their parents. Make sure you have handouts and homework in hand. Talk to the child about their pets, get excited and you both can form a team to get mom and dad on board with getting what’s needed for the pet. Often, they are more attentive than adults to the very subtle changes reptiles display when ill.

The “Owner” by Default may also walk into your office. First time guilt-driven reptile purchases, adoptions, or even the parent of a child heading off to college! These owners have an attachment to the animal and an investment in their well-being. The may not know all there is to know about husbandry. Provide resources and a community for these owners, turn their fear into pride about their new acquisitions! The Executive Herper is the owner whose pet is a reflection of status. They take pride in ownership. They expect top of the line and expect you to offer it. Surprise them with how much you know and celebrate their love for their pet. The HardCore Herper or “herpetologist” is the owner whose been neglected by veterinarians for years and has taken most measures into their own hands for medical care. In this case, you will have to abandon your vet ego. They DO know more about the animal’s natural history than you do. They have been caring for herps without help for years. Befriend them, learn from them and instill trust in them. If they own a business, that trust will set you up to care for collections.

Zoo keepers and curators
These are the ultimate patient advocates. The relationships can be rewarding but are ultimately met with challenges. Keepers can view themselves as the animal’s owner and decline or refuse your treatment recommendations. The reasons for this can be variable, but usually involve changing their trust and interaction dynamic with the patient, which can be dangerous for the keeper and the animal. Curators have an extremely difficult job, they sometimes have to make a decision based on prognosis and animal value, and their decisions affect all animals in the zoo. Be patient and offer unique and collaborative ways to manage patient care. This means you will also have to become friends with compounding pharmacists who have experience with unique species.

Mistakes to avoid
It’s important to remember assigning your value or a monetary value to the animal does not reflect the human animal bond values. Owners form strong bonds- no matter the species, be prepared to offer them all options. It may also be wise to set up “husbandry consultation” appointments that are separate from “recheck/consult” or “wellness exam” appointments. Many exotic pet owners are excited to meet vets that love their animals as much as they do, and this can lead to lengthy appointment sessions. Provide the owners with many options but carefully outline them with “specific time allotments” so that they do not feel dismissed because of your clinic schedule.
Managing Head Trauma in Exotic Pets
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Philadelphia, PA

Traumatic brain injury results from some compressive and blunt force to the skull. Often the trauma has affected other parts of the body as well. When triaging an exotics patient, the initial goals are to stabilize the extracranial disease, then proceed to stabilize intracranial injury while critically monitoring neurological status. In animals that have sustained trauma, extracranial trauma may include chest injuries, abdominal injuries, airway obstruction, compromise of oxygenation via ventilation or primary lung injury, and intravascular volume compromise, secondary to hemorrhage. When the extra-cranial conditions have been stabilized, the next steps to triage intracranial injury include (1) optimizing oxygen delivery to the brain, (2) maintaining cerebral perfusion pressure or CPP, (3) treating increased intracranial pressure or ICP, and (4) consistent neurologic monitoring.

The pathophysiological consequences of head trauma are often broken into two categories. First, primary injury results from concussive forces to the skull physically disrupting intracranial structures, causing a brief loss of consciousness that results from parenchymal hemorrhage and edema. Contusions can result in losses of consciousness for longer periods of time. The most severe insults occur in cases of parenchymal laceration in small mammals, which result in axial and extra-axial hematomas that cause brain compression. These primary injuries can cause cerebral edema, increased CSF volume due to obstruction of flow by edema or clot formation, compromise of the blood-brain barrier, vasospasm, infection, and seizures, which can all cause neuronal death.

Secondary injuries result from a cascade of inflammatory processes and biochemical events that signal neuronal cell death. These biochemical events may result from systemic insults (hypotension, electrolyte imbalances, acid-base imbalances, hypoxemia, hyper and hypocapnia, hyper and hypoglycemia, hyperthermia) and/or intracranial insults. After impact, there is a substantial release of excitatory neurotransmitters that cause an influx of calcium and sodium into the neurons. The mechanisms of removal are overwhelmed, and this leads to intracellular damage and ATP depletion. Reactive oxygen species, nitrous oxide, and catecholamines also contribute to a continued biochemical insult to injured neurons.

The uninjured brain can tolerate a variance in mean arterial blood pressure (50-150 mmHg), yet maintain cerebral perfusion pressure through auto-regulatory means. When the auto-regulatory mechanism is lost due to brain injury, hypoperfusion and local tissue acidosis can occur. Cerebral blood flow correlates linearly to systemic blood pressure when the CPP goes above or below the aforementioned range. Cerebral perfusion pressure (CPP) is the difference between MAP and ICP. Therefore, slight increases or decreases in MAP, coupled with expected increases in ICP, significantly affect CPP. The CNS ischemic response, or Cushing’s reflex, may be an initial response to decreases in MAP, whereby there is a reflex increase in MAP and a decrease in heart rate. This is seen with life-threatening increases in ICP are present and merit aggressive treatment to reduce ICP immediately.

**Triage**
On presentation, vital signs should be assessed and when possible, completing a modified Glasgow Coma Score and Animal Triage Trauma Score can help qualify the animal’s prognosis during initial assessment. The MGCS can be found at http://bvns.net/wp-content/uploads/2016/09/Neurotransmitter-2.0-MGCS-final.pdf. The animal triage trauma score can be found at http://www.k9tecc.org/assets/Animal_Trauma_Triage_Score.pdf.

For exotics patients, including reptiles, the patients should initially receive oxygen support during the visual and physical examination. During triage, the attending clinician should aim to qualify the animal’s mentation, access HR, RR, perform a thorough evaluation of the skull and cranial nerve exam, obtain an estimate of systemic blood pressure, and obtain an estimate of hydration status. In addition to attending to any additional extra-cranial injuries that have occurred, obtaining a weight or weight estimation will be key. The patient should then be placed on heat and oxygen support as needed in preparation for stabilization procedures and diagnostics. When safe to collect, a biochemistry screen and acid-base evaluation will be imperative. The author often monitors urine chemistries for changes in USG, pH, ketones, blood and glucose, in lieu of repeated venipuncture attempts to follow trends and assess the patient’s metabolic and hydration status. Radiographs, CT, and MRI can be pursued when the patient is stable. More often than not, the authors require 24–48 hours of stabilization minimal before attempting imaging diagnostics. When permissible, CT is preferred for speed of image acquisition, reduction in handling (patient is put in a small induction chamber with oxygen and no anesthetic), and the image detail acquired across all species orders (patients as small as 100 grams).

**Mentation**
For exotic animal patients, a qualification of their mentation may be difficult, as several prey species are adept at hiding signs of illness. Owner report will be important in assessing their status, however if the patient is indifferent to your presence, has acute vision deficits, does not respond to handling, and does not engage in exploratory behaviors, you can begin to qualify their status as altered, obtunded, and in severe cases, moribund for unresponsive patients.
Skull exam & cranial nerves
Evaluation of the skull will be key in pre-emptive diagnosis of potential skull fractures. In avian and reptile species, the eyes often take the brunt of rostral concussive injuries. Structurally, the presence of scleral ossicles further supports their large orbit and the skull. Any ocular trauma in these species merits a good evaluation of the skull. In avian species, the presence of green bruising may be appreciated, and on the dorsal aspect of the head, the skull sutures can be distinctly seen through the skin, which can help rule out the presence of displaced frontal bone fractures. In certain lizard species, auricular hemorrhage can be noted upon visualization of the tympanum. Small herbivores often sustain ocular injury secondary to head trauma, however head position abnormalities appear to be a common finding. Seizure activity may also alter the mentation in the post-ictal phase. Owner report of the animal’s immediate mentation after injury and any report of seizure activity will be important to acquire prior to assessment. The cranial nerve examination for small mammals, avian, and reptile patients is relatively comparable with that of cats and dogs with few exceptions.

Evaluation of the pupil position and size will help the clinician qualify prognosis. Small mammal prognostic indications for lesion localization have been used for avian and reptile species with success. In birds and reptiles, a consensual PLR is not appreciated due to differences in optic nerve desiccation and the response to light is present and controlled, but not voluntary. These is due to the presence of skeletal muscle in the ciliary bodies, as compared to smooth muscle in mammals. In all cases, abnormal PLRs, the absence of PLRs, and abnormal pupil position and size all follow the similar prognostic indicators. It is important to note that the normal PLR response of small herbivores and prey species will likely appear slow in comparison to that of cats and dogs. In the author’s experience, anisocoria can occur several days after head trauma insults in birds, which may be less likely in small mammal medicine, provided additional injuries are not occurring after the primary insult.

*Pupil size, reactivity, and prognostic

<table>
<thead>
<tr>
<th>Pupil Position</th>
<th>Response to Light</th>
<th>Level of Lesion</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midposition</td>
<td>Normal</td>
<td>---</td>
<td>Good</td>
</tr>
<tr>
<td>Bilateral Miosis</td>
<td>Poor to none</td>
<td>Cannot localize</td>
<td>Variable</td>
</tr>
<tr>
<td>Unilateral Mydriasis</td>
<td>Poor to none</td>
<td>Cranial nerve III</td>
<td>Guarded to poor</td>
</tr>
<tr>
<td>Unilateral mydriasis with ventromedial strabismus</td>
<td>Poor to none</td>
<td>Midbrain</td>
<td>Guarded to poor</td>
</tr>
<tr>
<td>Midposition</td>
<td>None</td>
<td>Pons, medulla</td>
<td>Poor to grave</td>
</tr>
<tr>
<td>Bilateral Mydriasis</td>
<td>Poor to none</td>
<td>---</td>
<td>Poor to grave</td>
</tr>
</tbody>
</table>

*Silverstein, Small animal critical care medicine. Elsevier Health Sciences; 2014

Hydration & blood pressure
In avian species, a quick evaluation of the ulnar wing vein distension will help qualify hydration status and perfusion. Healthy avian patients can normalize losses of 30% total blood volume before showing signs of shock. Avian patients regenerate quickly, and PCVs can normalize within 24-48 hours. In reptiles, assessment of the periorbital fat pad resilience, mucous membranes, and skin fold assessment may help qualify hydration status. In snakes, distension of the palatine vessel can be appreciated, but should be avoided to prevent additional head injuries. In small mammals, auricular and/or femoral pulse evaluation, and conjunctival color evaluation can help qualify hydration and estimated blood pressure. In rabbits, a compensatory tachycardia may not be appreciated in response to severe hypotension, as vagal nerve innervation is found near the carotid bodies. In many cases, subjective assessments will be required as objective monitoring equipment may be limited due to the animal’s size and/or lack of validation for the species. Monitoring for subjective trends, in conjunction with HR, will be labor intensive yet pivotal in enabling the clinician to respond to the patient’s status.

Treatment
After qualifying the severity of neurologic compromise, the treatment regimen mirrors those steps taken in small animal medicine. Extra-cranial therapy will include oxygen support, maintaining airway patency, heat support, reduction of environmental stressors (loud noises, bright lights, predators), and temporary stabilization of other wounds. Airway patency in small herbivores will be contingent upon reduction of compromise to the nasal passages, as many are obligate nasal breathers. In avian and snake patients, air sac cannulation may be warranted if there is obstructive upper airway disease. In small mammals, pleural effusions may need to be addressed to help reduce hyperventilation and thoracoocentesis has been described. When possible, during initial stabilization, the animals head should be elevated to help reduce cerebral blood volume, increasing venous drainage and safely decreasing ICP. This can be achieved with using towels arranged in a cage that would mirror a slant board for cats and dogs, which are used to provide a 30-degree slant. When the patient’s head is positioned, it is important to make sure that there is no compression or kinking of the neck or jugular vessels, as this could prevent drainage from the head and increase ICP.

Fluid therapy, electrolyte monitoring, oncotic support, and intravascular therapies aimed to reduce increased ICP will be essential for intracranial therapy. Exotic animal practitioners are faced with the challenge of providing these options without causing injury of exacerbating existent disease. In most all exotic species, vascular access will be necessary to provide emergency access for fluid
therapy, oncotic support, transfusion therapy, and treatment for increased ICP. Reviews for intravenous and intraosseous catheter placement are available.

Species specific crystalloid and colloid fluid rates are variable, however some general guidelines for dosages are listed below. Colloid support is used as that same rate across species. Hetastarch is provided at 5 ml/kg slow bolus over 15 minutes, not to exceed 20 mls/kg/day.

- **Avian Patients:**
  - Crystalloids: 50 ml/kg/day SC, IV, IO,
- **Reptiles:**
  - Normosol, Plasmalyte: 15-35 ml/kg/day depending on species water requirements
- **Small Mammals Crystalloid fluids:**
  - Rabbits, chinchillas, guinea pigs 100 ml/kg/day
  - Ferrets, rats, mice, sugar gliders, hamsters, hedgehogs: 60 mg/kg/day
  - Degus and gerbils: 50 ml/kg/day

The use of hypertonic saline or mannitol may be indicated in cases of apparent ICP. The author has used the following doses with guidance from a criticalist, in birds, reptiles and small mammals with success. Mannitol can be given, to help reduce ICP and improve CBP and increase oxygen delivery to the brain via osmotic reduction of brain edema. The author has had success with 25% mannitol, administered 0.5 g/kg over 15 minutes in mammals and 0.25-0.5 g/kg IV/IO over 15 minutes in birds. In cats and dogs, the range is 0.5 -1.5g/kg IV over 15 minutes. At high dosages, mannitol can cause a severe hypotension and has induced acute renal failure in humans. Additionally, it has been shown to leak into the brain parenchyma during prolonged use, and this can exacerbate edema. Mannitol must be followed with crystalloid and/or colloid therapy to maintain intravascular volume. Hypertonic saline is often used as a safer alternative to mannitol. As sodium does not freely cross the blood-brain barrier, it provides the same osmotic effect to reduce brain edema and provides intravascular volume without causing hypotension. The author has had success with 7.5% NaCl at a dose of 4 ml/kg or 3% NaCl at a dose of 5.4 ml/kg over 15-20 min IV or IO in birds and mammals.

In addition to fluid therapy and treatment of ICP, maintaining adequate glucose support will be necessary for exotics patients, as most have high metabolic demands. Dextrose supplementation (2.5-5%) in the crystalloid fluids may be necessary until the patient can recover enough to regain hunger. The use of anti-inflammatories may be considered provided the patient’s bloodwork supports’ safe usage. The author uses doxycycline as an alternative to NSAIDs in compromised patients. Steroids are contraindicated and have been shown to be associated with worse clinical outcomes in clinical trials. Furosemide is reserved for cases were pulmonary edema and/or oligoanuric renal failure are present. Furosemide is not used to reduced cerebral edema as a sole agent because it can cause a decrease in intravascular volume and subsequently decrease CPP. Seizure management will become vital in reducing continued hypoxemic threat during recovery. The use of phenobarbital as primary method may be species dependent and should be reserved for cases that are refractory to other anti-epileptics. Midazolam and keppra are commonly used to treat status and keppra is used as a first-line preventative agent. Doses for small mammals have been extrapolated from small animal medicine, which includes 60 mg/kg to treat status and 20 mg/kg q8 hrs for maintenance. Studies in birds reveal that therapeutic doses are achieved at 100 mg/kg q12h.

Pain management can be variable for different species. However, the authors often provide tramadol for avian (15-30 mg/kg q8) and reptile (5-10 mg/kg q24-48) patients. Gabapentin is also provided small mammal (5 mg/kg q8), and avian patients (15 mg/kg q8). The addition of gabapentin may also add in seizure management. In efforts to reduce nausea, Cerenia 1 mg/kg IV/SQ q 24 has been used in small mammal, avian, and reptile patients. Nutritional support is always provided; however syringe feeding is often implemented if need when the patients clinical signs are improving, which may take 24-48 hours.

**Long term consequences of traumatic brain injury**

If the patient shows improvements in blood pressure stabilization, hydration status, mentation, and oxygenation, the prognosis is good. If declines in neurologic status, systemic blood pressure, or additional injuries occur, this is associated with a poor to grave prognosis. On average, the author asks the owner to commit to a minimum 3-4 days of hospitalization, and more often it requires 7-10 days before the patient is released. Vision defects and/or persistent changes in mentation may occur after initial injury. Certain changes may be permanent. Seizure management may need to be maintained, as seizures may occur and/or recur weeks to months after initial injury. Young animals appear to have a better prognosis in recovering from severe traumatic brain injuries.

**References**


In herptile species, clinical signs of nutritional disease are like those seen in mammals. Calcium and phosphorus imbalances, diseases associated with hypercholesterolemia and obesity, vitamin D3 deficiencies, and vitamin A deficiencies remain common nutritional disorders in captive reptiles and amphibians. Many herptile caregivers have minimal information about the basic nutrition content of invertebrates or how to optimize it prior to feeding herptiles. This review will cover basic herptile nutrition, invertebrate diet preparation, and common nutritional diseases. To help make nutrition content easier to present to caregivers, invertebrate nutritional information is compared to common human foods. Additional information about insect composition and nutritive value can be found in the attached article (Updates on Amphibian Nutrition and Insect Nutritive Value).

What we know
Invertebrate nutrient composition has been formally studied for more than 50 years. Research on invertebrates for reptile and amphibian (herptile) nutrition is most extensive for the following invertebrates:

- Domestic cricket (*Acheta domesticus*)
- Earthworm (*Lumbricus terrestris*)
- Silkworm (*Bombyx mori*)
- Mealworm (*Tenebrio molitor*)
- Soldier fly larvae (*Hermetia illucens*)
- Superworm (*Zoophobas morio*)
- Madagascar hissing cockroach (*Gromphadorhina portentosa*)
- Butterworm or tebo worm, (*Chilecomadia moorei*)
- Turkistan or red rusty cockroach (*Blatta Lateralis*)
- Fruit fly (*Drosophila melanogaster*)

Although not formerly studied, enthusiasts also use the following as invertebrate feeders:

- Dubia cockroach, (*Blaptica dubia*),
- Hornworm (*Manduca quinquemaculata*),
- Pill bugs (*Armadillidium vulgare*)
- Springtails (*Collembola sp.*)

What we don’t know
The National Research Council (NRC) provides recommended minimum nutrient requirements for domestic and farmed animals based on comprehensive reviews of nutrition studies. No such database exists for herptile species and nutritionists often utilize the minimum requirements of laboratory rats or carnivores for insectivorous herptiles. The following is a review of major nutrient components and compares invertebrate composition to recommendations for rats, consistent with much of the herptile insectivore literature. This summary is based on the Nutrient Requirements of Laboratory Rats (http://www.nap.edu/openbook.php?record_id=4758&page=R1), unless otherwise stated.

Energy needs, fat, protein
Metabolizable energy is defined as the amount of net energy gained from food less the energy for digestion and absorption of the meal. This is influenced by species, age, activity, and environmental temperature. ME is measured as kilocalories per kilogram (kcal/kg). Invertebrate feeders typically range from 0.7 - 2.7 kcal ME/g, below rat minimal maintenance energy of 114 kcal ME/BWkg^{0.75} per day or 3.6 kcal ME/g.

The diet protein content reflects the energy concentration based on amino acid composition and availability. For rats fed a natural-ingredient diet, a minimum of 50 g/kg protein content is required. [The crude protein amount of larval insects may exceed the minimum requirement for rats by 2 - 4 times. The digestibility and bioavailability of insect protein is poorly researched, even in well-studied species. In one study, phoenix worms were passed undigested by mountain chicken frogs (*Leptodactylus fallax*), as the exoskeleton prevented digestion. When the larvae were macerated, the bioavailability improved. In addition, chitin in the exoskeleton and cuticle is not bioavailable to herptiles and amino acids in these structures vary in bioavailability.]

Fat (lipid content) provides a concentrated energy source, aids in fat-soluble vitamin absorption, provides essential fatty acids, and usually increases diet accessibility or palatability. The optimal lipid requirement for growing rats is 5% or 50 g/kg of diet fed, which also assures adequate vitamin A absorption. The total fat content of many larval insects is 3 - 6 times the optimal lipid requirement for rats.
Vitamins & minerals

Reptilian and amphibian vitamin and mineral requirements and absorption physiology may be considerably different than mammals.

Active forms of vitamin A include retinol, β-carotene, and retinyl esters. The absorption of Vitamin A, as a fat-soluble vitamin, corresponds with an adequate lipid content in the diet. Hypovitaminosis A is a common clinical problem in herptiles and can result in vision loss, epithelial hyperplasia, squamous metaplasia and keratinization of mucosal epithelium, growth failure, dermal ulcerations, and bone defects. In captive anurans, an inability to use the tongue effectively for prey apprehension, called “short tongue syndrome” is recognized. The minimum retinol requirement for rats is 2,300 IU/kg. Most invertebrate species contain less than 300 ug/kg or 1,000 IU/kg of retinol. Silkworms have the highest content at 1580 IU/kg.

Dietary supplementation has been reported by administering 0.1 mg liver from frozen food rodents (20 ug liver = 66 IU vitamin A) PO once a week to treat periorbital squamous metaplasia secondary to hypovitaminosis A in a Tiger salamander (Ambystoma tigrinum). Topical absorption studies have been performed in yellow and blue poison arrow frog, (Dendrobates tinctorius), New Guinea tree frog (Litoria infrafrenata), African foam nesting frog (Chiromantis xeramplina), and Puerto Rican crested toad (Peltophryne lemur). Collective results suggest that topical application of water miscible vitamin A palmitate (Aquasol A® parenteral) at 50 - 100 USP for frogs less than 20 gram and 100 - 150 USP for frogs greater than 20 grams resulted in resolution of short tongue syndrome and dermal ulcerations and, in the New Guinea tree frogs, significant increases in serum vitamin A levels. Parental supplementation must be provided with care, as iatrogenic hypervitaminosis A can occur and has been reported with the use of 10,000 IU/kg intramuscularly.

Vitamin E is often described as the body’s antioxidant vitamin. In rats, 42 umol/kg of α tocopherol is required in diets containing less than 10% fat. This is approximately 18 mg/kg (27 IU/kg diet). Surprisingly, some invertebrate feeders meet this requirement; adult houseflies (29.7 mg/kg), adult crickets (19.7 mg/kg), waxworms (13.3 mg/kg), and butterworms (13.0 mg/kg).

Calcium absorption relies on several factors, including UVB supplementation, dietary vitamin D3 levels, oral calcium availability, and health status of the gastrointestinal tract, kidneys, integument, and musculoskeletal system. Endocrine regulation of calcium in herptile species shares similarities with mammalian species. However, the nutrient sensitivity profiles differ based on the mode of vitamin D3 acquisition. The photobiosynthesis of provitamin D3 (7-dehydrocholesterol) to previtamin D3 requires UVB supplementation in diurnal reptiles. The herbivorous green iguana has historically been recognized as the poster-child for metabolic bone disorders secondary to UVB deficiency. However, some insectivorous species are at risk as well.

While some species absolutely require UVB supplementation for vitamin D3 production, others may not. In crocodilians and snakes, many authors suggest that the “nutritional completeness” of the vertebrate, whole prey diet has altered or decreased the photobiosynthetic demand for vitamin D3. Interestingly, increases in serum cholecalciferol levels did occur when diurnal corn snakes (Elaphe guttata) were exposed to UVB light (Acierno 2008). However, the study did not determine if UVB was required for adequate calcium absorption in the species. Nocturnal species are often described as not needing UVB exposure. However, one study of the crepuscular house gecko (Hemidactylus larcicus) determined that specialized integument provided increased ability to absorb UVB at dusk compared to diurnal species. A similar study has shown comparable results in a shade-tolerant species of Jamaican anole (Anolis lineotopus merope). It is therefore difficult to generalize regarding standards for UVB requirements in crepuscular and nocturnal insectivores.

In rat diets, vitamin D3 levels of 25 ug/kg (1,000 IU/kg diet) are required to prevent disruption in calcium homeostasis. Severe calcium deficiencies can lead to a host of disorders (e.g., growth retardation, pathological fractures, osteopenia, tetany, post-ovulatory stasis, seizures). In many invertebrate, levels under 25 IU/kg are reported.

There are few studies of dietary and/or photobiosynthetic vitamin D3 needs of insectivores. Providing UVB exposure that is comparable to natural environment levels is often recommended, as dietary supplementation alone can be problematic. Dietary supplementation is typically provided via dusting feeder insects with a vitamin D3 powder. This is imprecise and the actual amount of supplement ingested is difficult to quantitate. For example, insects may groom off powder within minutes and the amounts of vitamin D3 in marketed reptile products can vary widely from reported product amounts. Vitamin D3 toxicity is reported and over-supplementation also needs to be avoided.

Calcium absorption relies on several factors including vitamin D3 levels, oral calcium availability, and is dependent on the health status of the gastrointestinal tract, kidneys, integument and musculoskeletal system. Calcium requirements vary with age and life-stage. For instance, reproductive activity and egg development in female reptiles will increase calcium utilization. The largest calcium reserve is the skeleton and nutritional secondary hyperparathyroidism is a major concern if nutritional deficiencies exist. Some herptile species, like Rhacodactylid geckos and some amphibians, store calcium in specialized lymphatic sacs as well.

The minimum calcium requirements for several herptile species have been extrapolated based on the clinical onset of diseases associated with hypocalcemia. In rats, 3.5 - 5 g/kg calcium is required for maintenance. Most insects do not have calcium contents that come close to this requirement. Two insect species are exceptions to this rule. The phoenix worm naturally contains 9.3 g/kg without dietary supplementation and the wood louse (Porcellio scaber) contains 14% calcium on a dry matter basis and has an 11.79 Ca:P
ratio. Rat diets easily meet minimum phosphorus requirement of 3g/kg diet. In most invertebrates, this minimal requirement is met or exceeded and values range from 1.5 - 3.7 g/kg. Many prey items have inverse calcium to phosphorus ratios and are unable to meet herptile calcium needs without calcium supplementation.

**How to prepare your invert feeder for consumption**
Optimizing the herptile’s nutrition begins first with appropriate invertebrate prey choice and by optimizing the prey’s nutrition. Most invertebrates are shipped, stored at low temperatures to prevent molting, and sold without water. Travel substrates are generally not designed for optimal nutrition, apart from silkworms that will only eat mulberry leaves and come in an appropriate substrate. Generally, invertebrate prey should be given at least a few days to eat and rehydrated after shipment or purchase from a pet store. In some cases, establishing breeding colonies of prey items may be warranted. Feeding healthy prey items that have been fed appropriate diets, will help ensure appropriate nutrition for insectivorous herptile species.

Altering the nutritive quality through external supplements (“dusting”), can be difficult to reliably achieve. Powders with calcium and vitamin D₃ are commonly used as a dusting agent prior to consumption. Some supplements come with other vitamins or micronutrients as well. Although helpful, it is difficult to accurately measure the dosing effects, as mineral attachment can vary based on insect grooming behavior and particle size. It is important that the prey item is consumed quickly to maximize supplement ingestion.

Altering the insect’s internal nutritive quality is a more reliable way to improve the prey’s nutritional content. Gut-loading refers to act of providing invertebrate prey with a nutrient (e.g., calcium) dense diet for a specific time interval before the prey is fed to the intended insectivore. Several studies have shown a linear correlation between the intestinal contents of certain invertebrate prey and the feeding substrate provided. If the prey item is not ingested in a timely manner, the ingesta will pass from the insect and reduce the effectiveness of this supplementation method.

For most larval insects, you can feed them a diet that contains at least 9% calcium for 24 hours prior to feeding the insect to the reptile. Levels exceeding 40.7% calcium have been associated with dietary avoidance in crickets. The author has examined the calcium and phosphorus content of mealworm and superworm larvae given wheat millings, avian starter diet, high calcium cricket feed, and organic avian seed mash. The calcium content of mealworm and superworm larvae rose to calcium to phosphorus ratios that were 1.31:1 and 1.47:1 respectively; these nearly meet the NRC recommendation for growing rats (1.66:1).

**Sources for insectivore diet guidelines**
Herpetological journals, nutritional journals, comparative endocrinology and physiology journals, and Zoo Biology offer frequent articles that overview species-specific husbandry and diet information for captive insectivores. Online forums (e.g., www.herpconbio.org hosted by the Herpetological Conservation and Biology Society, HerpDigest) can also provide enthusiasts with free access to articles. Consulting with local herpetologists and reptile collection curators may provide access to diet guidelines for unique species. The Nutrition Advisory Group (NAG), a group of veterinary nutritionists who specialize in zoo nutrition, provides free access to conference proceedings and information on the nutrient composition of several prey items at http://www.nagonline.net/. NAG serves as the scientific nutrition advisory group for Association of Zoos and Aquariums (AZA).

**Comparative nutrient content of common invertebrate feeders**
Most commercially reared insects are larval worms of beetles or moths that are high in protein, low in calcium, and high in phosphorus. In efforts to improve their nutritive profile, we recommend gut-loading the larval species with a diet high in calcium and Vitamin A prior to serving it to insectivorous reptiles. Earthworms, silkworms, pill bugs, and katydids have a good nutritional profile and can be used as staple bug options for carnivorous species. See the table below for fun comparisons.

<table>
<thead>
<tr>
<th>Table 1.</th>
<th>Insect is known For</th>
<th>Human Food Comparison</th>
<th>Staple or Treat?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Domestic Cricket</td>
<td>Low protein, high fat (22.8%)</td>
<td>French Fries</td>
<td>Staple if gut loaded, otherwise a treat</td>
</tr>
<tr>
<td>Roaches</td>
<td>Lean protein with variable fat (14 - 50%)</td>
<td>Hamburger to Steaks</td>
<td>Staple</td>
</tr>
<tr>
<td>Silkworms</td>
<td>Some vitamin A, lean protein source</td>
<td>Turkey</td>
<td>Staple</td>
</tr>
<tr>
<td>Earthworms</td>
<td>Protein, good micronutrient content</td>
<td>Big Mac® + multi-vitamin</td>
<td>Staple</td>
</tr>
<tr>
<td>Phoenix Worms</td>
<td>Calcium content high</td>
<td>Tums® tablet</td>
<td>Staple, macerate prior to feeding</td>
</tr>
<tr>
<td>Wood Louse</td>
<td>Calcium (14.38%)</td>
<td>Tofu</td>
<td>Staple</td>
</tr>
<tr>
<td>False Katydid</td>
<td>Vitamin A (retinal esters) &amp; lean protein</td>
<td>Salmon</td>
<td>Staple</td>
</tr>
<tr>
<td>---------------</td>
<td>---------------------------------</td>
<td>--------</td>
<td>-------</td>
</tr>
<tr>
<td>Butterworms</td>
<td>High ME and fat content (29% DM)</td>
<td>2 Hotcakes with sausage (25%)</td>
<td>Treat, recovery diet</td>
</tr>
<tr>
<td>Mealworm larvae</td>
<td>Fat (31%)</td>
<td>2 McDouble Hamburgers ®</td>
<td>Treats</td>
</tr>
<tr>
<td>Mealworm beetles</td>
<td>Fat (17.7%)</td>
<td></td>
<td>Staple</td>
</tr>
<tr>
<td>Superworm Larvae</td>
<td>Fat (40.8%)</td>
<td>Two Sausage Egg &amp; Cheese Biscuits (46%)</td>
<td>Treats</td>
</tr>
<tr>
<td>Superworm beetles</td>
<td>Fat (14.3%), Protein (68.1%)</td>
<td></td>
<td>Staple</td>
</tr>
<tr>
<td>Waxworm larvae</td>
<td>More fat (51.4%), can try to gut load with calcium</td>
<td>Butter (53% fat)</td>
<td>Treat, recovery diet</td>
</tr>
<tr>
<td>Freeze-Dried or Canned</td>
<td>Variable moisture and high in fat</td>
<td>Doritos® Slim Jim®</td>
<td>Treat, use sparingly</td>
</tr>
</tbody>
</table>

**Take home points**

1. Larval insects are usually HIGH in fat, poor in vitamin and minerals, and have variable bioavailable protein content.
2. Adult beetles, earthworms, and roaches tend to have good protein levels and are low in fat.
3. Most insects have poor calcium levels; calcium content of the insect can be increased by “gut-loading” on a high calcium diet at least 24 hrs prior to feeding.
4. Calcium-rich insects include phoenix worms and the wood lice. To increase digestibility, these insects can be macerated (cuticle broken) prior to feeding insectivores.
5. For commercial diets, check the first ingredient to ensure that an appropriate animal protein product is listed. “Guaranteed” analysis minimums and maximums do not provide exact percentages.
6. Ensure that prey items are well-hydrated prior to consumption, as water is a major nutrient and many insectivores depend on prey moisture content to maintain hydration.

**References**


The ‘Phys’ Behind the Physical Exam in Reptiles
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Most of the reptile physical examination begins with a thorough evaluation of the husbandry and history, even before a visual examination should be tackled.

Husbandry sheets should be prepared for dispersal to your clients. This means doing homework for the species that are most likely to walk through your door. Which might they be? Head to a pet store nearby and take note of what is being sold. Where is a good place to start? Red-eared sliders, anoles, ball pythons, cornsnakes, bearded dragons, iguanas, and Russian tortoises. Visit reptilemagazine.com, chat with a few friends that own these animals and make your own husbandry sheets. Making the sheets will make you remember. This gives them a reference for proper reptile care. This also reassures your owners that you can offer compassionate veterinary care for their reptile. This is very important! Get a Detailed History: Diet, cage type, cage design, lighting and heating, bedding, cage location, behavior, prior medical history, other pets in household. This information prepares you for what to specifically evaluate on physical, and what to expect.

The exam room should have supplemental heating source, a pen light, Doppler, cotton tip applicators, warm ambient environment, and as few on-lookers as possible.

The following systems can be evaluated WITHOUT disturbing the animal: Ocular, aural, hydration status, proper occlusion, overall body condition score, obvious musculoskeletal abnormalities, RR, CNS/Mentation, and integument.

The rest of the “physical” physical should take about 10 minutes. This is important because handling will stress your patient out. Save oral exam for last, this will be the most stressful part of the physical exam.

Oropharyngeal
Start by palpating the mandible. If you can feel “bumps” along the maxilla or mandible, you may be palpating abscesses. Is the maxilla and mandible properly occluded? Do you see exposed gingiva? Does the rostral skull bend when you apply mild pressure to the snout? Fibrous osteodystrophy secondary to metabolic bone disease? Use a lubricated guitar pic or cotton tip applicator and a light source to visualize the entire dental arcade, gingival, tongue, glottis, assess mucus membranes and hydration status. Dry and/or ropey oral secretions indicates dehydration. Snakes have lovely palatine vessels, assess their distension and provide evidence for adequate or lack of perfusion.

Ocular
Do the eyes appear dull or appear sunken, this can indicate severe dehydration. They may dazzle, but will not menace. Tortoises, crocodilians and lizards control their PLRs, skeletal muscle control of the iris. This means atropine will not be affect in dilating pupils to evaluate the back of the eye. Check the medial aspects of lids in testudines and lizards, spectacles in snakes. Retained sheds? Check conjunctiva & 3rd eyelid mucus membrane color. Any ocular Discharge? Periocular swelling? Photophobia? Characterize their problems the SAME way you would a mammal. Chemosis? Corneal Defects? Anterior Uveitis?

Aural
Testudines have a tympanum, evaluate for swelling, which may indicate auricular abscesses. For lizards, evaluate their tympanums. Check for evidence of fluid or blood behind the window.

Respiratory
Get a respiratory rate BEFORE you handle the reptile. Remember that defensive postures and displays can affect rates. RR 12-36 bpm at rest is normal. For aquatic testudines: abnormal buoyancy is HIGHLY suggestive of pneumonia. Open mouth breathing in ANY REPTILE is an emergency.

Cardiovascular
Turtles, all lizards EXCEPT varanids: can be located in the thoracic window. For turtles, place Doppler on the Jugular vein OR behind the arm in the axillary pit. For lizards, place the Doppler probe in between the arms on the ventrum of the reptile. For our lovely monitor lizards, the varanids, place the Doppler in on the ventrum on the caudal thorax. For snakes, the heart is located on the ventrum 1/3rd the length of the body caudal to the head. For large snakes, the Doppler can be placed on the spectacle. The heart rate is heat and PAIN dependent. HR≤ 60 bpm can be normal. Elevations can be attributed to increased activity and/or most PAIN. Check for arrhythmias and murmurs. Unsure? Use an ultrasound to evaluate contractility and chamber size. It’s a beautiful pump, they can shunt in extreme circumstances, but the heart functions just like a mammals when the environmental conditions are right.
Gastrointestinal
The best way to evaluate the GI is to take a look at what came out of it. Fecal evaluations and a history of stool production are important. The GI, when palpated, is not as easy to distinguish when compared to mammals. They all have a stomach, small intestines, liver, gallbladder, and their colons vary in size based on diet. Our herbivores have large ones, the carnivores have short ones. You may be able to palpate ingesta and/or fecal matter. IN turtles, you can palpate the femoral fossa to rule out obstipation. As many reptiles have unique coloration of their oral mucus membranes, it is important to evaluate the color of the cloacal membranes.

Urogenital
In turtles, the kidneys are located caudodorsally along the carapace. You cannot palpate them. Femoral fossa palpation may allow you to feel urinary stones. Male turtles have a phallus, do not excrete it but evaluate it IF it is displayed or traumatized. In iguanas, the kidneys are located in the pelvic canal. You can perform a cloacal exam to rule out renomegaly. In other lizards, you can visually locate hemipene sacs. Unlike in pet parrots, we have many species that are sexually dimorphic! Agamids, geckos, iguanas, males have large femoral pores. In bearded dragons, do not mistake uterine horns for coelomic fat pads in agamids. In snakes, you should not be able to palpate kidneys unless there are severe gross abnormalities. In boids, there have remnant pelvic “legs” that are actual short bones that articulate with the rib cage. They are called “spurs, and can be flexed and extended during courtship. Some use pelvic spur size as a tool for sexing snakes, males having larger spurs. For sexing non-boids, you can use a small, lubricated red rubber French catheter to probe hemipene sacs. Avoid probing needles. Males have a deeper hemipene sac length. Mark how far the probe was placed in the sac. Then count the number of scales that accounts for the marked length distal to the anal scale. Scale depths greater than 7-9 scales indicate the sex is male. Females usually have a hemipene sac scale depth of less than 5 scales. This can vary dependant on the species.

Integument
Assess the skin turgor. If there is prolonged tenting and lack of elasticity, the reptile is dehydrated. What is their shed cycle? Aquatic Turtles shed scutes commonly. Lizards shed skin in a piecemeal fashion. Snakes shed in one sock-like piece INCLUDING the spectacles. Snakes undergoing an active shed should be handled carefully, as they are physiologically dehydrated and cannot see well. Dysecdysis is a very significant finding. It signals severe deficits in hydration status. Note any scars, vesicular dermatitis, or lacerations and characterize them.

Musculoskeletal
Palpate long bones, spine, and hips and examine the normal posture and their ability to ambulate on padded exam floor. The body condition score in all reptiles can be assessed by a visual and physical evaluation of masseter and temporal musculature, normal or atrophied. In iguanids and geckos, evaluate the tail as they have bands of fat stored in between the coccygeal musculature bands. You should NOT be able to feel lateral processes of the coccygeal vertebrae in a well-conditioned lizard. Coelomic fat pads can be palpated in longitudinal bands in bearded dragons and spiny tailed lizards.

Nervous
Start by carefully evaluating their mentation. What does the owner say, is this normal? Are they observing your actions? Trying to get away? Are they threat displaying, are they looking around? Are they trying to escape? Are they trying to move away? Are they flicking their tongues? For the Cranial Nerves exam, you can perform a quick assessment. Assess dazzle, PLRs, eye position, facial symmetry, hearing. Most reptiles can appreciate noises that have a low decibel level. Tongue and gag reflex is checked during the oral exam. Palpate the spine, check panniculus, check CP by placing and avoid hemi-walking and checking peripheral reflexes unless indicated.

With practice, you will start to have fun learning about the unique differences and celebrating them with a contagious enthusiasm that their owners have! Treating reptiles mean you get the opportunity to learn something new each time- have a great time!
GI Stasis: Oryctolagus cuniculi, the domestic rabbit, has a very specialized gastrointestinal system. These hindgut fermenters rely heavily on the cecum and colon to ferment long stem grass hays and plant material. The rest of the GI system and the other biological systems of the rabbit are at the mercy of the functionality of these organs. When GI motility slows downs or stops completely, it is a medical emergency for the rabbit or any small herbivore. Simple treatments can be implemented to quickly resolve the condition and afford a practitioner the time needed to identify the underlying cause— which could be pain or diseases that are not specifically related to the GI tract.

It is important to first stage the severity of GI stasis. Is the rabbit still passing small feces or has clinical signs persisting for more than 12 hours? We classify GI stasis cases as mild, moderate and severe.

- Mild: Potential dietary indiscretion, stress incident, unrelated mild illness. Patient is well hydrated, still eating, and fecal production is slowly decreasing, but has not stopped.
- Moderate: Patient has not produced feces in > 8 hour period, stopped eating, presents dehydrated and painful.
- Severe: Patient is severely dehydrated, painful, hypotensive, hypothermic and requires emergency fluid therapy and stabilization.

**GI stasis treatment approach for herbivores**

The treatment approach should include fluid therapy, analgesics, syringe feedings and tests to outline the underlying cause. Fluid therapy requirements range from 100-150 mls/kg/day. Give 50mls/kg SQ every 12 hrs if mild to moderate and if there are no indications of cardiac disease or hypoproteinemia. For severe cases, warm IV therapy and crystalloid and/or colloid shock boluses may be indicated. Fluid boluses can be 10mls/kg over 20 minutes, check SAP and modulate therapy as needed If there is a response. Colloid boluses of 5mls/kg over 20 minutes may be indicated if the patient is bleeding or hypoproteinemic. Buprenorphine 0.01-0.03 mg/kg SQ/IV q8-hrs should be given as needed for moderate to severe cases. Tramadol 5mg/kg PO q8-12 can be used in recovering animals or in mild cases that require continued supportive care at home. Syringe feeding is the mainstay of GI stasis therapy.

Cecal motility and colonic motility respond to 0.5mm fiber particles that stimulate their mechanoreceptors. Without this, the process halts. Getting the fiber BACK into the gut is the goal of treatment to re-stimulate normal motility. Critical care feedings can be arduous but are a vital part of the treatment process. Patients require 20mls/kg per feeding, with a feeding occurring every 6-8 hours, until the animal begins eating on its own. When patients are recovering, stimulate them to explore their environment provided their underlying ailment does not prevent physical motility. Lastly, identify the underlying cause of the stasis. Perform a thorough exam is key— trust your hands and your observations! If possible, perform a CBC, PCV, TS, and chemistry screen in your bunnies. Radiographs may be indicated. In most cases, metoclopramide and cisapride, which can induce side effects themselves in herbivores and in people, are not warranted.

**Diagnosing dental disease**

It’s important to know that you can diagnose dental disease in small herbivores via visual and physical exam before attempting an oral exam. Molar disease is the usual culprit that gets missed on many exams. Palpate the ventral aspect of the mandible, it should be smooth! If you feel bumps, these may represent reserve crown extensions or abscession. The same can be performed on maxilla palpation, particularly on the medial aspect of the zygomatic arch. The incisors should be properly occluded, if they aren’t there’s a high likelihood that the molars are mal-occluded. “Incisor-disease only” can occur but represents a small portion of rabbits with dental disease, therefore incisor adjustments alone do not always treat disease. Radiographs are commonly indicated to help qualify dental disease. There is an excellent reference for classifying radiographic evidence of disease in herbivores (E. Boehmer; D. Crossley: Objective interpretation of dental disease in small mammals. 2009), please email for a copy. Treatment options for dental disease vary, and may require clinical crown adjustments, diet modification (reducing pellets, increasing long stem grass hay) and in severe cases with abscession, antibiotic therapy (Duo-Pen 50,000 IU/kg SQ q 7 days to cover infections-anaerobic), and surgery. Pain medications can include Meloxicam 0.5mg/kg PO q12hrs and Tramadol 5-10mg/kg PO q8-12 hrs.

**The head tilt: What is the cause?**

The young or old rabbit that presents with vestibular signs should be evaluated the same way one would evaluate a cat or dog. Distinguishing if an animal has peripheral or central signs is imperative. Despite what the old references tell us, E.cuniculi (ECUN)— a microsporidian parasite that can infect rabbits, dogs, and humans, does NOT cause peripheral vestibular signs. Unfortunately, central vestibular signs can be subtle and missed. More often than not, ECUN can cause cerebral signs, which may include only slight mentation changes. In severe cases, cerebral disease disease can cause cortical blindness and seizures and this is easier to diagnose.
Central vestibular signs, which include axial rolling, tight circling, abrupt hemiparesis, CP deficits, severe ataxia, truncal swaying, horizontal, rotary or vertical nystagmus, are easily diagnosed but not always reflective of ECUN infections. In a case review of 230 post mortem exams performed on rabbits at PennVet from 1985-2012, *E. cuniculi* spores that caused active CNS inflammation (secondary to neuronal cell rupture) were identified in 13% of the cases, with only one rabbit that had severe inflammation in the brainstem that may have caused central vestibular signs. Studies now confirm that the degree of inflammation on post does not correlate with clinical signs (Csokai 2009). Commonly, rabbits > 4 years of age can present with acute thromboembolic events that may resolve in as little as 48 hours or as long as 1-2 weeks with supportive care. Supportive care includes protective caging, assist feeding, and anti-inflammatoryatories (meloxicam and/or doxycycline). Ruling out otitis, ototoxicity, and trauma becomes essential to hasten treatment for patients that have severe peripheral signs. True *E. cuniculi* cases can present with acute disease in newly infected or chronically affected rabbits. Renal failure secondary to repeat infections and damaged caused by spore migration can occur. CNS involvement occurs in chronically infected rabbits and can be difficult to diagnose antemortem. A review of the disease and treatment options can be found in this free access summary article. (Latney et al, *Encephalitozoon cuniculi in pet rabbits: diagnosis and optimal management*. 2014). Fenbendazole does not cure chronically infected animals with CNS inflammation and case reports reveal that its use causes severe liver disease and failure in rabbits (Graham, *Benzimidazole Toxicosis in Rabbits: 13 Cases from 2003 to 2011*, 2014).

**Chinchillas**

**GI stasis prognosis**

Unlike our lagomorph patients, chinchillas afford owners an even narrower window to respond. Clinically, we appreciate that of the small herbivores, chinchillas develop severe metabolic disturbances secondary GI disruption. Ketosis occurs rapidly. It is not uncommon to see chinchillas present with severe metabolic derangements secondary to GI stasis. It is imperative to get a urine sample to assess for the presence of ketonuria to qualify prognosis. If a blood sample can be obtained, assessing for the presence of lipemia is also imperative. If the patient was previously overweight (>500grams) and has hepatic lipodiosis, diabetic ketoacidosis is highly likely to occur within as little as 8 hours from not eating. The patient has a grave prognosis if ketonuria, glucosuria, and lipemia are noted and, even with aggressive care. If the patient presents muscle wasted/semaciated, a ketoacidosis may be present secondary to prolonged period of anorexia and muscle catabolism. If a ketonuria is noted, in the absence of lipemia, glucosuria, and/or hyperglycemia, the patient may recover with aggressive support care.

**Revisiting the murmur**

In the past, clinicians thought murmurs in chinchillas were a “normal” finding on physical exam. In a multi-institutional study evaluating cardiac disease in 260 chinchillas, murmur prevalence was found to be 23% (Pignon 2012). Chinchillas with a grade 3 murmur or greater 30 times higher chance of having echocardiograph disease than chinchillas without murmurs, and those with a grade 1 or 2 were 10 times more likely to have echocardiographic abnormalities. Mitral valve insufficiency, right ventricular outflow obstruction, tricuspid valve regurgitation, and left ventricular hypertrophy were among the specific cardiac diseases identified in 8 of 15 chinchillas that received full echocardiographs. Subsequently, we recommend monitoring chinchillas closely for murmur intensity and urge owners to pursue an echo if a Grade 3 is noted OR if the intensity of the murmur increases.

**Guinea pigs**

**Revisiting respiratory distress in the guinea pig**

Respiratory distress in the guinea pig comes with a long differential diagnoses list. Apart from *Streptococcus pneumoniae* and *Streptococcus zooepidemicus*, viral infections, like guinea pig adenovirus, can cause severe pneumonia. *Bordetella bronchiseptica*, commonly found as in rabbits and dogs, can cause acute respiratory distress and death in guinea pigs. Antibiotics for *S. pneumoniae* and *Bordetella* can be managed with enrofloxacin and chloramphenicol. *S. zooepidemicus* can cause an acute respiratory syndrome, which can be difficult to cure OR it can cause localized abscessation of the cervical lymph nodes. Medical management is the goal, as *S. zooepidemicus* is a normal flora in GPs. Although penicillins is an effective treatment for *S. zooepidemicus*, it will cause a dysbiosis in the guinea pig, even if given SQ. Perform cultures to rule out other infectious causes. Consider starting with enrofloxacin or chloramphenicol. Oral fluoroquinolones may cause mild GI upset and pigmenturia in some patients.

In some cases dental disease may be the cause of severe respiratory distress. Evaluation of the oral cavity is very important in this case, as treatment requires dental emergency adjustments. Their mandibular molar teeth can become overgrown, causing enamel point bridging that can entrapp the tongue. Their normal occlusal surface is a steep 30 degrees, causing the lingual points to angle down toward the tongue. They have a palatal ostium that connects the oropharynx to the trachea, so they can literally occlude their airways with struggling to unenrap the tongue. In addition to dental disease, guinea pigs can present in severe respiratory distress secondary to cardiac disease, primary pulmonary neoplasia, mass effects from severe gastric dilatation or ovarian cyst enlargement. For these reasons, a thorough, safely staged examination should be performed carefully in the respiratory pig, as the cause of presentation can be quite variable.
Updates on antibiotic and analgesia doses for herbivores (off-label references)

- Tramadol: 5-10 mg/kg PO q8-12h for pain
- Buprenorphine: 0.01-0.05mg/kg q8-12h for pain
- Tonazuril: 2.5 mg/kg PO once for Eimeria infections in rabbits
- DuoPen: 50,000 IU/kg SQ every 7 days for anaerobic dental disease, skin disease, chronic respiratory disease, Treponema infections in rabbits
  - Do NOT use in guinea pigs, some report use in chinchillas
- Enrofloxacin: 15-20mg/kg q24h- this is a dose dependent drug, smaller doses given q12 potentiates resistance patterns and reduces drug efficacy
- Metacam: 0.5 mg/kg PO/SQ q12h
- Maropitant: 1 mg/kg SQ/IV q 24h to reduce nausea and in severe GI stasis cases
- Cyanocobalamin: 20 mcg/kg PO/SQ q24h for liver and regenerative anemia support
- Keppra (Levetiracetam): 60 mg/kg to treat status epilepticus, 20 mg/kg SQ/IV q8h
- Thiamine: 20mg/kg PO/SQ q24h for chronic liver disease

Ferrets

Diarrhea – Why we treat aggressively

As the experimental model, ferrets provided us with the scientific proof that Helicobacter can cause gastric ulceration and primary disease of the gut. In practice, it is common to see ferrets develop diarrhea secondary to any underlying disease. Immunosuppression and stress-induced clinical Helicobacter infections can occur, no matter the age of your patient. There is strong evidence that Helicobacter mustelidarum is endemic in the ferret pet trade. Unfortunately, other pathogens can potentiate GI disease in ferrets as well. Infectious ferret viral diarrhea (epizootic catarhral enteritis), eosinophilic inflammation, metabolic disease, parasites (coccidians in young ferrets), proliferative bowel disease (Lawsonia intracellularis) and neoplasia are other differentials. Enzootic catarhral enteritis, a viral disease, can cause mild diarrhea in young kits and prove fatal in naïve geriatric ferrets. Commonly, diarrhea results in a hypoproteineinemia, hypoglycemia, and severe dehydration that is not solely responsive to subcutaneous fluid therapy.

For the dehydrated diarrhea ferret, muster your courage and place an IV catheter. Give shock fluid bolus as if for a cat. An assessment of their glucose, PCV/TS, and renal values is imperative. We recommend providing potassium supplemented IV fluid therapy for at least 12-24 hours at maintenance rate (60mls/kg/day). Also factor in their dehydration deficit and try to replace this in the first 8 -12 hours safely. Consider administering Hetastarch if the total protein (albumin) is low, as GI protein loss can be severe. Hetastarch boluses can be administered at 5ml/kg over 15min. In severe cases, a CRI can be maintained at A 15-20ml/kg/day. Patients on hetastarch CRIs for longer than 7 days are at risk for developing a coagulopathy (coagulation factor VIII). Obtain radiographs and if possible, ultrasound-flash the abdomen. Ferrets with ECE may appear obstructed on radiographs due to ileus and gas distension of intestines. Antibiotic treatment for diarrhea is based on Helicobacter studies and include the following: Amoxicillin PO (20mg/kg q12h), or ampicillin IV (20-22mg/kg q8h), Metronidazole PO or IV (20 mg/kg PO/IV q12h), Famotidine or Omeprazole IV or SC (0.5mg/kg IV/PO q12h), Carafate PO (20 mg/kg PO q6, 20 minutes prior to feeding). Begin syringe feeding them watered a/d every 4 hours BY hand. Resolution of complete clinical signs of a Helicobacter flare or ECE may take up to 2 weeks. Perform diagnostics to rule out additional causes of disease. Lawsonia intracellularis infections require PCR confirmation or silver stains performed on tissue samples, and chloramphenicol (50mg/kg q12h for 10-14d) is indicated for treatment.

Rats

Revisiting pneumonia

Rats suffer from an arsenal of respiratory pathogens and there are factors that can hasten and exacerbate disease in their delicate lungs. The 5 recognized pathogens are: Mycoplasma pulmonis, S. pneumoniae, Cornebacterium kutscheri, Sendai virus and cilia-associated virus. Rats may live their life of 2-2.5 years with signs of chronic respiratory tract infections. Once established in the lower airways, Mycoplasma pulmonis causes a chronic bronchitis, progressing to bronchiectasis and bronchiolectasis, and pulmonary abscessation. Acute disease may occur with signs of nasal discharge, ocular discharge and respiratory distress. Radiographs may reveal consolidation of lung lobes with abscessation. When approaching the dyspneic rat, qualify severity first. If they are open-mouth breathing, have a RR >100 bpm, have blue/cyanotic foot pads, and/or a severely increased respiratory rate, place them in an oxygen cage and assess for responsiveness. Warm saline nebulization may be necessary and may help during recovery. Airway support may be given with the use of a bronchodilator (Albuterol inhalation, aminophylline, or Terbutaline 0.1mg/kg SQ). If the patient is responsive, organize your treatments to administer quickly. Antibiotic therapy with enrofloxacin @ 15 mg/kg PO or SC q 24 hr (if giving SC need to dilute out in 5-10 ml sterile saline) and Doxycycline @ 5 mg/kg PO q 12 hr. Do not use the clear doxycycline hyclate for injection- this causes severe tissue necrosis. The depot formulation (Vibrafenos) of doxycycline can be given carefully, SQ, at a dose of 75-100 mg/kg, and will last for 7 days as the patient can tolerate oral medications. Adjunctive therapy may also
include Azithromycin 15-30 mg/kg PO q 24 h. Provide fluid therapy and nutritional support during recovery, as these patients can become severely dehydrated secondary to respiratory disease and severely hypoglycemic.

Sincere efforts should be made to reduce the potentiation of clinical signs and this can be modulated by the pet owner. Rats require adequate ventilation and low dust, non-aromatic bedding. Encourage owners to maintain room temperatures from 65 to 80º F, humidity between 40 to 70%, and clean the cage frequently to minimize ammonia fumes. A reduction in obesity may also help reduce the clinical onset of disease.

**Neurologic presentations**

Pituitary adenomas are not uncommon in geriatric pet rats (Mayer 2011). It is seen more commonly in female rats, however males can present with signs as well. Affected rats present with CNS signs such as blindness, circling, head pressing, "spacey" appearance or severe depression. They are reluctant to move from the cage wall and owners may report that they have fallen or developed balancing issues at home. Pituitary adenomas are usually prolactin secreting and mammary development can be seen. This disease can be painful and very distressing. Stabilizing treatment includes fluid therapy and steroid medications (Dexamethasone SP 0.25mg/kg-0.5mg/kg once or oral prednisolone 0.5 mg/kg q24h) pending the acquisition of cabergoline. Cabergoline, a dopamine receptor agonist, has been reported to decrease tumor size in per rats at 0.6mg/kg PO q72h and has been used to manage disease for 8.5 months in a select case (Mayer 2011). Fluid therapy is warranted, as affected rats are display hypodipsic hypernatremia syndrome due to the hypothalamic space-occupying mass. Nutritional support is also necessary during stabilization, as patients are usually very painful when eating solid foods.

**Mammary fibroadenoma and pituitary tumor prevention**

Although females develop mammary fibroadenomas, both sexes develop an increased risk as they age (> 1 year old). The incidence is clinically high in females, however 2-16% has been reported experimentally in males (Mayer 2013). Mammary tumors and pituitary tumors frequency is significantly lower in 18- to 24-month-old ovariecromized rats (4%) than in sexually intact rats (mammary tumors, 49%; pituitary tumors, 59%). Decreased frequency of mammary tumor development could be related to the decreased frequency of prolactin-secreting pituitary tumors (Hotchkiss 1995). Based on these findings, it is recommended that rats undergo ovariecotomy or neuter prior to 90 days of age to reduce the risk of mammary and pituitary tumor development. For patients that cannot undergo surgery, off-label use of deslorelin acetate (4.7mg SQ implant), a GnRH agonist, has been used to delay estrus for 1 year in rats (Alkis 2011). This may be helpful in modulating prolactin-secretion and reduce disease occurrence. Leuprolide acetate has been experimentally used to suppress gonadotropin and testosterone levels for 5 weeks in rats.