**Introduction:**

Metabolic bone disease (MBD) has become a very important issue in the South African lizard market over the last few years. MBD is defined as a syndrome including a range of bone diseases associated with metabolic diseases, e.g. secondary hyperparathyroidism, rickets and osteoperosis. It is generally a chronic, or long-term result, or combination of a dietary deficiency of calcium or vitamin D, a negative dietary calcium to phosphorus ratio or the lack of exposure to ultraviolet light. A less common cause includes disruption of vitamin D metabolism due to kidney, liver, intestinal, thyroid or parathyroid disease.

The disease is most commonly seen in young growing lizards without adequate calcium (Ca) supplementation or inadequate ultraviolet (UV) lighting. Adults, especially gravid females are also at risk.

Diagnosis of MBD is primarily made from dietary history and clinical signs.

**Physiology:**

Calcium is an important macro mineral in the body of vertebrate species. It is responsible for neurological and muscular function and normal bone formation. Bone aids in the homeostasis of blood calcium levels by serving as a calcium and phosphorus reservoir. Up to 99% of an individual’s Ca is stored in bone. Adult animals that developed normal bone or have adequate bone mass are more resistant to MBD. Dietary Ca and P is absorbed from the small intestine in conjunction with activated vitamin D3 (1,25-dihydrocolecalciferol). After absorption these minerals are distributed via the blood to bones and teeth where it is deposited in a crystalloid form. The balance between calcium incorporation and resorption (releasing Ca back to the blood) are under hormonal control. Both calcitriol

**IN SHORT:** Calcium (a mineral) is needed for normal bone growth, muscle contraction and nerve conduction. The only source of calcium is dietary intake. Gut absorption of this mineral is dependant on activated vitamin D3 and the ratio and interaction of phosphorus. Dietary vitamin D3 precursors are activated by UV radiation. Non-absorbed calcium is excreted via the faeces. When inadequate levels of calcium are absorbed the body uses its calcium stores,
and parathyroid hormone plays a role. The dietary Ca:P ratio is also critical for absorption. A ratio of 1:1 – 2:1 is recommended. Excessive dietary Ca is excreted via the feces.

In animals the precursor of vitamin D (7-dehydrocholesterol) is taken in via the diet. By the action of UV eradication, dehydrocholesterol is converted to cholecalciferol (inactive vitamin D3) in the skin. Both the D2 (from plants) and the D3 form (from animal origin) can also be absorbed. After absorption all these forms are carried in the blood attached to a “vitamin D binding protein” (DBP) where it is transported to the liver. In the liver cholecalciferol is converted to 25-hydroxycholecalciferol. 25-hydroxycholecalciferol is converted to activated (1,25-dihydroxycholecalciferol) and another inactivated (24,25-dehydroxycholecalciferol) form in the kidneys. Under conditions that require additional calcium and phosphorus (i.e. growth or egg development) the activated form are mainly produced. 1,25-dehydrocholecalciferol aids in the absorption of Ca and P from the GIT and in the release of parathyroid hormone (PTH).

[Diagram of the basic metabolism of Vitamin D]

Figure 1: Basic metabolism of Vitamin D

Blood calcium and phosphorus levels need to be kept constant in the body. Physiological or homeostatic mechanisms are responsible for minimizing changing blood levels.
Decreased blood calcium levels (hypocalcaemia) triggers the release of PTH. This hormone is responsible for the activation of osteoclastic activity, which ultimately causes bone resorption and the release of Ca and P into the blood. It is also responsible for the stimulation of the synthesis of 1,25-dihydrocolesleiferol which facilitates Ca absorption form the small intestine and the correction of the Ca:P ratio by increasing P excretion via the kidneys. All the mechanisms activated by hypocalcaemia and PTH release is responsible for increasing blood calcium levels to normal (eucalaemia). Prolonged osteoclast activity my lead to a decrease in bone mass and bone weakness.

Increased blood Ca (hypercalcaemia) triggers the release of calcitonin, a PTH antagonist. This hormone inhibits Ca resorption from bone and is ultimately responsible for decreased blood calcium levels.

**Figure 2: Summery of calcium & phosphorus metabolism**

**Dietary History:**

Insects such as crickets and cockroaches have a negative Ca:P ratio and need Ca supplementation. Insects should be fed foods with high Ca, two to three days prior to being used as food (so called “gut loading”) or dusted with a suitable Ca powder just prior to being used as food. Chicken starter mash or growing mash, sometimes used as maintenance food for crickets are deficient in Ca and should be supplemented (also see the crickets section for more info). Other gut loading foods include greens and vegetables such as lucerne/alfalfa (*Medicago sativa*), carrots, carrot greens and parsley. Fish flakes can also be used but dusting with appropriate calcium is still necessary.

Herbivorous diets often contain little Ca and excess P. Most fruits and lettuces are low in Ca and need Ca supplementation. Many commercial reptile diets are Ca deficient. Ca can be supplemented by adding powdered calcium to the food or by adding powdered or
Clinical Signs:

Clinical signs can broadly divided into classic MBD with symptoms related to the skeletal system and hypocalcaemic MBD where symptoms is secondary to hypocalcaemia. The most common form, namely the classic form is more common in growing animals, while the hypocalcaemic form is more prevalent in adult animals. Table 1 summarizes the most common clinical signs seen with each form.

Table 1: Most common clinical signs seen with classic & hypocalcaemic forms of MBD

<table>
<thead>
<tr>
<th>Classic MBD</th>
<th>Hypocalcaemic MBD</th>
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<tbody>
<tr>
<td>Partial to complete lack of truncal lifting</td>
<td>Intermitted muscle tremors, seizures &amp; tettany</td>
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<tr>
<td>Pliable mandible or maxilla</td>
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<tr>
<td>Retention of the rounded infantile skull</td>
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<tr>
<td>Lameness or reluctance to move</td>
<td>Flaccid paresis</td>
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<tr>
<td>Kyphosis, lordosis &amp; scoliosis</td>
<td></td>
</tr>
<tr>
<td>Reduced appetite (or unable to eat) &amp; weight loss</td>
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Clinical signs will only start a few weeks after the introduction of a sub-optimal diet or husbandry flaw. The first signs to appear are usually related to the skeletal system. Affected animals may be unable to lift their bodies from the ground, may have soft, pliable jaws and bilaterally thickened fore- and hind legs. Animals may or may not show decreased appetite. These early signs are usually only observed by experienced herpers or vets. Observant keepers will also be able to recognize these changes if they are aware of the syndrome.

In advanced cases animals will show jerky movements, tremors and malformed bodies. Signs may progress to complete lameness and eventually death. Complications of MBD include bone fractures, drying of the mucous membranes of the mouth and the inability to walk or eat.

If there are any signs that may suggest MBD the animal must be handled with the utmost care to prevent fractures and injury. Veterinary diagnosis and treatment are usually indicated.

Complications Of MBD:

Fractures are the most common complication of MBD. Falling or climbing from heights that would normally pose no problems for normal calcified bones is a common cause. Drying of mucous membranes of the mouth due to inability to keep the mouth close and inability to walk and eat are also commonly seen. Egg production may also be affected in females.

Diagnosis & Treatment:

When any of the clinical signs are seen the animal should immediately be treated by a reptile friendly vet or an experienced herpetologist. One recommended treatment is parenteral calcium administration repeated as described by the vet. Treatment can be life saving. The diagnosis can be made on the history of inadequate calcium
supplementation and/or insufficient exposure to UV radiation and clinical signs alone, no radiographs are indicated. The prognosis depends on the severity and the future cooperation of the owner. In early cases where supplementation and husbandry are improved the outcome is usually favourable.

Husbandry:
Calcium can only be optimally absorbed in the presence of activated vitamin D3 and/or adequate ultraviolet (UV) radiation. UV rays (are part of a group of rays, collectively known as the light spectrum. The three groups important here are UV rays, visible light rays and infra red (IR) rays. The wavelength of UV rays is shorter than, and that of IR are longer than visible light. IR rays are responsible for heat transmission and visible light includes all the colours visible to the eye. Ultraviolet can further be subdivided into UVA and UVB. UVA rays are apparently needed for lizards to see optimally. UVB rays are needed for the activation of vitamin D3 and subsequent calcium absorption.

Diurnal lizards, i.e. Bearded dragons (*Pogona vitticeps*) and Iguanas need natural or artificial UV radiation. It is recommended that animals without the correct UV lighting should be exposed to natural sunlight for at least (preferably more than) 2 hours a day. Important things to keep in mind when exposing animals to sunlight are:

- Always provide *ad lib* water and shade for in case animals get overheated.
- Never put animals in closed containers, i.e. aquariums, desert dens, etc. in direct sunlight as it will heat up and mortalities can occur.
- Never put animals outside when the temperature are not within the correct temperature range for the specific species.

Artificial sunlight can be supplied by various sizes of commercially available UV florescent lamps. It is important to use the biggest/longest possible lamp for the vivarium or container. Two important factors of a lamp is its output, indicated as a percentage or number, and the spectrum, UVA and UVB. The percentage output is the amount of rays the lamp emits. The popular percentages include 2.0 (2%), 5.0 (5%) and 8.0 (8%). Desert reptiles obviously need more radiation, i.e. 8% where in some cases tropical species can get away with 5%. Although most lamps are indicated to transmit both UVA and UVB it is important to get a lamp that at least transmits UVB. As ultraviolet lamps doesn't emit sufficient heat (i.e. IR rays), they should be used in conjunction with the correct heating. Important things to keep in mind when exposing animals to artificial sunlight are:

- Use the correct fitting. Fluorescent fittings differ according to length (usually indicated in foot / ft.) or wattage.
- Never use a UV fluorescent lamp for more than 6 months. Old lamps can emit cancer causing rays.
- UV lamps should be mounted as close as possible to 30cm / 12" from the basking spot of the lizard.

Nocturnal lizards, i.e. Leopard geckos (*Eublepharis macularius*) which do not need UV radiation must be supplemented with a suitable calcium supplement including adequate
activated Vit. D3. See supplementation for more info.

**Supplementation:**

Supplementation refers to the addition of extra elements to a diet to make the diet more balanced. The two most important supplementation categories are calcium and multi vitamin/mineral supplements. Various commercial multi vitamin/mineral combinations are available from specialized pet shops and reptile friendly vets. It is important to note that adequate calcium supplementation is not included in multi vitamin/mineral combinations and should be supplemented separately.

The more important additions to calcium supplementation combinations are phosphorus and vitamin D3. For adequate calcium absorption and bone incorporation the calcium to phosphorus ratio should always be between 1.2:1 and 2:1. Theoretically Vit. D3 combinations are only required in nocturnal species such as Leopard geckos (*Eublepharis macularius*) which doesn't have UV requirements. Calcium/Vit. D3 combinations are obviously more expensive than regular calcium supplementation. Where correct UV lighting are supplied dietary Vit. D3 precursors will be converted to vitamin D3 and only calcium need to be supplied.

Calcium supplements are usually sold in a powdered form and are available from reptile friendly vets or specialized pet shops. Calcium should be supplemented at least twice a week and three times a week to growing lizards and gravid females. Make sure to read added instructions for further additions that might apply to specific supplementations.

There are various ways to supplement reptiles. Methods include dusting food, adding supplement to water and supplying *ad lib* calcium. As many lizard species rely more on food for their water requirements than fresh water, the recommended way to supplement calcium is by dusting its food. Both crickets and herbaceous foods can be dusted. As Leopard geckos (*Eublepharis macularius*) are known to lick calcium according to need, *ad lib* powdered calcium/Vit. D3 in a separate shallow container are adequate for supplementation.

It is important to note that calcium supplementation should not be done by gut loading crickets. Gut loading of crickets is ideal for multi mineral/vitamin supplementation, but the amount of calcium needed for adequate calcium supplementation are known to cause mortalities in crickets.

Also see the Dusting & Gut Loading Feeder Insects Prior To Feeding section for more info.

**Conclusion:**

MBD is a less serious disease and can be treated, but even though this is the case it should not be ignored. Although quality of life are still optimum treated animals will physically never be the same than before the disease. As with all diseases prevention is
obviously better than cure. With MBD prevention is simply following the correct husbandry instructions i.e. supplementation and UV lighting.

"If you think I should add more information to this section or think that something is incorrect, contact me and let me know. I would love to hear your ideas or methods you might use that is different than ours."