

Komodo dragons J.Nijboer

**UV-B and vitamin D<sub>3</sub> metabolism in juvenile Komodo dragons (*Varanus Komodoensis*)**

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### Abstract

The aim of this research project was to assess the vitamin D status in juvenile Komodo dragons held in captivity in Rotterdam Zoo. In addition, the effect of interference with UV-B on the serum levels of vitamin D metabolites and on the serum calcium concentrations were investigated in three Komodo dragons. Supplying 450 IU vitamin D<sub>3</sub> /kg feed orally did not increase 25-hydroxyvitamin D<sub>3</sub> (25-(OH)D<sub>3</sub>), the 24-hydroxylated metabolite of vitamin D (24,25-(OH)<sub>2</sub>D<sub>3</sub>), 1,25-dihydroxyvitamin D<sub>3</sub> (1,25-(OH)<sub>2</sub>D<sub>3</sub>) and calcium levels.

In contrast, exposing the Komodo dragons to UV-B altered the levels of vitamin D metabolites. The amount of 25-(OH)D<sub>3</sub> increased in komodo dragon 1 (K1) (18 to 195 nmol/ml) and in komodo dragon no 2 (K2) (31 to 291 nmol/ml). The amount of 1,25-(OH)<sub>2</sub>D<sub>3</sub> did not change significantly in both komodo dragons (139.5.6 to 235.3 pmol/l).

Measurement of 24,25-(OH)<sub>2</sub>D<sub>3</sub> in K2 showed a dramatic improvement after exposing to UV-B; the amount of 24,25-(OH)<sub>2</sub>D<sub>3</sub> rose (7.5 to 448.1 ng/ml).

Komodo dragon 3 (K3) was sent to Gran Canaria where it received natural UV-B. The level of 25-(OH)D<sub>3</sub> improved from 18 to 272 nmol/l. The amount of 1,25-(OH)<sub>2</sub>D<sub>3</sub> did not increase either.

In all komodo dragons the calcium level remained stable and within the range 3.18 to 4.44 mmol/l.

The present study documents for the first time the levels of three vitamin D<sub>3</sub> metabolites and their regulation by UV-B in Komodo dragons. According to literature low levels of 25-(OH)D<sub>3</sub> have caused bone defects in juvenile Komodo dragons. The current data show a clear effect of UV-B on the 25-(OH)D<sub>3</sub> levels and a concomitant rise in serum 24,25-(OH)<sub>2</sub>D<sub>3</sub> levels while 1,25-(OH)<sub>2</sub>D<sub>3</sub> levels remained constant. Although we have no data on the bone metabolism in our 3 Komodo dragons it is tempting to speculate in view of the published improvements of bone after UV-B treatment, that 24,25-(OH)<sub>2</sub>D<sub>3</sub> is involved in bone metabolism in Komodo dragons. This would be in line with data obtained in chicken and human showing a positive effect on bone.

UV-B measurements of a UV-B radiating lamp shows that the amount of UV-B declines rapidly during time. Also the decay rate differs from lamp to lamp. If "UV-B" lamps are used for synthesising vitamin D<sub>3</sub> through the skin the UV-B radiation should be measured regularly and the lamp should be replaced in time before the UV-B radiation is too low for his synthesising purposes.

This study shows clearly that, although this is a preliminary study, there is a dramatic change in vitamin D metabolites when using feeds with vitamin D versus UV-B light in juvenile komodo dragons.

### Introduction

Komodo dragons (*Varanus komodoensis*) are rare animals, which only inhabit the islands of Komodo, Rintja, and the western half of Flores in Indonesia. Reports of animals on smaller islands nearby, including Padar and Gili Montang are probably based on observations of movement of transient animals by swimming to these islands. Komodo dragons live in the tropics on 8 Degrees Southern latitude which means that the intensity of sunlight is much higher there than in Western Europe for example. In nature Komodo dragons bask in the morning, from 15 minutes to more than 3 hours (Auffenberg 1981).

These animals are opportunistic carnivores, at the top of the food chain on these Indonesian islands. It has been suggested that Komodo dragons as alpha predators can survive on these islands because they are ectotherm, which means that they require less food than mammals in other parts of the world at the top of the food chain. As adaptation to survival during long periods of low prey density, a Komodo dragon in one meal can consume up to 80 % of its own body weight. They feed on live prey as well as on carrion. They are capable of taking

down deer, wild boars and water buffalos. When necessary they do not feed for months at a time. Young Komodo dragons feed on insects, small birds and mammals and on other reptiles which may be more readily available throughout the year (Walsh 1999).

Komodo dragons are listed on Cites Appendix 1 by IUCN. The wild population can be considered as several thousands of animals. The major threats include habitat alteration, poaching of prey species and perhaps tourism.

The total captive population as of November 1998 was 272 animals which consisted of 65 males, 50 females and 157 of unknown sexes in 49 institutions. In Indonesian zoos live 160 animals, in North America 82, in Europe 14 and in Asia (- Indonesia) and Australia both 8 animals. About a dozen successful breedings have been recorded world wide. Zoos in Europe which maintain Komodo dragons are Thoiry in France, Chester in United Kingdom, Lisbon in Portugal, Reptillad on the Canary Islands in Spain, Zoo Berlin in Germany, Pilzen in the Czech Republic and Rotterdam Zoo in the Netherlands.

In captivity an adult Komodo dragon eats 1.5 –3.0 kg of rats a week, depending upon the size of the lizard and the time of the year. In general no vitamins and minerals are supplemented. A diet of whole animals combined with access to hot spots up to 40° C and natural or artificial UV-B light are thought to be adequate to promote healthy growth and development for adult Komodo dragons.

Hatchlings are fed daily for the first eight months and then every third day throughout the next year. In captivity they live on a diet comprising 20 % of whole mice and 80 % of chopped beef or lamb to which a vitamin and mineral supplement is added (Walsh 1999). Bone problems in (juvenile) Komodo dragons

It was reported by Allen *et al.* (1994) that nine of the twelve hatched Komodo dragons in Washington Zoo had long bone fractures which were discovered at about two months of age. It was discovered that the 25-dihydroxivitamin D<sub>3</sub> (25-(OH)D<sub>3</sub>) level, which is one of the intermediaries metabolics in the Vitamin D<sub>3</sub> synthesis, was low. After exposing the animals during two months to UV-B the 25-(OH)D<sub>3</sub> level increased significantly. It was presumed that rapid growing animals have increased requirements for calcium (Ca), phosphorus (P), and vitamin D<sub>3</sub> and that non-reproductively active adults may be more tolerant to low levels of Ca, P and/or vitamin D<sub>3</sub>, or low exposure to UV-B.

In October 1995 Rotterdam Zoo obtained three juvenile Komodo dragons which were born in the National Zoo in Washington D.C. (USA). From Washington it was known that the juveniles received UV-B light in order to synthesize vitamin D<sub>3</sub> and to prevent bone problems. At first it was decided that the young Komodo dragons did not receive UV-B or extra vitamin D at all. At a later stage it was decided to add vitamin D<sub>3</sub> to their diet and months later to expose them to an UV-B emitting lamp. One of the juvenile dragons was sent to Gran Canaria in Spain in June 1999.

#### Vitamin D<sub>3</sub> metabolism

Rachitis is a deficiency disease of vitamin D, which appears to have been a problem recorded in ancient times; evidence shows that rickets occurred in the Neanderthal man about 50.000 BC. (Machlin, 1990). The major effects of vitamin D are to increase the active absorption of Calcium-ion from the proximal intestine and to increase the mineralisation of bones. Vitamin D represents a group of closely related compounds that possess anti rachitic activity. (Machlin 1990).

A diagram depicting the synthesis and initial step of metabolism via 24-hydroxylase activity is shown in Figure 1. There are two sources from which vitamin D<sub>3</sub> (cholecalciferol) is normally provided: it is produced in the skin and it is taken up via the diet.

In the skin 7-dehydrocholesterol is photo chemically converted by UV-B to provitamin D<sub>3</sub> that then isomerizes to vitamin D<sub>3</sub>. Vitamin D<sub>3</sub> from the intestines and from the skin vitamin D<sub>3</sub> is bound to vitamin D binding protein and moves to the liver where it is hydroxylated at the carbon 25 position by the enzyme 25-hydroxylase to form 25-hydroxyvitamin D<sub>3</sub> (25-(OH)D<sub>3</sub>). Finally, in the proximal tubules of the kidney the biologically most active vitamin D<sub>3</sub> metabolite, 1,25-dihydroxyvitamin D<sub>3</sub> (1,25-(OH)<sub>2</sub>D<sub>3</sub>), is formed. A second metabolite of vitamin D<sub>3</sub> is produced in the kidney, namely 24,25(OH)<sub>2</sub>D<sub>3</sub>. Generally, 24,25-(OH)<sub>2</sub>D<sub>3</sub> has been considered to be the first step in the degradation pathway of 1,25-(OH)<sub>2</sub>D<sub>3</sub> and 25-(OH)D<sub>3</sub>. However, several human and animal studies demonstrated a positive contribution of 24,25-(OH)<sub>2</sub>D<sub>3</sub> either alone or in combination with other hormones to bone metabolism (van Leeuwen *et al.*, 2001) Recent studies in chickens suggest that 24,25-(OH)<sub>2</sub>D<sub>3</sub> together with 1,25-(OH)<sub>2</sub>D<sub>3</sub> treatment improves fracture healing and that 24,25-(OH)<sub>2</sub>D<sub>3</sub> serum levels are correlated to fracture healing (Kato 1998, Seo 1997).

The synthesis of 1,25-(OH)<sub>2</sub>D<sub>3</sub> is tightly controlled in order to maintain the calcium homeostasis. The major stimulators of 1,25-(OH)<sub>2</sub>D<sub>3</sub> formation are low serum calcium, parathyroid hormone and low serum phosphate levels. Increased serum calcium levels (hypercalcemia) inhibits formation of 1,25-(OH)<sub>2</sub>D<sub>3</sub>. Most interestingly, 1,25-(OH)<sub>2</sub>D<sub>3</sub> itself inhibits its own formation but stimulates 24-hydroxylase activity and the formation of 24,25-(OH)<sub>2</sub>D<sub>3</sub> and 1,24,25-(OH)<sub>3</sub>D<sub>3</sub>. Thus the metabolic clearance of 1,25-(OH)<sub>2</sub>D<sub>3</sub> is enhanced. By these regulatory mechanisms toxic effects of hypercalcemia (too much calcium) in the blood is prevented.

Figure 1: Diagram of the vitamin D<sub>3</sub> synthesis. Details are described in the text above.

- insert figure 1 here-

#### UV and UV-B meter

The spectrum of irradiance of wavelengths that reach the earthly atmosphere from the sun is approximately from 100 to 3200 nanometer (nm). Molecules in the atmosphere absorb certain wavelengths, so that the solar spectrum is attenuated when the radiation reaches the surface of earth. Some of the solar radiation is partly absorbed by ozone, oxygen, carbon dioxide and water. It means that life on earth is in principal exposed to Ultra Violet (UV), Visible Light and Near Infra Red. The wave length of Near Infra Red is longer than 700 nm. The Visible Light has a wavelength from 400 to 700 nm. UV can be divided into UV-C with a range from 100-280 nm, UV-B with a range from 280-315 nm and UV-A with a length of 315-400 nm. As mentioned before UV-B plays a major role in converting 7-dehydrocholesterol into provitamin D<sub>3</sub> in the skin with a maximum conversion at 297 plus or minus 3 nanometer. (Bernard, 1995)

Depending on the degrees latitude and the time of the year in some places it is not possible for humans to produce provitamin D<sub>3</sub> by natural light. In locations on 52 degrees North latitude, for example Edmonton, in Canada no provitamin D<sub>3</sub> will be produced from October until the beginning of April (Holick 1997). Berlin, Warschau and Rotterdam in Europe are also situated on the same latitude. Tests in Boston (42 degrees North), have confirmed that no provitamin D<sub>3</sub> was produced from November until February. The European cities of Barcelona and Rome lie on 42 degrees North. Tests have demonstrated that at the latitude of Los Angeles (34 degrees North) enough provitamin D<sub>3</sub> is produced throughout the year. 34 degrees North is the same latitude as places in Morocco and Northern Syria in the old world! The data described tests related to humans but probably similar considerations can be made on the effect of provitamin D<sub>3</sub> synthesis in reptiles, for example Komodo dragons. Tests have also proven that through normal windows UV-B coming from outside is absorbed. If Komodo dragons rely on the availability of UV-B for their synthesis, it can be questionable if