Two Hypotheses

Rabbit dental health isn't fully understood. Currently there are two prevailing hypotheses that may explain the current prevalence of dental disease among the pet rabbit population. These are:

Hypothesis 1. Rabbits chew whole loose hay (which is more abrasive) differently than they chew pellets. The belief is that to prevent dental disease rabbits must eat a diet composed primarily of loose hay.

This is based upon the belief that rabbits teeth constantly grow so that they must eat a diet that is course enough to properly wear them down or they will overgrow. It is also believed that rabbits chew hay differently than pellets. Specifically it is believed that rabbits chomp up and down on pellets rather than using their normal figure 8 side-to-side chewing pattern and that this leads to improper wear.

Hypothesis 2. Metabolic bone disease (unbalanced dietary levels of calcium and phosphorous) is the root cause of dental disease that leads to malocclusion and improper tooth wear (tooth spurs on cheek teeth).

This is based upon the observation that rabbits with dental disease commonly have metabolic bone disease that weakens the bone supporting the orientation of the teeth. As a result the normal pressure of chewing causes the roots to become elongated and the teeth to become crooked not allowing them to properly wear.

Research by a Rabbit Veterinarian

Frances Margaret Harcourt-Brown, a recognized specialist in rabbit medicine and surgery in the UK, performed a detailed radiological and morphological study of 172 prepared skulls and 315 skull radiographs of pet rabbits with the collection of rabbit skulls from the Natural History Museum in London.

She also examined the skulls and radiographs, gender and breed details of 1254 pet rabbits presented for veterinary care, 465 of which required dental treatment.

She clearly **documented that the loss of alveolar bone preceded changes in shape or structure of the crowns and root elongation** in pet rabbits.

Despite the wide variation in breeds analyzed (and therefore differences in skull shapes and sizes) she found no statistically significant correlation except for gender where slightly more rabbits with Acquired Dental Disease were male.

She concluded that Metabolic Bone Disease plays a major role in the development of Acquired Dental Disease in pet rabbits.

Dental Health
in small herbivores

Metabolic Bone Disease
(induced by a low-calcium diet)
as a major player in the development of Acquired Dental Disease

A summary and analysis of recent published research

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Metabolic Bone Disease and Dental Health

Metabolic bone disease first affects the alveolar bone (location of teeth sockets) because it is actively growing tissue. If it is not optimally mineralized, then the pressure of chewing becomes increasingly likely to cause the teeth to shift within the alveolar bone. Once the teeth become slightly maloccluded then improper wear can occur (this is the pattern identified as the cause of dental disease in the study performed by Frances Margaret Harcourt-Brown).

Dental disease is well known to be associated with the curvature of the cheek teeth. Curved cheek teeth (malocclusion) are not worn properly because they do not come in proper contact with other teeth (dental attrition) and as a result they are not directly involved in chewing food (dental abrasion). This is likely due to metabolic bone disease which provides a mechanism to cause the teeth to become curved. Improper wear occurs after the teeth have shifted within the alveolar bone.

To test the first hypotheses Müller et. al. studied the rates of tooth growth and tooth wear in rabbits fed exclusively on either whole timothy grass hay, or one of four complete pelleted diets (isocaloric and isonitrogenic) based either on alfalfa, timothy grass, timothy grass with rice hulls (more abrasive than timothy grass), or timothy grass with rice hulls and sand (2014).

The results: Müller et. al. clearly documented that **the rate of tooth growth varies according to the rate of tooth wear** (i.e. higher rates of tooth wear subsequently increases the rate of tooth growth). They also found that the rate of cheek tooth wear varies in a gradient depending upon the position of the tooth in the cheek tooth row (distal teeth are worn faster than proximal because they have more contact with food). Interestingly all diets had similar wear patterns with no signs that would indicate the development of dental disease except the diet containing sand which showed dental abnormalities because of excessive wear on distal teeth.

They also found that the "chewing action of cheek teeth is more independent of whether the diet is offered whole or pelleted." As a result they concluded that the previous belief that rabbits spend more time chewing pellets vertically (chomping pellets believed to be the cause of tooth spurs on cheek teeth because of improper wear) instead of the horizontal 'figure 8' type motion used to chew hay was INCORRECT. Instead their research suggests that rabbits actually chew hay and pellets the same way with their cheek teeth and that the wear patterns were not different.

In their own words: "Based on these findings, it is our opinion that diet alone may be less likely to cause dental problems in pet rabbits, due to flexible growth that reacts to wear. Other causes such as mineral imbalances or genetics should be considered when diagnosing dental disease, and a minerally balanced diet and breeding hygiene (not allowing affected animals to reproduce) may be the most promising prophylactic approaches." This means that they concluded that metabolic bone disease (hypothesis 2) is more likely the cause of dental disease in rabbits.

In contrast another report published less than a year later by Meredith et. al. concluded that a low intake of forage was primarily responsible for dental disease and that metabolic bone disease was not implicated in the development of dental disease (2015). They compared feeding rabbits a grass hay only diet, an extruded diet with hay, a grain-based muesli mixed diet with hay, and a muesli mixed diet only (without hay).

Regarding tooth wear they found very similar results clearly showing that tooth growth rate correlated strongly with tooth wear rate and that the effect on each tooth was independent. However they found that the muesli diet (with or without hay) lead to dental abnormalities and tooth spurs. Their conclusion was that the muesli diet did not wear the tooth surface properly and that abnormal tooth wear was implicated in the development of dental disease and not metabolic bone disease. To support their conclusion they calculated the effect that selective feeding of the muesli diet had on the balance and the percentage of dietary calcium and phosphorous.

However, I feel that their study actually supports the likelihood that metabolic bone disease was responsible for the observed dental disease. This is because the authors conclusion was not based upon measuring the actual calcium consumed and absorbed. If the authors had quantified the amount of food/hay consumed then a simple estimate of actual bioavailable calcium consumed (relative to protein/energy) would likely identify that the rabbits were indeed deficient in calcium and that the cause of the dental abnormalities are due to metabolic bone disease.

For example, the diets they fed reported to provide a total calcium of no more than 0.6%. This is borderline deficient in calcium because optimal bone mineralization requires diets at least as high as 1.0% (Noris et. al. 2001). Furthermore the alveolar bone (teeth sockets) is the most prone to being affected by a low-calcium diet. The authors did not verify the manufacturer's claim by measuring the actual calcium content of the diets they fed. Neither did they consider the effect of the bioavailability of the calcium (some of the dietary calcium is expected to be bound to plant material or the added dietary phosphorous which was high relative to the calcium in the diet). Neither did they consider the amount of food consumed among the different treatments (rabbits eat less volume when eating concentrated diets) and how it alters the total amount of calcium consumed (relative to dietary protein/energy).

Works Cited

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