SYMPTOMS AND CONSEQUENSES OF CONGENITAL FETAL HYPOTHYROIDISM IN MULE DEER (*Odocoileus hemionus*) POPULATIONS.

Judy Hoy, 2858 Pheasant Lane, Stevensville, MT 59870

DEVELOPMENTAL MALFORMATIONS ON MULE DEER FAWNS

Can you see any developmental malformation on this female mule deer fawn? Answer on page 15 of this document.

A. THE IMPORTANCE OF THYROID HORMONES IN FETAL DEVELOPMENT.

1. Proper levels of thyroid hormones are needed to direct normal growth of brain cells, nerves, bones, muscles, reproductive organs, eyes, ears, the immune system, especially the thymus, and other organs on a developing mule deer fawn.

2. Thyroid hormones from both the maternal and fetal thyroid glands are like production managers for fetal development. If the production managers are compromised or interfered with in some way, the final product (the fawn) is likely to be flawed in some way.

3. Malformations and other adverse health symptoms in newborns, including mule deer fawns as a result of disrupted thyroid hormones during development, is commonly called Congenital Fetal Hypothyroidism (CFH).

4. Malformations as a result of Congenital Fetal Hypothyroidism are not genetic, the changes are epigenetic.

B. OTHER HORMONES BEING DISRUPTED BY ENVIRONMENTAL TOXINS.

According to multiple studies sex hormones, including aromatase, are being disrupted by a number of environmental toxins. Vitamin D3, actually a hormone is disrupted by some chemicals used for pesticides (umbrella term), as is retinoic acid (Vitamin A). Some hormone disrupting toxins disrupt the normal functions of glutathione, essential for cellular respiration. The disruption of those hormones, which are absolutely necessary for normal fetal development, is likely the cause of the drastic increase in epigenetic changes to fetuses during development in the womb.

C. WHAT DOES EPIGENETIC MEAN?

Epigenetics is the study of changes in gene activity that do not involve alterations to the genetic code. Epigenetic changes can be passed on for one or several generations. The new patterns of gene expression are regulated by cellular material called the epigenome. The prefix, epi-, means above. The epigenome sits on top of the genome and just outside of it. These epigenetic “markers” on the genome tell the genes when to switch on or off and/or dictate the strength of the gene expression. Gene expression in a young animal during its development is influenced by various environmental factors such as nutrition, radiation, chemical exposure and diet of the mother. Epigenetic changes can be passed to the next generation or for several

generations, but the DNA of the genes is not thought to be affected. Thus, epigenetic changes to individual organisms represent a biological response to an environmental factor or factors, but do not change the DNA of the organism.

All cell types on one animal – brain cells, muscle cells, nerve cells, keratin cells, bone cells, etc.– contain the exact same DNA. Epigenetic switches silence certain gene sequences and activate others, so that embryonic cells can differentiate. If the switches do not work correctly, the cells may begin to continuously reproduce, resulting in cancer.

Or premature cell death may occur, resulting in failure of organs to develop or function correctly. It takes only the addition of a methyl group, one carbon atom attached to three hydrogen atoms, to change an epigenome. When a methyl group attaches to a specific spot on a gene, it can change the gene's expression, turning it off or on, lessening its expression or increasing it. Results of such epigenetic changes to developing young can range from deadly to bizarre and sometimes beautiful.

D. THE MOST COMMON EPIGENETIC CHANGES CAUSED BY CONGENITAL FETAL HYPOTHYROIDISM (CFH) ON RUMINANT SPECIES, INCLUDING MULE DEER.

1. Mild to severe Brachygnathia Superior (BS), which is underdevelopment of the bones of the upper face, especially the premaxillary bone. Also called underbite, it is the most common CFH symptom on ruminants, including mule deer.

2. Mandibular Brachygnathia (MB) is the underdevelopment of the lower jaw, resulting in overbite. This does not appear to be as common on ruminants as BS, but has a prevalence of over 5% on mule deer in Montana. Any malformation with a prevalence of over 5% is suppose to be significant.

3. Death just prior to birth or miscarriage sometime during fetal development.

4. Inability to maintain heat and energy, resulting in failure to get up and suckle after being born, resulting in death soon after birth.

5. Umbilical hernias resulting in death sometime after being born.

6. Contracted tendons in leg joints, causing difficulty standing, walking and running, usually resulting in the death of the affected newborn.

7. Limbs or portions of limbs, especially the digits, not developed at all, only partly developed, crooked or otherwise malformed often resulting in death soon after birth.

8. Small eyes and occasionally, problems with sight or total blindness, resulting in death.

9. Disrupted immune system because of underdeveloped or damaged thymus causing newborns to be susceptible to infections and/or parasites, which can cause death.

10. Damage to internal organs, especially the heart, lungs and liver.

11. Enlarged right heart ventricle sometime with dilated vessels on the heart surface.

12. Fluid build-up in the lungs causing damage to lung tissue.

13. Disrupted liver function affecting newborn ruminant's ability to detoxify chemicals and affecting ability to digest milk, resulting in diarrhea, dehydration and often death.

14. Dished face with eyes facing more forward than normal for a ruminant species.

15. Underdeveloped skull bones or rounded skull.

16. Rolled or floppy ears.

17. Underdeveloped male genitalia, including no, short and/or misaligned scrotum and short penis sheath on the external skin.

18. Abnormal pedicle placement and/or abnormal antlers or horns on male ruminants.

19. On live animals, brittle hair and hair loss is a common symptom of severe thyroid hormone disruption.

20. On live animals, the inside of the eyelids (the conjunctiva) should be checked for inflammation. Conjunctiva that is reacting to airborne toxins is red and swollen. The conjunctiva on dead animals should always be checked for inflammation, especially if the animal dies of "natural causes." Being poisoned by toxins can't really be considered a natural cause.

E. BRACHYGNATHIA SUPERIOR AND MANDIBULAR BRACHYGNATHIA.

• Brachygnathia Superior is the underdevelopment of the upper facial bones, especially the premaxillary bone forward of the premolars.

• Brachygnathia Superior is commonly called underbite because the lower incisors are forward of the premaxillary pad on ruminants and forward of the upper incisors on other mammals, including humans.

• Mandibular Brachygnathia is the underdevelopment of the lower jaw forward of the premolars and is commonly called overbite.

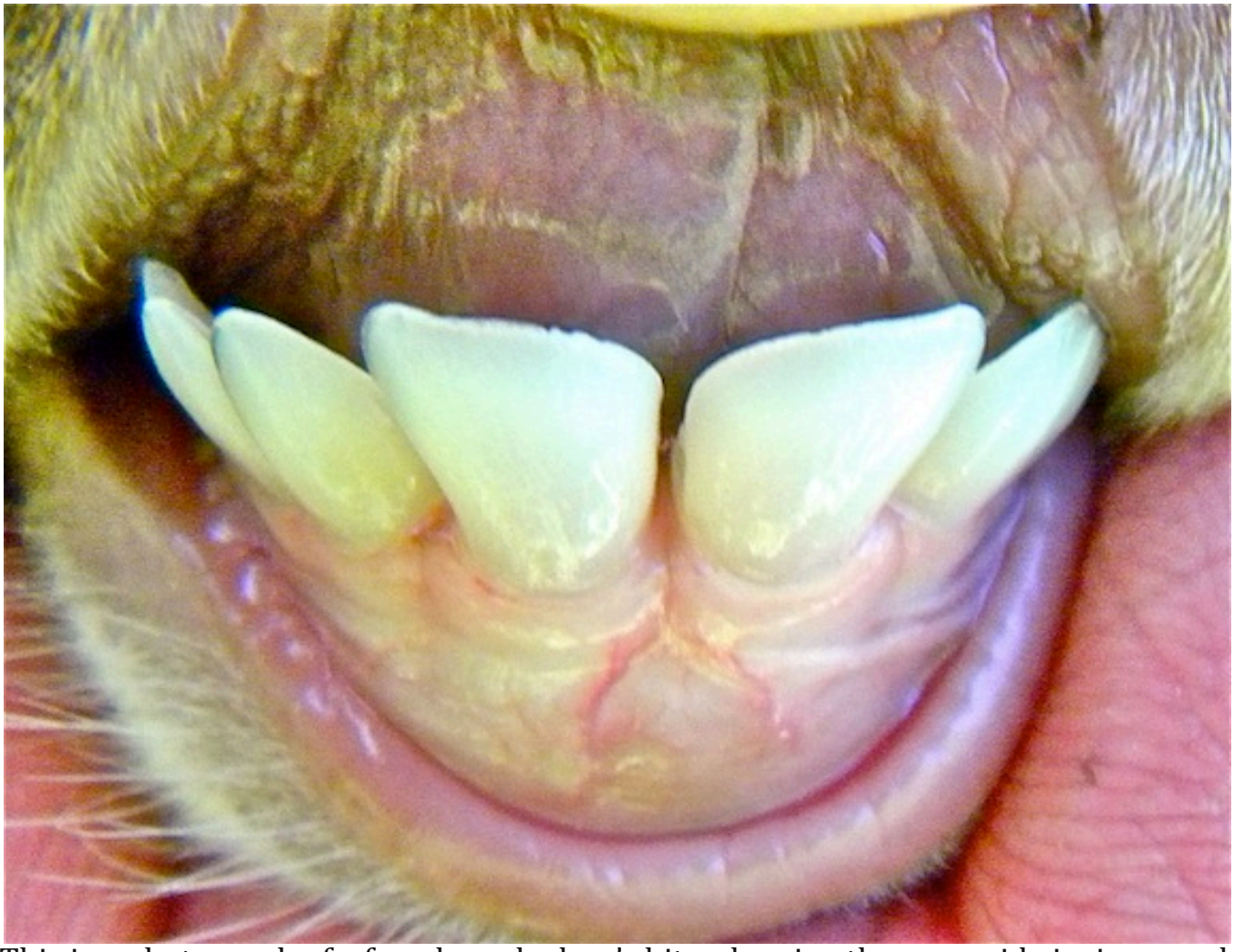
• Many newborn ruminants, including white-tailed deer and mule deer have abnormally shaped, crooked incisors, often wider than normal for the species.

• High rates of ungulates are being born each year with BS in Montana and other western states. These include elk, mule deer, white-tailed deer, pronghorn antelope, big horn sheep, bison and domestic animals such as goats, cattle, horses, sheep, llama and alpaca. A few individuals of some ruminant species are born with MB, but not at nearly as high in prevalence as BS. It is the opposite on humans, with MB and underdeveloped chin presently being much higher in prevalence than underbite, according to dentists consulted.

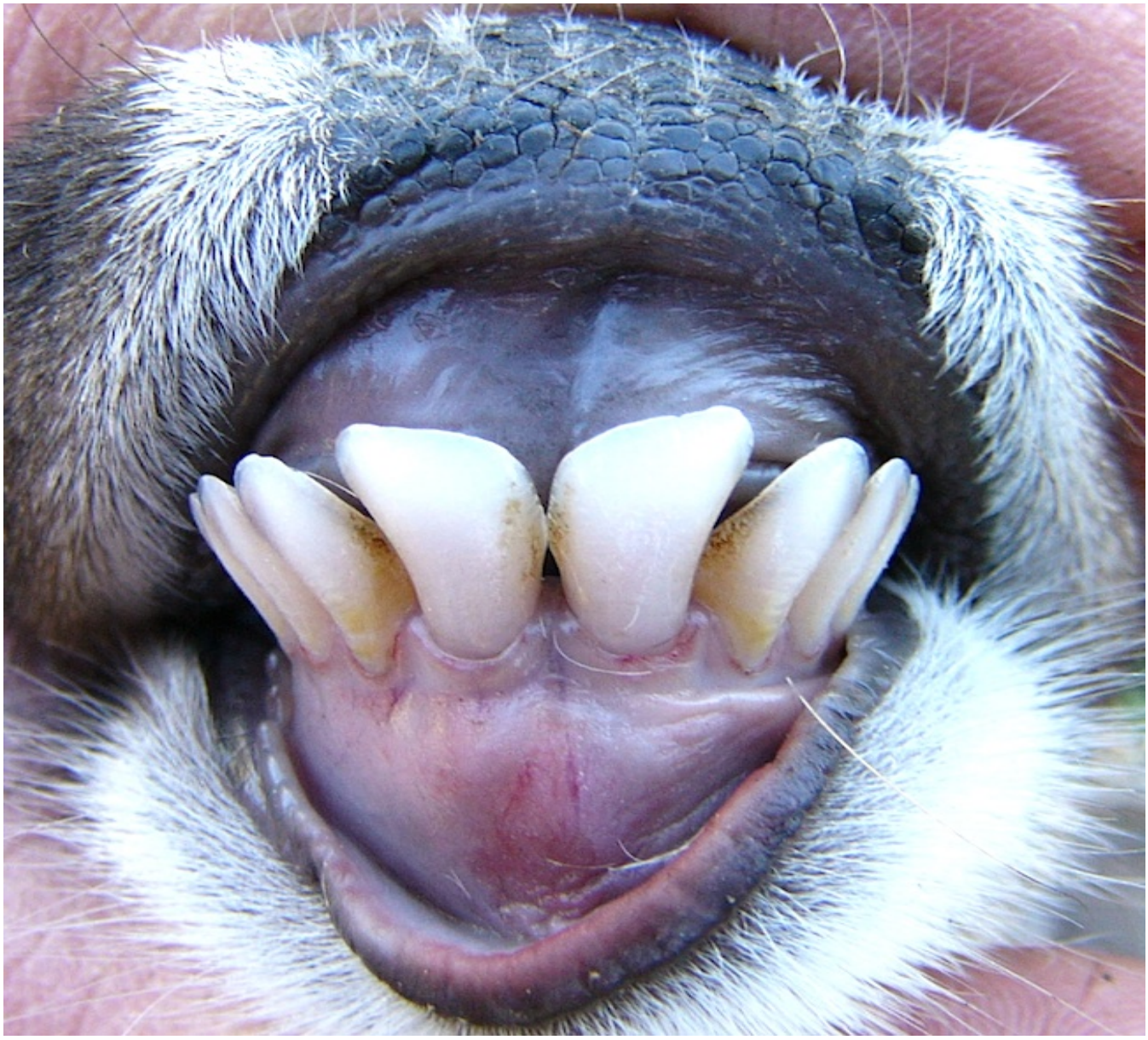
• BS, MB and malformed lower incisors appear to be caused by disruption of the epigenetic programming directing normal calcium uptake in the facial bones that have growth adversely affected during development.

• CFH symptoms which result in mortality prior to or soon after birth, have been at least partly responsible for the declines in wild ruminant populations, including/especially mule deer, because of non-survival of the young. There are not enough young surviving to be born, or surviving after being born to provide enough mule deer for all the predators, including human hunters and still maintain adequate population levels. See our 2011 study for discussion of the many factors affecting the ruminant populations and the domino effect the factors have on populations. This study is on Google. (J.A. Hoy, G.T. Haas, R.D. Hoy and P. Hallock, Observations of Brachygnathia Superior in wild ruminants in Western Montana, USA. (December 2011)Wildlife Biology in Practice.) doi:10.2461/wbp.2011.7.13

F. PHOTOS OF BRACHYGNATHIA SUPERIOR ON MULE DEER FAWNS



This is a photograph of a female mule deer's bite, showing the very wide incisors and the narrow premaxillary bone. On a fawn with a normal bite, the lower incisors all contact the premaxillary pad, so the deer can bite off forage.



This is the front view of another mule deer fawn with very severe brachygnathia superior, widely spaced lower incisors with doglegged middle incisors.

To determine if a captured animal or a dead animal has brachygnathia superior and abnormal lower incisors, just pull the lips apart and look. It takes only seconds and can tell a biologist volumes concerning the health and survivability of the animal. During examination of the head of a captured or dead animal, also check the inside of the eyelids of the eyes (the conjunctiva) for inflammation. This tells you if the immune system of the animal is reacting to toxins and/or bacteria. On a dead animal, the tissue can be collected and checked for bacteria. If there is no bacterial infection, the animal is reacting to environmental toxins. One would think this would be standard procedure because it tells a great deal about an animal's health. What is causing the inflammation of the conjunctiva on a fawn that is just found dead, with no apparent reason for the death, is important in determining the true cause of death.

This live female mule deer fawn has an obvious underbite as evidenced by the lower lip being even with the upper lip. On a deer with a normal bite, the lower lip is tucked in behind the upper lip. This condition is easily observed with binoculars on fawns in the wild. If the fawn is photographed, the fairly severe underbite is easily determined. However, a fawn can have a very mild underbite and the lower lip will not be even with or anterior to the upper lip, so only fairly severe brachygnathia superior is determinable by using binoculars or photography.

To determine if a captured animal or a dead animal has brachygnathia superior and abnormal lower incisors, just pull the lips apart and look. It takes only seconds and can tell a biologist a great deal concerning the health and survivability of the animal.

During examination of the head of a captured or dead animal, also check the inside of the eyelids of the eyes (the conjunctiva) for inflammation. This tells you if the immune system of the animal is reacting to toxins and/or bacteria. On a dead animal, the tissue can be collected and checked for bacteria, there is significant

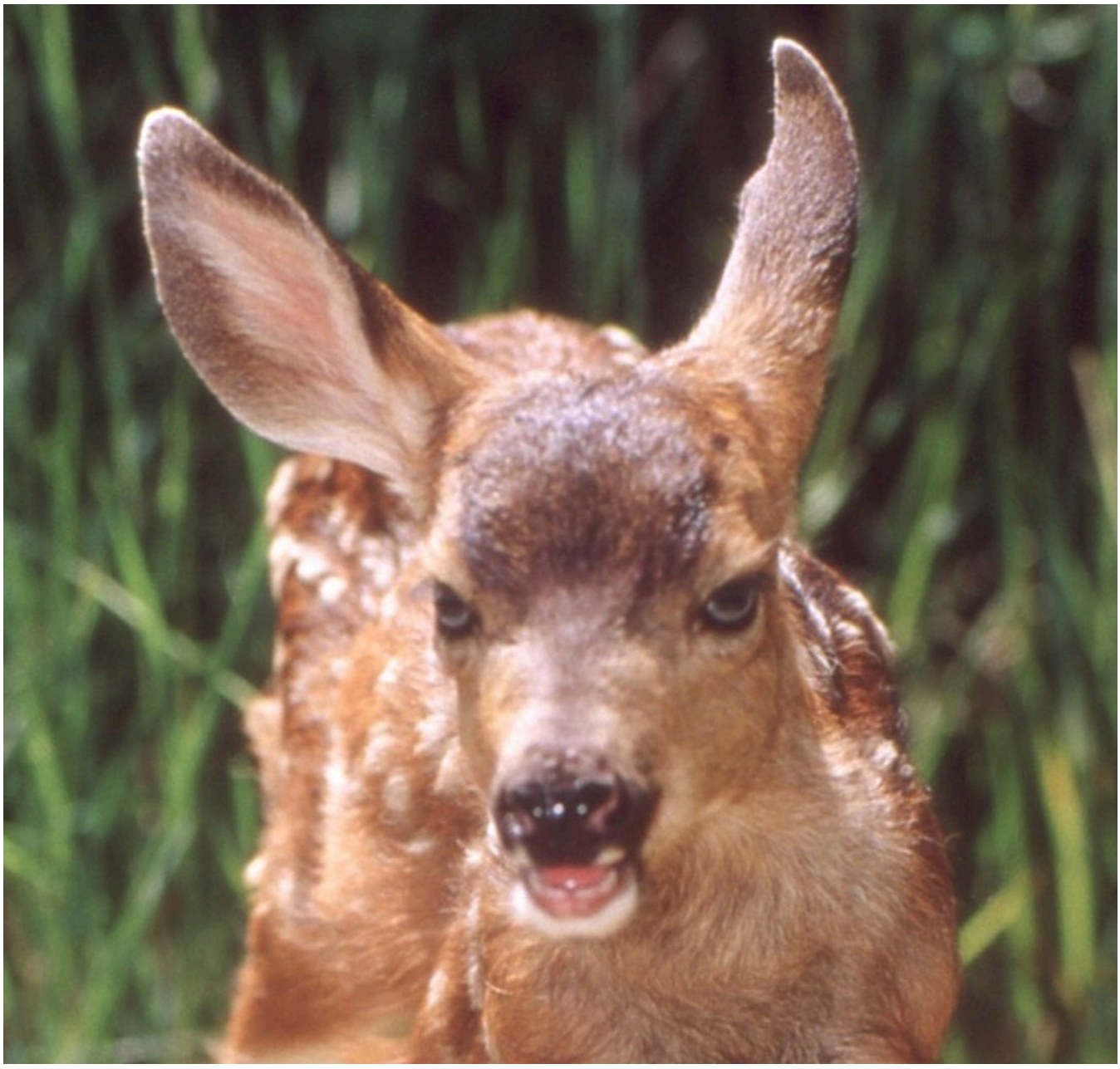
inflammation. If there is no bacterial infection, the animal is reacting to environmental toxins. One would think this would be standard procedure because it tells a great deal about an animal's health. What is causing the inflammation of the conjunctiva on a fawn that is found dead, with no apparent reason for the death, is important in determining the true cause of death.

If the right ventricle of the heart is enlarged, especially with dilated vessels on the surface, the lungs are inflamed or have fluid build-up as evidenced by the uneven lung surface or white areas in the lung tissue and the thymus is underdeveloped or has red spots throughout, the fawn was born with CFH. Those are all definitive symptoms of CFH.

This male fawn was brought for rehab because of a shattered right femur incurred when hit by a vehicle. This photo was taken 16 days after the bone was broken in the

accident. The bone was completely healed in 12 days and the fawn walked and ran with no limp when this photo was taken on the 16th day. This fawn had brachygnathia superior/underbite and the lower lip is almost even with the lower lip.

The four live fawns in the photos on pages 5, 8, 9 and 10 were received for rehabilitation. I examined the bite on all four fawns by lifting the lips and directly observing the underbite. All the photos of live mule deer fawns were of different fawns in different years.

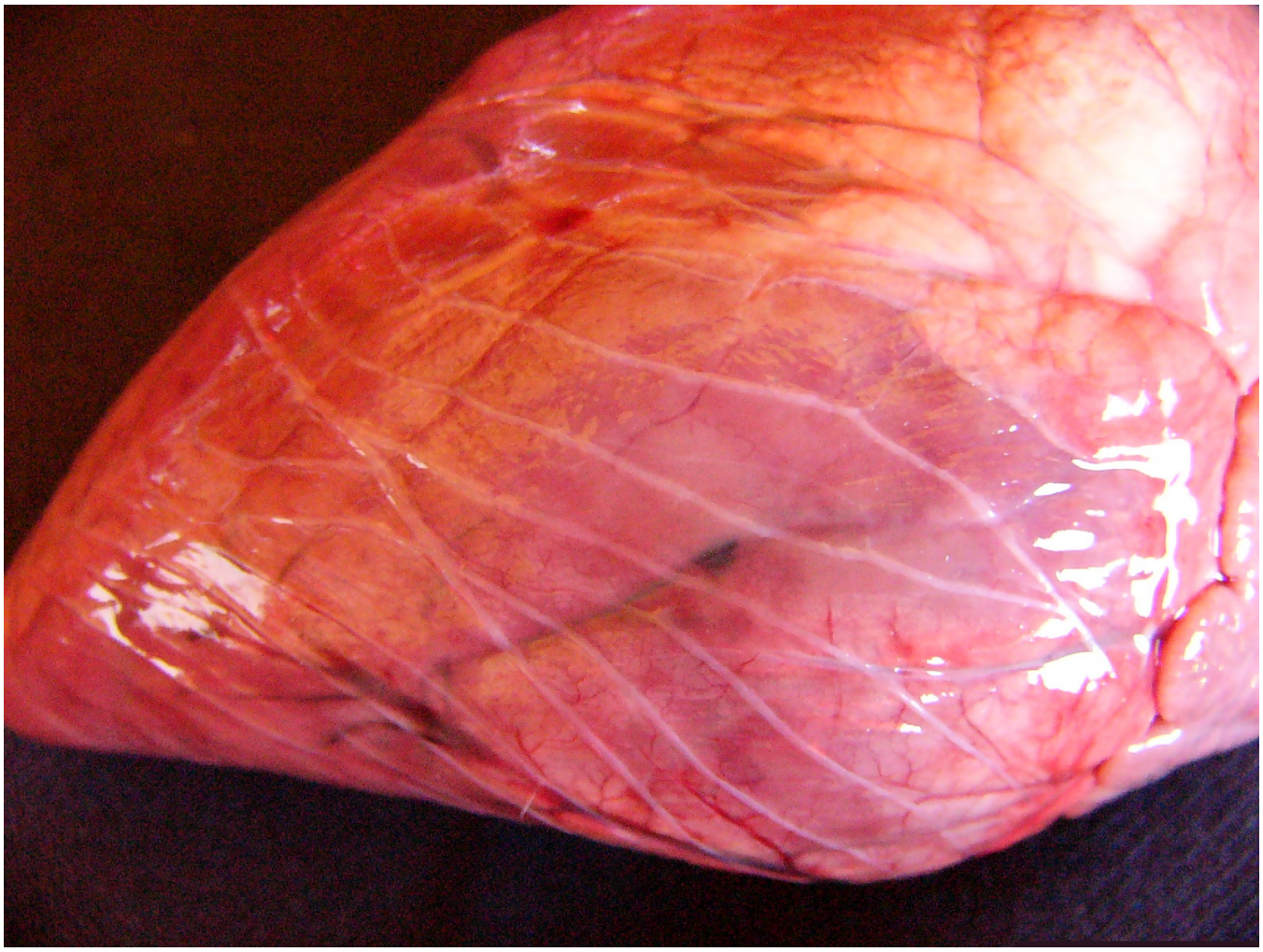


This female mule deer fawn has fairly severe underbite and the incisors are wider than the dental pad. One reason for the wide incisors was the middle incisors have a wide gap between them as well as the incisors being wider than what used to be the norm. Her incisors completely surrounded the dental pad with no contact.

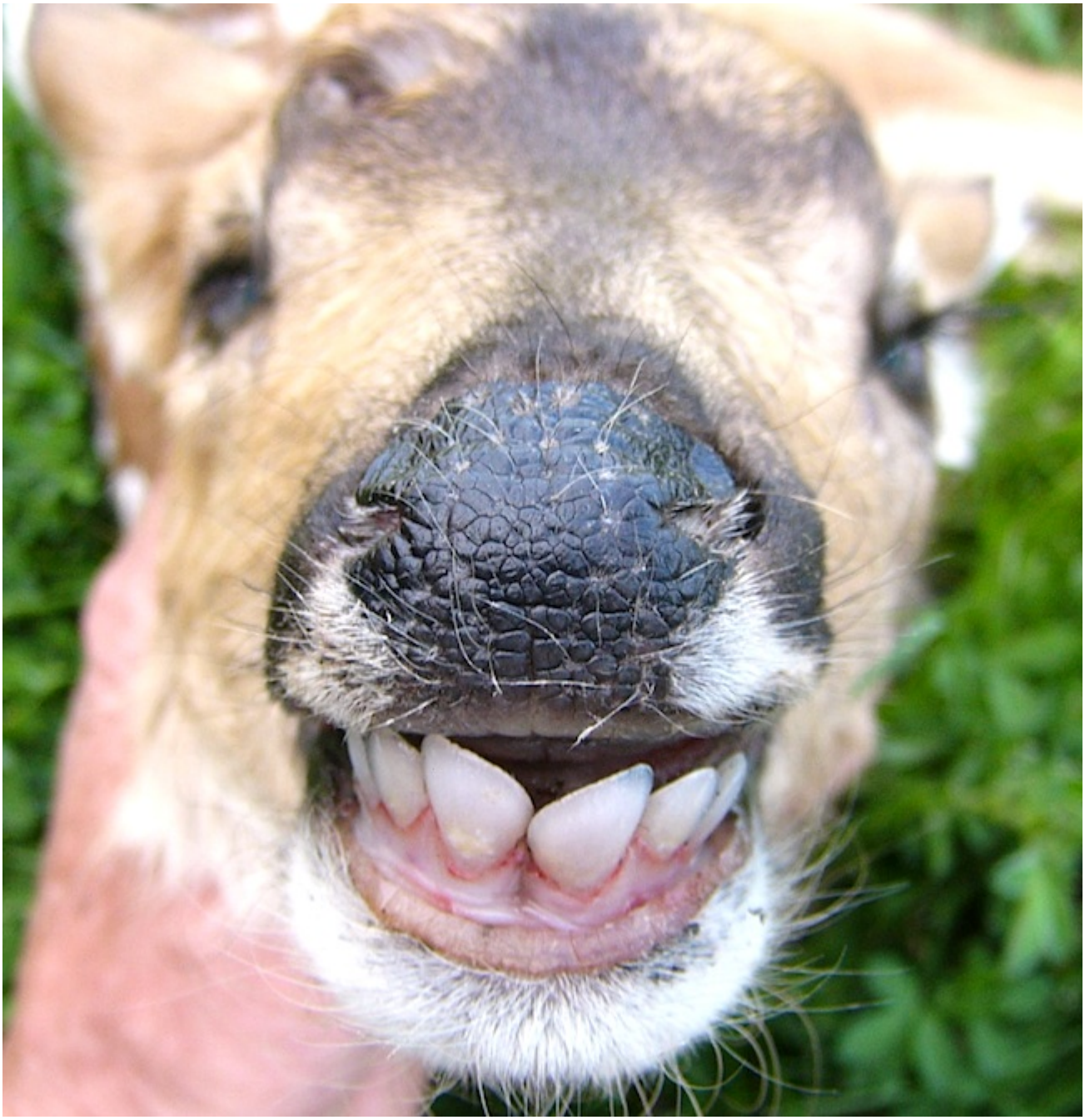
This is a white-tailed deer fawn with all the incisors contacting the premaxillary pad in what has always been considered a normal bite on a ruminant for comparison with the mouth of the mule deer with underbite on page 12.

The mule deer fawn in the two photos on page 12 has a very narrow dental pad, much narrower than the incisors. The middle incisors are doglegged in shape being much wider at the top than at the bottom.

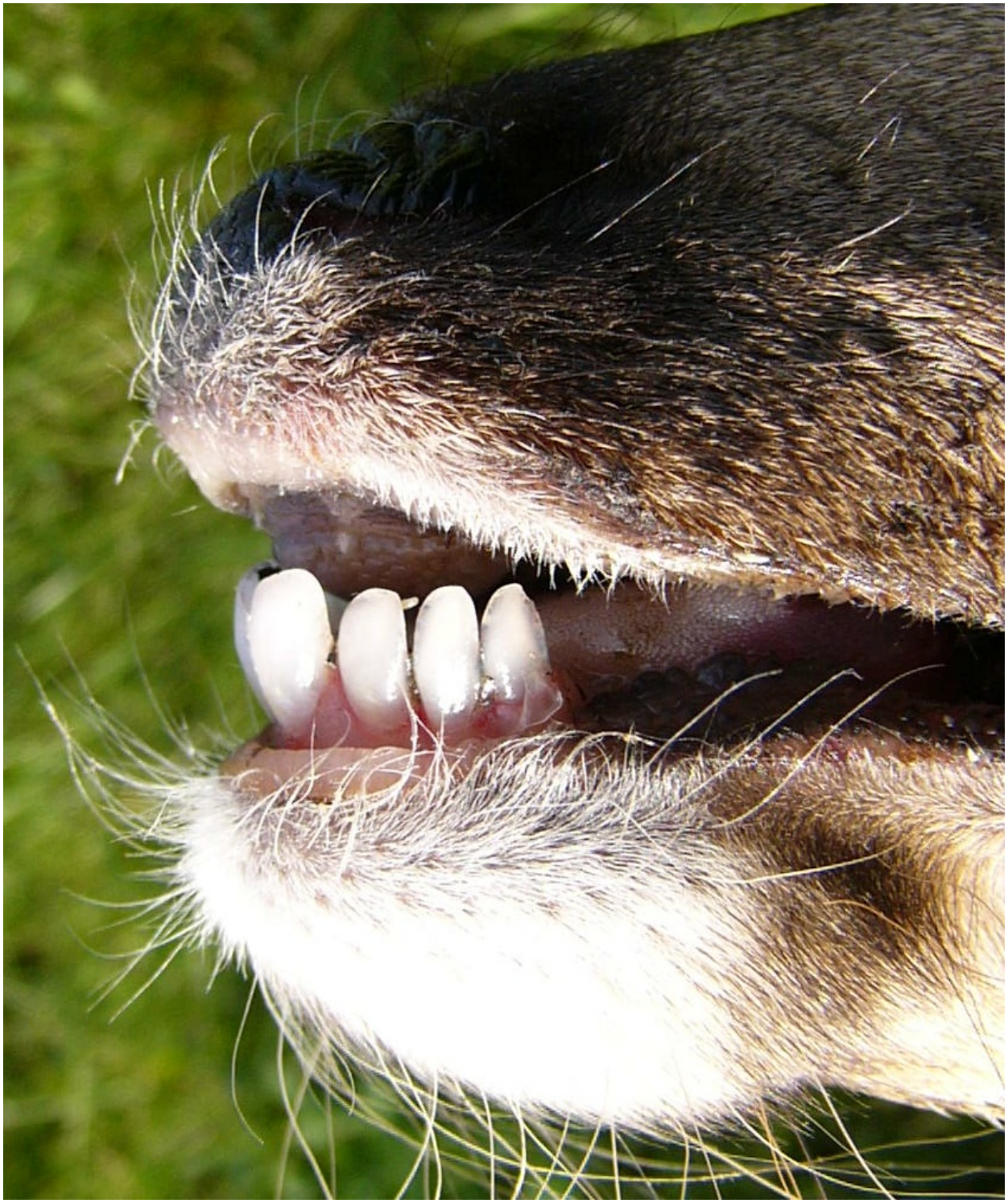
The bottom photo shows the side view of the same fawn and the significant underbite with the lower incisors forward of the dental pad rather than contacting it like in the photo of the fawn above. It is easy to examine a dead animal or an animal that has been captured for research for brachygnathia superior/underbite and for blepharitis. Brachygnathia superior is a definitive symptom of Congenital Fetal Hypothyroidism. Blepharitis indicates the animal is being affected at the time of examination by exposure to toxins to which it is sensitive.



This is the heart from the mule deer fawn on page 12, showing the very severely dilated vessels on the heart's surface. No one has been able to tell me what causes this condition. Between 2007 and 2009 most young animals, including many birds, I necropsied had dilated vessels on the heart surface. Brachygnathia superior more than doubled in 2007 to over 70% on white-tailed deer fawns from a prevalence of 33% in spring of 2006. What happened to cause the severe disruption of the thyroid hormones in so many young animals in spring of 2007 is unknown. Testing should be done to find what caused this, as the big game populations have declined significantly since 2007. Unfortunately, wolves, mountain lions and bears are being blamed, not the real culprit, which is Congenital Fetal Hypothyroidism.



This photo shows the protruding lower incisors on a female mule deer fawn that died from multiple symptoms of Congenital Fetal Hypothyroidism. Note the incisors, especially the middle incisors are crooked and very wide. All of the incisors are much wider than the dental pad and forward of the front of the dental pad as shown in the next photo on page 15, a side view of this fawn's underbite. This fawn also was narrow between the eyes and had smaller than normal eye openings. She had an enlarged right ventricle of the heart and an underdeveloped thymus.



Side view of the above female fawn's underbite. The incisors completely encircled the dental pad without touching it at any point. This would have made it hard to bite off foliage if this fawn had survived to be old enough to eat plants. Congenital Fetal Hypothyroidism causes high mortality in mule deer fawns.

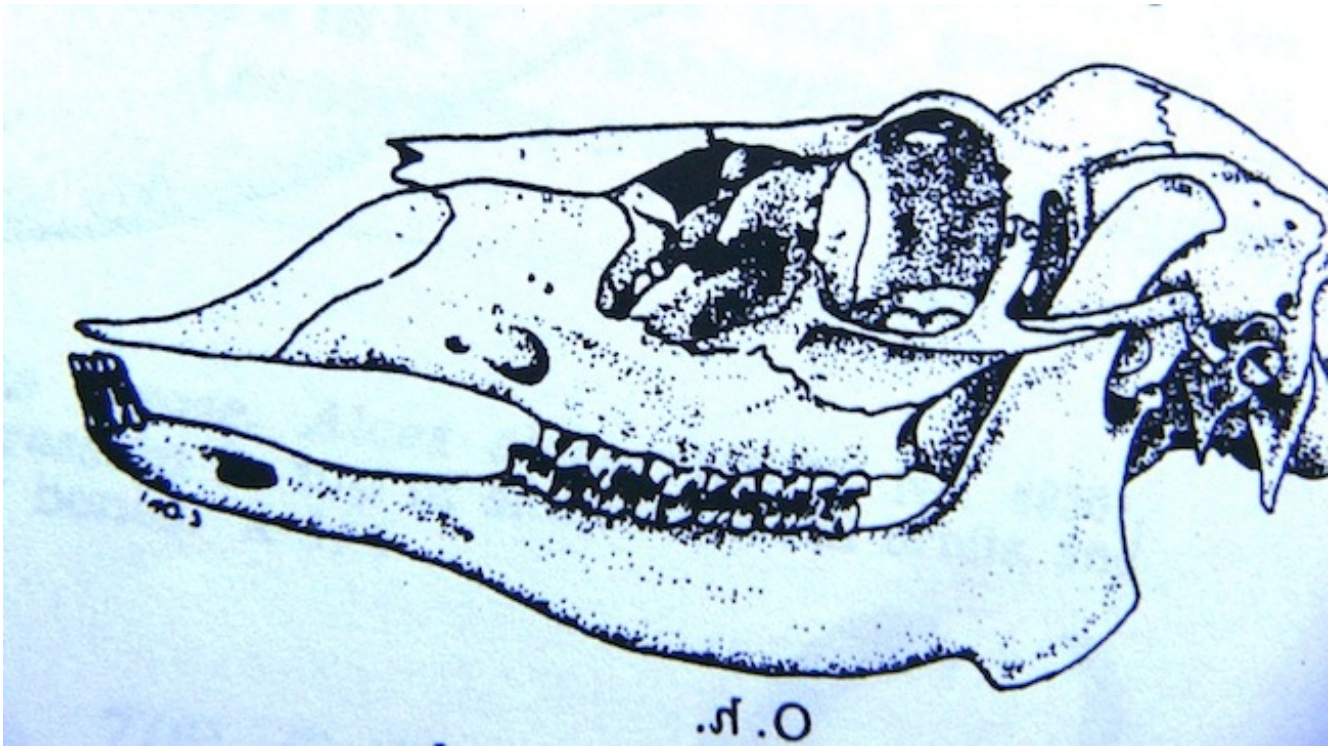
Answer to question on page 1. The lower lip is forward of the upper lip, with the pink inside of the lower lip showing. This observation indicates the fawn has an underbite/brachygnathia superior, and this fawn did have a fairly severe underbite as I found with direct examination.

DEVELOPMENTAL MALFORMATIONS ON ADULT MULE DEER

Since many CFH symptoms on fawns result in early death, the most common symptoms seen on adult deer are underbite, overbite, malformed teeth, malformed hooves and no, half or misaligned hemiscrota, with ectopic testes and/or short penis sheath on the external skin. Adult deer also have enlarged right heart ventricle, fluid in the lungs or white areas in the lung tissue, inflammation of the inside of the trachea and blepharitis.

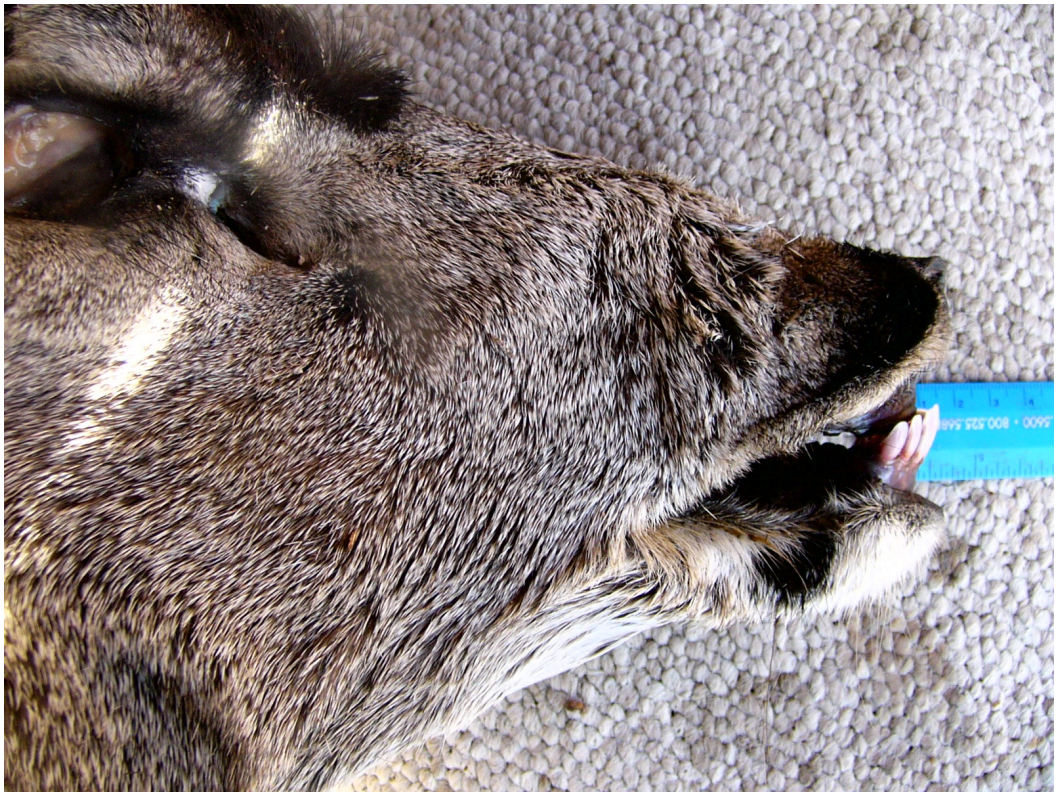
A. PHOTOS OF BRACHYGNATHIA SUPERIOR ON ADULT MULE DEER

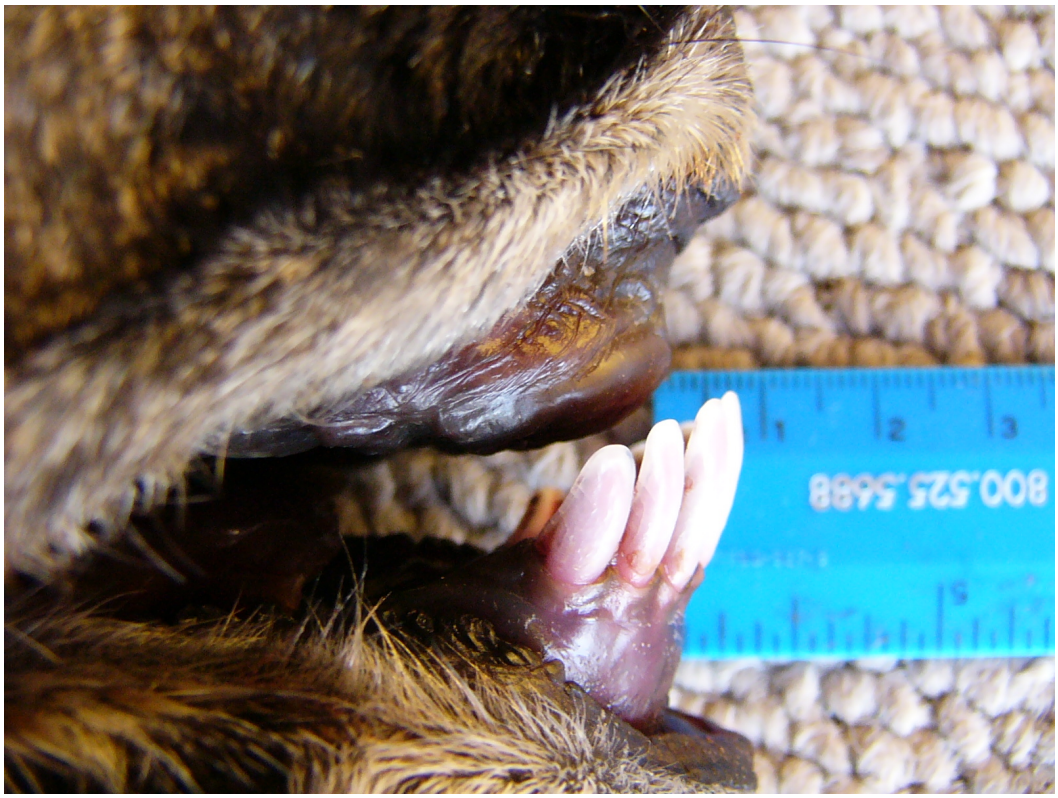
This photo of a cleaned mule deer skull shows a front view of the overgrown incisors with abnormal wear on the top because of the plant material being pulled across the top of the incisors when the deer tried to bite off the foliage. Also note the somewhat disintegrated bone on the front of the lower jaw and the absence of bone around the roots of the incisors. This condition is likely caused by being exposed to toxins on the foliage that are calcium blockers. Calcium disruption, in addition to depletion of important minerals in the foliage the deer eats, results in this type of bone loss.



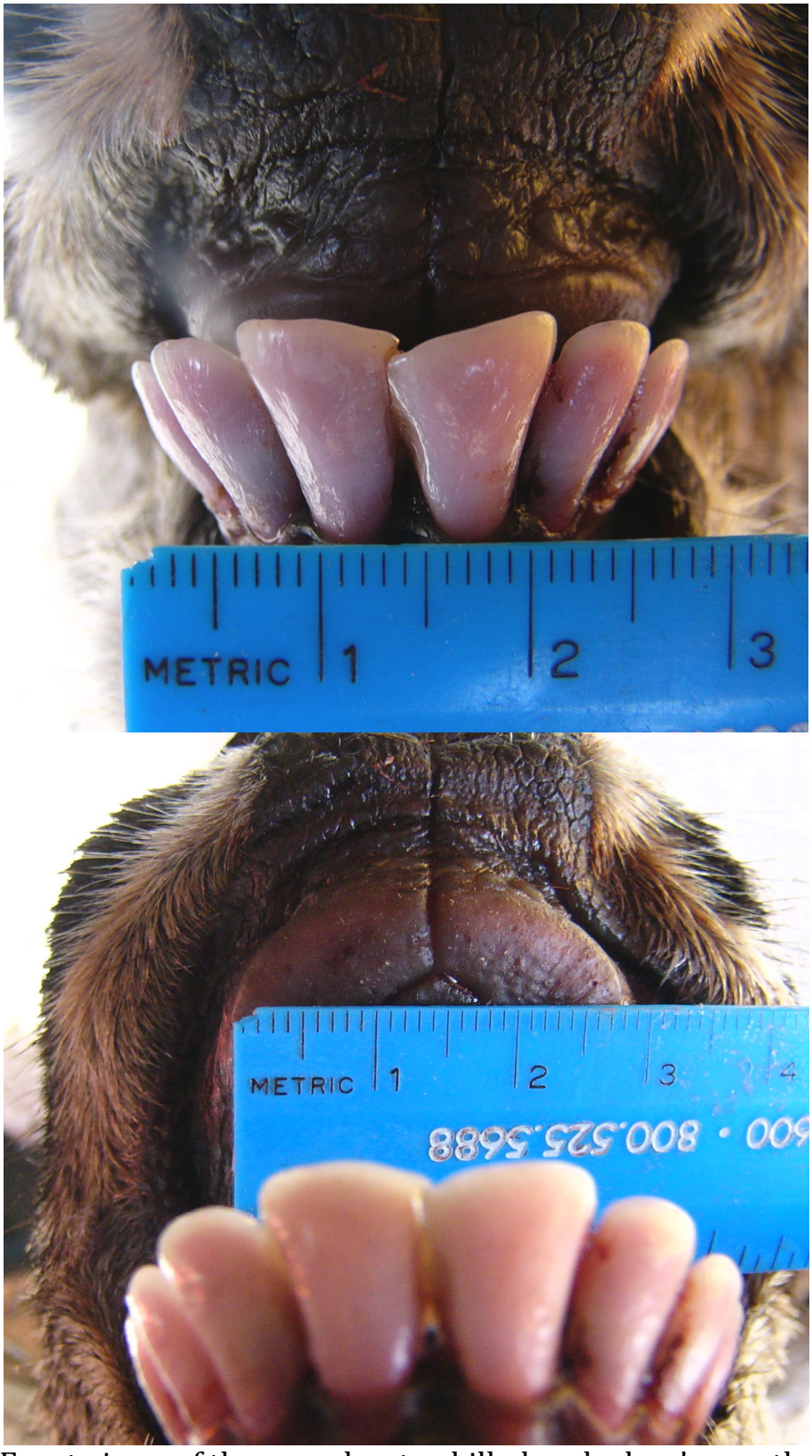
A drawing of an adult mule deer skull from a college biology textbook published prior to 1995. The drawing shows the premaxillary bone is normal in length, so the lower incisors would contact the premaxillary pad when the soft tissue was present.

This is the cleaned skull of an adult male mule deer hunter-killed after 1995, showing the short narrow premaxillary bone with the lower incisors surrounding the anterior of the bone rather than contacting it.

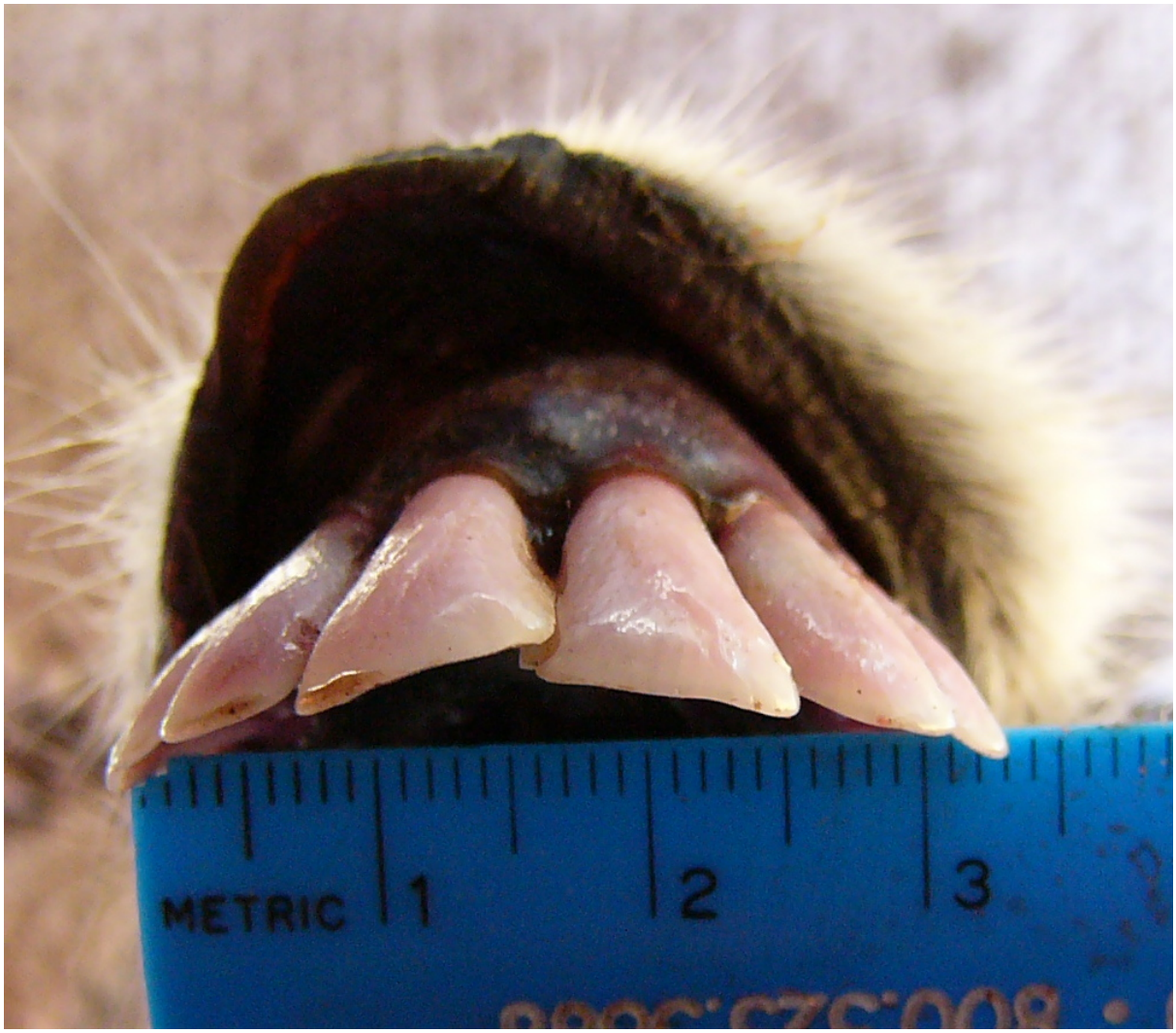




These are photos of the whole head and the mouth of a hunter-killed mule deer from Eastern Montana. Note how far forward of the dental pad the lower incisors were. For our study, I measured from the anterior of the dental pad to the top of the middle incisors. The under bite measures 8 mm from where the incisors should contact the dental pad if the bite was normal. The normal contact point is approximately 2 mm posterior to the front edge of the dental pad on an adult mule deer.



Front views of the same hunter-killed mule deer's mouth showing the crooked middle incisor and that the incisors were significantly wider than the dental pad. This deer had serious brachygnathia superior.



This photo shows the exact measurement of the width of the lower incisors, which is 3.3 cm. The dental pad measures 2.9 cm, a difference of 4 mm. Thus the lower incisors completely encircled the dental pad, making it difficult for this deer to get adequate nutrition.

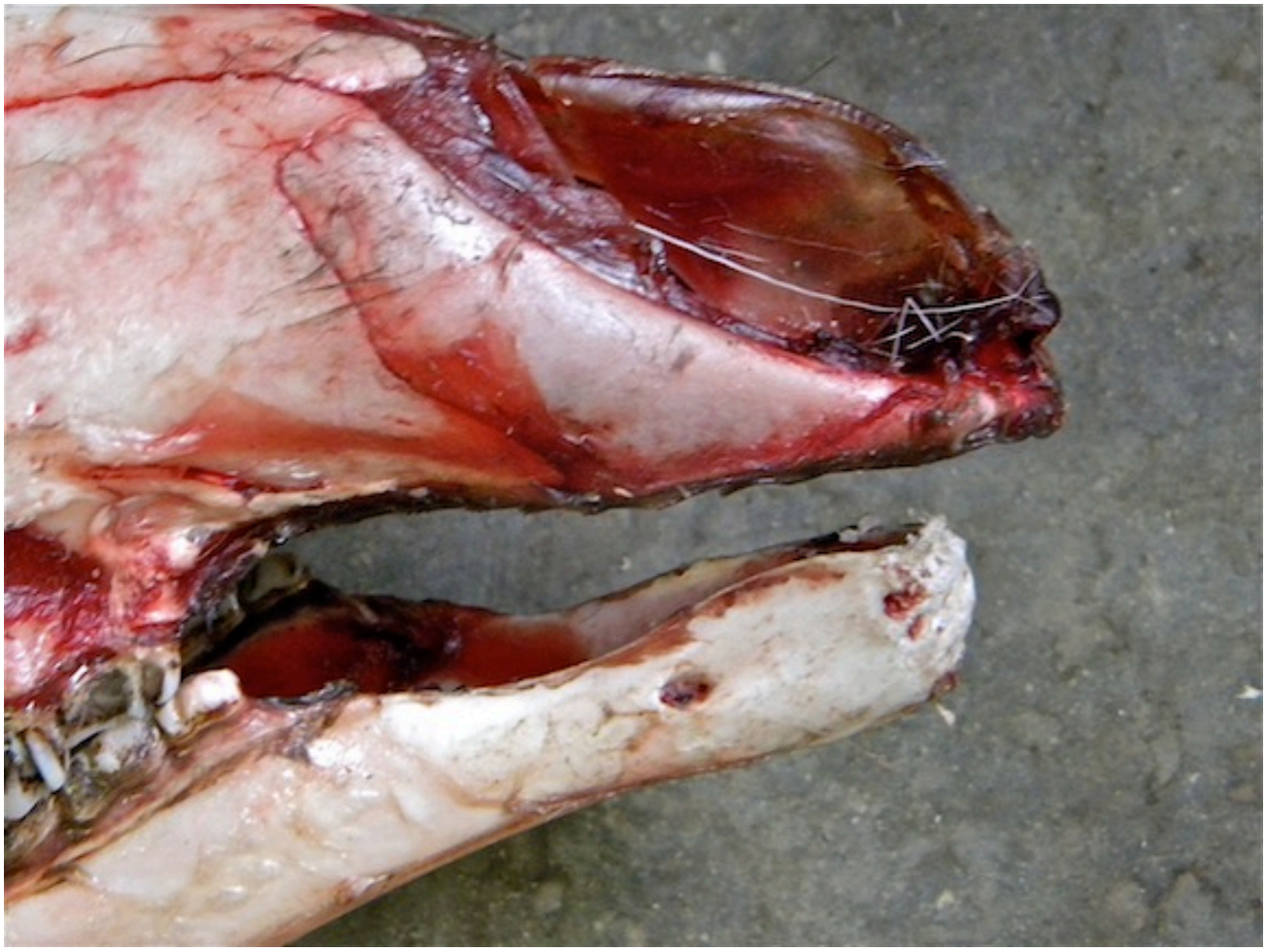
Another hunter-killed male mule deer from Eastern Montana with slight underbite and normal shaped lower incisors. The lower incisors are not wider than the dental pad.

B. PHOTOS OF MANDIBULAR BRACHYGHATHIA ON ADULT MULE DEER.

Mandibular brachygnathia is the underdevelopment of the lower jaw forward of the premolars. The prevalence on hunter killed male mule deer from Montana is 8%. This deer was killed by another male mule deer in a fight in the fall of 2011.

The bottom photo on page 22 shows the side view of the severe overbite on this male mule deer. The photo below shows where the incisors contacted the roof of the mouth. It also shows that the wider than normal incisors are considerably wider than the dental pad so incisor three and the canine tooth on each side do not contact the pad. This strongly indicates that the lower incisors are programmed by an epigenetic change during fetal development to be wider than normal as is shown in the previous photos of brachygnathia superior on mule deer fawns. This same abnormally wide incisor malformation is also evident on elk, white-tailed deer, domestic goats and individuals of most other ruminant species, wild and domestic.

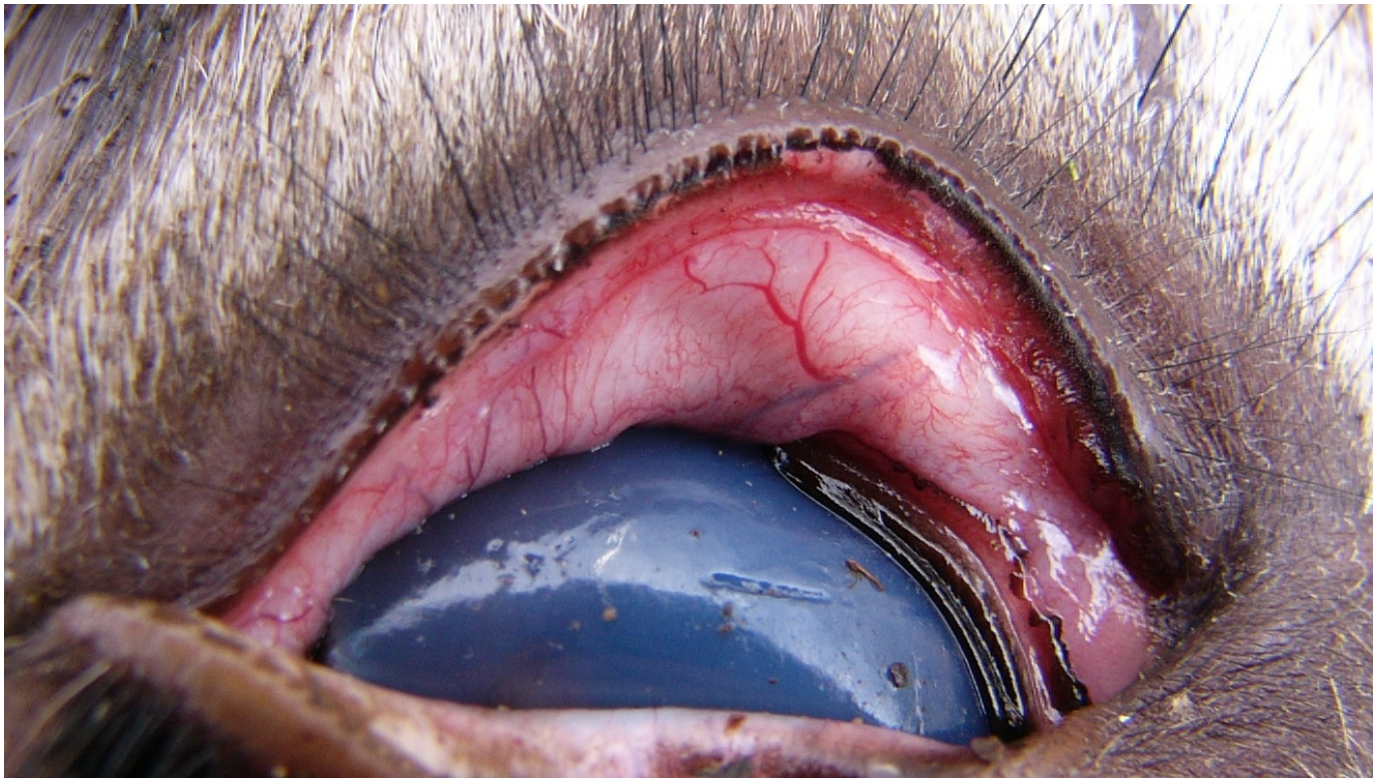
These conditions as well as blepharitis could easily be checked for on heads collected from hunters or on accident-killed animals with the heads undamaged, but usually are not assessed for by wildlife professionals. An example of blepharitis of the eyelids on a deer is shown on page 25. The head can tell a great deal about the animal and what was affecting its health just prior to being killed by hunters or accident, if these factors are examined.



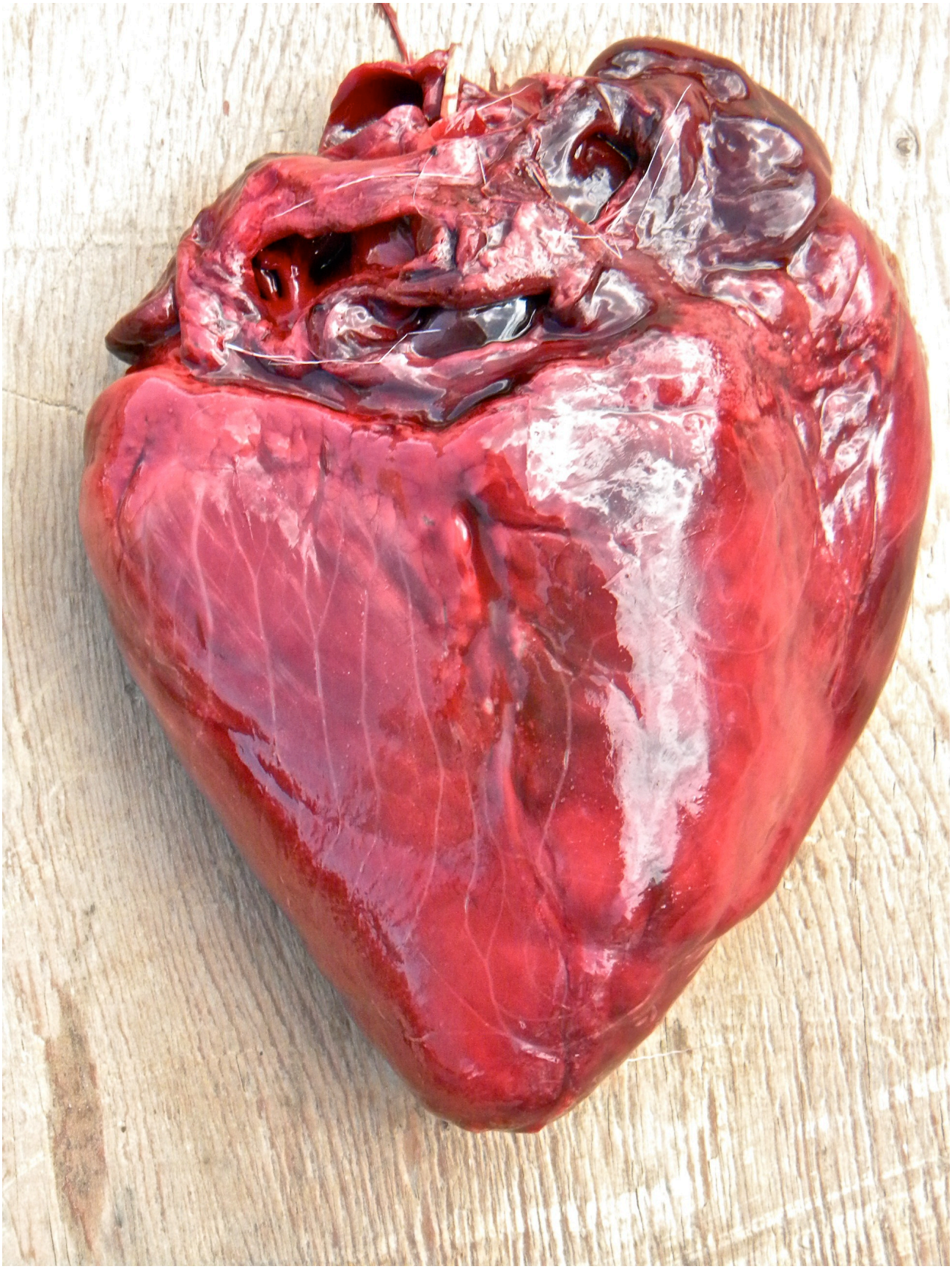
Another male mule deer hunter-killed in fall of 2011, which had very severe mandibular brachygnathia (MB). The whole front of the lower jaw was not properly developed, with no incisors present. How this deer got enough to eat is a mystery. It likely bit off foliage with the premolars by using the side of the mouth to chew off the plants. Mandibular brachygnathia is much more prevalent on animals from Eastern Montana, including white-tailed deer, antelope and mule deer than on animals from Western Montana. This is likely due to exposure to somewhat different environmental toxins during development, thus turning off the epigenetic switch for the growth of the lower jaw bone forward of the premolars, rather than the epigenetic switch for development of the premaxillary bone.

The underdevelopment of the facial bones on vertebrates, including the bones of the upper or lower bills on birds is an epigenetic change, not a genetic change affecting the DNA. We have proven that on many animals and birds. If you are interested in that information, just email me and ask for it. This document is just addressing how to recognize the epigenetic changes. How to make some changes grow to normal on a newborn or newly hatched animal is another document.

C. PHOTOS OF OTHER CFH SYMPTOMS ON ADULTS.



This is a photo of moderate blepharitis of the conjunctiva of a white-tailed deer. Mule deer also often have blepharitis. On a dead animal, conjunctiva unaffected and normal would be white, not red, with inflammation.



This heart was from an adult male mule deer. The right ventricle on the right side in the photo was slightly enlarged and there were visibly dilated vessels on the heart surface, most visible on the left side in the photo. A vehicle killed the deer.

D. PHOTOS OF ABNORMAL ANTLERS ON ADULT MALE MULE DEER.

A mule deer with very abnormal antlers had his photo taken in Colorado Springs, Colorado. This type of growth is usually the result of undescended testes causing a lack of testosterone, thus the antlers do not loose the velvet or drop. As new branches and tines grow out each year, the antlers become very large and bizarre in configuration. This mule deer does not appear to have underdeveloped facial bones and underbite, thus would be able to eat normally. However because the testes were likely cryptorchid, this deer would have not been able to produce viable sperm. This is a very serious birth defect.

Misplaced pedicles on deer and elk have been observed occasionally since the developmental malformations began in 1995. These two late summer mule deer with antlers in the velvet were photographed by Eugene Beckes on the National Bison Range north of Missoula, MT. The upper deer has a misplaced right pedicle that is closer to the center of the forehead than normal.

This young hunter-killed male mule deer killed in Ravalli County, Montana had testes undescended through the inguinal rings, which are called “cryptorchid” testes. Consequently, the antlers had remained in the velvet. The deer also had obvious brachygnathia superior/underbite. Cryptorchid testes appear to be more common on male mule deer than on male white-tailed deer in Montana. Of hundreds of male white-tailed deer examined, none were found to have cryptorchid testes. All testes on white-tailed deer were descended through the inguinal rings into the hemiscrota if the hemiscrota was formed on the external skin. Or the testes were descended into a horizontal position between the body wall and the external skin, which is referred to as “ectopic” in the literature. On those animals, very short or no hemiscrota were formed during fetal development, a very serious birth defect.

One other male mule deer besides this deer, of approximately 10 adult male mule deer examined had one testis, the right testis, “cryptorchid” or up by the kidney. That cryptorchid testis was much smaller than normal. The left testis was normal in size and in the left hemiscrota. That mule deer had only the left hemiscrota formed thus had half of a scrotum.

E. PHOTOS OF ABNORMAL GENITALIA ON ADULT MALE MULE DEER.

This mule deer photographed in Western Montana on the National Bison Range by Eugene Beckes has a normal bilateral scrotum that hangs straight down containing the testes away from the body wall. This has always been the normal configuration for a scrotum on a mule deer and other male ungulates.

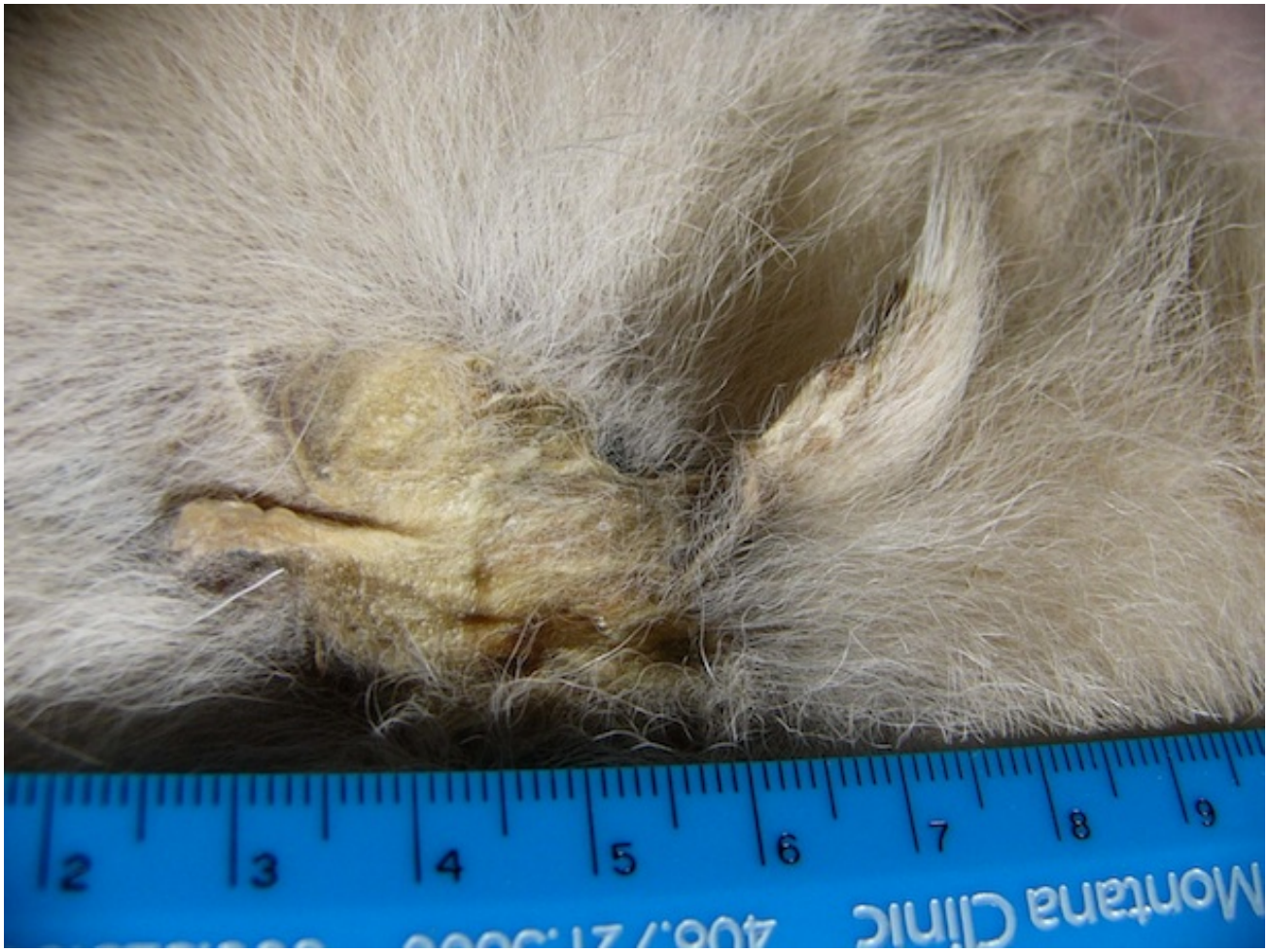
A mule deer that was hunter-killed in Western Montana in November of 2000 had the left hemiscrota directly forward of the right hemiscrota and the scrotum somewhat shorter than the testes. This deer had a normal length penis sheath (on the left in the photo).

Many male mule deer have misaligned hemiscrota with the left testis and hemiscrota directly forward of the right testis and hemiscrota. This malformation occurs early in fetal development when the testes descend to form the scrotum on the malleable fetal skin. This scrotal condition has been observed and photographed on many live male mule deer, but the prevalence of this malformation in the mule deer population has not been determined. It is over 70% on male white-tailed deer in Western Montana and mule deer appear to be more severely affected by Congenital Fetal Hypothyroidism than white-tailed deer. On hunter-killed male deer checked for brachygnathia superior, 48% of the adult white-tailed deer had jawbone malformations 38% with underbite and 10% with overbite. On hunter-killed mule deer males, 75% had jawbone malformations, 67% with underbite and 8% with overbite. Brachygnathia superior and mandibular brachygnathia are definitive symptoms of Congenital Fetal Hypothyroidism.

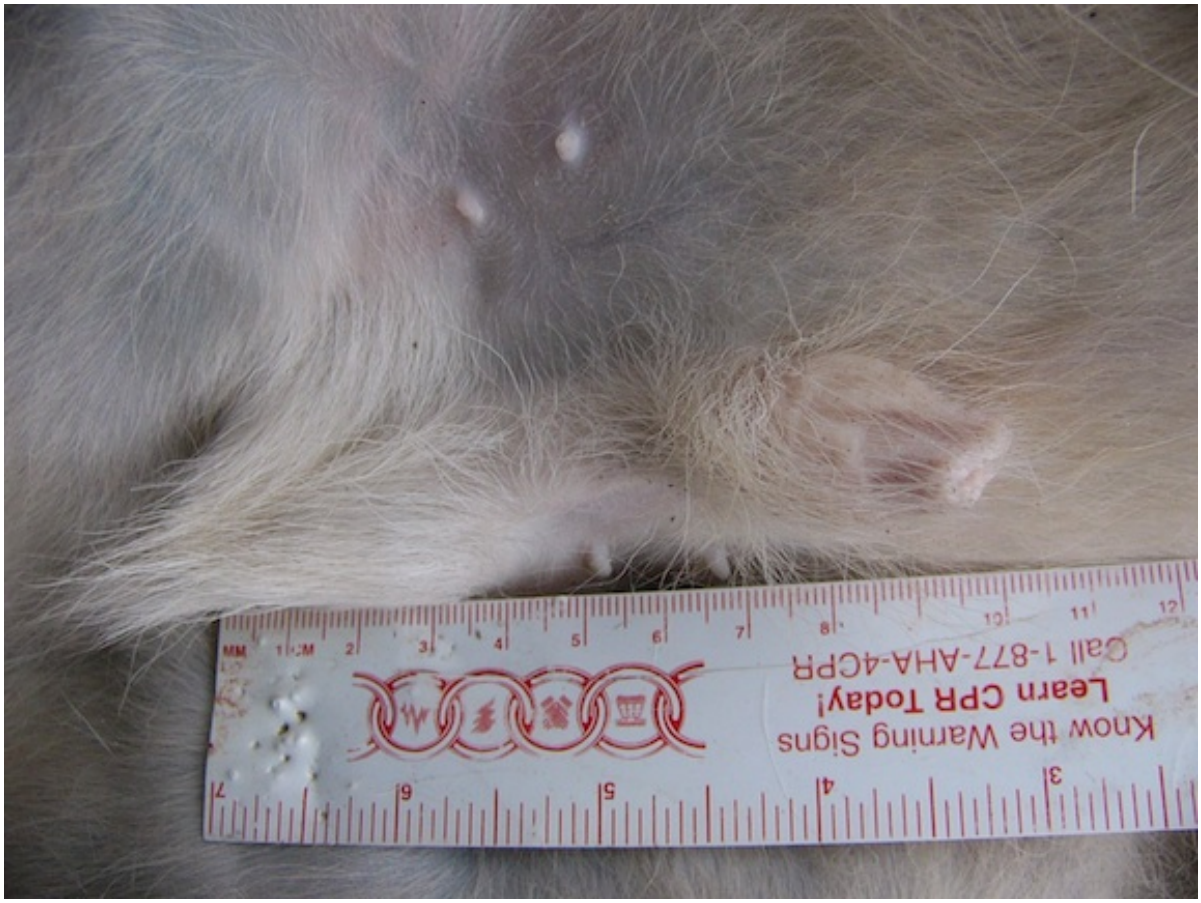
Since mule deer have a higher prevalence of jaw malformations than white-tailed deer, it can be assumed that male mule deer likely have at least as high a prevalence of misaligned hemiscrota as male white-tailed deer. On male white-tailed deer fawns since 2007, the prevalence of misaligned hemiscrota is around 70%, making this scrotal configuration now the norm. This may possibly be the fastest evolution of a mammal organ ever documented in human history.

The scrotal sac on this yearling male mule deer contains the testes well away from the body and is bilateral as used to be the norm on male deer.

The scrotum on this live adult mule deer appears to be misaligned because the left testis can barely be seen in front of the right testis, which is clearly visible. This older mule deer's scrotum is somewhat short, compared to the normal scrotum on the deer in the previous photo of a live yearling mule deer. This photo was taken by Eugene Beckes on the National Bison Range.



This is the dried genitalia of a newborn mule deer fawn. The two hemiscrota on this fawn consisted of two separate tiny flaps of skin where the scrotal sac should have been (directly above the 3 on the ruler). The normal sized testes were horizontal in a bilateral position between the skin and the body wall, thus were ectopic. Both testes were normal in size for a newborn fawn. A short flap of empty skin is the same as no scrotal sac with regard to affects on function. The flap of skin as can be seen had the left hemiscrota directly forward of the right hemiscrota, but the hemiscrota were much too short to contain the testes.



This mule deer fawn had a very short, bilateral scrotum and a somewhat short penis sheath. The teats were in the normal position between the front of the scrotum and the rear of the base of the penis sheath.

A short penis sheath is a very concerning malformation, because being born with the entire penis very short or with hypospadia is becoming much more prevalent on human male newborns. This has been shown and discussed in several studies, especially those done by Dr. Shanna Swann. Wild animals are test subjects, by which we can determine likely effects on human newborns. They are the canaries in the coalmine.

According to the dentists I consulted in our area, overbite with underdeveloped chin bone is the most prevalent facial bone malformation on human newborns and children. Interestingly, in 2001, the news media reported an epidemic of underbite in children throughout the world, including the U.S.

The other common symptoms in studies of animals with CFH deliberately caused by exposure to cyanide or other thyroid hormone disrupting toxins are diabetes, obesity, high cholesterol, heart defects and respiratory disorders at birth. Those are the health problems reported in the news media each day to be epidemic in human children, especially those under a year old. Autism has now been found by studies to be present on other vertebrates and have been shown to be the result of an epigenetic change to the brain during development of the fetus, just like the other epigenetic changes addressed in this document.