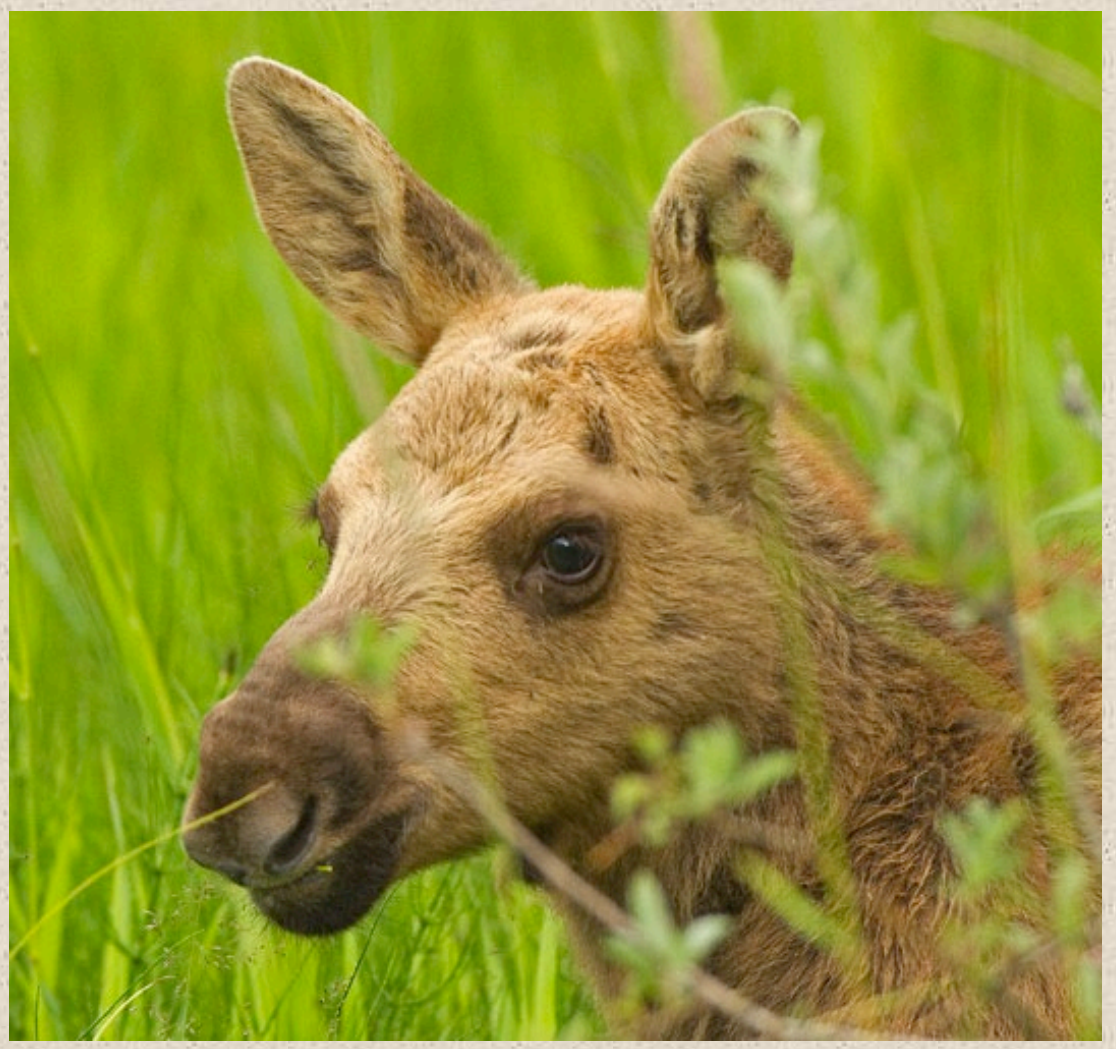
Observations of Birth Defects on Moose (Alces alces)

The most common birth defect on all ages and sexes of ungulate species is Brachygnathia Superior, commonly called underbite.
It is the result of the premaxillary bone and dental pad being underdeveloped.
This moose calf has an underbite as evidenced by the lower lip being slightly forward of the upper lip.



Produced by Judy Hoy, Independent Wildlife Research

Common symptoms of mineral deficiencies and congenital fetal hypothyroidism on ungulates.

Many of these symptoms, but not all, have been observed on moose in Montana and Wyoming, because not many moose have been directly examined.

1. Mild to severe Brachygnathia Superior (BS), resulting in underbite. This is caused by underdevelopment of the bones of the upper face, especially the premaxillary bone forward of the premolars, possibly caused by mineral deficiencies.

2. Mild to severe Mandibular Brachygnathia (MB), resulting in overbite. This is caused by underdevelopment of the lower jaw forward of the premolars.

3. Underdevelopment of the male genitalia, especially the scrotal sac, most common is genital hypoplasia possibly caused by caused by Zinc deficiency and and misaligned hemiscrota, possibly caused by disruption of specific genes.

4. Inability to maintain heat and energy, resulting in failure to stand and suckle.

5. Premature birth.

6. Umbilical hernias resulting in a painful death after birth.

7. Underdevelopment of the thymus so the immune system is compromised.

8. Underdevelopment or non-development of organs, including the brain.

9. Curled ear tips or unformed external ears.

10. Small eye openings, no eyes formed, problems with sight and/or blindness.

11. Limb bones or portions of limb bones not completely developed, resulting in crooked legs.

12. Dilated lymphatic vessels on the heart surface and enlarged right ventricle on both newborns and adults.

13. Contracted tendons and/or curved joints resulting in difficulty in standing and walking at birth.

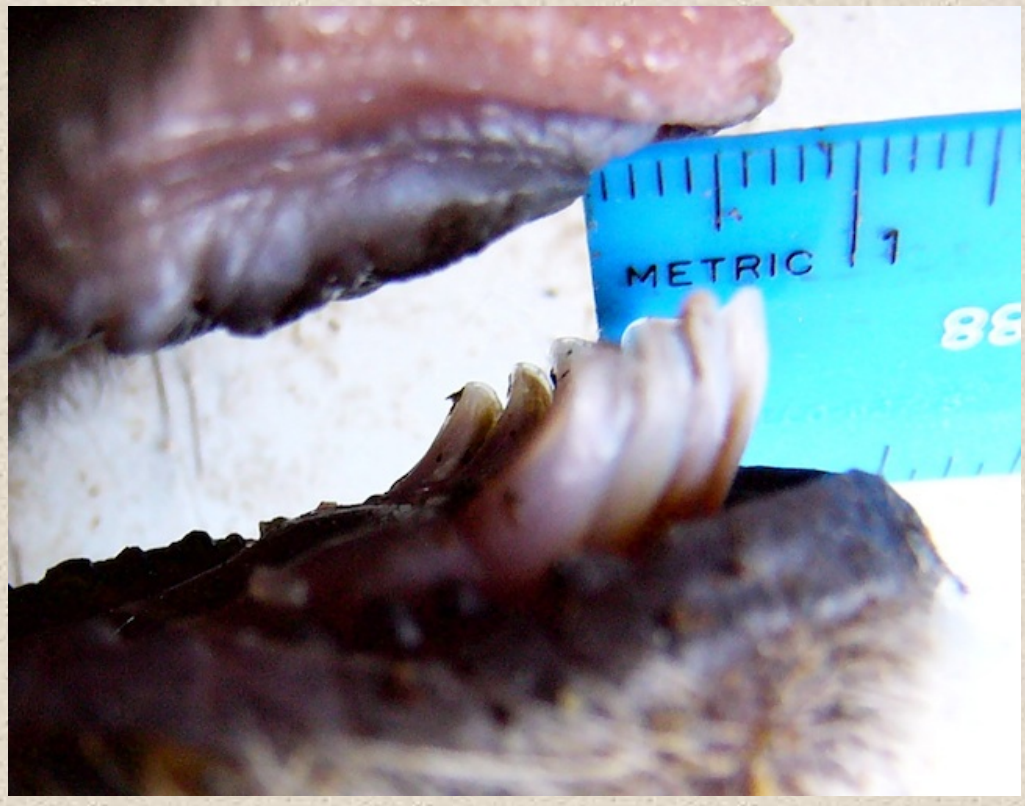
Brachygnathia Superior (underbite)

Brachygnathia superior is the result of underdevelopment of the bones of the upper face, particularly the premaxillary bone forward of the molars. Multiple studies have shown this birth defect to be caused on ungulates by mineral deficiencies, particularly manganese deficiency and by fetal hypothyroidism. Since spring of 1995, the premaxillary bone has been both narrower and shorter than normal, on multiple species of wild and domestic ungulates. Brachygnathia superior results in underbite of various amounts from mild to severe and sometimes small eye openings. A grazing animal with underbite is often not able to procure adequate nutrition, especially in winter. This is especially true of female moose with other health problems, such as laminitis, high tick loads or other health issues. Consequently, pregnant females with brachygnathia superior usually have young with even more severe adverse health effects consistent with mineral deficiencies and congenital fetal hypothyroidism (CFH). This is because nutritional deficiencies in pregnant females, added to and as a result of maternal and fetal exposure to environmental toxins, can cause a cumulative effect of more symptoms and more serious symptoms of CFH in subsequent generations as a result of inherited epigenetic changes.

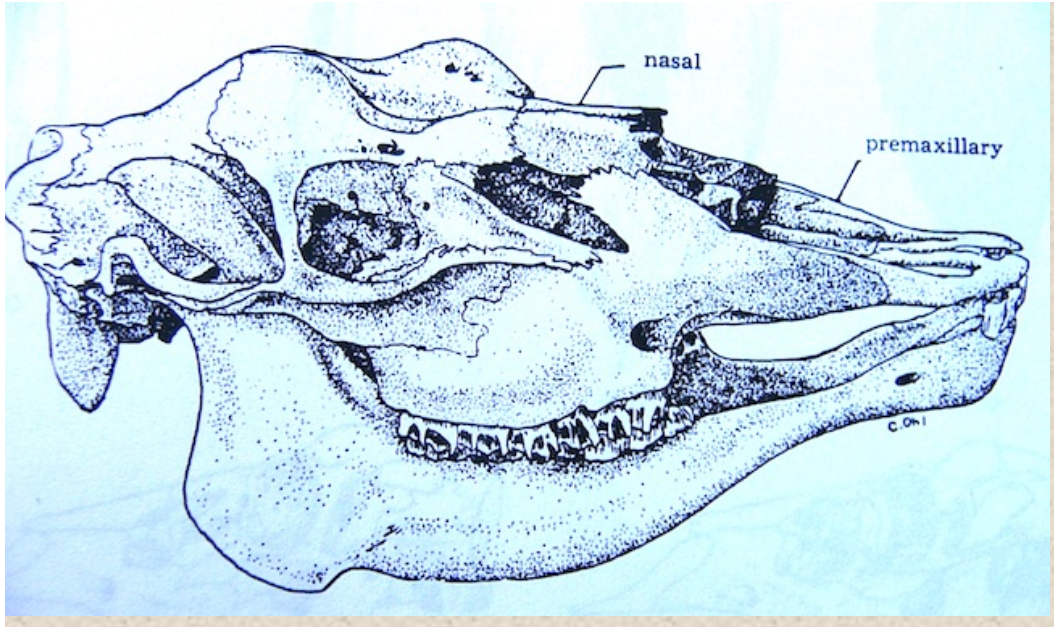
A normal bite appears the same on deer as on a moose or other ruminant.

On a ruminant species all of the lower incisors should contact the dental pad posterior to the extreme anterior of the pad. Prior to spring 1995, all ungulates and other animals, including birds, examined, observed or studied in Western Montana had a normal bite.

Measuring Brachygnathia Superior on cervids.



Measurements of underbite were taken on ruminants with a ruler, from the front of the dental pad to the top of the middle incisors as shown in the above photo. The photo upper left shows where the lower incisors contact with a normal bite on a white-tailed deer. The photo lower left shows a fairly severe underbite on a mule deer.



A drawing of a moose skull from a biology book.

In the photo on the bottom left is an adult male moose skull with underbite. Compare the position of the incisors in relation to the anterior of the premaxillary bone with the same in the biology book drawing. The photo on the top right is the same moose skull from the front showing the incisors and disintegrated bone at the anterior of the lower jaw holding the roots of the two middle incisors.

These photos show various amounts of underbite on five hunter-killed adult male moose from Montana and Idaho. The moose in photo #4 had five incisors on the left side. and only a slight underbite. The #2 moose had a slight underbite and had the middle incisors missing or possibly removed.

Reproductive birth defects on ungulates;

misaligned hemiscrota and genital hypoplasia.

Genital Hypoplasia on examined male ungulates greatly increased in some years and decreased in others (see Figure 1.). Maternal mineral deficiencies, particularly zinc deficiency, prior to conception and/or early in the gestation period have been shown to cause genital hypoplasia. The male genitalia is formed early in fetal development, beginning with the testes, followed by the other internal and external sex organs. Cellular zinc levels have a strong influence on the 5-alpha reductase inhibitor, which converts testosterone into dihydrotestosterone (DHT). DHT levels are instrumental in the normal growth and development of the external male reproductive organs because DHT bonds to androgen receptors more effectively than do other natural androgens. Thus the length of the hemiscrota and penis sheath on the external skin are determined by zinc and DHT levels. The years when the penis sheath and hemiscrota on WTD fawns are short would indicate years when maternal zinc levels are low, indicating possible exposure to mineral chelating toxins.

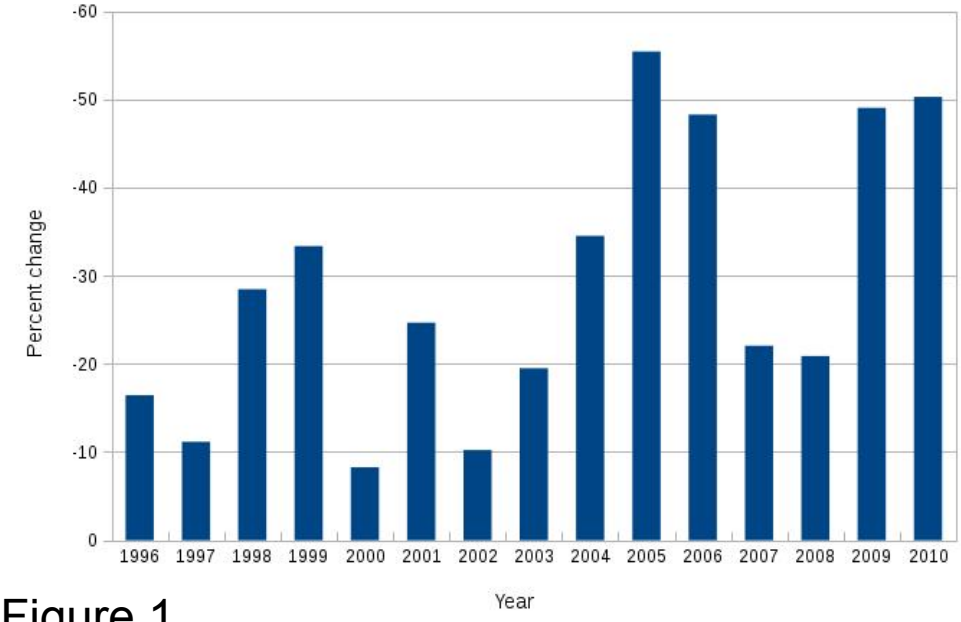


Figure 1.

Normal versus misaligned hemiscrota on an ungulate.

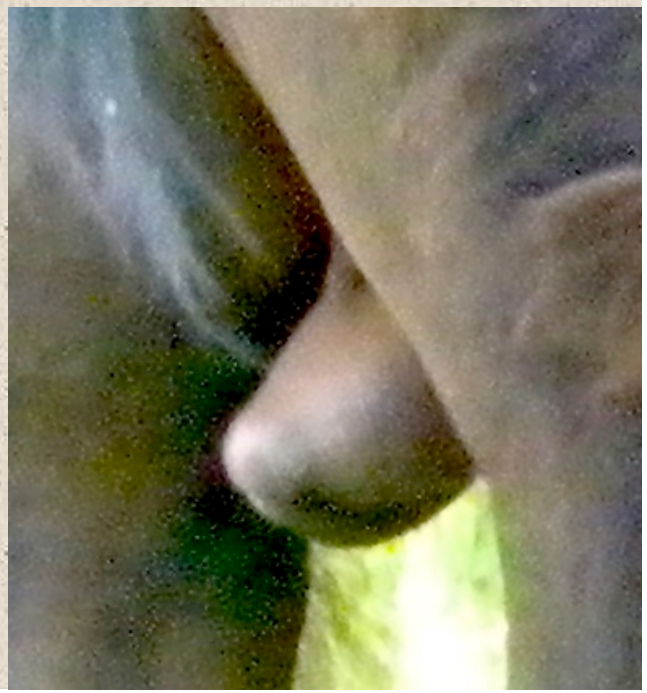
using photos of our study animal, white-tailed deer.

Normal bilateral hemiscrota on an adult.

Misaligned, tipped back hemiscrota.

A moose with extremely short misaligned hemiscrota photographed in 2014 in Glacier National Park.

The hemiscrota on a male moose with a normal scrotum would be bilateral and hang down away from the body wall much further than on this moose. Below, a photo of an adult bison photographed in YNP, shows a similar odd scrotal configuration.



1. A normal WTD scrotum with bilateral hemiscrota that hold the testes away from the body wall. 2. A normal scrotum and penis sheath shown from the side. The teats are somewhat forward causing the scrotum to tip back slightly. 3. A bilateral scrotum on a young fawn that is tipped far backward and that is very short, as is the penis sheath.

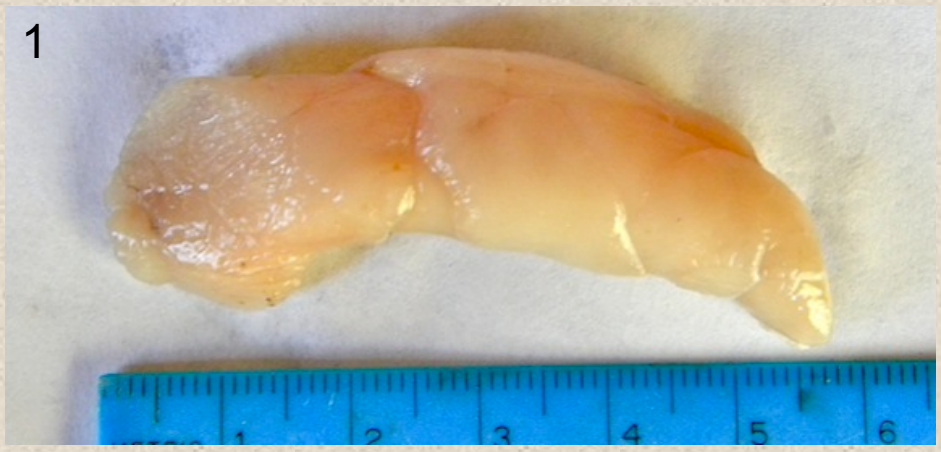
4. A misaligned, tipped back scrotum from an adult male WTD. 5. Genitalia from an older WTD fawn showing a tipped back misaligned scrotum. The penis sheath is close to normal length. 6. The genital of a newborn fawn showing a misaligned almost double scrotum and short penis sheath.

1. An adult WTD genitalia with no scrotum formed and short penis sheath.
2. An adult WTD genitalia with misaligned hemiscrota tipped very far back forming a long narrow short scrotum. The penis sheath was damaged by a bird.

3. A newborn fawn with hemiscrota almost not formed and what is formed is misaligned.
4. A close up photo of newborn fawn genitalia with hemiscrota misaligned and separated. The penis sheath is on the right and short on the fawns in photos 3 and 4.

An adult male bison with scrotum so short it is almost not visible. The arrow point is at the bottom of the scrotal sac. There are a large number of such male bison in the Lamar Valley in YNP. It is difficult to photograph large numbers of male moose. Genital hypoplasia appears to be epidemic on males of several ungulate species.

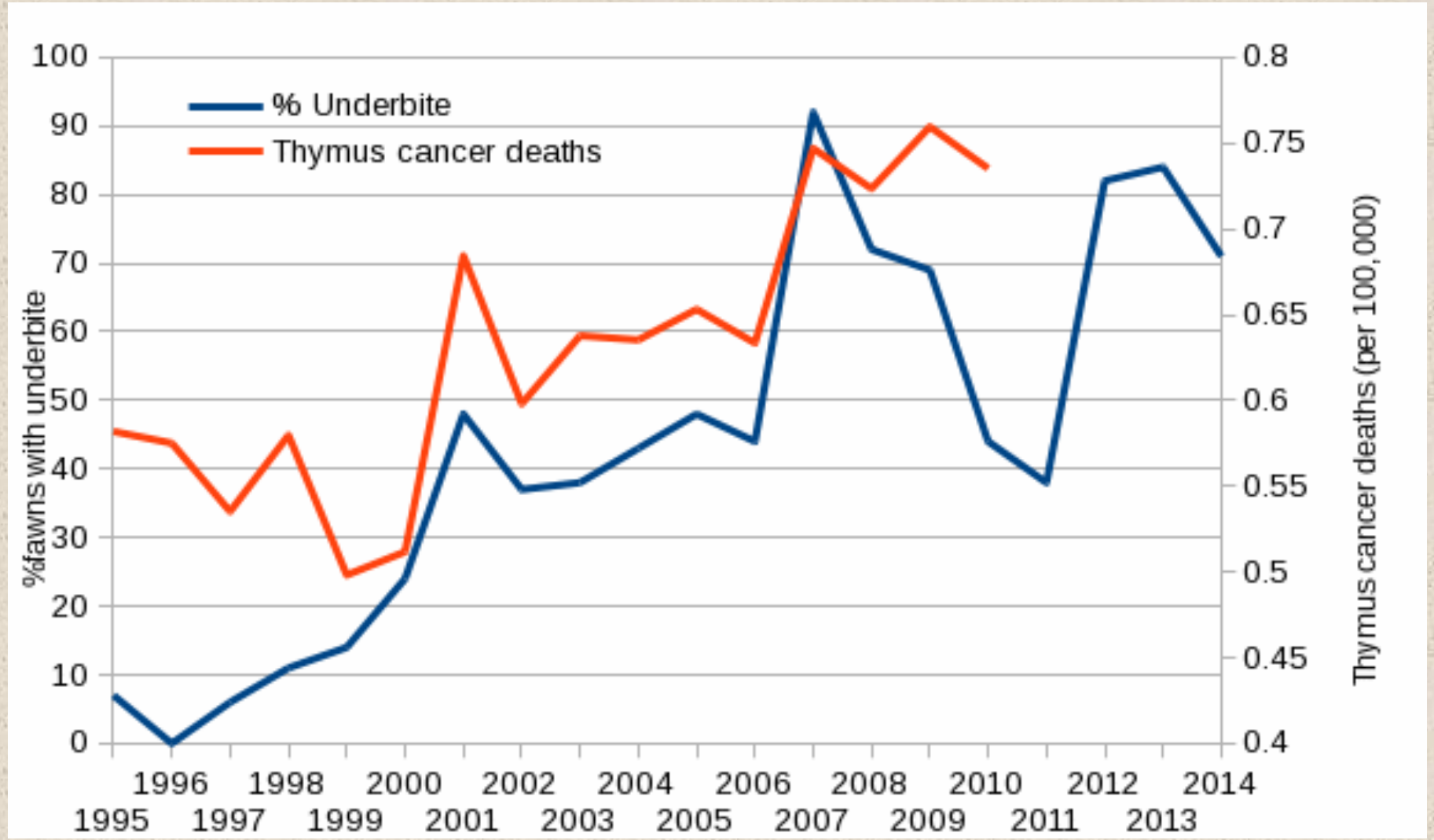
Damaged and/or underdeveloped thymus – a serious birth defect on newborn ungulates.



1. A thymus that is normal in color and size, 5.7 cm long and 1.6 cm wide.
2. A thymus that is short, only 3.7 cm long and 1.4 cm wide, but almost normal color.

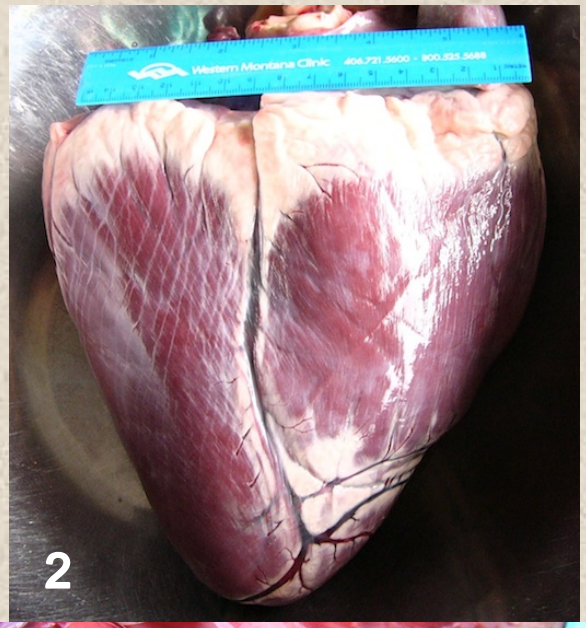
3. A very narrow thymus that is inflamed. It is only 0.8 cm wide but normal length.
4. A short, narrow inflamed thymus. All thymuses are from newborn white-tailed deer.

AN INTERESTING CORRELATION BETWEEN HUMAN CANCER DEATHS AND BRACHYGNATHIA SUPERIOR/UNDERBITE ON WHITE-TAILED DEER FAWNS

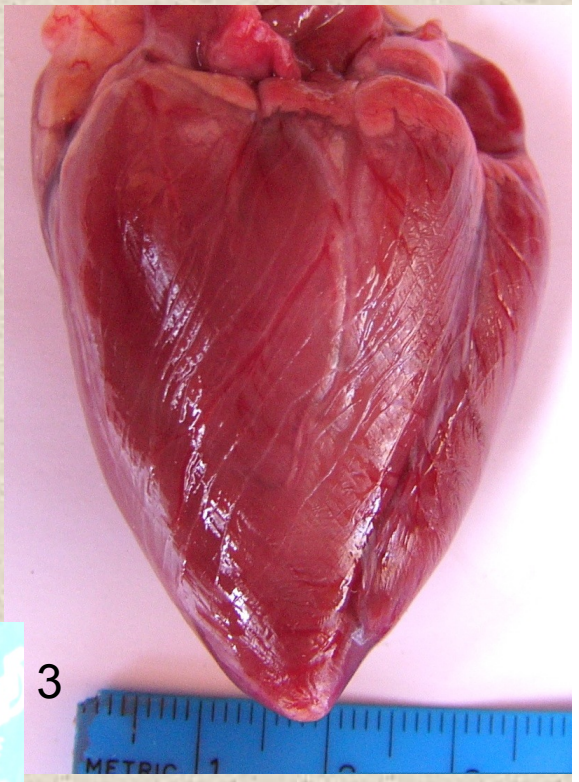


Health Problems Affecting Ruminant Hearts

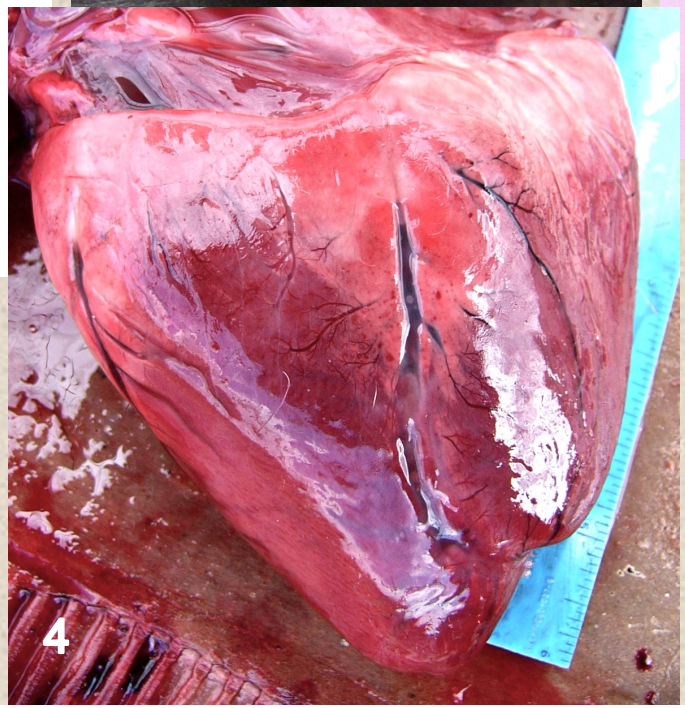
1. A normal white-tailed deer (WTD) heart.



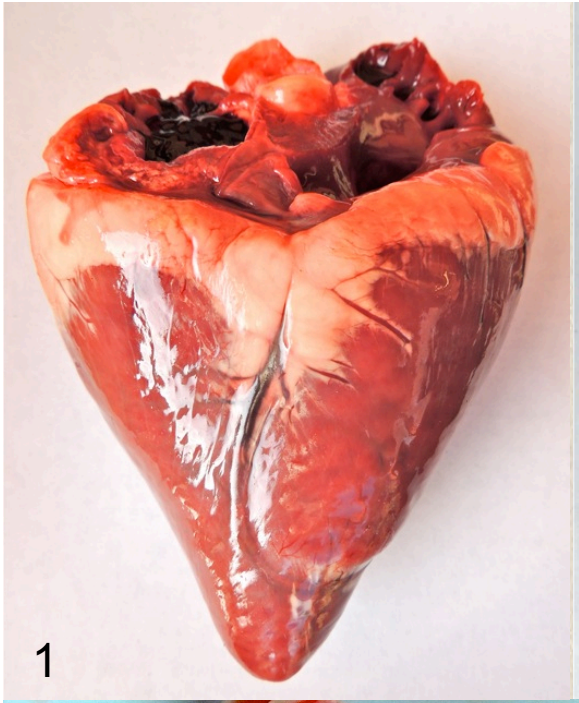
2. A WTD heart, normal in shape but with dilated lymphatic vessels (DLV) on the heart surface.



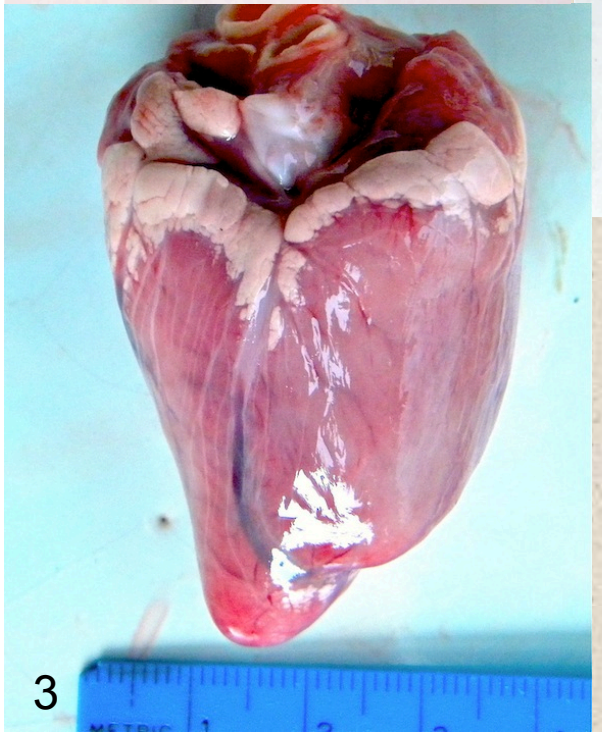
3. A WTD fawn heart fairly normal in shape, with fairly severe DLV on surface.



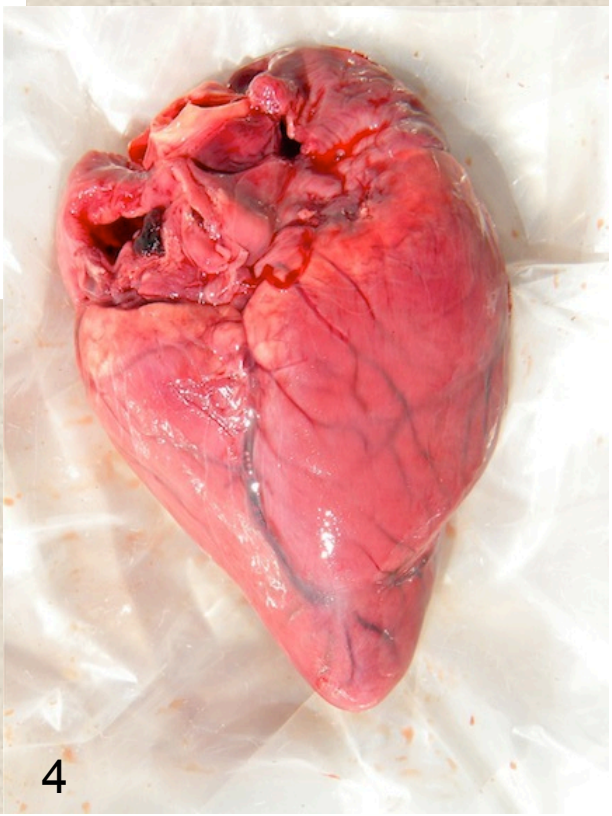
4. An adult male WTD with a very enlarged right ventricle.



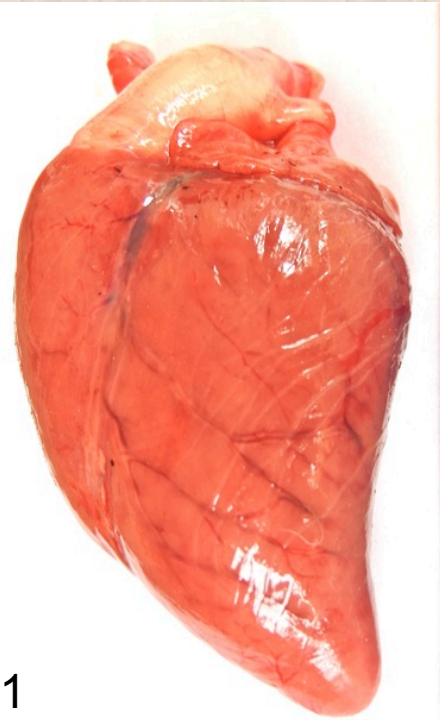
1. A female WTD fawn heart with slightly enlarged right ventricle.
2. Male yearling WTD heart with severely enlarged right ventricle and some DLV on left side.

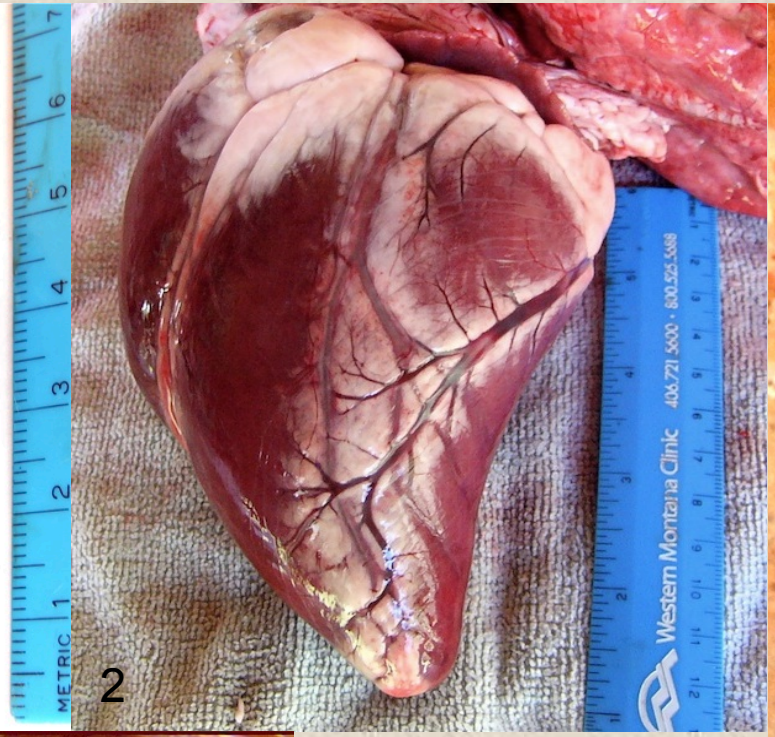


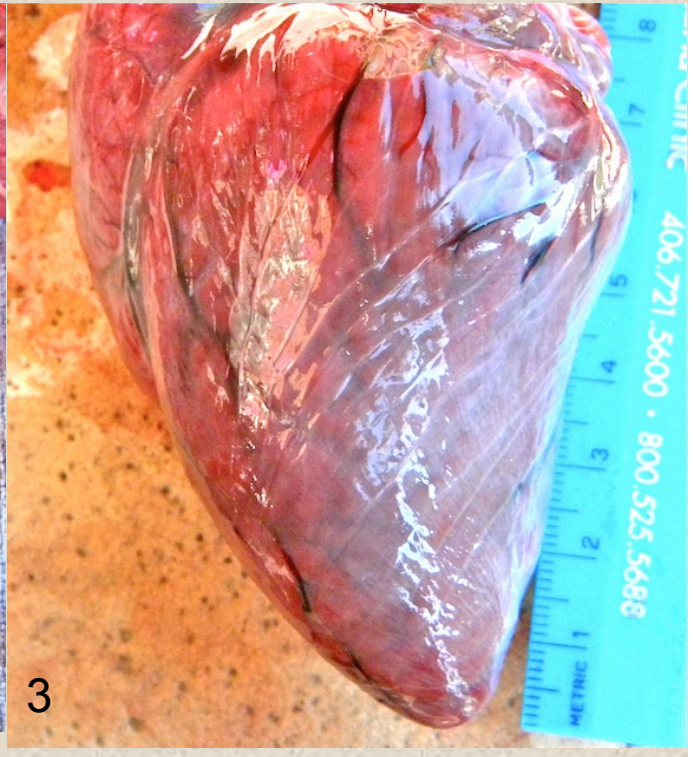
3. Female WTD fawn heart with severely enlarged right ventricle and DLV.
4. Male WTD fawn heart with fairly severe enlargement of right ventricle but not as severe as number 3.



Abnormalities on Hearts of Newborn Ruminants



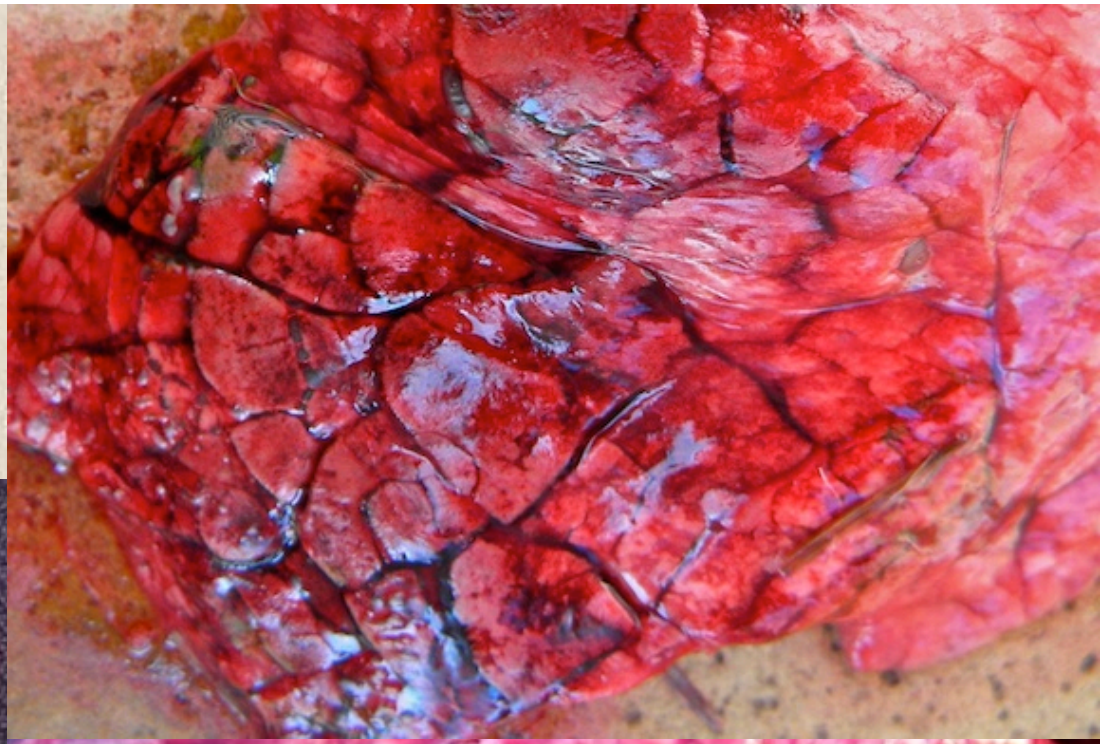




1. The heart of a female WTD fawn heart with DLV and an odd shape, narrow and curved.
2. The heart of a female elk calf with similar narrow curved shape and some DLV.
3. The heart of a male WTD fawn with very severe DLV with similar shape but not as curved.
4. A close up view of a male mule deer fawn with severely dilated lymphatic vessels.

Inflammation in Ruminant Lungs

Inflammation of the lungs like shown on the right is now common in ruminant species. The lung below illustrates a normal lung.



Overgrown Hooves, Laminitis and Lameness on Moose

Moose and other wild ungulates have devastating disruption of the growth of the keratin of the hooves causing malformed hooves, severe lameness and laminitis. Disrupted keratin growth often results in mortality when the affected animal can no longer walk, graze or escape from predators. Laminitis has also been increasing on horses throughout the United States. The keratin of the hooves of ungulates has a significant amount of cholesterol sulfate in their composition as shown in tests of horse hooves. A primary toxicity path of glyphosate, the active ingredient in Roundup is significant impairment of cholesterol sulfate synthesis. Roundup is a herbicide. It has the highest application rate of any herbicide or other pesticide in North America. Roundup has multiple adverse health effects including disruption of endocrine, retinoic acid, CYP-450, and liver functions. It also chelates many minerals essential to cellular functions, especially normal fetal cellular growth. The number of ungulates with birth defects, damaged hearts and lungs, lameness and other health issues has gone up in close correlation with the continuing increase in use of Roundup on Roundup Ready crops since 1996. In 1994, millions of pounds of fungicides, especially Chlorothalonil began being used on potato blight. The multiple birth defects on wild and domestic ungulates began in spring 1995, immediately after many millions of pounds of Roundup and Chlorothalonil were applied throughout the U.S., with use of Roundup increasing almost yearly since.

- If we do not stop the use of what is causing the serious symptoms consistent with mineral deficiencies and congenital fetal hypothyroidism, some wild ungulate species may not survive. Vehicle impact, infectious diseases and parasites (more damaging because of impaired immune system caused by endocrine disruption) and predators, including human hunters are killing far more animals each year than are being replaced by viable, healthy, normal young. The significant averse affects on fetal development is a sure path to extinction.
- Ungulates, rodents, all bird species, most amphibian species and multiple beneficial insect species are being harmed to the point that many are on the verge of extinction now or soon will be. Is a biologically devastated planet really what we want to leave to our children and grandchildren?

All of us should strive for a return to having young of all species born or hatched with normal characteristics, like this beautiful, normal moose calf, rather than being born or hatched with debilitating congenital defects.

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