Gross Causes of Neonatal Mortality in Devon Rex and Persian Kittens

by
Holly Omoto

A THESIS

submitted to

Oregon State University

Honors College

in partial fulfillment of
the requirements for the
degree of

Honors Baccalaureate of Science in Animal Sciences
(Honors Scholar)

Presented May 16, 2019
Commencement June 2019

AN ABSTRACT OF THE THESIS OF

Holly Omoto for the degree of Honors Baccalaureate of Science in Animal Sciences presented on May 16, 2019. Title: Gross Causes of Neonatal Mortality in Devon Rex and Persian Kittens.

Abstract approved: _____

Michelle Kutzler

Post-mortem examinations were performed on 29 deceased Persian kittens from 13 different litters, and 30 deceased Devon Rex kittens from 18 different litters in order to identify gross anatomical abnormalities. Fifteen kittens were stillborn (8 Devon Rex, 7 Persian), 17 kittens died during the first day of life (16 Devon Rex, 1 Persian), 14 kittens died later in the first week of life (5 Devon Rex, 9 Persian), and 13 kittens died during the second week of life (1 Devon Rex, 12 Persian). The average age at death excluding stillborn kittens was 5.2±4.7 days for both breeds, 1.8±2.0 days for Devon Rex kittens, and 8.5±4.2 days for Persian kittens. The most common sign reported by the cat breeders before death was dyspnea, with 12 kittens affected (11 Devon Rex, 1 Persian). Postmortem examination findings included pyothorax (9 Devon Rex kittens), pigmenturia (4 Devon Rex, 2 Persian kittens). It is estimated that at least 22.0% (13/59) of neonatal mortalities were due to infectious etiologies, at least 6.8% (4/59) were due to noninfectious etiologies, (n=7), and up to 71.2% (42/59) were classified as idiopathic.

Key Words: Devon Rex, feline, kitten, mortality, necropsy, neonatal, perinatal, Persian

Corresponding e-mail address: holly.omoto@gmail.com

©Copyright by Holly Omoto
May 16, 2019

Gross Causes of Neonatal Mortality in Devon Rex and Persian Kittens

by

Holly Omoto

A THESIS

submitted to

Oregon State University

Honors College

in partial fulfillment of
the requirements for the
degree of

Honors Baccalaureate of Science in Animal Sciences
(Honors Scholar)

Presented May 16, 2019
Commencement June 2019

Honors Baccalaureate of Science in Animal Sciences project of Holly Omoto presented on May 16, 2019.

APPROVED:

_____

Michelle Kutzler, Mentor, representing Animal and Rangeland Sciences

_____

Jana Gordon, Committee Member, representing Carlson College of Veterinary Medicine

_____

Tina Chittick, Committee Member, representing Everafter Devon Rex Cattery

_____

Toni Doolen, Dean, Oregon State University Honors College

I understand that my project will become part of the permanent collection of Oregon State University, Honors College. My signature below authorizes release of my project to any reader upon request.

_____

Holly Omoto, Author

Table of Contents

List of Figures

List of Appendices

I. Introduction

I. A. Kitten mortality

Although breeding management and veterinary intervention can prevent some newborn kitten losses, overall kitten mortality rates remain high. As many as 9.7% of kittens may be stillborn and up to 16% of kittens may die before weaning (Holst and Frössling, 2009; Fournier et al., 2017). However, other reported kitten mortality rates are lower, with as few as 7.2% of kittens being stillborn and only 8.3% of kittens dying in the first 12 weeks of life (Sparkes et al., 2006; Holst and Frössling, 2009). Kittens are most vulnerable when they are newborn; over half of all kitten deaths occur in the first two weeks of life (Casal, 2010; Holst and Frössling, 2009; Sparkes et al., 2006). With an average litter size of four kittens, one kitten may be stillborn and or one kitten may die before reaching two weeks of age, leaving only two or three live kittens (Fournier et al., 2017; Holst and Frössling, 2009; Sparkes et al., 2006).

I. B. Causes of kitten mortality

There are many causes of kitten mortality. A broad categorization of causes of kitten mortality would be to divide them in to infectious versus noninfectious etiologies. Non-infectious causes of kitten mortality include: trauma, congenital defects, nutritional imbalances, immune-mediated diseases, degenerative disorders, and poor husbandry. Infectious causes include viruses, bacteria, and parasites. Of kittens dying within the first 14 days of life, it is estimated that 50% die from infectious causes, 19% from idiopathic causes, 12% from immune-mediated causes, 9.5% from congenital causes, 4.8% from nutritional causes, and 4.8% from traumatic

causes (Cave et al., 2002). Immune-mediated disease, husbandry-related death, congenital defects, and infectious disease will be discussed below in more detail.

I. B. 1. Non-infectious causes of mortality

I. B. 1. a. Immune-mediated disease

As previously stated, 12% of kittens that die between the ages of 0 and 14 days die an immune-mediated disease, specifically neonatal isoerythrolysis (Cave et al., 2002). This is the most common noninfectious cause of mortality in kittens 14 days and younger (Cave et al., 2002). Neonatal isoerythrolysis occurs when kittens ingest alloantibodies, acquired from the queen, in colostrum that act against their own blood type during the first day of life. For the first 72 hours of life the alloantibodies can be absorbed from the digestive tract into the bloodstream (Casal et al., 1996).

There are three feline blood types: A, B, and AB. Cats have naturally occurring alloantibodies to red blood cell antigens other than their own. This means a blood type A cat has antibodies against blood type B and vice versa. The major concern is when blood type A or AB kittens receive antibodies from the colostrum of a blood type B queen. This is because blood type B cats have strong titers of naturally occurring antibodies against type A red blood cells (Giger and Casal, 1997). On the other hand, blood type A cats have weak titers of naturally occurring antibodies against type B red blood cells (Giger and Casal, 1997). Finally, blood type AB cats do not have anti-A or anti-B antibodies. Thus, blood type A cats have low anti-B activity while blood type B cats have high anti-A activity.

Blood type is genetically inherited in cats so the cat breeder should consider the cats' blood types when selecting the queen and tom to avoid this condition. As it was stated earlier, a cat can have one of three different blood types: A, B, or AB. There are also three different alleles: A, B, and AB. A is dominant over AB, and AB is dominant over B. Blood type A cats have at least one A allele, blood type AB cats have either a pair of AB alleles or one AB allele and one B allele, and blood type B cats have two B alleles. In this way, it is possible for a type B queen and a type A tom to have a type A kitten.

There are certain cat breeds wherein blood type B is very rare and blood type A is very common. Thus, the risk of a blood type A kitten receiving antibodies from a blood type B queen and developing neonatal isoerythrolysis is low. However, there are other breeds in which blood types A and B are both common, and thus the risk of neonatal isoerythrolysis is increased. Turkish Van, Turkish Angora, Devon Rex, and British Shorthair cats have the highest frequency of blood type B at 40 to 60% (Giger and Casal, 1997; Silvestre-Ferreira and Pastor, 2010). There are also geographical differences, with type B blood more common in Australia and certain parts of Europe than it in the United States (Giger and Casal, 1997; Silvestre-Ferreira and Pastor, 2010).

In terms of prevention, it is desirable to mate a blood type B queen with a blood type B tom. If a blood type B queen is to be mated with a blood type A tom, the kittens can be separated from the queen until they are 24 hours old, after which point the kittens will not absorb immunoglobulins (Casal et al., 1996). At-risk kittens can receive milk or colostrum from a type A foster queen or can be fed milk replacer.

Signs of neonatal isoerythrolysis include: cessation of nursing, sudden death, hemoglobinuria (i.e. presence of hemoglobin from destroyed red blood cells in the urine), icterus (i.e. yellow coloring of the mucous membranes), anemia, weakness, lethargy, tachycardia, tachypnea, collapse, hypoglycemia, and metabolic acidosis (Silvestre-Ferreira and Pastor, 2010). Death may occur rapidly or after a few days (Silvestre-Ferreira and Pastor, 2010). Suspicion of neonatal isoerythrolysis can be confirmed with blood typing of the queen and the kitten (or blood crossmatching if blood typing is not possible). Treatment begins with removing affected kittens from their mother if they are less than 24 hours old. Treatment may also include administering intravenous fluids, warming kittens up, and or performing a blood transfusion with blood from the queen since the antibodies from the queen's colostrum will not react with her blood (Silvestre-Ferreira and Pastor, 2010).

I. B. 1. b. Husbandry-related deaths

There are specific factors to consider when selecting the queen or the tom that have the potential to increase or decrease kitten mortality. Queens that have had multiple litters before tend to lose fewer kittens (Lawler and Monti, 1984). However, older queens can also have increased stillbirth rates (Holst and Frössling, 2009). Another factor to consider is the size of the queen. While smaller queens may give birth to fewer kittens, they tend to experience fewer kitten deaths (Lawler and Monti, 1984). Also in regards to size, overweight queens have a higher kitten mortality rate (Lawler and Monti, 1984). Unfortunately, there are no correlations between the tom's characteristics and kitten mortality that have been researched and published.

Once the kittens are born, hypoglycemia, hypothermia, and dehydration are major concerns in the first few weeks of life. Clinical hypoglycemia in a kitten is defined as a blood glucose level of less than 50 mg per dL (3 mmol per L; Little 2011). Newborn kittens are prone to hypoglycemia because they are born with minimal glycogen reserves and without full liver function, the organ largely responsible for glucose homeostasis (Little, 2011). Thus, a kitten can easily become hypoglycemic after vomiting, diarrhea, sepsis, hypothermia, or inadequate nutritional intake (Little, 2011). Signs of hypoglycemia include lethargy, weakness, and anorexia (Little, 2011). Treatment of hypoglycemia may include administration of dextrose orally by gastric tube, intravenously, or intraosseously (Little, 2011).

Severe hypothermia in a kitten is defined as a rectal temperature less than 94° F (34.4 ° C; Little, 2011). Normal body temperature for a newborn kitten, by comparison, is 97 to 98° F (Little, 2011). Newborn kittens are prone to hypothermia because they are born with a limited ability to maintain their internal body temperature (Little, 2011). Hypothermia has been associated with depressed respiration, impaired immunity, bradycardia, and ileus (Little, 2011). This can be confirmed by taking the kitten's rectal temperature. Kittens with low body temperatures should be gradually warmed to avoid further problems stemming from an increase in metabolic demand that results in dehydration, hypoxia, and loss of cardiovascular integrity (Little, 2011). To treat hypothermia, an incubator or oxygen cage can be utilized (Little, 2011). If those are not available, warm water bottles and heating lamps can be utilized with care (Little, 2011).

Dehydration is another concern with kittens because their bodies are made of 80% water compared to 60% in adult cats (Little, 2011). This increased water composition, higher proportion of surface area, higher metabolic rate, and lower level of body fat causes kittens to have higher fluid requirements than an adult cat of the same size (Little, 2011). Further, a kitten's kidneys are not fully developed at birth, causing them to excrete a higher volume of water per unit of body weight compared to an adult cat (Little, 2011). Thus, a kitten can easily become dehydrated if it has diarrhea, vomiting, or reduced fluid intake (Little, 2011). One way to quickly check a kitten's hydration status is to examine its mucous membranes and determine its capillary refill time. If the former appears pale and the latter is delayed, the kitten is at least 10% dehydrated (Little, 2011). In addition, a dehydrated kitten's urine will be darker in color and have a specific gravity greater than 1.020 (Little, 2011). If a kitten is only slightly dehydrated and does not have any other health issues, it can be treated with warmed subcutaneous or oral fluids. However, if a kitten is severely dehydrated, it will require intravenous or intraosseous Lactated Ringer solution (Little, 2011). Care should be taken to not overhydrate neonatal kittens because they do not have full renal excretory function (Little, 2011).

I. B. 1. c. Congenital defects

Congenital defects occur when kittens do not form properly *in utero.* As many as 14.3% of pedigree litters may have at least one congenital defect present (Sparkes et al., 2006). With regards to mortality, congenital defects may cause up to 10% of neonatal kitten losses (Cave et al., 2002). While some congenital defects are readily

visible at birth (e.g. cleft palate), others may remain unknown until the kitten dies and is submitted for necropsy (e.g. hiatal hernia). Examples of common congenital defects include: eyelid coloboma, cleft palate, ocular dermoids, flat chest defect, gastroschisis, pectus excavatum, syndactyly, and umbilical hernia, (Little, 2005; Little 2011). Hydrocephalus and urinary dysgenesis are examples of congenital defects that can cause kitten mortality (Cave et al., 2002).

There are many different causes of congenital defects. A congenital defect may be the result of genetic inheritance, infection *in utero,* exposure of the queen to a teratogen, hyperthermia of the queen, poor intrauterine environment, nutritional factors, or an interaction between environmental and genetic factors (Little, 2005). An example of an infectious cause of a congenital defect is the panleukopenia virus, which can cause cerebellar hypoplasia in kittens (Little, 2005). An example of a teratogen is griseofulvin, which is a drug used to treat ringworm infection. When administered to a gestating queen, griseofulvin can cause cleft palate in kittens (Little, 2005). A nutritional factor that can cause congenital defects is taurine deficiency, which is linked to various musculoskeletal defects (Little, 2005).

1. B. 2. Infectious causes of mortality

An analysis of 274 kitten deaths identified infectious disease in over half of the kittens (Cave et al., 2002). This rate was consistent within the neonatal age group, with one half of all losses occurring in the first 14 days due to infectious causes (Cave et al., 2002). Breaking this down further, of the neonatal kittens that died from infectious causes, half died from viral infections while the other half died from

bacterial infections (Cave et al., 2002). In terms of specific infectious agents, all of the neonatal kittens with viral disease were determined to have feline herpesvirus or feline calicivirus via histopathological testing (Cave et al., 2002). A different analysis of 168 deaths found that one third of the kittens died from infectious causes (Mossi-Dieth et al., 1990). In that study, bacterial infections were confirmed in twice as many kittens as viral and parasitic infections combined (Mossi-Dieth et al., 2002). Specific bacterial pathogens that were isolated from these kittens included *Escherichia coli*, *Streptococcus spp., Pasteurella spp., Plesiomonas, Proteus spp., Bordetella, and Salmonella* (Mossi-Dieth et al., 1990). Finally, a review reports that feline embryonic and fetal loss is more commonly caused by viral infection than bacterial or protozoal infection (Daniel Givens and Marley, 2008). In particular, feline infectious peritonitis virus can cause abortion, stillbirth, and high mortality in the first week of life; and feline panleukopenia virus can cause abortion, stillbirth, and cerebellar hypoplasia in kittens (Daniel Givens and Marley, 2008).

The most common types of bacteria isolated in cases of pneumonia, pleuritis, myocarditis/endocarditis, or meningitis/encephalitis were *Streptococcus spp*. and *Escherichia coli* (Mossi-Dieth, 1990). Other common causes of upper and lower respiratory tract disease in kittens include; feline herpesvirus-1, feline calicivirus, *Bordetella bronchiseptica*, *Mycoplasma spp*., and *Chlamydophila spp.*(Little, 2011). In addition, panleukopenia virus (parvovirus), coliform bacteria, *Tritrichomonas foetus, Giardia spp., Isopora spp., Ancylostoma spp.*, and *Toxocara spp*. are common causes of gastrointestinal tract disease in kittens. As for systemic disease in kittens, feline leukemia virus (FeLV), feline immunodeficiency virus (FIV), *Toxoplasma*

spp., Gram-positive bacteria (e.g. *Streptococcus spp*., *Staphylococcus spp*.), and Gram-negative bacteria (e.g. *Escherichia coli, Salmonella spp*.) are common causes (Little 2011).

I. C. Cat breeds

I. C. 1. Devon Rex

According to The International Cat Association and The Cat Fanciers' Association, the Devon Rex is a relatively new cat breed, originating from Devon, England in the late 1950's. All Devon Rex cats descend from Kirlee, the offspring of a feral tom cat with a curly coat. Thus, cat breeders created the Devon Rex breed by perpetuating a natural mutation. Today, Devon Rex cats are known for their short, curly coats (**Figure I-1**). This coat does shed and it can come in a variety of colors and patterns. The face of a Devon Rex cat is dominated by its large ears and eyes. In terms of personality, Devon Rex cats are described as having intelligent, playful, and active personalities. As for popularity, the Cat Fanciers' Association lists the Devon Rex as number seven in terms of the number of cats registered in 2018.

Devon Rex kittens are prone to developing neonatal isoerythrolysis, which was described earlier. The risk of neonatal isoerythrolysis is increased in cat breeds that have a high prevalence of type B blood, including the Devon Rex breed which has a 59:41 split between the A and B blood types (Silvestre-Ferreira and Pastor, 2010). Using Hardy-Weinberg equilibrium, it is estimated that 14% of kittens from random Devon Rex matings would be at risk of developing neonatal isoerythrolysis (Silvestre-Ferreira and Pastor, 2010). Reported stillbirth rates for Devon Rex kittens

range from 4.2 to 8.5%, which is approximately average for purebred cats (Holst and Frössling, 2009; Sparkes et al., 2006). Reported mortality rates for Devon Rex kittens during the first week of life range from 4.1-4.4%, which is slightly below average for purebred cats (Holst and Frössling, 2009; Sparkes et al., 2006).

Figure I-1. Devon Rex kitten. Photograph by Tina Chittick, used with permission.

I. C. 2. Persian

According to The International Cat Association and The Cat Fanciers' Association, the Persian is one of the oldest cat breeds, but its centuries-long history is, unfortunately, unrecorded. Some hypothesize that longhair cats spread westward via caravans carrying spices and jewels from modern-day Iran. The Persian cat has a long coat that ranges in texture from silky to cottony. Round eyes and a snub nose adorn the Persian cat's face (**Figure I-2**). As for personality, Persian cats are known

to be intelligent, sweet, and gentle. In terms of popularity, the Cat Fanciers' Association lists the Persian as number four in terms of the number of cats registered in 2018.

Persian cats are prone to conjunctivitis and upper respiratory tract disease. One half of Persian and Exotic Shorthair breeders reported conjunctivitis, and one third of Persian and Exotic Shorthair breeders reported other upper respiratory signs in their cats (Holst and Frössling, 2009). By comparison, only one third of all breeders reported conjunctivitis, and only one sixth of all breeders reported other upper respiratory signs in their cats (Holst and Frössling, 2009). Additionally, the prevalence of respiratory tract diseases in Persian cats is approximately twice as high as for all pedigree cats (Vapalahti et al., 2016). One possible explanation for the increased risk of conjunctivitis and upper respiratory tract disease in Persian cats is poor nasolacrimal drainage and narrowed nasal passages due to the brachycephalic conformation of their heads (Schlueter et al., 2009). Reported stillbirth rates for Persian kittens range from 10.8 to 12.2%, which is slightly above average for purebred cats (Holst and Frössling, 2009; Sparkes et al., 2006). Reported mortality rates for Persian kittens during the first week of life range from 8.6 to 12%, which is also slightly above average for purebred cats (Holst and Frössling, 2009; Sparkes et al., 2006).

Figure I-2. Persian kitten. Photograph by Laura Peters, used with permission.

I. D. Hypothesis and objectives

The purpose of this research was to identify gross anatomical abnormalities in Devon Rex and Persian kittens that died within the first two weeks of life and determine the cause of mortality, when possible. Studying two different breeds of kittens also allowed for comparison of causes of mortality between breeds. It was hypothesized that the two cat breeds would die from different conditions at different rates.

II. Materials and methods

Post-mortem examinations were conducted on 59 purebred kittens. Thirty deceased Devon Rex kittens and 29 deceased Persian kittens were received from two catteries. This project started as a collaboration between the Oregon State University Theriogenology Laboratory and a Devon Rex cat breeder located in Iowa. To expand the scope of this project, a search was conducted via The Cat Fanciers' Association (CFA) Cat Breeder Referral Search (https://secure.cfa.org/Search.aspx). Through this search, a Persian cat breeder located in Oregon was identified, and the collaboration was expanded to include this breeder.

To aid in the determination of each kitten's cause of death, the following information was requested from each cat breeder (Appendix A):

- The identification of the dam and the sire for pedigree analysis
- The dam's and sire's blood types
- The age of the kitten at death
- Any complications related to the kitten's birth
- Any medications or treatments administered to the dam or the kitten (including if the kitten was euthanized)
- Any signs present before the kitten's death (changes in body temperature, breathing, appetite, weight, appearance, etc.)

This background information was utilized to provide additional information into the causes of death and to compare signs present before death between different breeds and different ages.

The gross appearance of each kitten's external and internal organs was assessed and pictures were taken for reference. The design of the post-mortem

examination was based on the method described by Löhr 2011. For this project, the post-mortem examination consisted of:

- Weighing the kitten
- Assessing the kitten's general appearance (e.g. mummified, edematous)
- Examining the kitten's umbilical cord (or stump) and urogenital area for abnormalities
- Examining the placenta if it was included and freezing it for future tissue analysis
- Examining the kitten's extremities for abnormalities (e.g. maternal cannibalism of hind feet)
- Examining the kitten's mouth, tongue, and palate for abnormalities (e.g. cleft palate)
- Making a ventral medial incision and a perpendicular medial incision near the diaphragm to expose the organs of the thoracic cavity
- Describing the appearance of the heart and lungs and noting any thoracic cavity abnormalities (e.g. pleuropneumonia, hiatal hernia)
- Floating tissue from the right caudal lung lobe to evaluate lung inflation and freezing tissue from the left caudal lung lobe for future analysis
- Evaluating the appearance of the liver and freezing a tissue sample of the liver for future analysis; the tissue sample was taken from one of the left lobes of the liver unless those were damaged by freezing of the kitten's body or handling during the post-mortem examination
- Evaluating the appearance of the stomach and the spleen

- Aspirating fluid from the bladder or the urachus
- Examining the kidneys and adrenal glands externally and bisecting the kidneys for internal examination and tissue collection

III. Results and discussion

III. A. Sample demographics

Fifty-nine kittens from 31 litters that died within the first fourteen days of life were received from two catteries: a Devon Rex cattery located in Iowa and a Persian/Himalayan cattery located in Oregon. The kittens were all born between December 2015 and February 2019. There were 29 deceased Persian kittens from thirteen litters, and 30 deceased Devon Rex kittens from eighteen litters. One Devon Rex kitten could not be fully examined because of its small size and mummified tissues, thus complete post-mortem examinations were performed on 29 Devon Rex kittens from eighteen litters. Four Devon Rex kittens were euthanized, so their causes of decline will be discussed as opposed to their causes of death. The average number of neonatal losses per litter was 1.9±1.3 for both breeds, 1.7±1.1 for Devon Rex litters, and 2.2±1.4 for Persian litters. The average age at death excluding stillborn kittens was 5.2±4.7 days for both breeds, 1.8±2.0 days for Devon Rex kittens, and 8.5±4.2 days for Persian kittens (**Figure III-1**).

Previous research has found that Persian and Exotic Shorthair kittens have a higher mortality rate compared to Devon Rex and Cornish Rex kittens for the first three weeks of life (Holst and Frössling, 2009). It was not possible to determine the mortality rate for kittens from the catteries used in the current study, but the average

age of the kitten at death (excluding stillbirths) was different for the two cat breeds: 1.8 days for Devon Rex kittens and 8.5 days for Persian kittens. This is in contrast to previous research that reported Persian kittens have a higher mortality rate during the first week than Rex breed kittens (Sparkes et al., 2006).

This difference in age at death could be due to a difference in cause of mortality. It has been shown that while trauma is a leading cause of mortality for kitten deaths occurring in the first day of life, viral and bacterial infections are a leading cause of mortality for kitten deaths occurring from one day of age to 14 days of age (Cave et al., 2002). However, another study found that noninfectious etiologies are more common in the first two weeks of life while infectious etiologies are more common after three to four weeks of life (Bücheler, 1999).

Further evidence for a difference in cause of mortality can be found in differing disease prevalence rates for Persian cats compared to Devon Rex cats. Persian and Exotic Shorthair cats have been shown to have a higher prevalence of respiratory tract disease than Devon Rex cats (Vapalahti et al., 2016). In addition, the percentage of Persian and Exotic Shorthair breeders reporting conjunctivitis and other upper respiratory signs in their cats is higher than the percentage of Devon Rex and Cornish Rex breeders (Holst and Frössling, 2009). One possible explanation for the prevalence of upper respiratory disease in Persian cats is that the brachycephalic conformation of their heads impedes drainage of the nasolacrimal duct, thus impeding normal defense mechanisms and making these cats more susceptible to secondary infections (Schlueter et al., 2009).

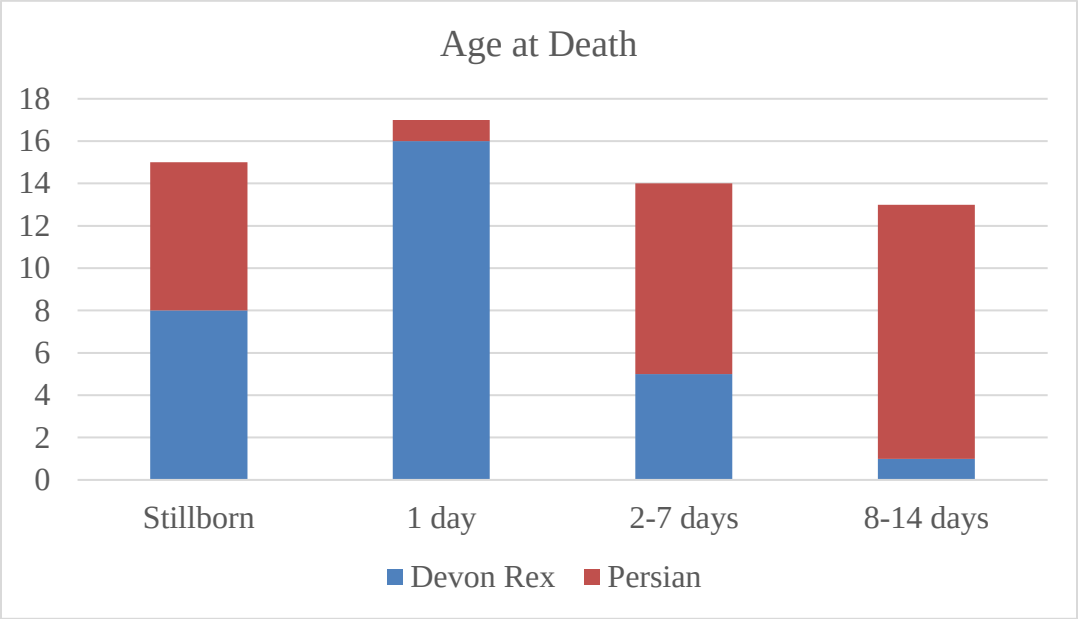


Figure III-1. Comparison of age at death in Devon Rex and Persian kittens. The average age at death excluding stillborn kittens was 5.2±4.7 days for both breeds, 1.8±2.0 days for Devon Rex kittens, and 8.5±4.2 days for Persian kittens.

III. B. Signs before death

There were five maiden litters, three maiden Persian litters and two maiden Devon Rex litters, and there were thirteen neonatal losses from those maiden litters. In total, eight kittens from six litters were affected by dystocia, five Persian kittens from three litters and three Devon Rex kittens from three litters. Only one Devon Rex kitten affected by dystocia was from a maiden litter. Four out of the eight kittens affected by dystocia were stillborn. Maternal neglect (kitten found isolated, kitten not groomed, etc.) was reported as a sign before death for five Persian kittens from five litters. Hypothermia was reported as a sign before death in five kittens from five litters, three Persian kittens and two Devon Rex kittens.

Clinical signs related to respiratory tract disease were reported in fifteen kittens, twelve Devon Rex kittens and three Persian kittens. Dyspnea was the most common abnormal clinical sign reported before death with thirteen kittens from ten litters affected. Only one Persian kitten exhibited dyspnea before death, compared to twelve Devon Rex kittens. Signs of upper respiratory tract disease were present in two fourteen-day-old Persian kittens from two litters: one kitten with nasal discharge and one kitten with ocular discharge. Additionally, there was a Persian kitten with discharge from the umbilicus.

Failure to gain weight was reported in seven kittens from five litters, five Persian kittens from three litters and two Devon Rex kittens from two litters. Of the two Devon Rex kittens that failed to gain weight, both experienced weight loss before death. Failure to nurse before death was reported in two Devon Rex kittens from two different litters; however, both of these kittens had been growing normally prior. Abnormal feces (i.e. white-colored feces, diarrhea, etc.) were reported in three kittens from two litters, two Persian kittens from the same litter and one Devon Rex kitten. These two Persian kittens also failed to gain weight, while the Devon Rex kitten also failed to nurse.

III. C. Postmortem examination findings

III. C. 1. External examination

There were four Devon Rex kittens that had undergone mummification, two of which were from the same litter. One mummified kitten was too small and desiccated for a post-mortem examination to be performed (**Figure III-2**). Two

kittens presented with meconium staining, one Devon Rex and one Persian (**Figure III-3**). One stillborn Persian kitten had external evidence of maternal cannibalism with its hind feet removed after birth (**Figure III-4**). Upon examination of the face, two kittens were found to have nasal discharge, one stillborn Devon Rex kitten and one fourteen-day-old Persian kitten (**Figure III-5**). This pair of kittens did not include the kitten reported by its breeder as having nasal discharge before death. Examination in the mouth revealed a cleft palate in a one-day-old Devon Rex kitten (**Figure III-6**). Abdominal bruising was present in seven kittens from five litters, five Devon Rex kittens from four litters and two Persian kittens from the same litter (**Figure III-7**). None of the kittens with abdominal bruising were reported to have been affected by dystocia. One stillborn Devon Rex kitten was missing the tip of its tail due to cannibalism, and one Persian kitten that died during the first day of life born without a tail **(Figures III-8 and III-9**). The Devon Rex kitten with a cleft palate and the Persian kitten born without a tail were the only two congenital defects detected.

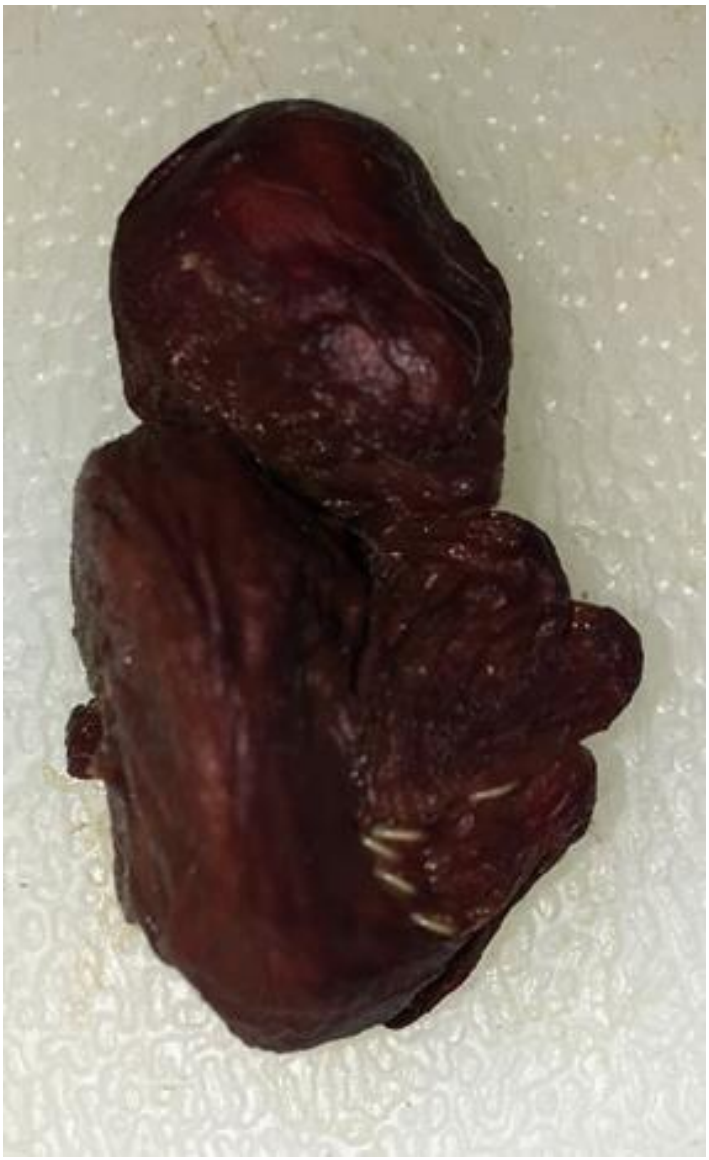


Figure III-2. Mummified kitten. There were four Devon Rex kittens that had desiccated tissues consistent with mummification.

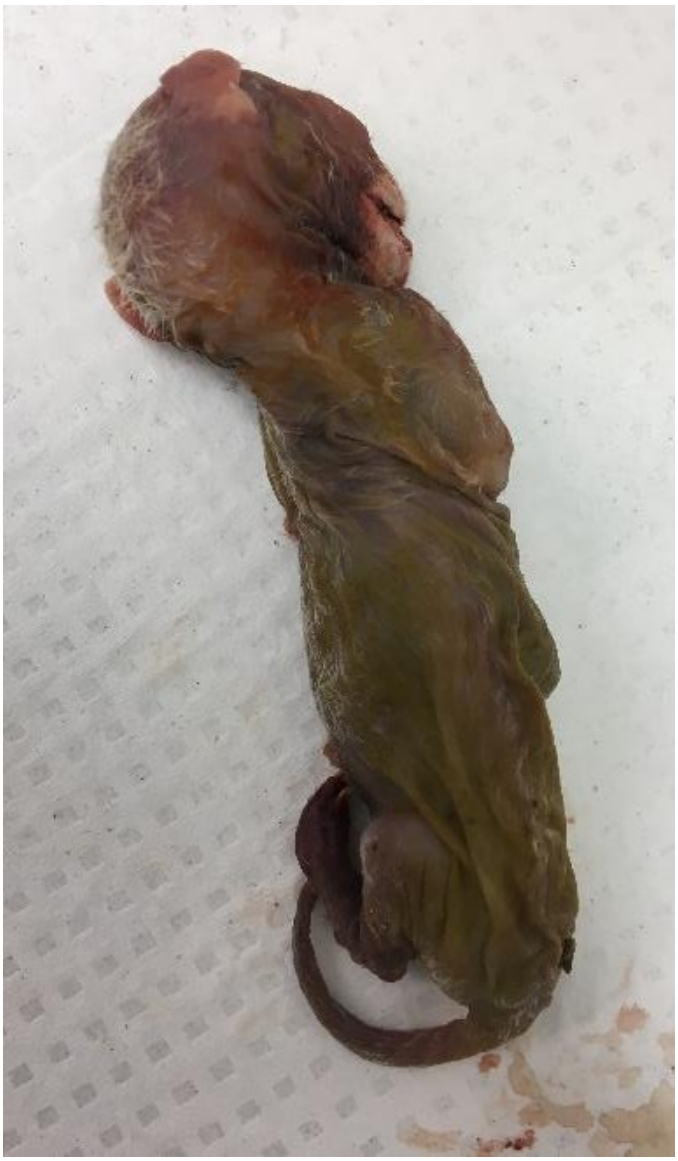


Figure III-3. Kitten with meconium staining. There were two kittens that presented with meconium staining (one stillborn Devon Rex kitten and one stillborn Persian kitten).

Figure III-4. Kitten that was affected by maternal cannibalism. Note how the back legs have been chewed off. The umbilical cord and placenta are still attached. There were two stillborn kittens with signs of maternal cannibalism (one Devon Rex kitten that was missing the tip of its tail and one Persian kitten whose back legs were chewed off).

Figure III-5. Kitten with nasal discharge. Two kittens were found to have nasal discharge during postmortem examination (one stillborn Devon Rex kitten and one 14-day-old Persian kitten).

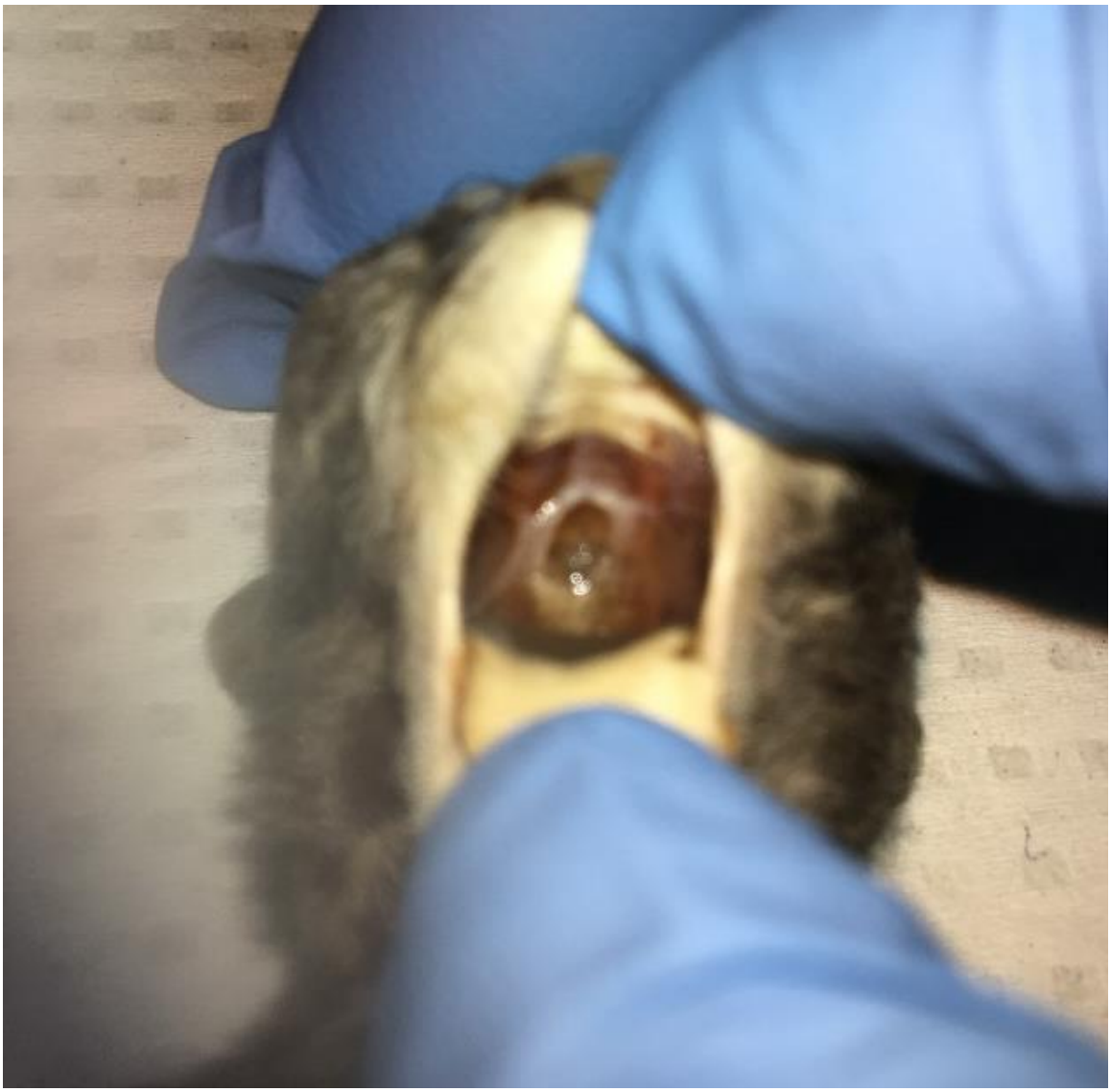


Figure III-6. Kitten with a cleft palate. There was only one kitten that was born with a cleft palate.

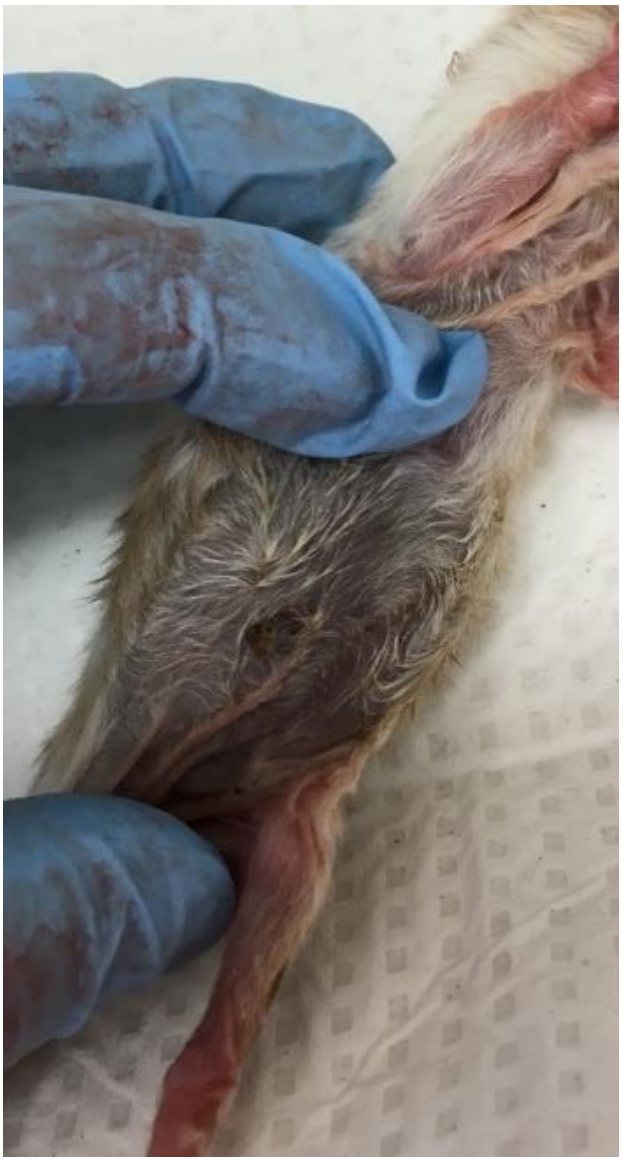
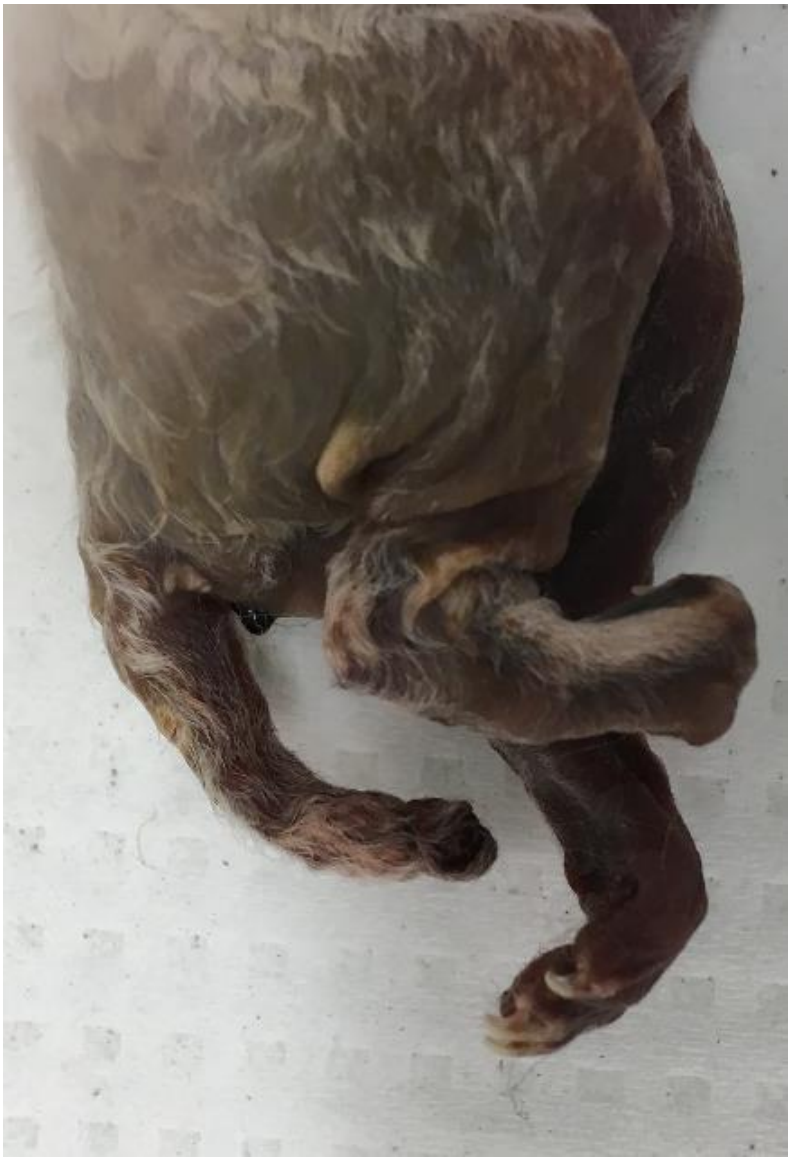


Figure III-7. Kitten with abdominal bruising. Abdominal bruising was observed in seven kittens (five Devon Rex kittens and two Persian kittens).



Figures III-8. Kitten missing the distal end of its tail. This was due to maternal cannibalism. There was only one Devon Rex kitten that was missing a part of its tail.

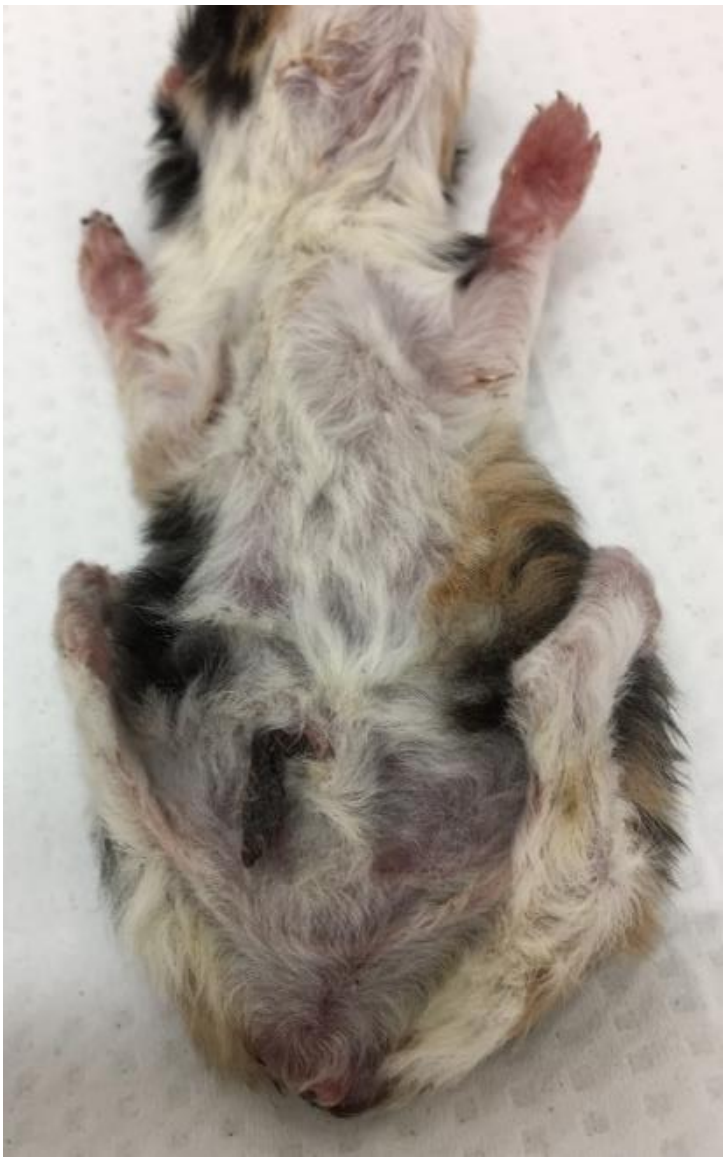


Figure III-9. Kitten born without a tail. There was only one Persian kitten that was born without a tail.

III. C. 2. Internal examination

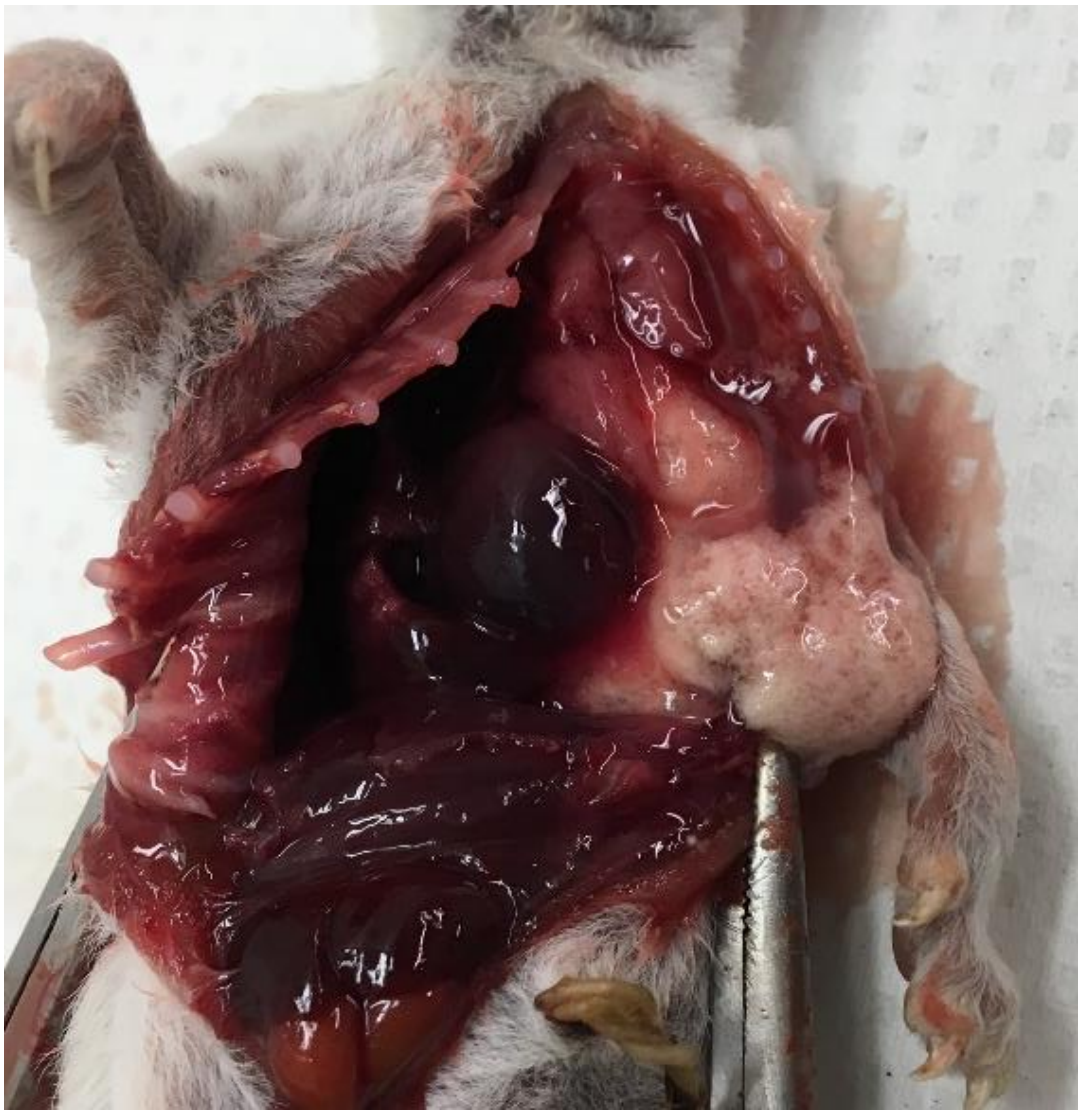
III. C. 2. A. Thoracic cavity

Upon internal examination, nine Devon Rex kittens from seven litters presented with thick, cream-colored material in their thoracic cavities suggestive of

pyothorax with inspissated pus (**Figure III-10**). The ages of these nine kittens ranged from one to three days. Of these nine kittens, seven exhibited dyspnea before death. Three of these nine kittens also presented with lung adhesions which is consistent with pleuropneumonia (**Figure III-11**). Thoracic wall penetration from a bite wound is the most common route of infection for feline pyothorax (Barrs and Beatty, 2008). Another possible route of infection could be injury due to tube feeding. Three Devon Rex kittens that presented with suspected pyothorax were reported to have been tube fed before death.

In addition, two Persian kittens from different litters had red-tinged fluid within their thorax which may be due to autolysis or hemothorax (**Figure III-12**). Examination of the heart revealed one Devon Rex kitten with gross lesions consistent with myocarditis (**Figure III-13**). Myocarditis in cats has been associated with several different pathogens, including feline immunodeficiency virus, panleukopenia virus, and *Bartonella henselae* (Rolim et al., 2016; Meurs et al., 2000; Varanat et al., 2011).

Examination of the lungs revealed 31 kittens with diffuse patches of pink and red lung tissue consistent with partial pulmonary congestion. Eighteen Persian kittens from ten litters and thirteen Devon Rex kittens from ten litters, which ranged from one to fourteen days of age, were affected (**Figure III-14**). There were also thirteen kittens with diffuse, dark red coloration of their lung tissue consistent with more severe pulmonary congestion, including ten Persian kittens from eight litters and three Devon Rex kittens from three litters (**Figure III-15**). Nine of the kittens with dark red coloration of the lungs were stillborn.



Figures III-10. Kitten with suspected pyothorax. Pyothorax was suspected in nine Devon Rex kittens, all of which died in the first week of life.

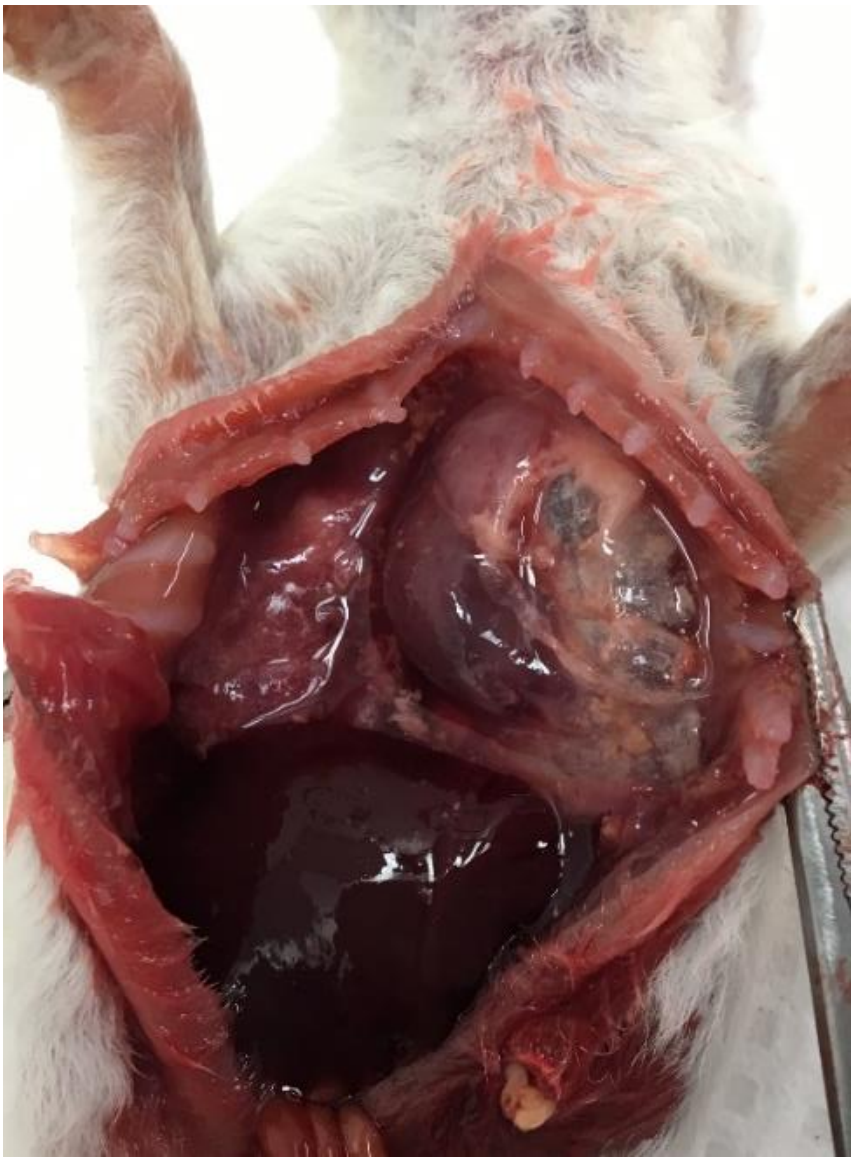


Figure III-11. Kitten with pulmonary adhesion. Three kittens suspected of having pyothorax also had pulmonary adhesions, which are consistent with pleuropneumonia.

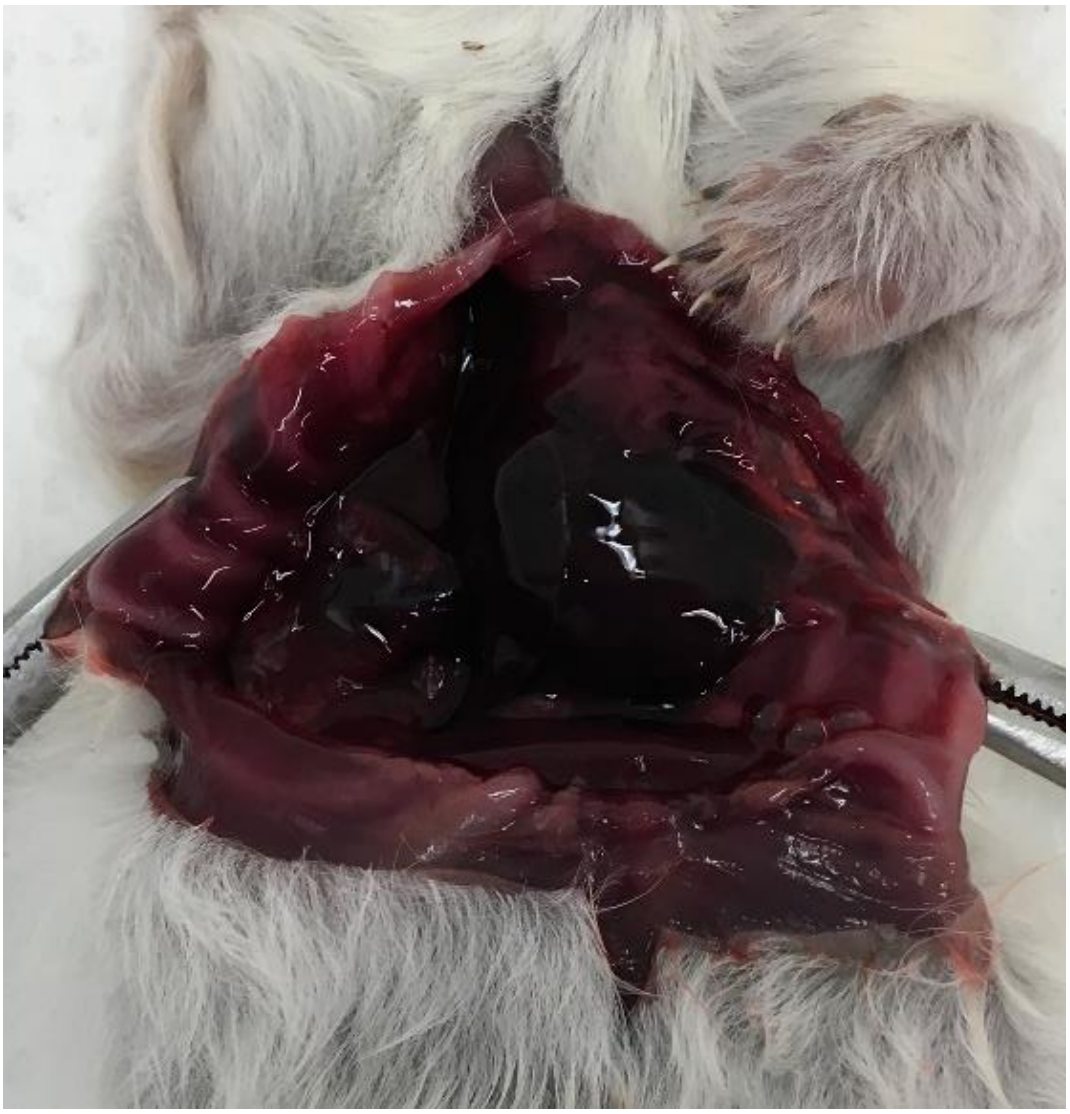


Figure III-12. Kitten with free blood in its thoracic cavity. There were two Persian kittens that had free blood in their thoracic cavities. This could be due to pre-mortem hemothorax or post-mortem autolysis.

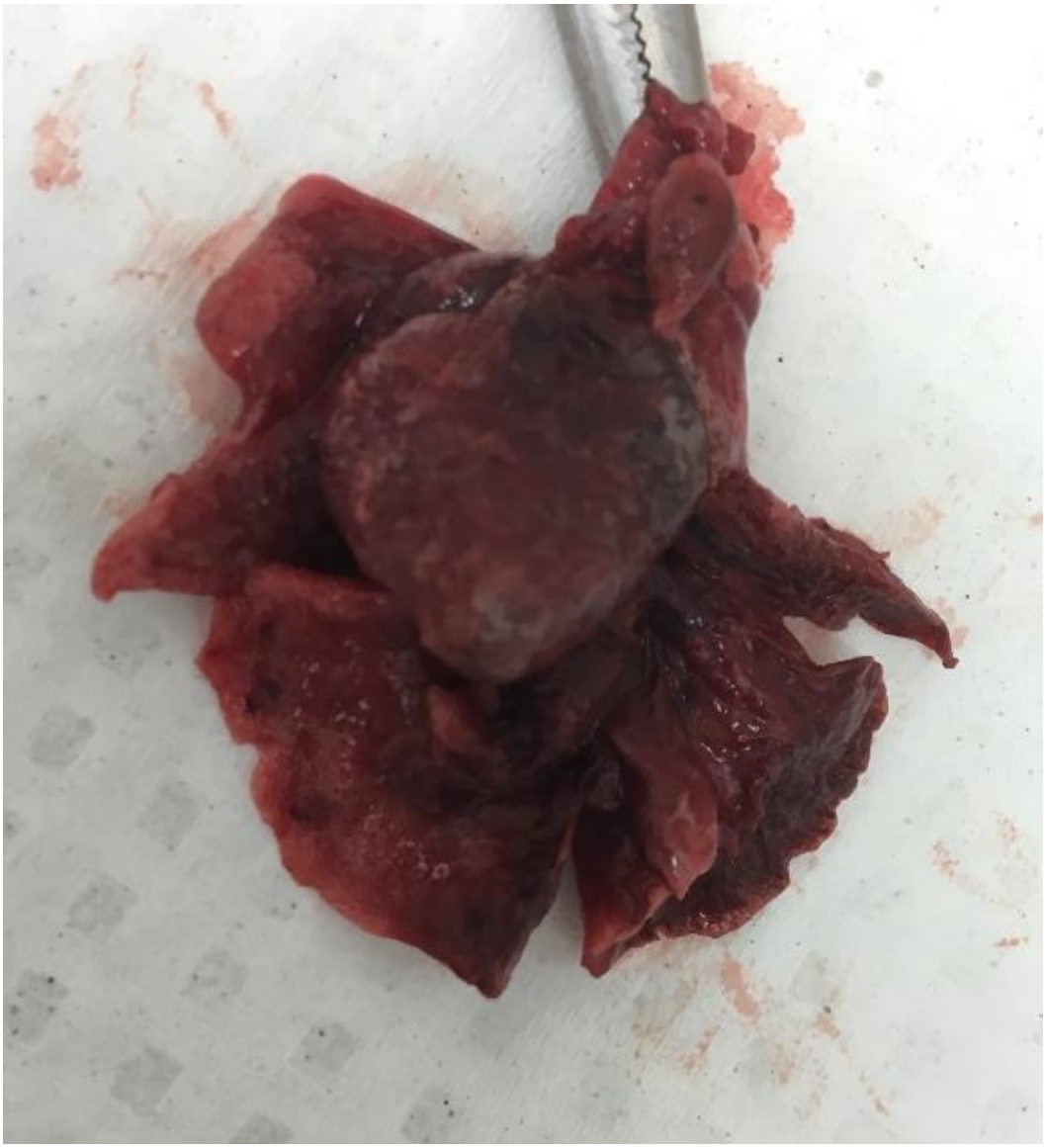


Figure III-13. Heart with suspected myocarditis. The heart and lungs removed from a kitten postmortem. The cardiac gross lesions are consistent with myocarditis. Myocarditis was suspected in one Devon Rex kitten.

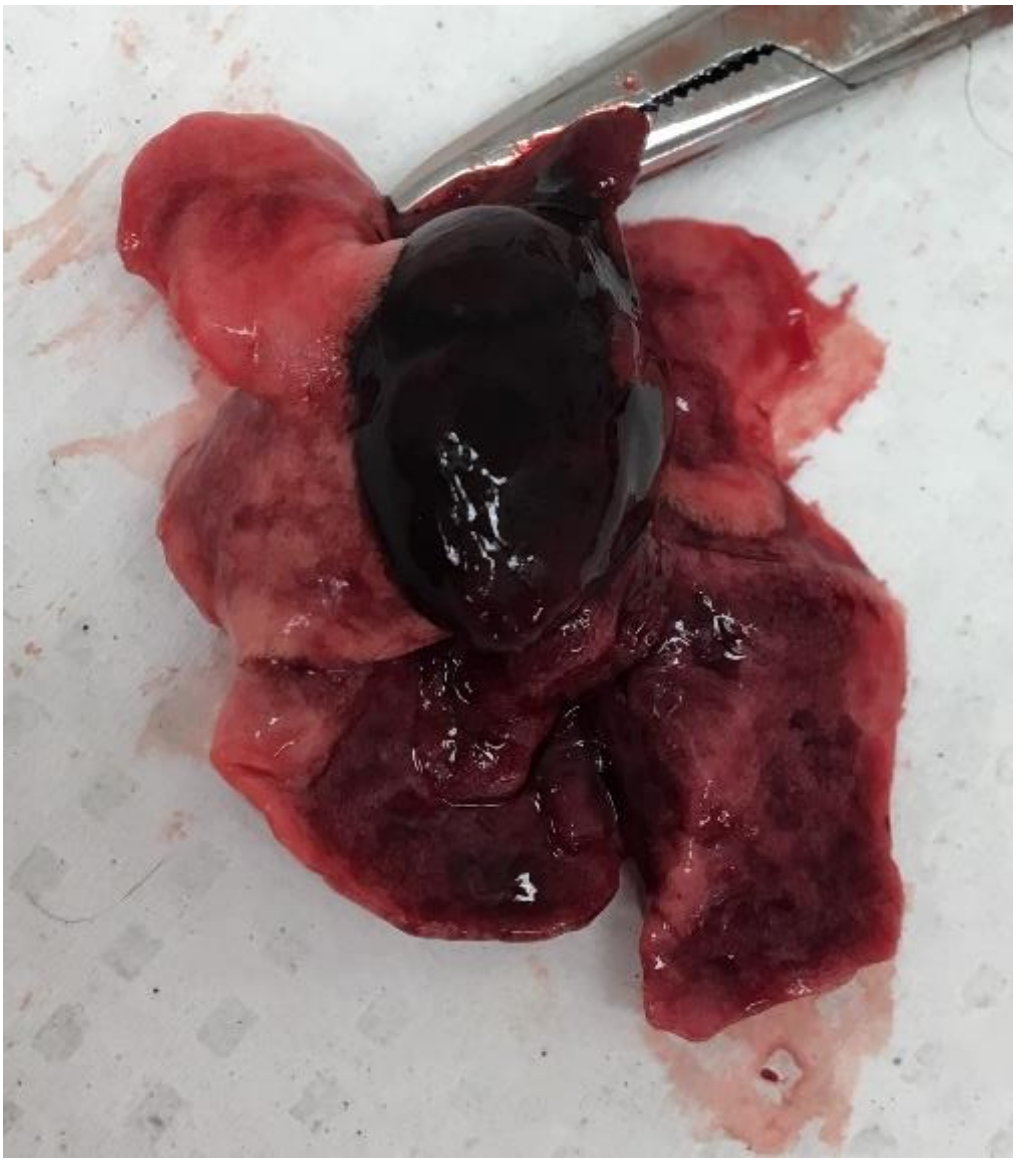


Figure III-14. Lungs with partial pulmonary congestion. The heart and lungs removed from a kitten postmortem, the lungs unevenly colored. Examination of the lungs revealed 31 kittens with diffuse patches of pink and red lung tissue consistent with partial pulmonary congestion (eighteen Persian kittens and thirteen Devon Rex kittens).

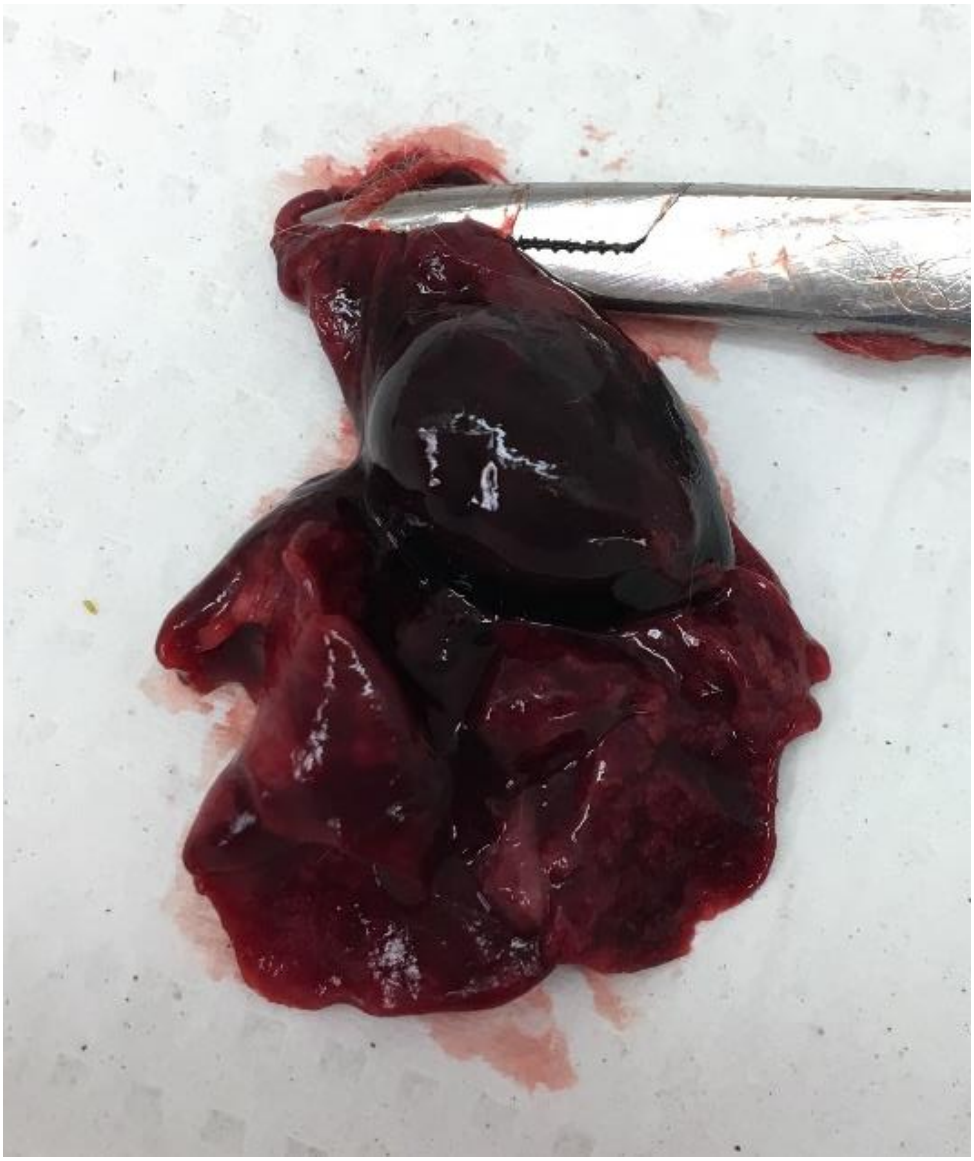


Figure III-15. Lungs with severe pulmonary congestion. The heart and lungs removed from a kitten postmortem, the lungs solidly dark red in coloration. There were thirteen kittens with dark red coloration of their lung tissue consistent with more severe pulmonary congestion, nine of which were stillborn.

Lung tissue from each left caudal lung lobe was evaluated for buoyancy in water indicating a live birth. Excluding the kitten that was too small and mummified to be fully examined, only one stillborn kitten had lung tissue that floated in water. This kitten presented with tissue desiccation that was consistent with early mummification so the density of the lung tissue could have been affected by tissue decomposition (**Figure III-16**). The other thirteen stillborn kittens had lung tissue that sank in water, indicating that their lung tissue was not aerated. In addition to the thirteen stillborn kittens, there were six other kittens (four Devon Rex, two Persian) that had lung tissue that sank in water, which could be due to partial or complete atelectasis, the presence of fluid or food in the airways, or changes due to decomposition **(Figure III-17**). These six kittens ranged in ages from one to fourteen days.

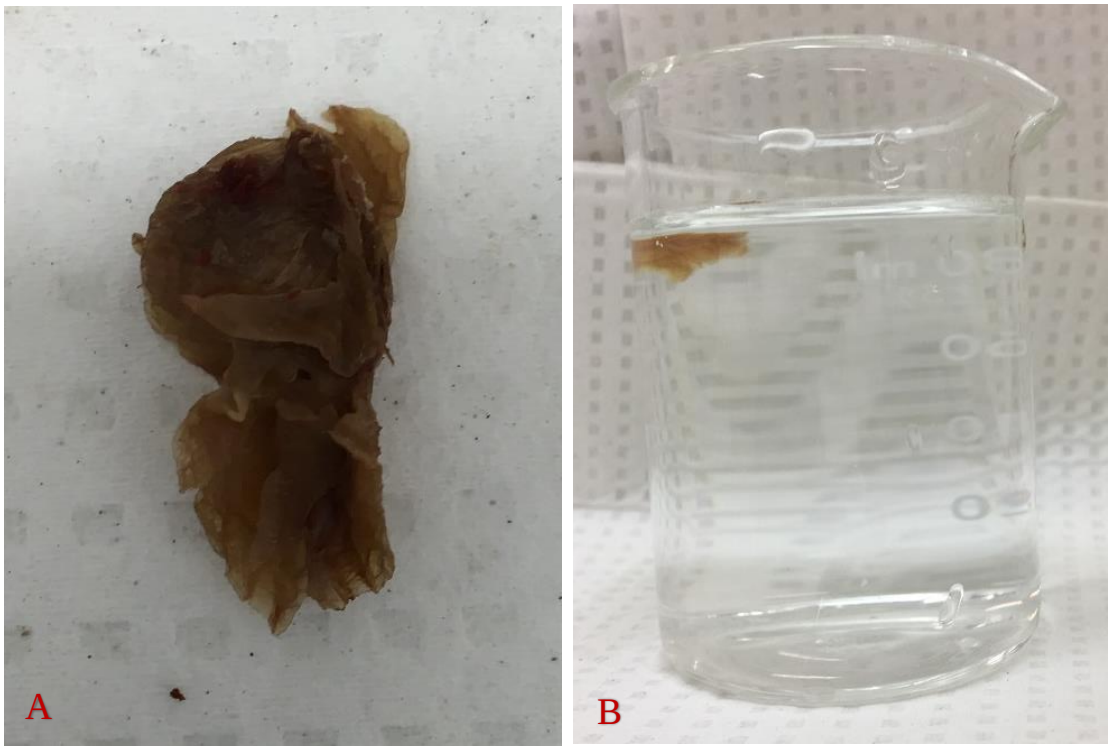
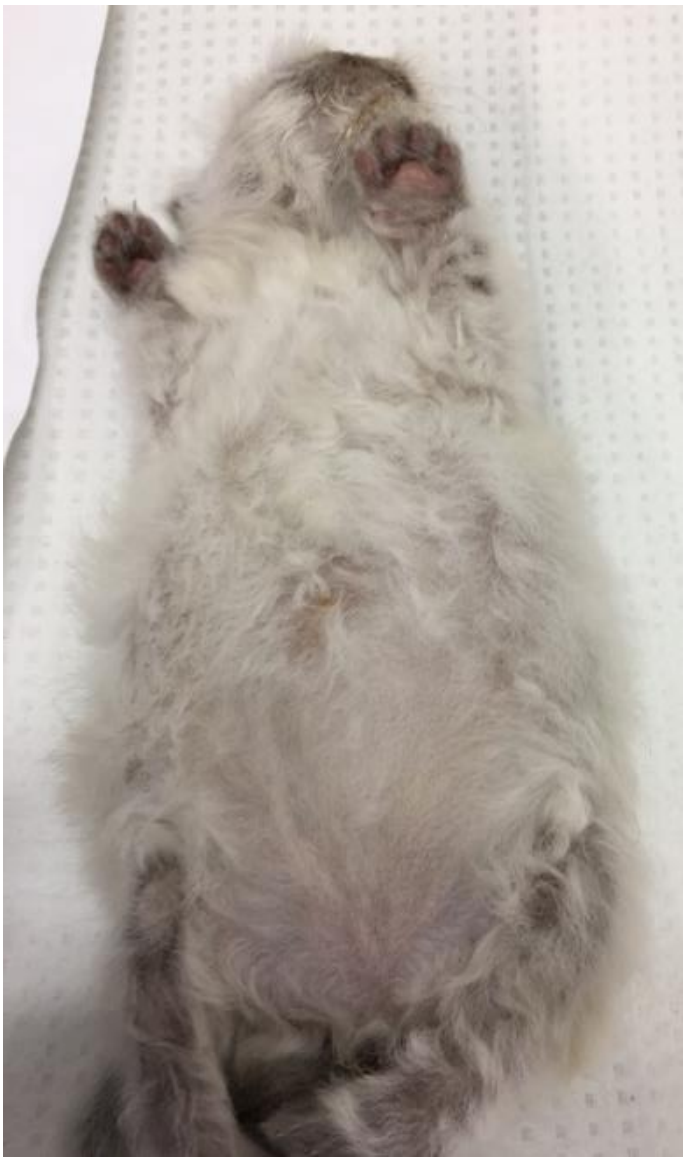


Figure III-16. Heart and lungs from mummified kitten. A: The heart and lungs from a stillborn tissue which appeared to have undergone the beginning of mummification. B: The desiccated lung tissue floating in water, presumably because of gases present in the tissue secondary to decomposition.

Figure III-17. Demonstration of lung floatation test and partial atelectasis. Three samples of lung tissue from a kitten, demonstrating partial atelectasis. Six kittens that were born alive exhibited partial atelectasis upon a lung tissue float test.

III. C. 2. B. Abdominal cavity

During abdominal examination, one Persian kitten and one Devon Rex kitten presented with ascites (**Figure III-18**). The liver of one Devon Rex kitten was unevenly colored consistent with partial hepatic congestion (**Figure III-19**). Pale coloration of the liver was present in six kittens, four Persian kittens and two Devon Rex kittens all from different litters (**Figure III-20**). Pale coloration of the liver has been associated with *Enterococcus hirae* infection affecting the hepatic ducts (Lapointe et al., 2000) Pale coloration of the liver has also been associated with low fat diets and high glutamic acid diets (Deady et al., 1981; Mackay, 1921). The ages at death for the kittens with pale coloration of the liver ranged from one to fourteen days. Three of the four Persian kittens with pale coloration of the liver also had pale coloration of the spleen (**Figure III-21**).



Figures III-18. Kitten with a distended abdomen from ascites. A total of two kittens presented with ascites (one Persian kitten and one Devon Rex kitten).

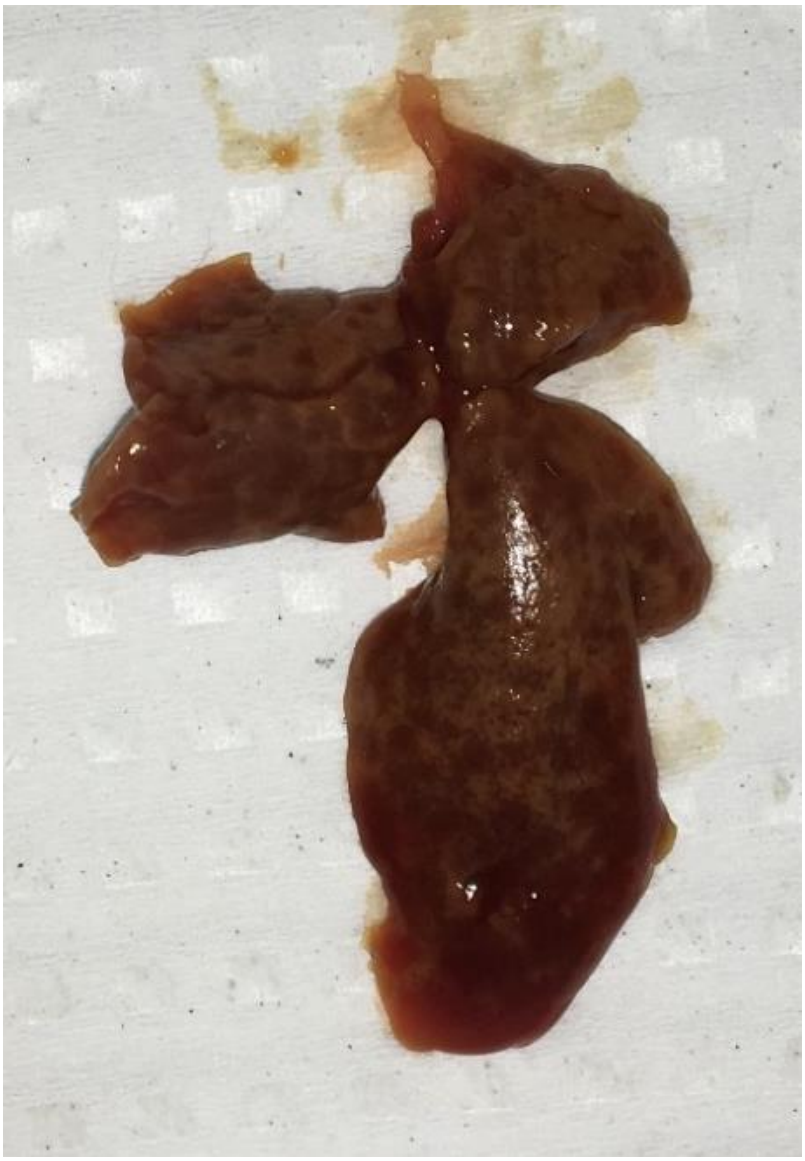


Figure III-19. Liver with partial hepatic congestion. A liver from a Devon Rex kitten that had uneven coloring, consistent with partial hepatic congestion

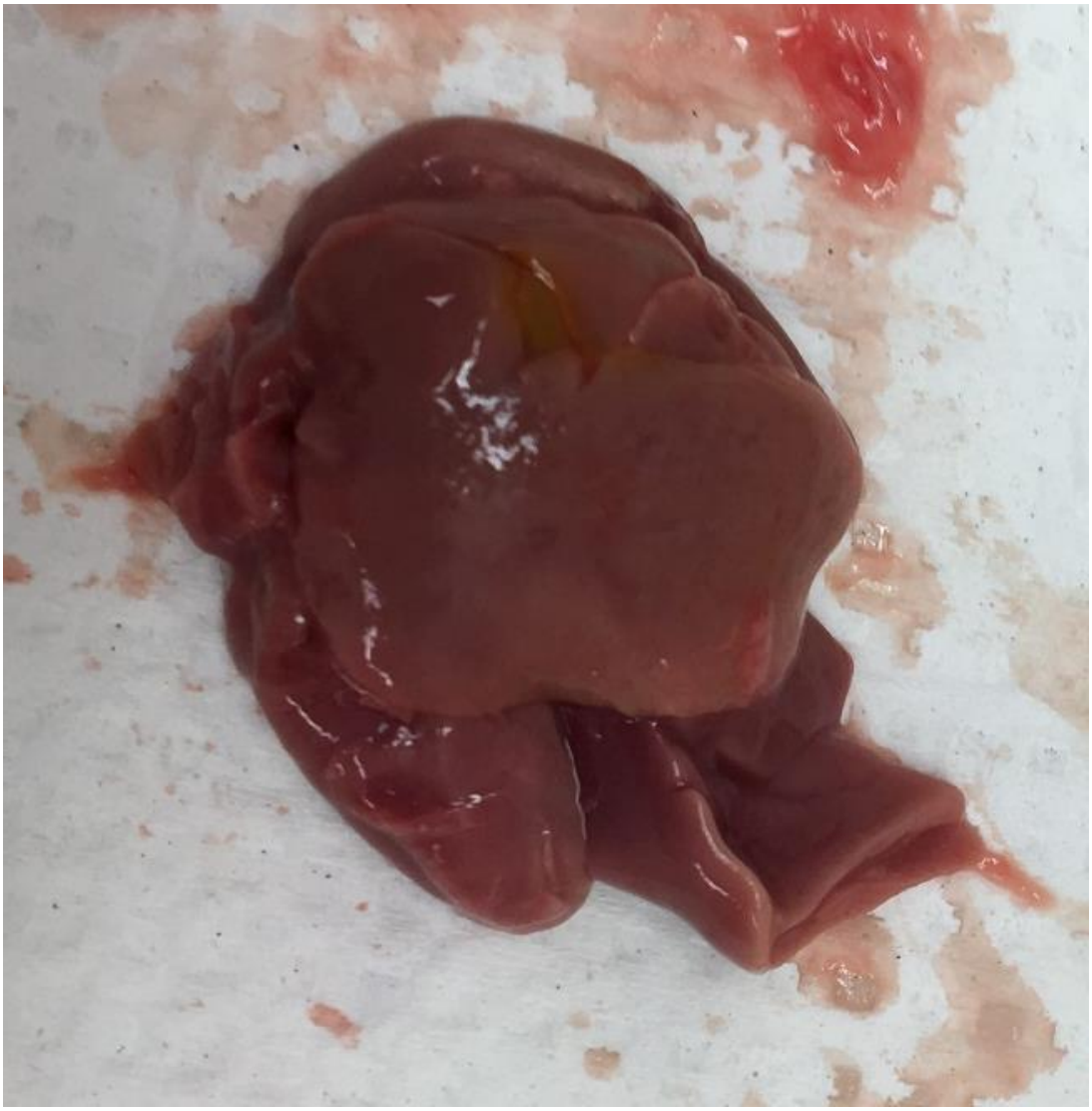


Figure III-20. Liver with pale coloration. A liver with pale coloration that was removed from a kitten during postmortem examination. Pale coloration of the liver was present in six kittens (four Persian kittens and two Devon Rex kittens).

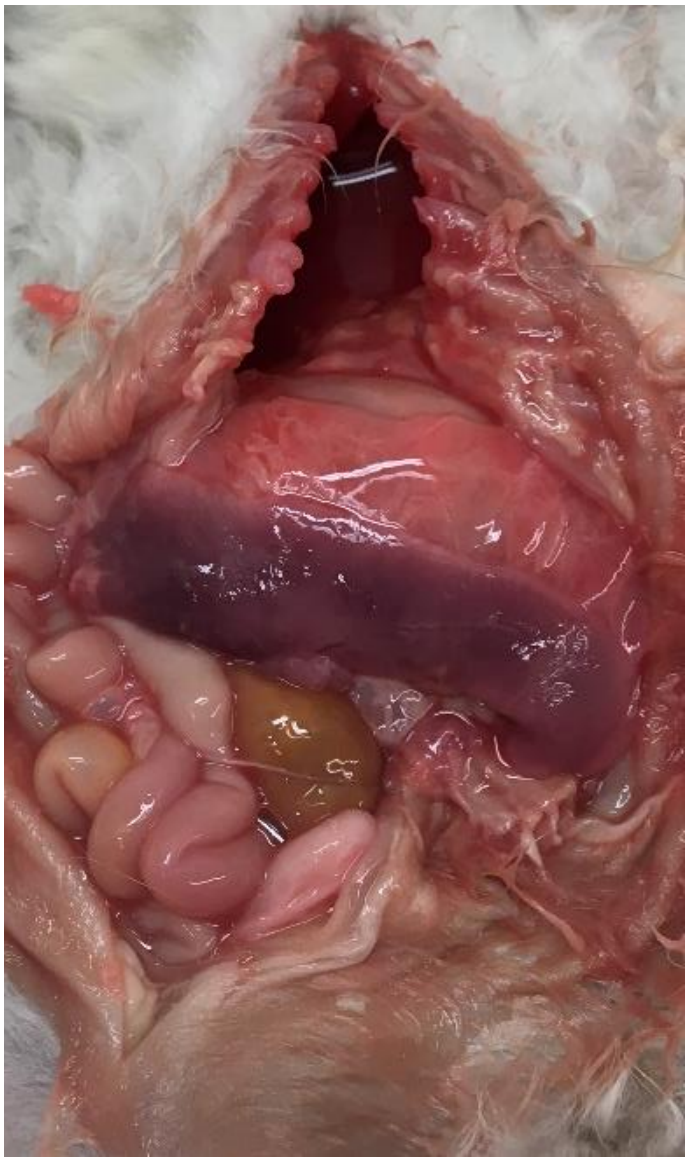
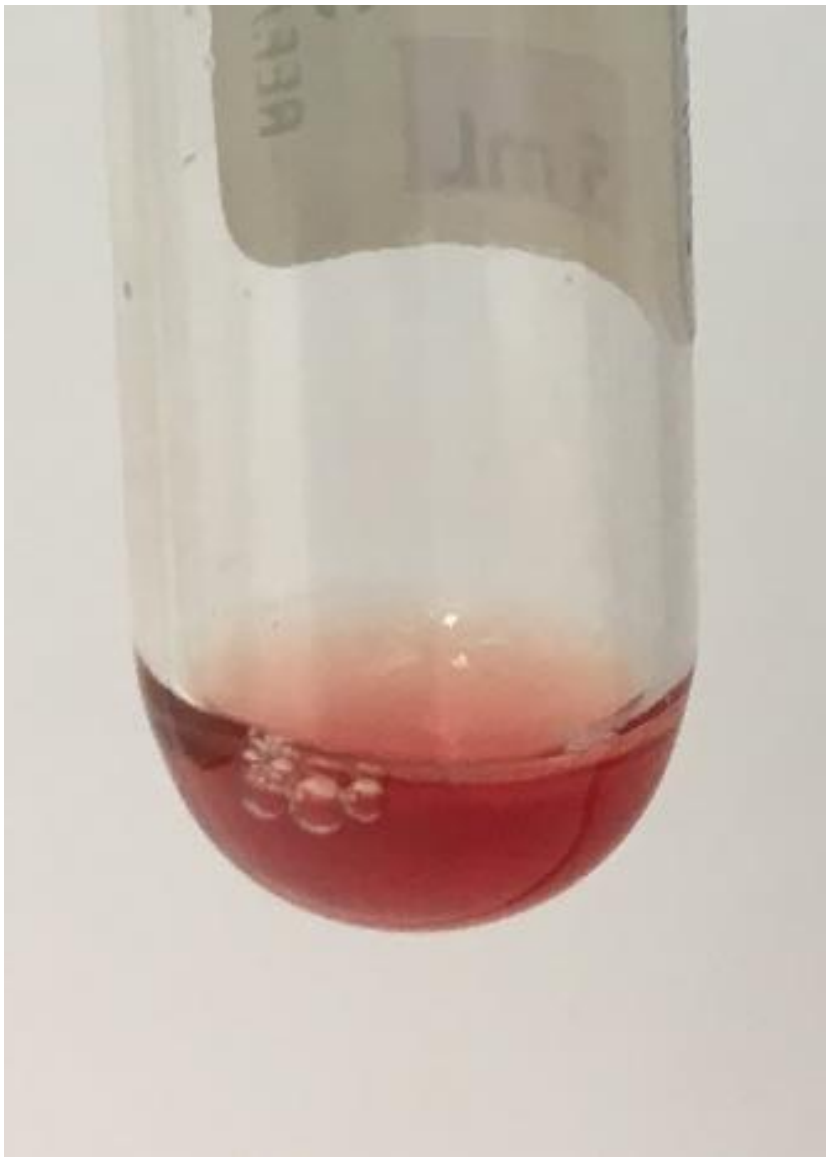


Figure III-21. Spleen with pale coloration. A spleen with pale coloration, in situ during a postmortem examination of a kitten. Pale coloration of the spleen was present in three Persian kittens that also presented with pale coloration of the liver.

When it was possible, urine was aspirated from the urinary bladder. Pigmenturia was identified in six kittens that died within the first two days of life (**Figure III-22**). Two were Persian kittens from the same litter, and the other four

were Devon Rex kittens from three litters. One Persian kitten had congestion of the superficial cortical kidney blood vessels (**Figure III-23**). Another Persian kitten had small hemorrhages present on the surface of its kidneys, which has been associated with feline infectious peritonitis (**Figure III-24**; Evermann et al., 1981). Two stillborn kittens from the same Devon Rex litter had adrenal glands that appeared to be enlarged, which could be indicative of prenatal stress (**Figure III-25**).

Possible causes of pigmenturia that are relevant to neonatal mortality include: premortem erythrocyte destruction and postmortem decomposition of urinary blood vessels. The four Devon Rex kittens that presented with pigmenturia resulted from matings between blood type A queens and blood type B toms. Because the naturally occurring anti-B alloantibodies in type A cats have weak hemolyzing and hemagglutinating activity, the risk of neonatal isoerythrolysis in kittens resulting from these matings is low but not impossible (Silvestre-Ferreira and Pastor, 2010). There were also two Persian cats from the same litter that presented with hematuria. Because blood type B is rare in Persian cats, the risk of Persian kittens being affected by neonatal isoerythrolysis is low but not impossible. Using Hardy-Weinberg equilibrium, it is estimated that 2% of random Persian matings would be at risk of developing neonatal isoerythrolysis, compared to 14% of random Devon Rex matings (Silvestre-Ferreira and Pastor, 2010). Finally, while pigmenturia can be caused by urinary tract infections in adult cats, urinary tract infections are rare in young kittens.



Figures III-22. Demonstration of pigmenturia. Red-colored fluid aspirated from the bladder, which is consistent with pigmenturia. Pigmenturia was identified in six kittens (four Devon Rex kittens and 2 Persian kittens).

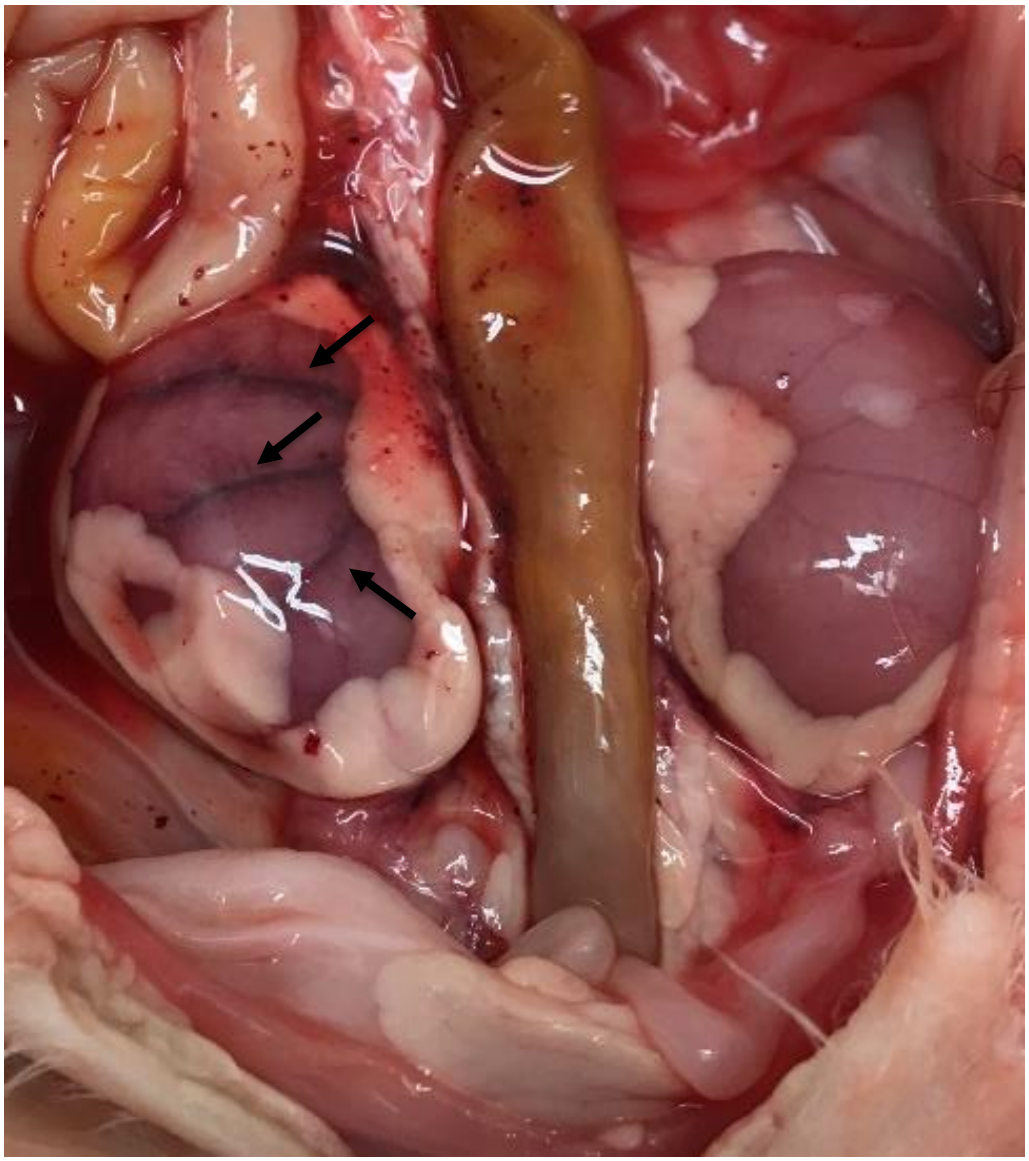


Figure III-23. Congestion of the right superficial cortical kidney vessels. Kidneys of a kitten that presented with congestion of the right superficial cortical kidney vessels (arrows). The superficial vessels on the left kidney are normal.

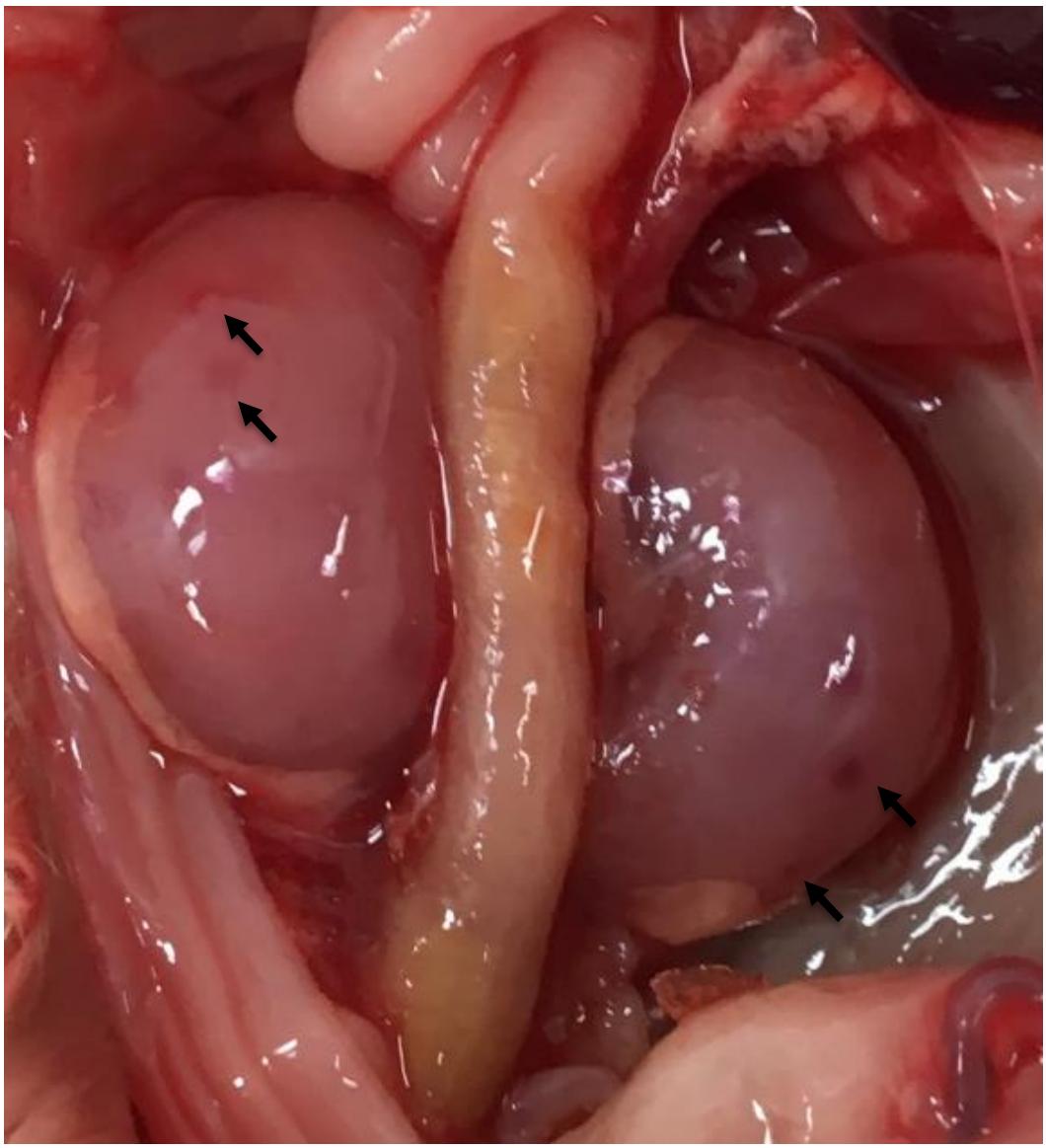


Figure III-24. Small hemorrhages on the kidneys. The kidneys of a Persian kitten, which had small hemorrhages on their surfaces.

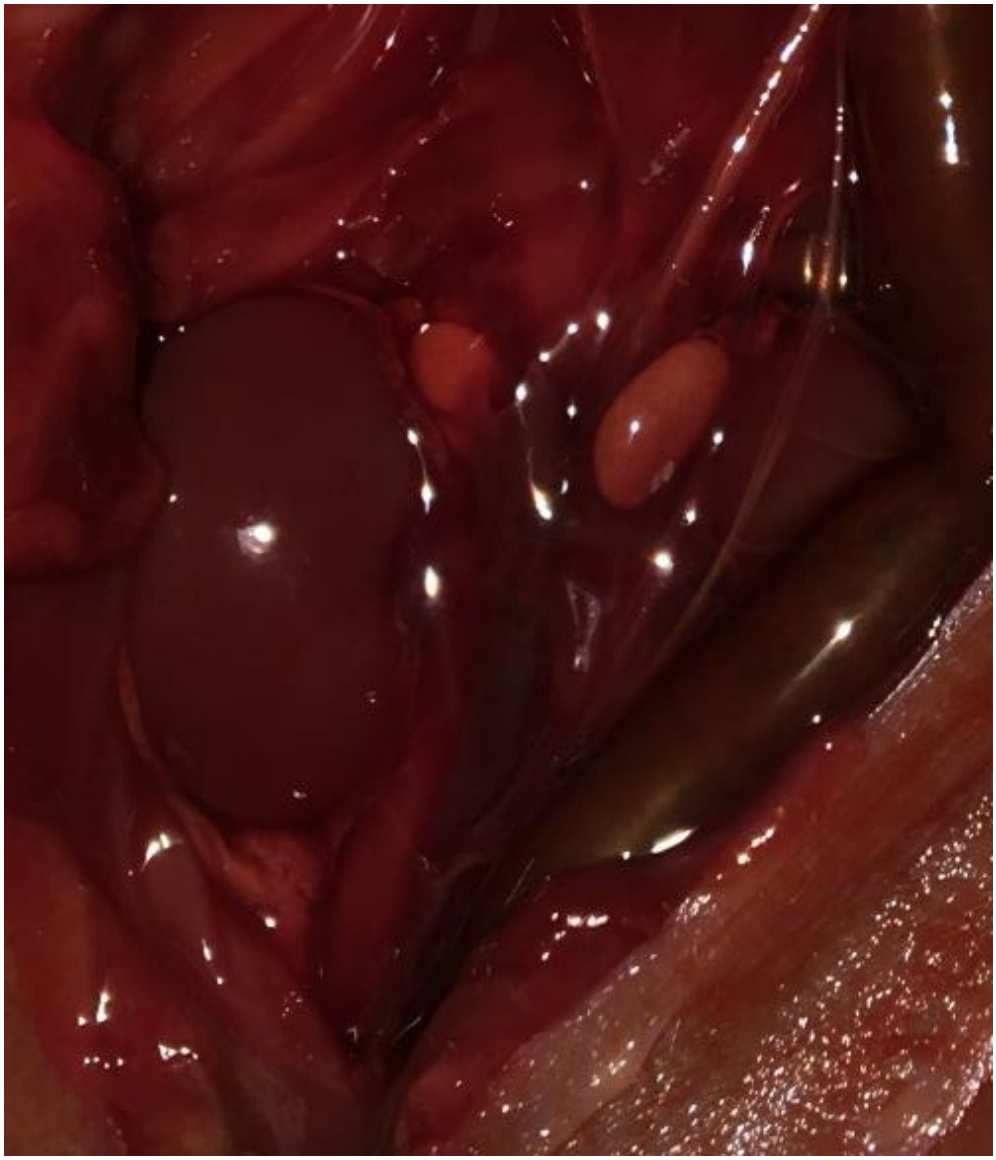


Figure III-25. Enlarged adrenal glands. The enlarged adrenal glands of a stillborn kitten. Adrenal glands that appeared enlarged were present in two stillborn Devon Rex kittens from the same litter.

III. D. Infectious versus noninfectious

There were three Persian kittens from different litters that showed signs of possible infection before death (i.e. nasal, ocular, or umbilical discharge; **Figure III-**

26). There were also nine Devon Rex kittens from seven litters that presented with suspected pyothorax upon post-mortem examination (**Figure III-27**). There was also one Devon Rex kitten that had gross lesions consistent with myocarditis. Counting kittens with signs of infection before death and kittens with suspected pyothorax, it is estimated that at least thirteen kittens (three Persian, ten Devon Rex) had possible infectious causes of mortality. Excluding stillborn kittens, it is estimated that at least 29.5% (13/44) of kitten deaths in the current study could have been due to infection.

In addition to kittens that showed signs of infectious disease before death or upon postmortem examination, stillbirths can be the result of infectious disease. In general, viruses cause feline reproductive loss more often than bacteria or protozoa (Daniel Givens and Marley, 2008). In particular, feline panleukopenia virus, feline leukemia virus, feline rhinotracheitis virus, and feline infectious peritonitis virus have been associated with abortion, stillbirth, neonatal death, and fetal mummification (Lefebvre, 2015). Feline immunodeficiency virus can also cause stillbirth (Daniel Givens and Marley, 2008). However, without further testing it cannot be determined how many stillbirths were due to infections.

There were four stillborn kittens (three Persian, one Devon Rex) that were affected by dystocia and presumably died from anoxia. However, there was no overlap between kittens affected by dystocia and kittens with abdominal bruising, pigmenturia, or free blood in their thoracic cavities. Counting stillbirths affected by dystocia, at least 26.6% (4/15) of stillbirths were due to noninfectious causes. This is supported by previous reports that 31% of deaths in the first two weeks of life and 33% of deaths in the first 12 weeks of life are from noninfectious causes (Cave et al.,

2002; Mossi-Dieth et al., 1990). However, these previous reports did not focus on stillbirths.

If it is assumed that a minimum number of kitten deaths or stillbirths had infectious etiologies (13/59, 22.0%) and a minimum number of kitten deaths or stillbirths had noninfectious etiologies (4/59, 6.8%), then there could be up to 42 kitten deaths or stillbirths with idiopathic etiologies (42/59, 71.2%). This is consistent with the previous report that gross abnormalities are not a major factor in neonatal kitten death (Lawler and Monti, 1984). Two other reports have found a 19% rate of idiopathic causes of mortality occurring in the first two weeks of life and a 33% rate of idiopathic causes of mortality occurring in the first 12 weeks of life (Cave et al., 2002; Mossi-Dieth et al., 1990).

Signs before death

	MAID	DYST	MAT NEG	HYPO	DYSP	NO WT
Total	13	8	5	5	13	7
By breed						
Persian	8	5	5	3	1	5
Devon Rex	5	3		2	12	2
By age						
Stillborn	8	4				
≤ 1 day		2		1	9	1
2 - 7 days	2	1	2	2	3	3
8 - 14 days	3	1	3	2	1	3

Figure III-26. Summary of antemortem symptoms. MAID = maiden litter; DYST = dystocia; MAT NEG = maternal neglect; HYPO = hypothermia; DYSP = dyspnea; NO WT = no weight gain.

Necropsy findings

	ABD BRU	PYO	ATE	PART PULM	PULM CON	PAL LIV	HEMA
Total	7	9	19	31	13	6	6
By breed							
Persian	2		9	18	10	4	2
Devon Rex	5	9	10	13	3	2	4
By age							
Stillborn	1		13		9		
≤ 1 day	3	7	4	10	1	1	5
2 - 7 days	3	2		10		1	1
8 - 14 days			2	11	3	4	

Figure III-27. Summary of postmortem examination findings. ABD BRU = abdominal bruising; PYO = pyothorax; ATE = atelectasis; PART PULM = partial

pulmonary congestion; PULM CON = pulmonary congestion; PAL LIV = pale coloration of liver; HEMA = hematuria.

IV. Conclusion and future directions

Without further diagnostic testing, it is impossible to recommend specific actions for cat breeders to take in order to reduce kitten losses. Nevertheless, the author recommends proper and thorough cattery hygiene, with special care taken to minimize the exposure of neonatal kittens to pathogens, which can occur by contact with other adult cats in the cattery. The author also recommends consultation with a veterinarian knowledgeable in feline pediatrics, who can provide guidance with vaccination schedules and neonatal care. Finally, the author advises caution with the practice of tube feeding kittens because of the possibility of causing injury and infection.

This research project has demonstrated the ability and desire of cat breeders to work with investigators in the interest of improving the health and welfare of neonatal kittens. Whether it is for cost, convenience, or other reasons, it appears some cat breeders lack access to veterinarians who are equipped for and experienced in postmortem examination of kittens.

This research project has also demonstrated the lack of current research in causes of kitten mortality. Recent investigations appear to focus on individual diseases that can cause kitten mortality or overall kitten mortality rates. In order for kitten losses to be prevented, the causes of kitten mortality must be studied. Further, there is a need for research that compares causes of kitten mortality between types

and breeds of cats. Previous studies have shown differing incidences of diseases in different breeds of cats, but these studies tend to focus on adult cats (e.g. Valpalahti et al., 2016; mean age of cats 5.4 years). In particular, there is a need for updated research into causes of kitten mortality that includes laboratory testing of tissue samples due to emerging feline infectious diseases.

V. References

Barrs VR, Beatty JA. Feline pyothorax – new insights into an old problem: part 1. aetiopathogenesis and diagnostic investigation. *The Vet J* 2008;179:163-170.

Bücheler J. Fading kitten syndrome and neonatal isoerythrolysis. *Vet Clin North Am Small Anim Pract* 1999;29(4):853-870.

Casal ML, Jezyk PF, Giger U. Transfer of colostral antibodies from queens to their kittens. *Am J Vet Res* 1996;57(11):1653-1658.

Cave TA, Thompson H, Reid SWJ, Hodgson DR, Addie DD. Kitten mortality in the United Kingdom: a retrospective analysis of 274 histopathological examinations (1986 to 2000). *Vet Rec* 2002;151:497-501.

Daniel Givens M, Marley MSD. Infectious causes of embryonic and fetal mortality. *Theriogenology* 2008:1-16.

Deady JE, Anderson B, O'Donnell JA, Morris JG, Rogers QE. Effect of level of dietary glutamic acid and thiamin on food intake, weight gain, plasma amino acids, and thiamin status of growing kittens. *J Nutri* 1981;111:1568-1579.

Evermann JF, Baumgartener L, Ott RL, Davis EV, McKeirnan AJ. Characterization of feline infectious peritonitis virus isolate. *Vet Pathol* 1981;18:256-265.

Fournier A, Masson M, Corbière F, Mila H, Mariani M, Grellet A, Chastant-Maillard S. Epidemiological analysis of reproductive performances and kitten mortality rates in 5,303 purebred queens of 45 different breeds and 28,065 kittens in France. *Reprod Domest Anim* 2017;52(Suppl. 2):153-157.

Giger U, Casal ML. Feline colostrum – friend or foe: maternal antibodies in queens and kittens. *J Reprod Fertil Suppl* 1997;51:313-316.

Holst BS, Frössling J. The Swedish breeding cat: population description, infectious diseases and reproductive performance evaluated by a questionnaire. *J Feline Med Surg* 2009;11:793-802.

Lapointe JM, Higgins R, Barrette N, Milette S. *Enterococcus hirae* enteropathy with ascending cholangitis and pancreatitis in a kitten. *Vet Pathol* 2000;37:282-284.

Lawler DF, Monti KL. Morbidity and mortality in neonatal kittens. *Am J Vet Res* 1984;45(7):1455-1459.

Lefebvre RC. Fetal mummification in the major domestic species: current perspectives on causes and management. *Vet Med: Res Rep* 2015;6:233-244.

Little S. Congenital defects of kittens. In: Tufts' canine and feline breeding and genetics conference; 2005.

Little S. Feline pediatrics: how to treat the small and the sick. *Vetlearn.com Compendium: Continuing Education for Veterinarians;* 2011.

Löhr CV. Postmortem examination of the puppy and kitten. In: *Small Animal Pediatrics: The First Twelve Months of Life* (Peterson ME and Kutzler MA, eds). Elsevier: St. Louis, Missouri. 2011; pp 276-287.

Mackay HMM. The effect on kittens of a diet deficient in animal fat. *Biochem J* 1921;15(1):19-27.

Meurs KM, Fox PR, Magnon AL, Liu S, Towbin JA. Molecular screening by polymerase chain reaction detects panleukopenia virus DNA in formalin-fixed hearts from cats with idiopathic cardiomyopathy and myocarditis. *Cardiovasc Pathol* 2000;9(2):119-126.

Mossi-Dieth V, Hauser B, Corboz L, Lutz H, Pospischil A. Todes und erkrankungsursachen bei katzenwelpen. *Schweiz Arch Tierheilk* 1990;132:587-594.

Rolim VM, Casagrande RA, Wouters ATB, Driemeier D, Pavarini SP. Myocarditis caused by feline immunodeficiency virus in five cats with hypertrophic cardiomyopathy. *J Comp Path* 2016;154:3-8.

Schlueter C, Budras KD, Ludewig E, Mayrhofer E, Koenig HE, Walter A, Oechtering GU. Brachycephalic feline noses: CT and anatomical study of the relationship between head conformation and the nasolacrimal drainage system. *J Feline Med Surg* 2009:11;891-900.

Silvestre-Ferreira AC, Pastor J. Feline neonatal isoerythrolysis and the importance of feline blood types. *Vet Med Int* 2010;2010:1-8.

Sparkes AH, Rogers K, Henley WE, Gunn-Moore DA, May JM, Gruffydd-Jones TJ, Bessant C. A questionnaire-based study of gestation, parturition and neonatal mortality in pedigree breeding cats in the UK. *J Feline Med Surg* 2006;8:145-157.

Vapalahti K, Virtala AM, Joensuu TA, Tiira K, Tähtinen J, Lohi H. Health and behavioral survey of over 8000 Finnish cats. *Front Vet Sci* 2016;3(70):1-16.

Varanat M, Broadhurst J, Linder KE, Maggi RG, Breitschwerdt EB. Identification of *Bartonella henseale* in 2 cats with pyogranulomatous myocarditis and diaphragmatic myositis. *Vet Pathol* 2011:49(4):608-611.

Appendix A: Form for cat breeders to fill out

1. **Does this kitten have a name? If so, what is it?** (This will help us to share the results of the necropsy with you).

2. **What identifying features does this kitten have?** (Color, coat type, etc.)

3. **How old was this kitten when it died? Was it stillborn?**

4. **What are the names of the queen/dam and tom/sire?** (This will help us to track any patterns we notice).

5. **What are the blood types of the queen/dam and tom/sire? If unknown, write "N/A."** (This will help us to evaluate any findings that suggest neonatal isoerythrolysis).

6. **What day(s) did the breeding occur?** (This will help us to track differences between premature, on time, and overdue litters).

7. **What medications (if any) did you administer to the queen/dam during her gestation?**

8. **Approximately when did the queen's labor start? When was the first kitten of this litter born? When was the last kitten of this litter born?**

9. **When was this kitten born?**

10. **What signs (if any) did you notice while the kitten was being born?** (Signs may include but are not limited to: duration of labor,

11. **What medications (if any) did you administer to the kitten? Was this kitten euthanized?**

12. **When did this kitten die?** (This will help us to evaluate changes we suspect are due to freezing, and it will help us to confirm age at death).

13. **What signs (if any) did you notice leading up to the kitten's death?** (Signs may include but are not limited to: changes in temperature, breathing, appetite, weight, appearance).

14. **What else would you like us to know? Do you have any concerns regarding this kitten?**

Appendix B: Form for conducting postmortem examinations

Age at death given by breeder

Signs before death (agonal breathing?)

Description (fur color, etc.)

Number (start with 125--use letter suffix for littermates)

WT=**Weight** in grams

📷Take picture of **kitten (whole body) with identification number**

📷General appearance: does kitten appear **mummified**?

Congenital abnormalities (umbilical cord, palate, urogenital area)

📷P+T=appearance of palate and tongue (**cleft palate**?)

📷UGA=appearance of **urogenital area**

📷UC=appearance of **umbilical cord**

📷PL=Appearance of **placenta** if provided (keep whole thing as sample)

📷Note any **bruising** on abdomen/groin

📷Note any **tail** abnormalities (especially if tip is missing)

📷HE=appearance of **heart** (note myocarditis)

Lung tissue (appearance, float test, sampling)

RCL sample=**Right caudal lung sample**

📷Appearance of lungs (uneven coloring?)

📷LCL float=**Left caudal lung float** test (hydrostatic test)

(Does not float→ suspect stillborn)

📷Is there any **pus in the thoracic cavity**? (Yes→ suspect pleuropneumonia)

📷Is there any **lung adhesion**?

Liver (sampling)

📷LI=**appearance of liver** (note jaundice/icterus)

Take **sample** from "split" lobe, which I think is the left lobe

📷SP=appearance of **spleen**

📷BA=**Bladder/urachus aspirate**

📷KI=appearance of **kidneys**

Note if perirenal **brown adipose tissue** (BAT) is present

BAT is also present along spine, can indicate age at death

📷Are **adrenal glands** obvious/large? (perinatal stress?)

📷**Sample from any abnormal organs**

DX=**diagnosis** (pleuropneumonia, stillborn, etc.)

College of Agricultural Sciences | Honors College

CAUSES OF NEWBORN MORTALITY IN DEVON REX KITTENS

Holly Omoto, Michelle Kutzler

Department of Animal and Rangeland Sciences

	Signs before death									
	No signs		Dyspnea		Hypothermia		Facial deformity with neck and abdominal edema		Dystocia	
Age (days)	1	2-3	1	2-3	1	2-3	1	2-3	1	2-3
Pathologies										
None found			2			1			1	
Pyothorax		2	3		1					
Icterus			1							
Cleft palate	1									
Hematuria	2		1				1			
Atelectasis	2	1							1	

Table 1. Table of signs before death compared with gross pathologies. Over half of the kittens (9/17, 52.9%) died from infectious causes (pyothorax and hematuria not associated with neonatal isoerythrolysis). Dyspnea was reported in four of the six kittens that presented with pyothorax. Note kittens with multiple signs before death or multiple gross pathologies: one one-day-old kitten with abnormal breathing and dystocia; one one-day-old kitten with abnormal breathing and facial deformity with neck and abdominal edema; one two/three-day-old kitten with pyothorax and atelectasis.

INTRODUCTION

- Even in well-managed catteries, up to 16% of kittens may be stillborn and **up to 20% of kittens may die before weaning**.[1]
- The objective of this study was to **identify gross pathology lesions** in newborn purebred kittens who died spontaneously.
- We hypothesized that lesions consistent with **congenital causes of mortality would be more common than infectious causes**.

METHODS

- Post-mortem examinations were performed on **17 newborn (up to 3 days old) Devon Rex kittens**. These kittens were from 12 litters.
- The **palate, heart, lungs, liver, spleen, kidneys, bladder, umbilical stump, and urogenital area** were examined for abnormalities.
- A sample of **lung tissue was floated in water** to evaluate atelectasis (collapsed lung), and **urine was aspirated from the bladder** to evaluate hematuria (blood in urine).
- The **age at death, the dam's and sire's blood types, and clinical signs present before death** were also recorded.

RESULTS

- **No gross pathological abnormalities were found in 17.6% (3/17) of the kittens.** Of these kittens with no abnormalities, signs before death were hypothermia (low body temperature; n=1), dystocia (difficult birth; n=1), and dyspnea (abnormal breathing; n=2).
- There were **two kittens with two signs before death**, one kitten with dystocia and dyspnea; one kitten with dyspnea and facial deformity with neck and abdominal edema (accumulation of fluid).
- **A cleft palate was found in one kitten (5.8%)**, and no other congenital defects were found in the remaining kittens.
- **No kittens died from neonatal isoerythrolysis.** Hematuria (a sign associated with neonatal isoerythrolysis, NI) was present in 17.6% (3/17) of the kittens, and icterus (discolored liver; another sign associated with NI) was identified in one kitten. However, **all four of these kittens were produced by blood type B sires,** indicating that NI was not the cause.[2]
- **Pyothorax (pus in chest cavity; indicative of infection) was present in 35.3% (6/17)** of the kittens, and dyspnea was reported in 4/6 of those kittens prior to death. One kitten with pyothorax had atelectasis.

DISCUSSION

- About 13% of Devon Rex litters have at least one congenital defect.[3] However, we found **one congenital defect in 17 kittens from 12 litters** (1/12, 8.3%).
- Our findings revealed that **52.9% (9/17) of the kittens up to 3 days of age died from infectious causes** (pyothorax and hematuria not associated with NI).
- Additional diagnostic testing is planned to **determine the etiologic agents** involved with the infectious causes of feline newborn mortality.

REFERENCES

1. Malandain É, Little S, Casseleux G, et al: Diseases of the neonate. In: Malandain É, editor. Practical guide to cat breeding. Aimargues(France): Aniwa SAS on behalf of Royal Canin; 2006 p. 164-179.
2. Silvestre-Ferreira AC, Pastor J: Feline neonatal isoerythrolysis and the importance of feline blood types. Vet Med Int 2010;2010:1-8.
3. Little S: Congenital defects of kittens. In: Tufts' canine and feline breeding and genetics conference; 2005

ACKNOWLEDGEMENTS

The authors would like to thank **Tina Chittick of Everafter Devon Rex Cattery** for her collaboration on this project. The authors would also like to thank the **Theriogenology Laboratory** for covering shipping costs.

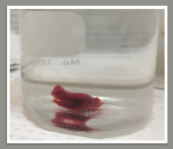


Figure 1. Demonstration of atelectasis. Atelectasis (collapsed lung) was present in 4/17 kittens (23.5%). One of the six kittens with pyothorax had atelectasis.

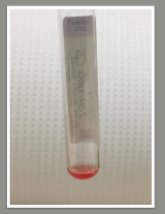


Figure 2. Demonstration of hematuria. Hematuria (blood in urine) was present in 3/17 kittens (17.6%). Hematuria can be a sign of neonatal isoerythrolysis or an infectious pathology. Neonatal isoerythrolysis was ruled out because the sires had type B blood.[2]

Figure 3. Demonstration of cleft palate. Cleft palate was present in 1/17 kittens (5.9%). Cleft palate is a congenital defect.

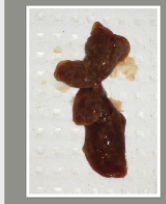


Figure 4. Demonstration of icterus. Icterus (discolored liver) was present in 1/17 kittens (5.9%). Icterus can be a sign of neonatal isoerythrolysis. However this was ruled out because the sire of the kitten had type B blood.[2]

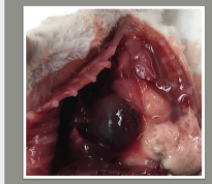


Figure 5. Demonstration of pyothorax. Pyothorax (pus in chest cavity) was present in 6/17 kittens (35.3%). Pyothorax suggests an infectious pathology.

Oregon State University

Appendix D: Theriogenology Conference abstract presentation

CAUSES OF NEWBORN MORTALITY IN DEVON REX KITTENS

Holly Omoto; Michelle Kutzler, DVM, PhD
Department of Animal and Rangeland Sciences
College of Agricultural Sciences
Corvallis, Oregon

Oregon State University

Introduction: Kitten mortality

Up to **16%** of kittens may be stillborn, and up to **20%** of kittens may die before weaning[1]

1. Malandain É, Little S, Casseleux G, et al: Diseases of the neonate. In: Malandain É, editor. Practical guide to cat breeding. Aimargues(France): Aniwa SAS on behalf of Royal Canin; 2006 p. 164-179.
Image: https://cdn.pixabay.com/photo/2016/08/10/14/44/cat-1583462_960_720.png

Introduction: Devon Rex breed

8.8% of Devon Rex kittens are **stillborn**[3]

- Compared to 8.5% for all breeds[3]

18.1% of Devon Rex kittens are born alive but **die within first 60 days of life**[3]

- Compared to 7.9% for all breeds[3]

3. Fournier A, Masson M, Corbière F, et al: Epidemiological analysis of reproductive performances and kitten mortality rates in 5,303 purebred queens of 45 different breeds and 28,065 kittens in France. Reprod Domest Anim 2017;52 (Suppl. 2): 153-157.
Image: http://upload.wikimedia.org/wikipedia/commons/3/33/Devon_rex.jpg

Introduction: Congenital defects

13% of Devon Rex litters have at least one congenital defect[1]

- Examples of congenital defects in Devon Rex kittens include flat chest defect, cleft palate, and umbilical hernia[4]
- Compared to 5-80% for other breeds (Norwegian Forest Cat, Contemporary Burmese)[4]
- 9.5% of kittens that die within first two weeks of life die from congenital causes[2]

2. Case TA, Thompson H, Reid SWJ, et al: Kitten mortality in the United Kingdom: a retrospective analysis of 274 histopathological examinations (1986 to 2000). Vet Rec Case Rep 2002;151: 497-501.
4. Little S: Congenital defects of kittens. In: Tufts' canine and feline breeding and genetics conference; 2005.

Hypothesis & Objective

Hypothesis: We hypothesized that lesions consistent with **congenital causes of mortality** would be more common than infectious causes

Objective: Our objective was to **identify gross pathology lesions** in newborn Devon Rex kittens who died spontaneously

Methods

Sample	Examination	Testing	Records
• Kittens: 17 • Litters: 12 • Age: ≤3 days old • Breed: Devon Rex	• Palate • Heart • Lungs • Liver • Spleen • Kidneys • Bladder • Umbilical stump • Urogenital area	• Hydrostatic test to evaluate atelectasis • Urine aspirate to evaluate hematuria • Lung, liver, and placenta samples for future analysis	• Age at death • Blood types of queen and tom • Clinical signs present before death • Medications administered to dam or kitten

Results: Idiopathic

No gross pathological abnormalities were found in 3/17 kittens (17.6%)

Signs before death for these three kittens were hypothermia (n=1), dystocia (n=1), and dyspnea (n=2)

One kitten had two signs before death, dystocia and dyspnea

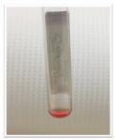
Results: Congenital

Cleft palate was found in 1/17 kittens (5.8%)

Only congenital defect found in all 17 kittens

Results: Immune-mediated

- Four kittens presented signs of neonatal isoerythrolysis
  - Hematuria was present in 3/17 kittens (17.6%)
  - Icterus was present in 1/17 kittens (5.8%)
- All four kittens were produced by blood type B sires[5]
  - **No kittens died from neonatal isoerythrolysis (NI)**

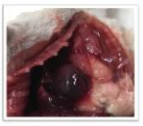


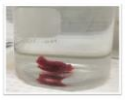
5. Silvestre-Ferreira AC, Pastor J: Feline neonatal isoerythrolysis and the importance of feline blood types. Vet Med Int 2010;2010:1-8.

Results: Respiratory

Pyothorax
- **Pyothorax was present in 6/17 kittens (35.3%)**
  - Dyspnea was reported in 4/6 kittens prior to death

Atelectasis
- **Atelectasis was present in 4/17 kittens (23.5%)**
  - One kitten with atelectasis had pyothorax





Discussion: Comparison with literature values

Idiopathic	Immune-mediated	Congenital	Infectious
No gross pathological abnormalities were found in 3/17 kittens (17.6%)	No kittens died from neonatal isoerythrolysis (NI)	Cleft palate was found in 1/17 kittens (5.8%); only congenital defect in 12 litters (1/12, 8.3%)	Pyothorax and hematuria (not associated with NI) were found in 9/17 kittens (52.9%)
19.0% of kittens ≤14 days die from idiopathic causes[2]	11.9% of kittens ≤14 days die from immune-mediated causes[2]	9.5% of kittens ≤14 days die from congenital causes[2]; 13% of Devon Rex litters have ≥1 congenital defect[4]	50% of kittens ≤14 days die from infectious causes[2]

2. Cave TA, Thompson H, Reid SWJ, et al: Kitten mortality in the United Kingdom: a retrospective analysis of 274 histopathological examinations (1986 to 2000). Vet Rec Case Rep 2002;151: 497-501.
4. Little S: Congenital defects of kittens. In: Tufts' canine and feline breeding and genetics conference; 2005.
6. Löhr CV: Postmortem examination of the puppy and kitten. In: Peterson ME, Kutzler MA, editors. Small animal pediatrics. St. Louis(Missouri): Elsevier Saunders; 2011 p.276-287.

Conclusion: Additional research

Testing
- Additional diagnostic testing is planned to **determine etiologic agents** involved with infectious causes of feline newborn mortality

Recruitment
- **Four additional cat breeders** who specialize in different breeds have been recruited for this project

Conclusion: Practice tips

1. Post-mortem kitten exams are cheap, fast, and easy
   - Working with veterinary pathologist is ideal but not always feasible
2. Hydrostatic test is simple diagnostic tool to consider
   - Can evaluate stillbirth, atelectasis
3. Cat breeders are interested in results
   - Opportunity to collaborate

Acknowledgements

The authors would like to thank: Tina Chittick of **Everafter Devon Rex Cattery** for her collaboration on this project; **Theriogenology Laboratory** for covering shipping costs

References

1. Malandain É, Little S, Casseleux G, et al: Diseases of the neonate. In: Malandain É, editor. Practical guide to cat breeding. Aimargues(France): Aniwa SAS on behalf of Royal Canin; 2006 p. 164-179.
2. Cave TA, Thompson H, Reid SWJ, et al: Kitten mortality in the United Kingdom: a retrospective analysis of 274 histopathological examinations (1986 to 2000). Vet Rec Case Rep 2002;151: 497-501.
3. Fournier A, Masson M, Corbière F, et al: Epidemiological analysis of reproductive performances and kitten mortality rates in 5,303 purebred queens of 45 different breeds and 28,065 kittens in France. Reprod Domest Anim 2017;52 (Suppl. 2): 153-157.
4. Little S: Congenital defects of kittens. In: Tufts' canine and feline breeding and genetics conference; 2005.
5. Silvestre-Ferreira AC, Pastor J: Feline neonatal isoerythrolysis and the importance of feline blood types. Vet Med Int 2010;2010:1-8.
6. Löhr CV: Postmortem examination of the puppy and kitten. In: Peterson ME, Kutzler MA, editors. Small animal pediatrics. St. Louis(Missouri): Elsevier Saunders; 2011 p.276-287.

Appendix E: Winn Feline Foundation grant proposal

RESUBMISSION COMMENTS

Dear Grant Review Committee:

In response to the comments we received during the previous submission cycle, we have made the following adjustments:

Adequacy of preliminary data: We have added another cattery to the study so that our preliminary data comes from two separate catteries, which represent different breeds that are located in Oregon and Iowa.

Adequacy of experimental design:

1. Since dependence on breeders sending in animals is an unavoidable limitation of voluntary participation, we will emphasize to breeders the importance of their full participation in sending in all newborn kittens that do not survive.
2. Upon grant receipt, we will solicit participation from breeders across the country using cat registries (e.g. Cat Fanciers' Association Cat Breed Referral Search).
3. Case selection will be focused on purebred catteries so that causes of kitten mortality can be compared between breeds.
4. Additional diagnostic testing will be conducted following the completion of histopathology.

Appropriateness of budget: In order to account for the costs of additional diagnostic testing following histopathology, the budget has increased by $2000.

Thank you very much for the constructive feedback and opportunity to resubmit this proposal,

Sincerely,

Michelle Kutzler, DVM, PhD, DACT

II. SCIENTIFIC SUMMARY

Even in well-managed cat colonies, up to 16% of kittens may be stillborn, and up to 20% of kittens may die before weaning. Despite the high mortality rate of kittens, the number of primary research articles investigating causes of kitten mortality is limited. Our preliminary findings revealed that 25% (6/24) of Devon Rex kittens died from infectious causes compared to only 7% (1/14) of Persian kittens. Therefore, it is hypothesized that more infectious perinatal and neonatal losses will occur in certain breeds compared to others. Post-mortem examinations will be conducted on kittens (n=100) originating from purebred catteries vaccinating against common feline infectious diseases (including feline leukemia). Because this project is focused on comparing the differing infectious and noninfectious etiologies of neonatal losses between cat breeds, only purebred kittens will be included. Both fresh and formalin-fixed tissue samples will be collected and submitted to the Oregon State University Veterinary Diagnostic Laboratory for bacteriology and histopathology. The occurrence of different infectious diseases and the organs affected will be categorized by etiology (e.g. viral, bacterial). In addition, noninfectious causes of death (e.g. congenital, immune-mediated) will also be categorized by etiology. Results will be analyzed using chi-squared statistic and odds ratio. If necessary and appropriate, a Fisher's exact test will be used. Significance will be defined as $p<0.05$. The results from this research will allow kitten caretakers to take the appropriate steps to prevent future kitten deaths.

III. LAY-LANGUAGE ABSTRACT

Newborn kitten death can be a significant problem even in optimal settings, with up to 20% of kittens dying before weaning. Even though a significant number of kittens do not survive to adulthood, there have been very few studies on the causes of kitten mortality. Furthermore, the focus of these studies has been on older (not newborn) kittens. Therefore, the purpose of the proposed research is to determine the causes of mortality of newborn kittens (including stillbirths). The results will allow kitten caretakers to take the appropriate steps to prevent future kitten deaths. To accomplish this purpose, one hundred purebred kittens that died in the first two weeks of life will be studied. The pedigree, treatments administered (if any), and age at death will be recorded for each kitten. In addition, each kitten will have a thorough post-mortem examination with collection of several organs for further testing (e.g. histopathology, bacterial aerobic culture). Our preliminary findings revealed that 25% (6/24) of Devon Rex kittens died from infectious causes compared to only 7% (1/14) of Persian kittens. Based upon these findings, we hypothesize that among other causes, infectious disease is a significant cause of newborn kitten death and may vary in incidence between breeds. The overall goal is to reduce perinatal and neonatal kitten mortality by identifying the most common causes of mortality and then making recommendations to breeders to prevent future losses.

IV. CONTINUATION STUDIES: Not applicable

V. STUDY PROPOSAL

1. Background:

Even in well-managed cat colonies, up to 16% of kittens may be stillborn and up to 20% of kittens may die before weaning (Malandain et al, 2006). Despite the high mortality rate of kittens, there has been relatively little research in this area. Reported common causes of perinatal (<one day) and neonatal (one to 14 days) kittens include infectious disease, idiopathic causes, congenital defects, and immune-mediated conditions (e.g. neonatal isoerythrolysis) (Cave et al, 2002). Certain diseases occur at a higher prevalence in different cat breeds. Valapalahti and coworkers (2016) studied several breeds of cats (mean age 5.4 years) and found that Persians and Exotic breeds have a higher prevalence of respiratory tract disease (15%) compared to all purebred and mixed breed cats (8%). This was supported by research of Holst and Frössling (2009) who reported a higher prevalence of upper respiratory signs in Persian and Exotic Shorthair cats (36%) compared to other breeds (18.6%). In addition, Giger and Casal (1997) reported that 41% of Devon Rex cats have type B blood, which may increase the risk of developing neonatal isoerythrolysis. However, none of these studies focused on causes of neonatal mortality.

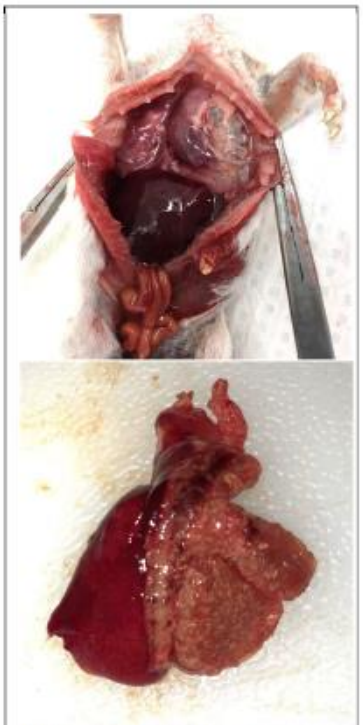


Figure 1. Pyothorax (top) with interstitial pneumonia (bottom) was present in 25% (6/24) of the Devon Rex kittens.

In our preliminary investigation, post-mortem examinations were performed on 24 Devon Rex kittens and 14 Persian kittens that were stillborn or died within the first two weeks of life. No gross pathological abnormalities were found in 25% (6/24) of the Devon Rex kittens and 29% (4/14) of the Persian kittens. Pyothorax with interstitial pneumonia was present in 25% (6/24) of the Devon Rex kittens (**Figure 1**) and multifocal pulmonary hemorrhages were present in 29% (4/14) of the Persian kittens. Congestion of superficial cortical renal vessels and petechial hemorrhages (gross pathologic evidence of feline herpes virus) were also identified and were each present in 7% (1/14) of Persian kittens. Twenty-one percent of the kittens (4/24 Devon Rex and 4/14 Persian) were stillborn based on atelectasis). Red discolored urine (**Figure 2**) and icterus (gross pathological evidence consistent with feline neonatal isoerythrolysis) were present in 13% (3/24) and 4% (1/24) of the Devon Rex kittens, respectively. Congenital defects were only observed in one (1/24, 4%) Devon Rex kitten (cleft palate, **Figure 3**).

Figure 2. Red discolored urine aspirated from urachus (an indication of feline neonatal isoerythrolysis) was present in 13% (3/24) Devon Rex kittens.

Based upon the increased prevalence of infectious causes of death in Devon Rex kittens in our preliminary data, it is hypothesized that more infectious perinatal and neonatal losses will occur in certain breeds compared to others. Therefore, the specific aim of the proposed research is to determine the specific causes of neonatal kitten mortality in purebred catteries. The overall goal is to reduce perinatal and neonatal kitten mortality by identifying the most common causes of mortality and then making recommendations to breeders to prevent future losses.

2. **Experimental design:** Post-mortem examinations will be conducted on kittens (n=100) originating from purebred catteries. This project is focused on purebred kittens so that causes of kitten mortality may be compared between different breeds of cats. We are confident based upon the 38 kittens we have received from two breeders and our relationship with other local cat breeders that we will be able to obtain the sample size needed to yield definitive results. The following information will be requested from each cat breeder to aid in the determination of each kitten's cause of death:

Figure 3. Cleft palate was present in one (1/24, 4%) Devon Rex kitten (normal on left; cleft palate on right).

- Identification (name) of the queen and tom of each kitten will be recorded for pedigree analysis;
- The dam's and sire's blood types (if known) will be recorded to identify kittens at risk of neonatal isoerythrolysis;
- Age at death (in hours or days)
- Complications related to birth
- Birthweight and daily postnatal weights
- Medications or treatments administered
- Clinical signs present before death

The post-mortem examination will consist of a complete external and internal examination of the organs as described by Löhr 2011. Both fresh (unfixed) and formalin-fixed tissue samples will be collected from every deceased kitten. Fresh samples from the placenta (if available), lung, liver, and stomach contents will be collected in individual sealable containers and submitted for aerobic bacterial culture at the Oregon State University Veterinary Diagnostic Laboratory (OSUVDL). In addition, placenta, lung, liver, kidney, heart, brain, thymus, spleen, thyroid gland, adrenal gland, and small intestine will be formalin-fixed for

histopathology. Paraffin-embedding, sectioning, and histological staining with hematoxylin and eosin will be performed at the OSUVDL. Interpretation and digital image capturing will be performed using bright field microscopy at 50X, 200X, and 400X magnifications using a DMI6000B fully motorized microscope (Leica Microsystems, Germany). Based upon histopathology results, additional virology testing will be performed to achieve a definitive diagnosis.

3. **Data Analysis:** Mossi-Dieth and colleagues (1990) investigated kittens <2 weeks of age and reported that 26% (18/69) died as a result of bacterial infection; whereas Cave and coworkers (2002) reported that 47% (10/21) of kittens <2 weeks of age died as a result of bacterial infections. Using the average number of kittens dying from bacterial infections from these two previous studies (36.5%) and the average number of kittens dying from all other causes (63.5%), the sample size was determined to be 106 kittens using an alpha = 0.05 and power = 80% (http://clincalc.com/stats/samplesize.aspx). The occurrence of different infectious diseases and the organs affected will be categorized by etiology (e.g. viral, bacterial). In addition, noninfectious causes of death (e.g. congenital, immune-mediate) will also be categorized by etiology. Results will be analyzed using chi-squared statistic and odds ratio. If necessary and appropriate, a Fisher's exact test will be used. Significance will be defined as $p<0.05$.

4. **Expected Outcomes:** It is anticipated that the present study will identify a variety of causes of kitten mortality. Since there have been few recent reports on causes of kitten mortality, it is expected that this project will identify causes of mortality that have not been discussed extensively in primary literature. The initial findings of this project include seven kittens (out of 38) that presented with an infectious cause of death (pyothorax or herpes virus). Therefore, it is expected that a high percentage of the mortalities investigated in this project will be caused by infectious disease. The results from this research will allow kitten caretakers to take the appropriate steps to prevent future kitten deaths from infectious diseases.

5. **Potential limitations and alternative approaches:** One concern is that the requested sample size will not be met. If it appears that insufficient numbers are available, online advertising will be utilized to recruit cat breeders. Another concern is that the kittens received thus far have originated from one Devon Rex cattery and one Persian/Himalayan cattery. In order to obtain a more diverse sample population and avoid any genetic or environmental bias, cat breeders will be recruited through cat breed registries (e.g. Cat Fanciers' Association, The International Cat Association). Decomposition related to time between death and receipt is another potential limitation. Participating cat breeders will be instructed to refrigerate deceased kittens and then ship to our laboratory overnight to minimize the risk of decomposition.

VI. TIMELINE. Samples collection will begin as soon as funding is awarded and will continue until March 2020. Cadavers will be examined immediately upon arrival and then samples will be processed by the Oregon State University Veterinary Diagnostic for bacteriology and histopathology. Histopathology slides will be reviewed within two weeks after being prepared. Results from this project will be analyzed by the end of April 2020 and then summarized in a manuscript submitted to the *Journal of Feline Medicine and Surgery* by May 2020. The project will be completed within one year of award receipt.

Date	June 2019 – December 2019	June 2019 – March 2020	March 2020 – April 2020	April 2020 – May 2020	May 2020
Event	Recruit participation from cat breeders	Conduct necropsies; collect tissue samples; send samples for bacteriology and histopathology	Complete data analysis and statistical analysis	Submit manuscript to *Journal of Feline Medicine and Surgery*	Complete project

VII. ITEMIZED BUDGET WITH JUSTIFICATION

	Description	Cost
Salaries		
Dr. Michelle Kutzler (PI)	<0.05 FTE will be spent on this project	
Holly Omoto (Undergraduate Honors student researcher)	$11/hour for 200 hours ($2200) plus benefits @ 8% ($176)	$2,376
Services		
Oregon State University Veterinary Diagnostic Laboratory	Research rate for aerobic bacterial culture and viral detection methods and histologic preparation ($155/kitten * 100 kittens)	$15,500
Federal Express	Shipping charges ($35/kitten * 100 kittens)	$3,500
SUBTOTAL		**$21,376**
Overhead	0% allowable	$0
GRANT TOTAL		**$21,376**

Personnel:

Principle investigator, Dr. Michelle Kutzler: Project coordination and design; conduct necropsies; collect tissue samples; send samples for bacteriology and histopathology; data analysis; final report and manuscript preparation. No salary requested.

Undergraduate Honors student researcher, Holly Omoto: Recruit participation and coordinate sample shipment from cat breeders; organize histopathology and bacteriology results; assist with data analysis and manuscript preparation. Salary requested: $11/hour * 200 hours ($2,200) plus benefits @ 8% ($176).

Supplies and services:

Oregon State University Veterinary Diagnostic Laboratory: Research rate for aerobic bacterial culture and viral detection methods and histologic preparation. $155 per kitten for 100 kittens

Federal Express Shipping Charges: $35 per kitten for 100 kittens

VIII. ANIMAL INVOLVEMENT JUSTIFICATION. No live animals will be used for the present study. The research subjects will be deceased kittens that have been provided by cat breeders in order to determine the cause of death.

IX. REFERENCES

Cave TA, Thompson H, Reid SWJ, Hodgson DR, Addie DD. Kitten mortality in the United Kingdom: a retrospective analysis of 274 histopathological examinations (1986 to 2000). *Vet Rec* 2002;151:497-501.

Giger U, Casal ML. Feline colostrum – friend or foe: maternal antibodies in queens and kittens. *J Reprod Fertil Suppl* 1997;51:313-316.

Holst BS, Frössling J. The Swedish breeding cat: population description, infectious diseases and reproductive performance evaluated by a questionnaire. *J Feline Med Surg* 2009;11:793-802.

Löhr CV. Postmortem Examination of the Puppy and Kitten. In: *Small Animal Pediatrics: The First Twelve Months of Life* (Peterson ME and Kutzler MA, eds). Elsevier: St. Louis, Missouri. 2011; pp 276-287.

Malandain E, Little S, Casseleu G, Shelton L, Pibot P, Paragon B. Diseases of the Neonate. In: *Practical Guide to Cat Breeding* (Malandain E, ed). Aniwa SAS on behalf of Royal Canin: Aimargues, France. 2006; pp 164-179.

Mossi-Dieth V, Hauser B, Corboz L, Lutz H, Pospischil A. Todes- Und Erkrankungsursachen Bei Katzenwelpen [Causes of death and disease in kittens]. *Schweizer Archiv für Tierheilkunde* 1990;132(10):587-594.

Vapalahti K, Virtala AM, Joensuu TA, Tiira K, Tähtinen J, Lohi H. Health and behavioral survey of over 8000 Finnish cats. *Front Vet Sci* 2016;3:70.