Comparative pathology of Neotropical deer: morphological and immunohistochemical evaluation

Dissertação apresentada ao Programa de Pós-Graduação em Patologia Experimental e Comparada da Faculdade de Medicina Veterinária e Zootecnia da Universidade de São Paulo para obtenção do título de Mestre em Ciências

Departamento:

Patologia

Área de concentração:

Patologia Experimental e Comparada

Orientador:

Prof. Dr. José Luiz Catão-Dias

De acordo: _____

Orientador

São Paulo 2017

PEDRO ENRIQUE NAVAS-SUÁREZ

Comparative pathology of Neotropical deer: morphological and
immunohistochemical evaluation

São Paulo

2016

PEDRO ENRIQUE NAVAS-SUÁREZ

Comparative pathology of Neotropical deer: morphological and immunohistochemical evaluation

Dissertação apresentada ao Programa de Pós-Graduação em Patologia Experimental e Comparada da Faculdade de Medicina Veterinária e Zootecnia da Universidade de São Paulo para obtenção do título de Mestre em Ciências

Departamento:

Patologia

Área de concentração:

Patologia Experimental e Comparada

Orientador:

Prof. Dr. José Luiz Catão-Dias

De acordo: _____

Orientador

São Paulo 2017

Obs: A versão original se encontra disponível na Biblioteca da FMVZ/USP

Autorizo a reprodução parcial ou total desta obra, para fins acadêmicos, desde que citada a fonte.

DADOS INTERNACIONAIS DE CATALOGAÇÃO NA PUBLICAÇÃO

(Biblioteca Virginie Buff D'Ápice da Faculdade de Medicina Veterinária e Zootecnia da Universidade de São Paulo)

T. 3425
FMVZ

Navas-Suárez, Pedro Enrique
Comparative pathology of Neotropical deer: morphological and immunohistochemical evaluation / Pedro Enrique Navas-Suárez. -- 2016.
179 f. : il.

Título traduzido: Patologia comparada de cervídeos Neotropicais: avaliação morfológica e imunohistoquímica.

Dissertação (Mestrado) - Universidade de São Paulo. Faculdade de Medicina Veterinária e Zootecnia. Departamento de Patologia, São Paulo, 2017.

Programa de Pós-Graduação: Patologia Experimental e Comparada.

Área de concentração: Patologia Experimental e Comparada.

Orientador: Prof. Dr. José Luiz Catão Dias.

1. Cause of death. 2. Deer. 3. Diseases. 4. Veterinary pathology. 5. Wild animals.
I. Título.

Parecer da Comissão de Ética

UNIVERSIDADE DE SÃO PAULO

FACULDADE DE MEDICINA VETERINÁRIA E ZOOTECNIA

Comissão de Ética no Uso de Animais

São Paulo, 09 de fevereiro de 2015
CEUA N 4271090215

COMPROVANTE DE SUBMISSÃO DE PROPOSTA À COMISSÃO DE ÉTICA NO USO DE ANIMAIS

CNP: 029.597.888-00

Título do projeto: *Patologia comparada de cervídeos neotropicais no Brasil*

Responsável: *José Luiz Catão Dias*

Telefone: 113091134

e-mail: *zecatao@usp.br*

A Comissão de Ética no Uso de Animais da Faculdade de Medicina Veterinária e Zootecnia da Universidade de São Paulo, terá o prazo máximo de 30 dias para emissão do parecer. Todo o processo poderá ser acompanhado no sistema (<http://orion.fmvz.usp.br/>) por meio da sua senha de acesso.

Profa. Dra. Denise Tabacchi Fantoni
Presidente da Comissão de Ética no Uso de Animais
Faculdade de Medicina Veterinária e Zootecnia da Universidade de
São Paulo

Roseli da Costa Gomes
Secretaria Executiva da Comissão de Ética no Uso de Animais
Faculdade de Medicina Veterinária e Zootecnia da Universidade de
São Paulo

SISBIO

Ministério do Meio Ambiente - MMA
Instituto Chico Mendes de Conservação da Biodiversidade - ICMBio
Sistema de Autorização e Informação em Biodiversidade - SISBIO

Autorização para atividades com finalidade científica

Número: 47858-1	Data da Emissão: 13/04/2015 16:16	Data para Revalidação*: 12/05/2016
* De acordo com o art. 28 da IN 03/2014, esta autorização tem prazo de validade equivalente ao previsto no cronograma de atividades do projeto, mas deverá ser revalidada anualmente mediante a apresentação do relatório de atividades a ser enviado por meio do Sisbio no prazo de até 30 dias a contar da data do aniversário de sua emissão.		

Dados do titular

Nome: José Luiz Catão Dias	CPF: 029.597.888-00
Título do Projeto: Patologia comparada de cervídeos neotropicais no Brasil	
Nome da Instituição : Faculdade de Medicina Veterinária e Zootecnia USP	CNPJ: 63.025.530/0019-33

Cronograma de atividades

#	Descrição da atividade	Início (mês/ano)	Fim (mês/ano)
1	projeto de mestrado	02/2015	02/2017

Observações e ressalvas

1	As atividades de campo exercidas por pessoa natural ou jurídica estrangeira, em todo o território nacional, que impliquem o deslocamento de recursos humanos e materiais, tendo por objeto coletar dados, materiais, espécimes biológicos e minerais, peças integrantes da cultura nativa e cultura popular, presente e passada, obtidos por meio de recursos e técnicas que se destinem ao estudo, à difusão ou à pesquisa, estão sujeitas a autorização do Ministério de Ciência e Tecnologia.
2	Esta autorização NÃO exime o pesquisador titular e os membros de sua equipe da necessidade de obter as anuências previstas em outros instrumentos legais, bem como do consentimento do responsável pela área, pública ou privada, onde será realizada a atividade, inclusive do órgão gestor de terra indígena (FUNAI), da unidade de conservação estadual, distrital ou municipal, ou do proprietário, arrendatário, posseiro ou morador de área dentro dos limites de unidade de conservação federal cujo processo de regularização fundiária encontra-se em curso.
3	Este documento somente poderá ser utilizado para os fins previstos na Instrução Normativa ICMBio nº 03/2014 ou na Instrução Normativa ICMBio nº 10/2010, no que especifica esta Autorização, não podendo ser utilizado para fins comerciais, industriais ou esportivos. O material biológico coletado deverá ser utilizado para atividades científicas ou didáticas no âmbito do ensino superior.
4	A autorização para envio ao exterior de material biológico não consignado deverá ser requerida por meio do endereço eletrônico www.ibama.gov.br (Serviços on-line - Licença para importação ou exportação de flora e fauna - CITES e não CITES).
5	O titular de licença ou autorização e os membros da sua equipe deverão optar por métodos de coleta e instrumentos de captura direcionados, sempre que possível, ao grupo taxonômico de interesse, evitando a morte ou dano significativo a outros grupos; e empregar esforço de coleta ou captura que não comprometa a viabilidade de populações do grupo taxonômico de interesse em condição in situ.
6	O titular de autorização ou de licença permanente, assim como os membros de sua equipe, quando da violação da legislação vigente, ou quando da inadequação, omissão ou falsa descrição de informações relevantes que subsidiaram a expedição do ato, poderá, mediante decisão motivada, ter a autorização ou licença suspensa ou revogada pelo ICMBio e o material biológico coletado apreendido nos termos da legislação brasileira em vigor.
7	Este documento não dispensa o cumprimento da legislação que dispõe sobre acesso a componente do patrimônio genético existente no território nacional, na plataforma continental e na zona econômica exclusiva, ou ao conhecimento tradicional associado ao patrimônio genético, para fins de pesquisa científica, bioprospecção e desenvolvimento tecnológico. Veja maiores informações em www.mma.gov.br/cgen .
8	Em caso de pesquisa em UNIDADE DE CONSERVAÇÃO, o pesquisador titular desta autorização deverá contactar a administração da unidade a fim de CONFIRMAR AS DATAS das expedições, as condições para realização das coletas e de uso da infra-estrutura da unidade.

Locais onde as atividades de campo serão executadas

#	Município	UF	Descrição do local	Tipo
1	SAO PAULO	SP	Faculdade de medicina veterinária e zootecnia - USP	Fora de UC Federal

Atividades X Táxons

#	Atividade	Táxons
1	Coleta/transporte de amostras biológicas ex situ	Blastocerus dichotomus, Mazama gouazoubira

Material e métodos

1	Amostras biológicas (Outros mamíferos)	Fragmento de tecido/órgão
---	--	---------------------------

Destino do material biológico coletado

Este documento (Autorização para atividades com finalidade científica) foi expedido com base na Instrução Normativa nº 03/2014. Através do código de autenticação abaixo, qualquer cidadão poderá verificar a autenticidade ou regularidade deste documento, por meio da página do Sisbio/ICMBio na Internet (www.icmbio.gov.br/sisbio).

Código de autenticação: 13812153

FOLHA DE AVALIAÇÃO

Autor: NAVAS-SUÁREZ, Pedro Enrique

Título: **Comparative pathology of Neotropical deer:** morphological and immunohistochemical evaluation

Dissertação apresentada ao Programa de Pós-Graduação Patologia Experimental e Comparada da Faculdade de Medicina Veterinária e Zootecnia da Universidade de São Paulo para obtenção do título de Mestre em Ciências

Data: ____/____/____

Banca Examinadora

Prof. Dr. _____

Instituição: _____ Julgamento: _____

Prof. Dr. _____

Instituição: _____ Julgamento: _____

Prof. Dr. _____

Instituição: _____ Julgamento: _____

AGRADECIMENTOS

In this section I took the liberty of addressing people in their native language.

Este párrafo de agradecimientos no tiene un orden preestablecido, cada una de las personas mencionadas ha cumplido un papel muy importante en mi formación profesional y personal.

Este es un agradecimiento que particularmente me hace sentir muy orgulloso y honrado. Eres la persona más especial para mí; has sido mi apoyo fundamental durante estos largos dos años. Así como yo, has reído, sufrido, llorado, peleado y compartido muchas experiencias. Fue una etapa que tuvimos la fortuna de vivir y me siento orgulloso de afirmar que logramos superar aquel “amor a distancia”. Sé que aún nos quedan muchas historias por vivir, aún tendremos que pasar por dificultades, alegrías, compromisos y mil cosas más que personalmente me siento ansioso de vivir si estás junto a mí. No tengo palabras para agradecer todos los sacrificios que has hecho para darme la posibilidad de crecer como profesional. Ahora comenzará nuestro crecimiento familiar. Gracias Amor de mi vida. Y futura esposa te amo Catalina Ospina.

A mi mamá Alicia Suárez Contreras, por haberme dado las herramientas que me permitieron inicialmente realizar mi práctica profesional y ahora esta pos graduación. Hoy más que nunca agradezco todo el tiempo y empeño que dedicó a mi formación en los primeros años de vida, todos aquellos libros que iban firmados con la frase “para mi pequeño veterinario”. Mamá muchas gracias, hoy en día soy quien soy por su dedicación. A mi padre Pedro Enrique Navas-García, pese a no haberlo podido conocer, todos los buenos comentarios tanto de su nivel profesional como su trato a las personas me han hecho esforzarme diariamente para ser el hijo que siempre quiso, gracias por todo padre. A mi grandioso hermano Sergio que ha sido un pilar fundamental en mi vida, desde pequeños jugando a lucha libre, fútbol, monopolio y aquellas extensas jordanas de risk, AOE y FIFA. Siempre he admirado su inteligencia y capacidad de resolver los problemas en tiempos ultra reducidos, gracias por ser mi apoyo en tiempos en los que he necesitado de un hermano. Tenga seguridad que si me dieran la posibilidad de escoger un hermano nunca dudaría en que es usted.

A mis tías y tíos Elda, Olga, Flor Oliva, Omar, Celemín (QEPD), Carlos, Hernando, Miguel y Germán (QEPD). De todos ustedes he aprendido innumerables lecciones. Siéntanse seguros que el Pedro de hoy tiene muchas facetas de ustedes. Tía Elda para usted un agradecimiento particular porque desde que tengo uso de razón siempre has sido mi apoyo espiritual y familiar, gracias por la confianza que has depositado en mí. Esa confianza me ha hecho ser fuerte cuando la situación lo amerita. Tía Olga, usted es como mi segunda mama, me ha consentido, dedicado tiempo y siempre está disponible cuando la necesito. Muchas gracias. Tío Omar, sin lugar a duda, aparte de tío mi padre, gracias por todos los consejos y conversaciones. Usted para mí es un ejemplo a seguir. A mis primos y primas, en especial Caro, Alfred, Edison y Edwin, cada uno de ustedes vivieron etapas diferentes de mi vida y de cada uno de ustedes tengo los mejores recuerdos y aprendizajes. Gracias por el apoyo en momentos en que lo necesité. A mis colegas de universidad por todos los momentos que compartimos, en especial, Ani, Piw, July, Nata, Naki, Vargas, Jessica, Fabra, Blain, checho, Diego, Juanfe, Germán, Sebastián y Martín. De la universidad, un agradecimiento particular al doctor Diego Soler-Tovar, mi mentor, quien me motivo a investigar, cuestionarme y siempre

buscar herramientas para responder mis preguntas y nunca darme por vencido. Doctor Soler, tenga la seguridad que mi vocación investigativa se la debo enormemente a usted, para mí usted es un ejemplo integral de persona, gracias por ser mi mentor, profesor, evaluador y ahora amigo. A la doctora Victoria Pereira, por permitirme iniciar en este mundo de los animales silvestres, gracias por la confianza depositada en mí. A las instituciones Parque Jaime Duque, Zoológico Santacruz, CRRFSOC y Fundación Bioandina Colombia, por permitirme desarrollar como profesional en el área de fauna silvestre. A mis colegas y amigos de São Paulo, Jilma, Sandy, Noelia, Angélica, Pablo, Gilbert, Carlos, Jairo, Nicolás, Danny, Camilo, John y Julián. Compartir con ustedes profesional y personalmente ha sido un placer, han hecho que en mi estancia en Brasil pueda decir que no solo tengo excelentes colegas, sino también grandiosos amigos.

Ao Fundação de Amparo à Pesquisa do Estado de São Paulo por ter me privilegiado com a bolsa N° 2015/04231-2 graças a vocês consegui fazer esta pesquisa e levar parte dos resultados a um dos melhores congressos na área de patologia.

Ao professor José Luiz Catão Dias, agradeço pela oportunidade de fazer estágio em 2012, e por mais tarde, aceitar me orientar no mestrado. O aprendizado foi imensurável. Você é um dos melhores profissionais que eu já tive a honra de conhecer. Além disso, é um grande ser humano, com altíssimo nível intelectual, bom líder, e possuir muitas outras qualidades, que se eu fosse citar tornaria essa dissertação interminável! Prezado professor, conhecê-lo me proporcionou uma nova visão de vida, que uma vez aplicada, me fez sentir um ser humano muito mais completo. Obrigado pela paciência, conhecimentos, bate papos e, obviamente, pelas tardes de café.

Professora Matu, você sempre foi muito boa comigo, desde o meu estágio. Sou muito grato por tê-la conhecido e por poder compartilhar com você conversas científicas, políticas e de temas gerais. Muito obrigado por aquele casaco e por toda as ajudas no desenvolvimento do meu mestrado.

Professores Bruno, Lilian, Paulo, Venâncio, Fernando, Grissi, Claudia, Ênio e Fred: obrigado pelos ensinamentos nas diversas disciplinas que cursei. Aprendi muito com cada um de vocês. Ao Cláudio, Luciano e Sândara: obrigado por me acolherem no laboratório de Histologia e me ensinar todas as técnicas de processamento. Graças a vocês aprendi muitas coisas importantes da rotina.

Aos meus colegas do LAPCOM: Carol, Samira, Valeria, Ju, Sândara, Silmara, Roberta, Gi, Cami, Angélica, Claudia, Marina, Kátia, Cátia, Laura, Cintia, Carlos, Gilbert, Marcelo, Marco, Ralph e Josué; e aos estagiários: Isabella, Mayara, Arícia, Giorgia, Giulia, Natália, Bruno, Edu, Renan e Julián: obrigado por todo o aprendizado. Vocês fazem com que nosso LAPCOM seja o que é. Muito obrigado ao Jorge, tão importante no universo lapconiano! Eu pessoalmente agradeço por todas as experiências que compartilhei com você: bate papos sobre política, cultura, nerd, ecologia, economia, evolução, etc. Você é uma excelente pessoa e um grande ser humano. Obrigado.

Ao pessoal do VPT, especialmente à Mariana, Jilma, Lu, Elena, Milena, Claudia, Adriana, Marcia, Cesar, Vagner, Edson, Rai e todos os demais, por me fazerem sentir parte da família VPT.

Especialmente quiero agradecer a Joel Stangle y Carol Ewbank por todo su tiempo en ayudarme con la gramática de esta disertación. Sin su incalculable ayuda este trabajo no tendría forma. MIL GRACIAS.

RESUMO

NAVAS-SUÁREZ, P. E. **Patologia comparada de cervídeos Neotropicais**: avaliação morfológica e imunohistoquímica. [Comparative pathology of Neotropical deer: morphological and immunohistochemical evaluation]. 2016. 179 f. Dissertação (Mestrado em Ciências) – Faculdade de Medicina Veterinária e Zootecnia, Universidade de São Paulo, São Paulo, 2017.

Os cervídeos neotropicais desempenham funções de importância para a sustentabilidade e sanidade dos ecossistemas que habitam, e a investigação dos processos patológicos, assim como dos agentes causais envolvidos, é uma parte crítica dos trabalhos de conservação envolvendo estes animais. Além disso, os animais selvagens em geral, e em especial os cervídeos, têm um papel importante na transmissão e disseminação de agentes infecciosos, alguns deles relevantes à saúde humana, para os animais de companhia e de produção. O presente estudo descreveu as características macroscópicas e histológicas dos processos patológicos de duas espécies de cervídeos brasileiros: cervo do pantanal (*Blastocerus dichotomus*) e veado catingueiro (*Mazama gouazoubira*) no estado de São Paulo por um período de 21 anos (1995-2015). As principais causas de morte identificadas em cervo do pantanal foram classificadas como: respiratória 53,3% (40/75), nutricional (4,0%; 3/75), trauma (4,0%; 3/75) e eutanásia (4,0%; 3/75). Já em veado catingueiro foram identificadas as seguintes causas: respiratória (25,2%; 33/131), eutanásia (12,2%; 16/131) e trauma (9,2%; 12/131). O presente trabalho detalha a importância do sistema respiratório nestas duas espécies de cervídeos brasileiros. É importante destacar que apesar de não ter identificado agentes etiológicos, os achados histopatológicos são altamente sugestivos de etiologias bacteriana, viral e fúngica. O presente trabalho fornece informações sobre a ocorrência de diversos processos patológicos nestas duas espécies de cervídeos, e sua correlação com o grupo etário, gênero e condição corpórea dos espécimes analisados.

Palavras-chave: Animais selvagens. Causas de morte. Cervos. Doença. Patologia veterinária.

ABSTRACT

NAVAS-SUÁREZ, P. E. **Comparative pathology of Neotropical deer:** morphological and immunohistochemical evaluation. [Patologia comparada de cervídeos Neotropicais: avaliação morfológica e imunohistoquímica]. 2016. 179 f. Dissertação (Mestrado em Ciências) – Faculdade de Medicina Veterinária e Zootecnia, Universidade de São Paulo, São Paulo, 2017.

Neotropical deer have important functions for the sustainability and health of their ecosystem. The investigation of pathological processes and the respective etiologic agents is a critical part of the conservation programs involving these species. In addition, wild animals and in particular deer, play an important role in the transmission and spread of infectious agents, some of them relevant to human health, companion animals and livestock health. The present study describes gross and histological lesions of brown brocket deer BBD (*Mazama gouazoubira*) and marsh deer MD (*Blastocerus dichotomus*) from São Paulo state (Brazil), collected during a 21-year period (1995-2015). Major causes of death in MD were respiratory (53.3% 40/75), alimentary (4.0%; 3/75), nutritional (4.0%; 3/75), trauma (4.0%; 3/75) and euthanasia (4.0%; 3/75). In BBD, the main causes of death were respiratory (25.2% 33/131), euthanasia (12.2%; 16/131) and trauma (9.2%; 12/131). This study shows the importance of respiratory disturbances in these two species of South American deer. In this work, despite no etiologic agents were identified, histopathological findings suggest the presence of infectious etiologies including bacterial, viral and fungal. We have described characterized and evaluated pathological processes in function with biological and epidemiological aspects.

Keywords: Cause of death. Deer. Diseases. Veterinary pathology. Wild animals.

LISTA DE FIGURAS

Figure 1 -	Artiodactyl evolution	25
Figure 2 -	Left), Male brown brocket deer; Right), Male marsh deer	27
Figure 3 -	Distribution and density of marsh deer (right) and brown brocket deer (left) populations.....	28
Figure 4 -	Annual distribution of Brazilian marsh deer cases between 1995-2015 .	50
Figure 5 -	Monthly distribution of Brazilian marsh deer cases between 1995-2015	50
Figure 6 -	Seasonality in mortality of Brazilian marsh deer cases between 1995-2015, and its relationship to annual distribution.....	51
Figure 7 -	Distribution according to reported clinical diagnosis of Brazilian marsh deer from 1995 to 2015	52
Figure 8 -	Annual distribution of Brazilian brown brocket deer submissions between 1995-2015	80
Figure 9 -	Monthly distribution of Brazilian brown brocket deer cases between 1995-2015	80
Figure 10 -	Seasonality in mortality of Brazilian brown brocket deer cases between 1995-2015, and its relationship to annual distribution	81
Figure 11 -	Distribution according to the reported clinical diagnosis.	82

LISTA DE TABELAS

Table 1 -	General epidemiological aspects of Brazilian marsh deer from 1995 to 2015	51
Table 2 -	Respiratory system: macroscopic findings, biological and epidemiological aspects in marsh deer	53
Table 3 -	Severity of major pulmonary hemodynamic disorders in marsh deer	54
Table 4 -	Lungs: histopathological findings, biological and epidemiological aspects in marsh deer	55
Table 5 -	Epidemiological aspects of pneumonia in Brazilian marsh deer from 1995 to 2015	56
Table 6 -	Trachea: histopathological findings, biological and epidemiological aspects in marsh deer	58
Table 7 -	Cardiovascular system: macroscopic findings, biological and epidemiological aspects in marsh deer	59
Table 8 -	Heart: histopathological findings, biological and epidemiological aspects in marsh deer	60
Table 9 -	Alimentary system: macroscopic findings, biological and epidemiological aspects in marsh deer	61
Table 10 -	Tongue: histopathological findings, biological and epidemiological aspects in marsh deer	62
Table 11 -	Rumen: histopathological findings, biological and epidemiological aspects in marsh deer	63
Table 12 -	Reticulum: histopathological findings, biological and epidemiological aspects in marsh deer	63
Table 13 -	Abomasum: histopathological findings, biological and epidemiological aspects in marsh deer	64
Table 14 -	Small intestine: histopathological findings, biological and epidemiological aspects in marsh deer	64
Table 15 -	Large intestine: histopathological findings, biological and epidemiological aspects in marsh deer	65
Table 16 -	Liver: histopathological findings, biological and epidemiological aspects in marsh deer	66
Table 17 -	Severity and morphological patterns of major hepatic hemodynamic disorders diagnosed in marsh deer	67
Table 18 -	Urogenital system: macroscopic findings, biological and epidemiological aspects in marsh deer	68

Table 19 - Kidney: histopathological findings, biological and epidemiological aspects in marsh deer	70
Table 20 - Urinary bladder: histopathological findings, biological and epidemiological aspects in marsh deer	70
Table 21 - Endocrine system: macroscopic findings, biological and epidemiological aspects in marsh deer	71
Table 22 - Adrenal glands: histopathological findings, biological and epidemiological aspects in marsh deer	72
Table 23 - Hematopoietic system: macroscopic findings, biological and epidemiological aspects in marsh deer	73
Table 24 - Spleen: histopathological findings, biological and epidemiological aspects in marsh deer	73
Table 25 - Lymphatic node: histopathological findings, biological and epidemiological aspects in marsh deer	74
Table 26 - Nervous system: macroscopic findings, biological and epidemiological aspects in marsh deer	75
Table 27 - Brain: histopathological findings, biological and epidemiological aspects in marsh deer	76
Table 28 - Integumentary system: macroscopic findings, biological and epidemiological aspects in marsh deer	77
Table 29 - Musculoskeletal system: macroscopic findings, biological and epidemiological aspects in marsh deer	78
Table 30 - Skeletal muscle: histopathological findings, biological and epidemiological aspects in marsh deer	79
Table 31 - Brown brocket deer: general epidemiological aspects	81
Table 32 - Brown brocket deer: general aspects of trauma	82
Table 33 - Respiratory system: macroscopic findings, biological and epidemiological aspects in brown brocket deer.....	83
Table 34 - Severity of major pulmonary hemodynamic disorders in brown brocket deer.	84
Table 35 - Lungs: histopathological findings, biological and epidemiological aspects in brown brocket deer	85
Table 36 - Epidemiological aspects of brown brocket deer with pneumonia	87
Table 37 - Trachea: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	87
Table 38 - Cardiovascular system: macroscopic findings, biological and epidemiological aspects in brown brocket deer	89

Table 39 - Heart: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	89
Table 40 - Alimentary system: macroscopic findings, biological and epidemiological aspects in brown brocket deer.....	90
Table 41 - Tongue: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	91
Table 42 - Esophagus: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	92
Table 43 - Rumen: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	92
Table 44 - Reticulum: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	93
Table 45 - Omasum: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	93
Table 46 - Abomasum: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	94
Table 47 - Small intestine: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	94
Table 48 - Large intestine: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	95
Table 49 - Liver: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	96
Table 50 - Urogenital system: macroscopic findings, biological and epidemiological aspects in brown brocket deer.....	99
Table 51 - Kidney: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	100
Table 52 - Urinary bladder: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	100
Table 53 - Endocrine system: macroscopic findings, biological and epidemiological aspects in brown brocket deer.....	101
Table 54 - Adrenal glands: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	103
Table 55 - Hematopoietic system: macroscopic findings, biological and epidemiological aspects in brown brocket deer.....	104
Table 56 - Spleen: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	106
Table 57 - Lymphatic node: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	106

Table 58 - Nervous system: macroscopic findings, biological and epidemiological aspects in brown brocket deer.....	107
Table 59 - Brain: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	108
Table 61 - Integumentary system: macroscopic findings, biological and epidemiological aspects in brown brocket deer.....	108
Table 60 - Skin: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	109
Table 62 - Musculoskeletal system: macroscopic findings, biological and epidemiological aspects in brown brocket deer.....	110
Table 63 - Skeletal muscle: histopathological findings, biological and epidemiological aspects in brown brocket deer.....	110
Table 64 - Causes of death in Brazilian South American deer from 1995 to 2015	112
Table 65 - Causes of death, biological and epidemiological aspects of marsh deer	113
Table 66 - Causes of death, biological and epidemiological aspects of brown brocket deer	116

LISTA DE FOTOS

Photo 1 -	BBD. Open fracture on the right hind limb	155
Photo 2 -	BBD. Hindlimbs, lacerations and alopecia.....	155
Photo 3 -	BBD. Note lacerations and hemorrhage (white arrowhead) around the eyes and lips	155
Photo 4 -	BBD. Multiple ticks on the skin of the zygomatic region (white arrowhead)	155
Photo 5 -	BBD. Hemorrhage of the semimembranosus and semitendinosus muscles	155
Photo 6 -	BBD. Ruminal bloat	155
Photo 7 -	BBD. Inguinal hernia	157
Photo 8 -	BBD. Hernial sac with hemorrhagic intestinal loops and mesentery.....	157
Photo 9 -	BBD. Comminuted fracture of the metatarsal bones	157
Photo 10 -	BBD. Phytobezoars in rumen	157
Photo 11 -	BBD. Respiratory system. Severe pulmonary edema	157
Photo 12 -	BBD. Respiratory system. Bronchopneumonia	157
Photo 13 -	MD. Trachea. Hemorrhagic tracheitis. H&E. Scale bar= 200µm	159
Photo 14 -	MD. Trachea. Mild moderate mononuclear and hemorrhagic tracheitis. H&E. Scale bar= 100µm.....	159
Photo 15 -	MD. Lung. Pulmonary thrombosis. H&E. 100µm.....	159
Photo 16 -	BBD. Lung. Severe pulmonary hemorrhage, compatible with infarct. H&E. Scale bar= 100µm.....	159
Photo 17 -	BBD. Lung. Mild moderate granulocytic bronchiolitis. H&E. 100µm	159
Photo 18 -	MD. Lung. Lungworm eggs. H&E. 50µm	159
Photo 19 -	MD. Lung. Nematode Larvae compatible with Strongylida Order. H&E. 50µm	161
Photo 20 -	MD. Lung. Early histiocytic infiltration of lung-worm on alveolar lumen. H&E. 50µm.....	161
Photo 21 -	MD. Lung. Parasitic granuloma. H&E. 50µm.....	161
Photo 22 -	MD. Lung. Pulmonary granuloma. H&E. 50µm.	161
Photo 23 -	MD. Lung. Fibrinosuppurative bronchopneumonia. H&E. 50µm	161
Photo 24 -	MD. Lung. Early stage of suppurative bronchopneumonia. H&E. 100µm	161

Photo 25 - MD. Lung. Fibrinosuppurative bronchopneumonia. H&E. 200µm	163
Photo 26 - MD. Lung. Fibrinonecrotic bronchopneumonia. H&E. 100µm	163
Photo 27 - BBD. Lung. Fibrinous bronchopneumonia. H&E. 100µm	163
Photo 28 - MD. Lung. Mild acute interstitial pneumonia. H&E. 50µm	163
Photo 29 - MD, adult, male (BD-22). Lung. Multifocal type II pneumocyte hyperplasia. H&E. 100 µm.....	163
Photo 30 - BBD. Lung. Mild diffuse mononuclear interstitial pneumonia. H&E. 50µm	163
Photo 31 - MD. Lung. Pulmonary mononuclear perivascularitis. H&E. 50µm	165
Photo 32 - MD. Lung. Pulmonary mononuclear perivascularitis. H&E. 50µm	165
Photo 33 - MD. Lung. Mixed pulmonary perivascularitis associated with bronchopneumonia. H&E. 100µm	165
Photo 34 - MD. Heart. Myocardial hemorrhage. H&E. 200µm	165
Photo 35 - BBD. Heart. Fibrofatty infiltration. H&E. 200µm.....	165
Photo 36 - BBD. Heart. Mycotic thrombosis and angioinvasion. PAS. 100µm.....	165
Photo 37 - BBD. Heart. Mycotic myocarditis. PAS. 50µm.....	167
Photo 38 - BBD. Tongue. Severe necrosuppurative glossitis. H&E. 200µm	167
Photo 39 - BBD. Tongue. Severe hemorrhagic ulcerative and necrotizing rumenitis. H&E. 200µm.....	167
Photo 40 - MD. Rumen. Necroulcerative rumenitis. H&E. 100µm.....	167
Photo 41 - MD. Rumen. Mycotic necrotizing rumenitis. H&E. 100µm	167
Photo 42 - MD. Rumen. Mycotic necrotizing rumenitis. H&E. 100µm	167
Photo 43 - MD. Rumen. Mycotic necrotizing rumenitis. H&E. 100µm	169
Photo 44 - BBD. Reticulum. Necrotizing reticulitis. H&E. 200µm.....	169
Photo 45 - MD. Colon. Hemorrhagic necrotizing colitis. H&E. 200µm	169
Photo 46 - MD. Liver. Macrovesicular steatosis. H&E. 50µm.....	169
Photo 47 - MD. Liver. Microvesicular steatosis. H&E. 50µm.....	169
Photo 48 - MD. Liver. Hepatic necrosis with multifocal coalescing hemorrhage. H&E. 200µm	169
Photo 49 - BBD. Liver. Thrombosis and centrilobular necrosis. H&E. 100µm	171
Photo 50 - MD. Liver. Massive hepatocellular necrosis. H&E. 200µm.....	171
Photo 51 - BBD. Liver. Periportal lymphohistiocytic hepatitis. H&E. 50µm	171
Photo 52 - BBD. Liver. Focal Periportal lymphohistiocytic hepatitis. H&E. 50µm...	171

Photo 53 - BBD. Liver. Portal lymphohistiocytic cholangiohepatitis. H&E. 50µm...	171
Photo 54 - BBD. Liver. Lymphohistiocytic hepatitis. H&E. 50µm	171
Photo 55 - BBD. Kidney. Focal mild-moderate lymphocytic-histiocytic perivascularitis. H&E. 50µm.....	173
Photo 56 - BBD. Kidney. Focal mixed interstitial nephritis. H&E. 50µm.....	173
Photo 57 - MD. Kidney. Diffuse mixed tubulointerstitial nephritis and leukocytic tubular casts. H&E. 50µm.....	173
Photo 58 - BBD. Kidney. Focal moderate proliferative glomerulonephritis. H&E. 50µm	173
Photo 59 - BBD. Kidney. Focal mild membranous glomerulonephritis. PAS. 50µm	173
Photo 60 - BBD. Kidney. Necrotic cylinders. H&E. 50µm	173
Photo 61 - BBD. Skeletal muscle. Rhabdomyolysis. H&E. 100µm	175
Photo 62 - BBD. Skeletal muscle. Thrombosis. H&E. 50µm.....	175
Photo 63 - MD. Skeletal muscle. Suppurative myositis. H&E. 50µm	175
Photo 64 - BBD. Skeletal muscle. Nonsuppurative perineuritis. H&E. 100µm	175
Photo 65 - BBD. Skeletal muscle. Focal Sarcocystid cyst. H&E. 50µm	175
Photo 66 - BBD. Adrenal gland. Cortical congestion of adrenal gland. H&E. 200µm	175
Photo 67 - MD. Adrenal gland. Adrenitis. H&E. 50µm.....	177
Photo 68 - MD. Adrenal gland. Focal nonsuppurative adrenitis. H&E. 50µm.....	177
Photo 69 - BBD. Brain. Chronic meningitis. H&E. 200µm.....	177
Photo 70 - BBD. Brain. Focally extensive severe meningeal fibroplasia. Masson. 200µm	177
Photo 71 - BBD. Brain. Focal mixed chronic meningitis. H&E. 50µm	177
Photo 72 - BBD. Brain. Focally extensive severe meningeal scar. IHQ - GFAP. 200µm	177
Photo 73 - BBD. Spleen. Hemosiderosis and extramedullary hematopoiesis. H&E. 50µm	179
Photo 74 - BBD. Skin. Focally expansive moderate-severe pyoulcerative dermatitis. H&E. 100µm.....	179
Photo 75 - BBD. Skin. Focally expansive moderate ulcerative dermatitis. H&E. 200µm	179

LISTA DE ABREVIATURAS

Deer species:

Marsh Deer (<i>Blastocerus dichotomus</i>)	MD
Brown Brocket Deer (<i>Mazama gouazoubira</i>)	BBD
White Tailed Deer (<i>Odocoileus virginianus</i>)	WTD
Fallow Deer (<i>Dama dama</i>)	FD
Red Deer (<i>Cervus elaphus</i>)	RD

Diseases

Malignant Catarrhal Fever	MCF
Bluetongue	BT
Bovine Viral Diarrhea	BVD
Hemorrhagic Epizootic Disease	HED
Infectious Bovine Rhinotracheitis	IBR

Virus

Bluetongue Virus	BTV
Hemorrhagic Epizootic Disease Virus	HEDV
Ovine Herpesvirus type II	OVHV-II
Caprine Herpesvirus type II	CaHV-II
Bovine Viral Diarrhea Virus	BVDV

SUMÁRIO

1	INTRODUCTION	23
2	OBJECTIVES.....	24
2.1	GENERAL OBJECTIVE	24
2.2	SPECIFIC OBJECTIVES.....	24
3	LITERATURE REVIEW.....	25
3.1	NATURAL HISTORY AND BIOLOGY OF SOUTH AMERICAN DEER	25
3.2	PATHOLOGY OF DEER SPECIES BY ORGAN SYSTEM	29
3.2.1	ALIMENTARY SYSTEM.....	29
3.2.1.1	Oral cavity.....	29
3.2.1.2	Salivary glands	29
3.2.1.3	Esophagus.....	30
3.2.1.4	Forestomachs.....	30
3.2.1.5	Abomasum	31
3.2.1.6	Intestines	31
3.2.2	HEPATOBIILIARY AND PANCREAS.....	31
3.2.3	URINARY SYSTEM.....	32
3.2.3.1	Kidney	32
3.2.3.2	Lower urinary tract.....	34
3.2.4	RESPIRATORY SYSTEM	34
3.2.4.1	Nasal cavity and sinuses	34
3.2.4.2	Pharynx	35
3.2.4.3	Larynx.....	35
3.2.4.4	Trachea	36
3.2.4.5	Lungs	36
3.2.4.6	Pleura	37
3.2.5	CARDIOVASCULAR SYSTEM.....	38
3.2.6	HEMATOPOIETIC SYSTEM	38
3.2.7	GENITAL SYSTEM	39
3.2.8	NERVOUS SYSTEM	39

3.2.9	INTEGUMENTARY SYSTEM.....	40
3.2.10	MUSCULOSKELETAL SYSTEM.....	40
3.3	NONINFECTIOUS DISEASES.....	40
3.4	INFECTIOUS DISEASES.....	41
3.4.1	Viral diseases.....	41
3.4.1.1	Infectious Bovine Rhinotracheitis (IBR).....	41
3.4.1.2	Malignant Catarrhal Fever (MCF).....	41
3.4.1.3	Epizootic Hemorrhagic Disease (EHD).....	42
3.4.1.4	Bovine Viral Diarrhea Virus (BVDV).....	43
3.4.1.5	Foot and Mouth Disease (FMD).....	43
3.4.2	Bacterial diseases.....	43
3.4.2.1	Mycobacterial diseases.....	44
3.4.3	Protozoal diseases.....	44
3.4.4	Helminthic diseases.....	44
3.4.5	Fungal diseases.....	45
3.4.6	Prionic diseases.....	45
3.4	DISEASES IN SOUTH AMERICAN DEER.....	45
3.4.1	Noninfectious.....	45
3.4.2	Infectious.....	46
4	MATERIALS AND METHODS.....	47
4.1	Materials.....	47
4.2	Methods.....	47
4.2.1	Histopathology.....	47
4.2.2	Histochemical analysis.....	48
4.2.3	Immunohistochemical analysis.....	48
4.2.4	Epidemiological analyses.....	49
5	RESULTS.....	50
5.1	Marsh Deer (<i>Blastocerus Dichotomus</i>).....	50
5.1.1	Biological and epidemiological aspects.....	50
5.1.2.	Pathologic analysis.....	52
5.3	Brown Brocket Deer (<i>Mazama gouazoubira</i>).....	80

5.3.1	Biological and epidemiological aspects	80
5.3.2	Pathologic analysis.....	82
5.4	Causes of death (CD).....	112
5.4.1	CD in marsh deer.	112
5.4.2	CD in brown brocket deer.	115
6	DISCUSSION	118
7	FINAL COMMENTS	130
	APPENDIX	154

1 INTRODUCTION

Hunting and habitat destruction, along with livestock-introduced diseases are the main threats to deer conservation in Brazil (DUARTE; REIS, 2012). Deer may serve as reservoir for many infectious agents, promoting its spread and maintenance in the environment. In addition, the consumption of venison has important implications for public health, because of potential transmission of toxoplasmosis, clostridiosis, Creutzfeldt-Jakob disease and hepatitis E (MIDURA et al., 1972; VOS, 1982; SACKS et al., 1983; BELAY et al., 2001; TEI et al., 2003). Health aspects of certain deer species have been extensively studied, with reports of infectious (i.e., bacterial, viral, parasitic and prionic diseases) and non-infectious diseases (e.g., toxic, nutritional-metabolic disorders, neoplasia) (DINKINES et al., 1992; NETTLES et al., 2002; VREELAND, DIEFENBACH; WALLINGFORD, 2004; MAWHINNEY et al., 2010). Health monitoring studies in South American deer have mainly focused on the exposure to several infectious agents, both in free ranging and in captive deer, but clinical and pathogenesis studies are also available (DRIEMEIER et al., 2002; UHART et al., 2003). Studies in white-tailed deer fawns (*Odocoileus virginianus*) do not show a common death pattern; instead both infectious and non-infectious pathologic processes have been reported, with no clear predisposition or frequency pattern (CARSTENSEN et al., 2009; VREELAND, DIEFENBACH; WALLINGFORD, 2004). Trauma is the leading cause of death in free ranging Swedish roe deer (*Capreolus capreolus*), followed by starvation, enteritis/gastritis, bacterial infections, parasitic infestations, systemic diseases, neoplasms and congenital disorders (AGUIRRE et al., 1999). In South American deer, mortality outbreaks associated with cat flea infestation (*Ctenocephalides felis*) have been reported in Brazilian MD (SZABÓ et al., 2000).

2 OBJECTIVES

2.1 GENERAL OBJECTIVE

To identify and characterize the macroscopic and microscopic features of pathological conditions together with the etiological agents possibly involved, and the causes of death of two species of South American deer referred to the Laboratory of Wildlife Comparative Pathology (LAPCOM), VPT-FMVZ-USP, over a 210-year period (1995-2015) with the aid of medical and necropsy records, and histopathological, histochemical and immunohistochemical techniques.

2.2 SPECIFIC OBJECTIVES

- ✓ To identify and characterize the main macroscopic manifestations via analysis of medical and necropsy records.
- ✓ To characterize the causes of death through the information gathered from medical and necropsy records, and histopathologic evaluation.
- ✓ To investigate the presence of selected infectious agents and special morphologic patterns through histochemical staining.
- ✓ To investigate the presence of selected infectious agents and special morphologic patterns through immunohistochemistry methods.
- ✓ To deduce on the impacts and consequences of these pathological processes in deer conservation, as well as human and animal health, based on the results obtained in the previous stages.

3 LITERATURE REVIEW

3.1 NATURAL HISTORY AND BIOLOGY OF SOUTH AMERICAN DEER

It is estimated that the evolution of deer took approximately 30 million years MY. It is believed that the antlers played an important role in their evolution (GEIST, 1998). Diacodexis was the first known artiodactyl, dwelling 50-55 MY in the Eocene. They are believed to be a common ancestor of the ruminants (ROSE, 1982).

Figure 1 - Artiodactyl evolution

Prothero (1994)

During the Oligocene, with the formation of the Alps and the Himalayas the first deer-like forms became extensively diverse. Fossil evidence from the Miocene registered the first members of the superfamily Cervoidea, described in Eurasia. The

Dicrocerus, Euprox and Heteroprox are considered the first antlered deer species. The first Cervinae appeared in Asia, during the late Miocene, around 7-9 MY. *Cervus* and *Dama* genres appeared about 3 MY, during an outbreak of cervid diversity observed in the Pliocene, caused by an increase in rangeland due to climate changes. The first cervid appeared in North America around 5 Mya (GENTRY, 1994; DONG; PAN; LIU, 2004; LUDT, 2004; GILBERT; ROPIQUET; HASSANIN, 2006).

In the late Pliocene, approximately 2.5–3 MY, the formation of the Isthmus of Panama, created the “Great American Interchange” between North and South American species, and is believed to be the method of entrance for the first deer to South America (STEHLI; WEBB, 1985; WEBB, 2000). The largest known cervids (Eucladoceros and Megaloceros) appeared in the Pleistocene (GONZALEZ; KITCHENER; LISTER, 2000). Central and South American deer can be allocated in two morphological groups. The first one includes the *Mazama* and *Pudu* genera, with small-bodied species, and males with unbranched spiked antlers – morphological adaptations to their ecosystem of tall forests and closed landscapes. The second group includes the *Odocoileus*, *Hippocamelus*, *Ozotoceros*, and *Blastocerus* genera, with bigger-bodied species and males with branched antlers. They inhabited open forested areas, grasslands, pampas and wetlands (DUARTE; GONZÁLEZ; MALDONADO, 2008).

The brown brocket deer (BBD; *Mazama gouazoubira*) is classified as a Small Solitary Forest Ruminant (SSFR). Considered a medium-sized deer, and weighing between 11 to 25 kg, the BBD is widely distributed along Argentina, Brazil, Bolivia and Uruguay (figure 2-3). The secretive nature and closed habitat in which these species dwell, limits our ability to study them. This species is of least concern in the IUCN (International Union for Conservation of Nature and Natural Resources) Red List of Threatened Species, and CITES (Convention on International Trade in Endangered Species of Wild Fauna and Flora) does not include it in any appendix. Further research for the conservation of BBD, including the health status evaluation of free ranging populations, are still needed in terms of livestock-wildlife interface (PAN, 2007; BLACK; VOGLIOTTI, 2008; BLACK-DÉCIMA, 2010; DUARTE; GONZALEZ, 2010; DUARTE; REIS, 2012).

The marsh deer (*Blastocerus dichotomus*) is the largest South American deer (figure 2), weighing over 150 kg, classified as vulnerable by the IUCN and is included

in the Appendix I of CITES. According to the Brazilian National Action Plan for Conservation of Endangered South American Deer, the major threats to their extinction are excessive hunting, conversion of wetlands for agriculture, and the construction of dams (figure 3). Diseases introduced by cattle, e.g., foot and mouth disease, brucellosis, babesiosis and ecto- and endoparasites, have also been recognized as important factors in their populations' decline (DUARTE; GONZÁLEZ, 2010; PIOVEZAN et al., 2010; DUARTE; REIS. 2012).

Figure 2 - Left), Male brown brocket deer; Right), Male marsh deer

Credits: Sergio Ramirez (2016)

Figure 3 - Distribution and density of marsh deer (right) and brown brocket deer (left) populations

Author: (IUCN, 2012).

3.2 PATHOLOGY OF DEER SPECIES BY ORGAN SYSTEM

3.2.1 ALIMENTARY SYSTEM

3.2.1.1 *Oral cavity*

Congenital abnormalities of the oral cavity, such as brachygnathia and prognathism have been widely reported in North American deer (WOBESER; RUNGE, 1973; BARRETT; CHALMERS, 1975; ROLLOR, 1993; HOY et al., 2011), in an Italian FD fawn has been reported Palatoschisis (MAZULLO, 2005).

In infectious pathology, experimental infections with vesicular stomatitis virus in deer species showed that despite the presence clinical signs (blisters, sores, and sloughing of skin in the mouth, on the tongue, on the muzzle and ears, and above the hooves) and gross lesions in the oral cavity, this disease has a fast course and a reduced viremia period. An enzootic cycle is suggested by the presence of antibodies in free ranging populations (KARSTAD; HANSO, 1957; WEBB et al., 1987; FLETCHER et al., 1991). It is important to notice there are no studies on the pathogenesis of this disease in deer available in the literature after the 90's.

Oral lesions have been observed in fawn species (*Odocoileus hemionus*, *O. virginianus*, *Antilocapra americana*) experimentally infected with poxvirus. Gross findings included elevated, multilobulated and sharply delineated lesions in the mucosa. Microscopically, acanthotic epidermis, epithelial peg formation and mononuclear cell infiltration of the dermis were observed (LANCE; HIBLER; DEMARTINI, 1983). Ulcerative and necrotizing stomatitis have been reported in viral (MCF, adenovirus, poxvirus) and protozoal infections (e.g., *Besnoitia tarandi*) in several deer species, as well as in American bison and reindeer (DIAZ; STÉEN; FABER, 1996; WOODS et al., 1999; WOODS et al., 2001; O'TOOLE et al., 2002; DUBEY et al., 2004).

3.2.1.2 *Salivary glands*

Infestation by *Elaeophora schneideri* may lead to salivary parotid impactions, in severe cases was reported gingivitis, loose of premolar and/or molar teeth, and lysis

of mandibular bone in North American WTD (COUVILLION et al 1986). In BVD multiorgan infection are reported immune-stain of viral antigen in salivary glands of WTD (CHASE et al., 2008).

3.2.1.3 *Esophagus*

There are reports of esophageal gongylonemiasis observed post-mortem in North American deer (SAMUEL; BEAUDOIN, 1966; BEAUDOIN; SAMUEL; STROME, 1970). In Swedish Roe Deer, there is reported ulcerative and necrotizing esophagitis associated to GI-tract necro-ulcerative inflammation, however, no etiology was isolated (DIAZ, STÉEN, FABER, 1996).

3.2.1.4 *Forestomachs*

Ruminal bloat has been reported in chemical restraining with Medetomidine/Ketamine in North American deer (CAULKETT; CRIBB; HAIGH, 2000). Traumatic reticulopericarditis has been reported in North American deer (FOREYT; LEATHERS, 1986). 'Ruminal drinking' (reticulo-ruminal milk accumulation) has been described in fawns of farmed white-tailed deer, with clinical signs including diarrhea, cachexia and sudden death. However, the only gross finding was necrotic and ulcerative rumenitis and the presence of fresh and coagulated milk in the rumen (ADETUNJI et al., 2016).

Rumenitis is considered an important cause of death in captive Canadian deer, either due to the consumption of food rich in carbohydrates or food shortage in free ranging animals (WOBESER; RUNGE, 1975; WOOLF; KRADEL, 1977; HATTEL et al., 2004; HATTEL et al., 2007). Necrotic and ulcerative rumenitis were reported in systemic adenovirus and poxvirus infections (WOODS et al., 1999; BOYCE et al., 2000; BAUGHMAN et al., 2011). Rumenitis is an important cause of death in captive Canadian deer, however, in other species has reported a lower frequency (HAIGH; BEREZOWSKI; WOODBURY, 2005).

3.2.1.5 *Abomasum*

There are few reports of abomasal ulceration in captive white tailed-deer associated with bacterial pneumonia, enterocolitis, intussusception, chronic diarrhea and capture myopathy. Morphological patterns are like those observed in calves; however, the pathophysiological mechanisms are still unknown (PALMER; WATERS; WHIPPLE, 2001).

3.2.1.6 *Intestines*

Enteritis is a well-recognized cause of death in deer, affecting free-ranging and captive animals of various ages and body condition (AGUIRRE; BRÖJER; MÖRNER, 1999; LAMARQUE, 1999; HAIGH; BEREZOWSKI; WOODBURY, 2005). The following bacterial pathogens have been implicated in enteritis and diarrhea in free-ranging and captive deer: *Escherichia coli*, *Clostridium* spp., *Salmonella* spp., *Yersinia* spp., *Lawsonia intracellularis*, *Mycobacterium paratuberculosis* (POWER; HAAGSMA; SMYTH, 1993; DROLET; LAROCHELLE; GEBHART, 1996; SATO et al., 2000; MACKINTOSH; HAIGH; GRIFFIN, 2002; HAIGH; BEREZOWSKI; WOODBURY, 2005; BALSEIRO et al., 2008). Viruses commonly implicated in cases of enteritis and diarrhea included: rotavirus, adenovirus, herpesvirus and parvovirus (WOODS et al., 1999; WOODS et al., 2001; KEEL et al., 2003; HAIGH; BEREZOWSKI; WOODBURY, 2005).

3.2.2 HEPATOBILIARY AND PANCREAS

Cystic lesions of the liver are reported in free-ranging North American WTD, at necropsy multiple cysts scattered throughout the liver, without other gross cystic lesions in the carcass. Histopathologically, cysts were lined by a smooth epithelium of cuboidal to columnar cells (PICONE et al 1981).

Hepatic necrosis is not considered a frequent cause of death in deer (HATTEL et al., 2004). Experimental infections with BTV caused hepatic hemodynamic

disturbances (e.g., congestion and hemorrhages) in white tailed deer (KARSTAD; TRAINER, 1967). Hepatic chronic degenerative/reparative processes (e.g., cirrhosis) caused by giant liver fluke (*Fascioloides magna*) has been described in some European species (NOVOBILSKÝ et al., 2007; KARAMON et al., 2015). Although some European and Asian deer appear to carry Hepatitis E Virus, infection is subclinical. Furthermore, this agent has been successfully transmitted to humans through meat consumption (TEI et al., 2003; BOADELLA et al., 2010; RUTJES et al., 2010).

Experimental studies of aflatoxin consumption have shown subclinical liver injury in white-tailed deer, characterized by an increase in alkaline phosphatase and hepatocellular degeneration (QUIST et al., 1997).

In British Roe deer, there is associated the presence of liver carcinomas with high levels of spruce in their diet, induced by the poor habitat quality (DE JONG et al., 2004).

Lymphocytic vasculitis, perivascularitis and fibrinoid arterial necrosis are observed in pancreatic blood vessels of North American WTD with MCF (LI et al., 2000).

3.2.3 URINARY SYSTEM

3.2.3.1 *Kidney*

Polycystic kidney disease, a congenital condition characterized by severe dilation of renal tubules has been found in European and North American stillbirths (PALMER; CARPENTER, 2004; BLUTKE et al., 2013). Renal dysplasia has been described in fetuses of North American white-tailed deer (WOBERSER; RUNGE, 1973).

The reported rate of renal urolithiasis occurrence in North American deer herds is approximately 1.3% (WOOLF; KRADEL; ROTHENBACHER, 1976). In WTD deer this disease has been associated with the presence of oxalate crystals and systemic aspergillosis (WYAND; LANGHEINRICH; HELMBOLD, 1971).

Drug-associated renal papillary necrosis has been reported in animals treated with nonsteroidal anti-inflammatory drugs (NSAID). The main histopathological findings described, included: coagulation necrosis of the renal medulla and papillae,

thrombosis and interstitial medullary edema (STERN et al., 2010). Experimental studies on the nephrotoxicity of bog asphodel (*Narthecium ossifragum*) in European deer have shown a marked increase of serum creatinine and urea, tubular epithelial cell degeneration, necrosis and regeneration (FLÅØYEN et al., 1999). Denholm and Westbury (1982) reported renal cortical infarcts in animals with MCF.

Studies of chronic cadmium poisoning in roe deer (*Capreolus capreolus*) conducted in Austria indicated that increased environmental concentrations may be an important cofactor in the pathogenic mechanisms of renal damage. The most important histopathological findings were vacuolar degeneration, necrosis, lymphohistiocytic infiltration, interstitial fibrosis, glomerular swelling, pigment deposition and thickening of Bowman's capsule (BEIGLBÖCK et al., 2002).

Capture myopathy or exertional rhabdomyolysis, is possibly one of the most studied pathological processes in deer. Some authors believe it is an important iatrogenic disease that affects a wide range of ungulates. It is widely found in American, European, Asian and African species, usually associated with stress factors such as captivity and immobilization. Capture myopathy is characterized by metabolic acidosis, muscle necrosis, myoglobinuria and subsequent renal failure and death. Four clinicopathological presentations have been recognized:

Capture Shock Syndrome (CSS): occurs during immobilization or within a short period after capture (1-6 hours). Gross lesions include hepatic and pulmonary congestion and pulmonary edema. The most commonly observed histopathological changes are characterized by small areas of necrosis in the skeletal muscle, brain, liver, heart, adrenal glands, lymph nodes, spleen, pancreas and renal tubules.

Ataxic Myoglobinuric Syndrome (AMS): Possibly the most commonly observed form of capture myopathy, occurring several hours to days post-capture. Upon necropsy examination, kidneys are swollen and dark, skeletal muscles (cervical, lumbar, and flexor and extensor limb muscles) contain pale, a linear pattern of soft and dry areas accentuated by small white foci, and occasionally, either an empty or slightly filled urinary bladder containing a small amount of brownish urine. Histological findings show marked tubular dilatation and necrosis with the presence of protein (myoglobin) casts, acute rhabdomyolysis.

Ruptured Muscle Syndrome (RMS): Manifests within 24 to 48 hours' post-capture. Muscular gross findings are similar to AMS, but more severe and widespread.

Histological lesions are characterized by marked necrosis, extensive sarcolemmal proliferation, fibrosis, and muscular regeneration

Delayed-Peracute Syndrome (DPS): Observed in animals held captive for at least 24 hours. Usually, it presents no gross pathological findings, however, rhabdomyolysis is found on histopathology.

(WOBESER et al., 1976; WALLACE; BUSH; MONTALI, 1885; BERINGER et al., 1996; PATERSON, 2007; CATÃO-DIAS; CAMARGO, 2010; DECHEN QUINN et al., 2014; NUVOLI et al., 2014).

3.2.3.2 *Lower urinary tract*

Hemodynamic disturbances (e.g., hemorrhage) have been extensively linked to MCF and adenovirus infections (SANFORD; LITTLE; RAPLEY, 1977; DENHOLM; WESTBURY, 1982; WOODS et al., 1999; SCHULTHEISS et al., 2007). Neoplastic disease processes including hemangioma, hemangiosarcoma, transitional cell carcinoma and urothelial carcinoma are reported associated with chronic ingestion of bracken fern (*Pteridium aquilinum*) in European FD (SCALA et al., 2014). Invasive poorly differentiated carcinoma has also been described in an adult male North American WTD (ROHER; NIELSEN; STONE, 1982).

3.2.4 RESPIRATORY SYSTEM

3.2.4.1 *Nasal cavity and sinuses*

Epistaxis in Chital (*Axis axis*) has been associated with the presence of carcinoma of the ethmoid mucosa and is a sign of poor prognosis (RAJAN; SREEKUMARAN; SULOCHANA, 1982). Epistaxis may occur in terminal courses of epizootic hemorrhagic disease (SHOPE; MACNAMARA; MANGOLD, 1960).

Rhinitis has been observed in Ovine Herpesvirus type 2 (Ov-HV-2), Bovine Herpesvirus Type 1 (BHV-1) and BTV infections in domestic ruminants, while catarrhal

and necrotizing rhinitis have been reported in deer with Ov-HV-2 and adenoviral infections, respectively (CLARK et al., 1972; LAMONTAGNE; SADI; JOYAL, 1989; WOODS et al., 1999; MACLACHLAN et al., 2009; DAS NEVES et al., 2010; HEADLEY et al., 2012; PALMER et al., 2013). Fibrinosuppurative rhinitis is observed in FD with septicemia caused by *Pasteurella multocida multocida*. Histologically, acute inflammation characterized by marked hyperemia, neutrophilic infiltrates, desquamated epithelium and occasional sub epithelial clusters of coccobacilli are frequently observed (ERIKSEN et al., 1999).

The most common pathologic process reported in nasal sinuses of black-tailed deer is the infestation by *Cephenemyia stimulator* (Diptera) larvae, occasionally associated with catarrhal sinusitis ((CALERO-BERNAL; HABELA, 2013).

3.2.4.2 Pharynx

Catarrhal inflammation may be seen in *Cephenemyia stimulator* (Diptera) larvae infestations in the pharynx and guttural pouch ((CALERO-BERNAL; HABELA, 2013). The following bacteria have been isolated from North American deer with necrotizing and suppurative pharyngitis: *Fusobacterium* sp., *Actinomyces pyogenes* and *Prevotella* sp. Primary adenoviral infections may favor secondary bacterial pharyngitis (WOBESER; RUNGE; NOBLE, 1975; WOODS et al., 1999; BOYCE et al., 2000).

3.2.4.3 Larynx

Viral infections by Rinderpest virus and Bluetongue Type 8 (BTV-8) virus may cause hemodynamic disturbances (congestion and hemorrhage) (FALCONI; LOPEZ-OLVERA; GORTAZAR, 2011). Necrotic laryngitis has been observed in North American deer with necrobacillosis (WOBESER; RUNGE; NOBLE, 1975). In roe deer, infestation by *Cephenemyia stimulator* larvae may cause catarrhal laryngitis (CALERO-BERNAL; HABELA, 2013).

3.2.4.4 *Trachea*

Reports of heat stroke in New Zealand farmed deer have been associated with severe acute congestion of the entire respiratory tract (BLACK; MITCHELL, 2009). Viral infections such as Adenovirus, EHDV, BTV and MCF cause hemodynamic disturbances, resulting in superficial fibrinous exudate, associated by secondary bacterial colonization. Histologically, epithelial hypertrophy and hyperplasia, and partial epithelial cell depletion of submucosal mucous glands are found. In adenovirus cases, it is possible to observe epithelial intranuclear inclusions (SHOPE; MACNAMARA; MANGOLD, 1960; TOMKINS et al., 1997; WOODS et al., 1999; SORDEN et al., 2000; FALCONI; LOPEZ-OLVERA; GORTAZAR, 2011).

Infection with cilia-associated respiratory bacillus (CAR-bacillus) has been reported in European deer. The main histopathological findings included multifocal infiltrates of lymphocytes in the lamina propria (BERGOTTINI et al., 2005). Caseonecrotic granulomas, hyperemia and necrosis of the mucosa were observed in *Mycobacterium bovis* infections (PALMER; WHIPPLE; OLSEN, 1999). Infection with *Dyctiocalus* larvae was described in European and North American deer. Major gross alterations included heavy tracheal and bronchial worm infestation, and pulmonary edema and emphysema (CHARLESTON, 1980; PANADERO et al., 2001).

3.2.4.5 *Lungs*

Atelectasis is an incidental finding in deer with epizootic hemorrhagic disease virus type 2 (EHDV-2) and invasive aspergillosis (JENSEN; JØRGENSEN; SCHØNHEYDER, 1989; NOON et al., 2002). Pulmonary emphysema has been reported in lungworm infestation, possibly caused by laryngospasms and lung migration of the first stage larvae (GOBLE, 1941; SUTHERLAND, 1976; NETTLES et al., 1977; CHARLESTON, 1980; RAHKO, SAARI; NIKANDER, 1992; MASON, 1994; MAWHINNEY; WOODGER; KNUDSEN, 2010).

Pulmonary edema, congestion, and less frequently hemorrhage, are commonly observed in North American, European and South American deer infected with BTV, EHDV and Adenovirus (SHOPE; MACNAMARA; MANGOLD 1960; KARSTAD; TRAINER, 1967; PRESTWOOD et al., 1974; WOODS et al., 1996; WOODS et al., 1997; SORDEN; WOODS; LEHMKUHL, 2000; LEHMKUHL; HOBBS; WOODS, 2001; NOON et al., 2002; FAVERO et al., 2013). Pulmonary congestion and interlobular edema are frequently observed in bacterial infections caused by *Cowdria ruminantium*, *Actinomyces pyogenes*, *Mycobacterium paratuberculosis* and *Mycoplasma* (DARDIRI; LOGAN; MEBUS, 1987; TURNQUIST; FALES, 1998; DYER et al., 2004; GONZÁLEZ-ACUÑA et al., 2011). Petechial hemorrhages and septal edema were described in a Norwegian roe deer with granulocytic ehrlichiosis (STUEN et al., 2001).

Pneumonia is one of the most relevant pathological conditions in routine cervid medicine, and is considered a major morbidity and mortality factor, both in captive and free-ranging deer (HATTEL et al., 2004; VREELAND, DIEFENBACH; WALLINGFORD 2004). The main bacteria isolated in cases of bronchopneumonia are: *Actinomyces pyogenes*, *Arcanobacterium pyogenes*, *Escherichia coli*, *Fusobacterium necrophorum*, *Klebsiella pneumoniae*, *Mannheimia haemolytica*, *Mycobacterium* sp., and *Streptococcus gallolyticus* (WOBESER et al., 1975; ORR 1991; RHYAN; SAARI, 1995; TURNQUIST; FALES, 1998; LUCIOLI et al., 2008; DA COSTA et al., 2008). The most commonly reported viral agents in respiratory cases are: BTV, ADV, EHDV, MFC and BVDV, Immunosuppression is a typical pathogenic mechanism in BVD cases (PRESTWOOD et al., 1974; BROWN; BLOSS, 1992; TOMKINS et al., 1997; WOODS et al., 1997; SORDEN et al., 2000; LEHMKUHL et al., 2001; WOODS et al., 2001; DRIEMEIER et al., 2002; PASSLER et al., 2012).

In North American WTD was reported pulmonary fibroblastoma, apparently, metastases from the skin tumors, suggesting that deer cutaneous fibromatosis can occasionally be a malignant disease (KOLLER, OLSON, 1971).

3.2.4.6 Pleura

Fibrinous pleuritis in housed Weaner deer, may be caused by: *Pasteurella* spp., *Arcanobacterium pyogenes*, protostrongylid infestations and pulmonary mycoplasmosis. Gross lesions include occasional fibrinopurulent pleuritis and pericarditis, areas of pulmonary necrosis and abscessation, and variable amounts of pleural yellow, serofibrinous, foul-smelling exudate (PRESTWOOD; SMITH; BROWN, 1971; EVE; KELLOGG, 1977; ORR, 1991; PALMER; WHIPPLE, 1999; DYER; KROGH; SCHAAN, 2004). Tuberculous pleuritis was described postmortem in New Zealand deer with tuberculosis (LUGTON et al., 1998).

Metastases to the pleura has been recognized in a Père David's Deer, Metastatic Adenocarcinoma of the mammary gland (VEATCH, CARPENTER, 1993), concurrent multicentric hemangiosarcoma (YOON et al., 1999), in WTD, lymphosarcoma (DEBBIE, FRIEND, 1967), and in an adult captive European FD with Pleural mesothelioma (BYERLY et al., 1989).

3.2.5 CARDIOVASCULAR SYSTEM

Myocarditis was found in a North American WTD with *Sarcocystis mehlhorni* infection, as well as in an Argentinian RD (CALERO-BERNAL et al., 2015; REISSIG et al., 2016). Vasculitis is a common finding in MCF (BROWN; BLOSS, 1992; TOMKINS et al., 1997; KEEL et al., 2003; LI et al., 2003; PALMER et al., 2013).

3.2.6 HEMATOPOIETIC SYSTEM

Lymphoid depletion is reported in a variety of viral and bacterial diseases, such as BVDV, Adenovirus and tuberculosis, among others (WELCH et al., 1983; WOODS et al., 1996; DUNCAN et al., 2008; PASSLER et al., 2012). SCHMITT et al. (1997), described the association between caseous lesions in lymph nodes and tuberculosis in North American Deer.

Multicentric T cell lymphosarcoma was diagnosed in an ataxic and severely debilitated female WTD. On necropsy examination emaciation, severe

lymphadenopathy of mesenteric and abdominal lymph nodes, nodules in the rumen, and brain metastasis were found (MADSON; OPRIESSNIG, 2009).

3.2.7 GENITAL SYSTEM

Information on reproductive parameters in both males and females is available in the literature. The scarce reports include metritis and dystocia (TURNQUIST; FALES, 1998; AGUIRRE; BRÖJER; MÖRNER, 1999)

3.2.8 NERVOUS SYSTEM

Intracranial abscesses are the most studied pathological process of the nervous system, and considered an important cause of death in North American deer (DAVIDSON et al., 1990; BAUMANN 2001; KARNS et al., 2009). Most frequently seen in males, this condition is caused by secondary bacterial colonization, mainly by *Arcanobacterium pyogenes* and *Actinomyces pyogenes*, following trauma or antler fracture. Bacterial meningoencephalitis caused by *Listeria monocytogenes* has been reported in European deer (ERIKSEN et al., 1998).

Most available studies regarding neurotropic viruses in deer were based on serology, but overall, reports of viral nervous diseases are scarce, serologic evidence of West Nile Virus and St. Louis Encephalitis Virus infection in WTD (FARAJOLLAHI et al., 2004), clinical evidence of rabies in a North American WTD (WILSON, 1949). Nevertheless, eastern equine encephalitis virus is considered a possible agent of neurologic disease in North American deer (TATE et al., 2005).

Neospora caninum is considered an important cause of non-suppurative encephalitis in fawns of European and North American deer (DUBEY et al., 1996; SOLDATI et al., 2004).

3.2.9 INTEGUMENTARY SYSTEM

Viral dermatitis caused by poxvirus has been identified in fawns; although lesions are less frequent in comparison with contagious ecthyma viruses in sheep (WILLIAMS et al., 1985). Skin tumors were observed in European deer affected by Cervid papillomavirus (CePV1) diagnosed by molecular techniques (ERDÉLYI et al., 2009; SCAGLIARINI et al., 2013).

Dermatophilus congolensis is the most important bacterial pathogen in deer dermatopathology. It has been extensively studied in North American deer and is characterized by desquamation of skin of the snout, antlers and ears and entire body covered with eruptions (GORDON; SALKIN; STONE, 1977).

Mite dermatitis, mainly caused by trombidiosis (e.g., *Eutrombicula* spp.) and *Demodex* sp., has been observed in North American deer species. In trombidiosis, skin lesions are characterized by chronic diffuse plasma cell dermatitis containing intralesional larvae of trombiculin mites. *Demodex* sp. infestation also presented dilated and filled hair follicles and sebaceous glands containing large numbers of mites, and no signs of associated inflammatory response (LITTLE; CARMICHAEL; RAKICH, 1997; GENTES; PROCTOR; WOBESER, 2007).

3.2.10 MUSCULOSKELETAL SYSTEM

Clostridial myositis causing acute necrosis, degeneration, hemorrhage, fibrin deposition and minimal inflammatory exudate was described in caribou (*Rangifer tarandus*) (HERRON et al., 1979).

3.3 NONINFECTIOUS DISEASES

According to Masters and Flach (2013), Capture myopathy (exertional myopathy) is a major pathological process caused by stress, trauma caused by vehicle

collision is another process of great importance, deer are known to be susceptible to temperature disorders (hyperthermia/hypothermia), hypothermia is especially important in youngsters due to their thermoregulatory issues; Finally, protein rich diets may cause ulcers in the abomasum and duodenum.

3.4 INFECTIOUS DISEASES

3.4.1 Viral diseases

Deer are affected by several viral diseases, but most importantly by BTV, BVDV, EHDV and IBR, with occasional reports of Parainfluenza-3 (PI-3), FMD and BRSV (LAMONTAGNE; SADI; JOYAL, 1989; SORDEN et al., 2000; DRIEMEIER et al., 2002; UHART et al., 2003; ARAUJO; NOGUEIRA; DUARTE, 2010; DUARTE; GONZÁLEZ, 2010). A brief pathological description for selected viral agents follows.

3.4.1.1 *Infectious Bovine Rhinotracheitis (IBR)*

There are numerous serological, pathological and molecular studies on IBR in European, Asian, and North and South American deer. No anti-Bovine Herpesvirus-1 (BHV-1) antibodies were detected in Chilean South Andean deer (*Hippocamelus bisulcus*), Bolivian BBD and Brazilian MD (DEEM et al., 2004; CORTI; SAUCEDO; HERRERA, 2013; ZIMPEL et al., 2015). Seroneutralization assays detected BHV-1-related antibody titers in Canadian WTD and caribou (*Rangifer tarandus caribou*), suggesting that this viral infection is endemic in the deer population (ELAZHARY et al., 1961; LAMONTAGNE; SADI; JOYAL 1989; SADI et al., 1991). BHV-1-seropositive Italian FD and farmed North American WTD have also been diagnosed respectively by Giovannini et al. (1988) and Ingebrigtsen, Ludwig and McClurkin (1966).

3.4.1.2 *Malignant Catarrhal Fever*

Outbreaks of MCF in deer include the follow viruses: OvHV-2, CpHV-2, AIHV-1.

- Ovine Herpesvirus type II (OvHV-II)

Major clinical signs described in experimental and natural OvHV type II infections included: profound depression, fever, anorexia, corneal opacity, uveitis, hypopyon, keratitis and watery to bloody diarrhea (12-24 hours ante mortem). Gross postmortem findings included: hypopyon, corneal opacity, hemorrhagic enterocolitis, petechial to ecchymotic hemorrhage in the kidneys, spleen, myocardium, lungs, lymph nodes and skeletal muscles (diaphragm and intercostal), occasionally presenting multifocal necrotic foci identified as pale areas in the myocardium, enlarged edematous and hemorrhagic mesenteric lymph nodes, pulmonary congestion and edema. Histological findings included: lymphocytic and plasma cell adventitial, and occasionally sub-intimal infiltrates (vasculitis and perivasculitis) in small to medium sized blood vessels of multiple organs, thrombosis and multiple hemorrhage (BROWN; BLOSS, 1992; TOMKINS et al., 1997; PALMER et al., 2013).

- Caprine Herpesvirus type II (CaHV-II)

Clinical signs included anorexia, hemorrhagic diarrhea, weight loss and alopecia around the mouth, eyes and limbs. Gross postmortem findings included: alopecia, skin thickening, crusting, hyperkeratosis and moderate focal ulceration of the distal extremities. On histopathology, cerebrum, meninges and cerebellum exhibited significant lymphocytic infiltration, rare plasma cells in the adventitia of small to medium sized blood vessels (less severe in mesentery cardiac and renal tissues). Lymphoplasmacytic and necrotizing arteritis in the rete mirabile and subcutis were also reported, along with other findings such as coagulation necrosis of the lymph nodes and multifocally ulcerated intracorneal epidermal eosinophilic pustules (KEEL et al., 2003; LI et al., 2003). In addition to OvHV-2, which is responsible for most outbreaks of MCF in deer, CpHV-2, AIHV-1, and MCFV–white-tailed deer (WTD) can also cause disease.

3.4.1.3 Epizootic Hemorrhagic Disease

The Orbivirus genus includes two viruses that cause hemorrhagic disease in deer: Bluetongue virus (BTV) and Epizootic Hemorrhagic Disease Virus (EHDV). BTV-1 and 8 serotypes has been widely studied in Europe. The environmental circulation of the virus has been demonstrated in Spain, where experimental infections have shown clinical and pathological findings like those observed in domestic ruminants (LÓPEZ-OLVERA et al., 2010; FALCONI et al., 2012; DROLET et al., 2013).

3.4.1.4 *Bovine Viral Diarrhea Virus (BVDV)*

BVDV may cause symptomatic or asymptomatic infections in North American and European deer. Although gross lesions are not commonly observed on postmortem examination, histopathological evidence of pneumonia has been detected in fawns. Non-cytopathic BVDV has been detected in animals with bacterial pneumonia (AGUIRRE et al., 1995; PASSLER et al., 2007; CHASE et al., 2008; RAIZMAN et al., 2009; PASSLER et al., 2012).

3.4.1.5 *Foot and Mouth Disease (FMD)*

FMD in wildlife can vary from completely unapparent to acutely lethal infection. Pathological findings in FMDV-infected white-tailed deer are similar to those seen in cattle, including vesicles on both oral and coronary band. In experimental infections viral antigen was detected in tissues of WTD. Studies suggest that cattle grazing in areas adjacent to National Parks present a risk for transmission to deer and vice versa. Cattle or wild ungulates can act as efficient disseminators of FMD viruses (MCVICAR et al., 1974; THOMSON; VOSLOO; BASTOS, 2003; ARAUJO; NOGUEIRA; DUARTE, 2010; MONIWA et al., 2012; JORI et al., 2014).

3.4.2 Bacterial diseases

There are several reports of bacterial diseases involving deer, including brucellosis, anthrax, clostridiosis (including tetanus), paratuberculosis, salmonellosis, tuberculosis and yersiniosis, among others. Most of them are of significance to public health. Clinical and pathological conditions are commonly similar to those observed in domestic ruminants (MASTERS; FLACH, 2013). A brief pathological description for selected bacterial agents follows.

3.4.2.1 *Mycobacterial diseases*

The genus *Mycobacterium* sp. has been extensively studied. *M. avium* ssp. *paratuberculosis* affects several deer species, causing proliferative enteritis mainly in the last portion of the small intestine, at the ileocecal valve (BALSEIRO et al., 2008; MACKINTOSH et al., 2010). Wildlife studies suggest lower prevalence, but with potential to remain in wild populations (O'BRIEN et al., 2002; PALMER et al., 2002; MARTÍN-HERNANDO et al., 2010).

3.4.3 Protozoal diseases

Neospora caninum has been described in European and North American deer. Its prevalence varies, and in general, animals do not express clinical signs. *Toxoplasma gondii* infections can cause greater impact on deer population if they get involved in the its sylvatic cycle. (WOODS et al., 1994; MALMSTEN, JAKUBEK, BJÖRKMAN 2011).

3.4.4 Helminthic diseases

Although cervids are affected by a wide variety of helminth parasites, infestations are usually asymptomatic. Severe helminthic infestation may lead to pathological disorders; however, in most cases, they represent an incidental postmortem finding. The most commonly reported helminth genera in the gastrointestinal tract of deer are: *Ostertagia*, *Trichostrongylus*, *Cooperia* and

Oesophagostomum (HOSKIN et al., 2000). *Dictyocaulus* and *Protostrongylus* are the most common helminths involving the respiratory system of deer. They have been linked to severe infestations and multiple granulomas in the lung parenchyma, emphysema, pulmonary edema, interstitial emphysema and fibrosis (CHARLESTON, 1980; RAHKO et al., 1992; HOSKIN et al., 2000; PANADERO et al., 2001; CHARLESTON, 2011).

3.4.5 Fungal diseases

Pneumocystis carinii has been described in asymptomatic European deer. Mucormycosis was also diagnosed in captive European deer; in this case, lesions were predominantly pulmonary and *Aspergillus fumigatus* was also isolated (JENSEN et al., 1989; LAAKKONEN 1998).

3.4.6 Prionic diseases

Chronic Wasting Disease is the most important transmissible spongiform encephalopathy reported in deer with proven occurrence in North America, and currently in Norway (MASTERS; FLACH, 2013).

3.4 DISEASES IN SOUTHAMERICAN DEER

3.4.1 Noninfectious

Like other deer species, capture myopathy is one of the major recognizable noninfectious processes; prevention is the best way to control it (CATÃO-DIAS; CAMARGO, 2010).

3.4.2 Infectious

Some viral agents have been explored in South American deer, among the most important include: hemorrhagic disease (BTV, EHDV), highly associated because of clinical signs and gross findings in deer of the genus *Mazama* in Brazil; foot and mouth disease (FMDV), some researches have been studied the presence of this virus, however, it has not been isolated in animals with clinical signs. And serologic evidence of Bovine Respiratory Syncytial Virus (BRSV), Parainfluenza (PI-3), Vesicular Stomatitis (VS) and Malignant Catarrhal Fever (MCF) (ARAUJO et al., 2010). The most important bacterial diseases include: Actinomycosis, brucellosis, campylobacteriosis, clostridiosis, leptospirosis, paratuberculosis, pasteurellosis, salmonellosis and tuberculosis (UHART et al., 2010). According to protozoal diseases there are reports of *Neospora caninum*, *Toxoplasma gondii* and *Sarcocystis* spp. (GENNARI et al., 2010).

4 MATERIALS AND METHODS

4.1 MATERIALS

Formalin-fixed, paraffin-embedded (FFPE) tissue samples of two species of South American deer: brown brocket deer BBD (*Mazama gouazoubira*) and marsh deer MD (*Blastocerus dichotomus*), were analyzed. The samples analyzed included tissues collected from regular necropsies performed during a 21-year period (1995-2015) by **(Department of Parks and Green Areas (DEPAVE), Deer Research and Conservation Center (NUPECCE))** that were archived in the LAPCOM/VPT/FMVZ/USP. A total of 206 cases were studied: 75 MD and 131 BBD. Samples were from different institutions and regions of the State of São, Brazil. The study was performed in compliance with the System Authorization and Information on Biodiversity (SISBIO) of the Brazilian Institute of Environment and Renewable Natural Resources (IBAMA) (license number: 47858-1) and the School of Veterinary Medicine and Animal Science - University of São Paulo (FMVZ-USP) Ethics Committee on Animal Use (CEUA) (license number: 4271090215). A large part of the free-ranging animals came from the (marsh deer conservation program led by the deer research and conservation center (NUPECCE), department of animal science, São Paulo state university (UNESP-Jaboticabal), Brazil.

4.2 METHODS

4.2.1 Histopathology

Tissue samples were processed as routine, embedded in paraffin and trimmed at 5µm with a rotary microtome. Slides were stained with hematoxylin & eosin (H&E).

Histopathological diagnosis and causes of death identified followed adaptation of Montali (CATÃO-DIAS, personal communication, 1993)¹.

4.2.2 Histochemical analysis

Special stains were employed in selected tissue sections in order to better characterize certain histopathological findings (PROPHET et al., 1992):

- **Congo Red:** used to stain amyloid.
- **Gram's method:** used to differentiate bacterial species into two large groups (Gram-positive and Gram-negative).
- **Grocott's Methenamine Silver Stain (GMS):** used as screening for fungal organisms.
- **Luxol Fast Blue (LFB):** used to evaluate myelin.
- **Masson's trichrome stain:** used to evaluate collagen and muscular fibers.
- **Periodic Acid-Schiff (PAS):** used to evaluate polysaccharides such as glycogen, and mucosubstances (e.g., glycoproteins, glycolipids and mucins).
- **Von Kossa stain:** used to reveal mineral deposits on tissue sections.

4.2.3 Immunohistochemical analysis

Immunohistochemistry was performed in order to evaluate myoglobin and glial fibrillary acidic protein (GFAP). These studies were done in collaboration with both the Immunohistochemistry Laboratory at the Adolfo Lutz Institute and Laboratory of Animal Models (VPT-FMVZ-USP). Sections (4 µm) were submitted to IHC streptavidin–biotin–peroxidase method (LSAB; Dako). The sections were dewaxed, rehydrated through graded alcohols and submitted to endogenous peroxidase inactivation by methanol with hydrogen peroxide (3%). Antigen retrieval was performed in a water bath at 98°C

¹ CATÃO-DIAS, J. L. DMV, Titular professor, Department of Pathology, School of Veterinary Medicine and Animal Sciences, University of São Paulo, Brazil.

in citrate buffer (pH 6.0) for 20 mins. The sections were incubated in a humid chamber at 4°C for 16–18 h with the following primary antibodies: anti-GFAP (rabbit polyclonal, 1:400; Dako), anti-myoglobin (1:45000 Adolpho Lutz Institute). Subsequently, the slides were incubated with biotinylated secondary antibodies (LSAB; Dako) followed by streptavidin–peroxidase (LSAB; Dako), both for 30 mins. The binding between antigens and antibodies was visualized using the chromagen diaminobenzidine (Vectastain®; Vector Laboratories) according to manufacturer's instructions, followed by slight counterstain with hematoxylin. Primary antibodies were substituted by homologous non-immune sera as negative controls. Sections of deer brain and kidney without histological alterations were used as controls. Anti-GFAP were used for astrocytic evaluations. Anti-myoglobin was used for identification of renal intratubular myoglobin in cases with a history of capture myopathy.

4.2.4 Epidemiological analyses

We analyzed epidemiological parameters, i.e., gender, age, species, body condition, trauma history and season, and gross findings by organ system available in necropsy reports and histopathological findings. Microsoft Excel was the software used for data collection and statistical analyses were performed based on descriptive statistics (FINNEY, 1952).

5 RESULTS

5.1 MARSH DEER (*BLASTOCERUS DICHOTOMUS*)

5.1.1 Biological and epidemiological aspects

Seventy-five cases were recorded between 1995 and 2015. Distributions per month, year, weather, season and general epidemiological aspects are recorded in figures 4 through 6.

Figure 4 - Annual distribution of Brazilian marsh deer cases between 1995-2015

Figure 5 - Monthly distribution of Brazilian marsh deer cases between 1995-2015

Figure 6 - Seasonality in mortality of Brazilian marsh deer cases between 1995-2015, and its relationship to annual distribution

Trauma and ectoparasitosis were poorly reported; 14.7% (11/75) and 17.3% (13/75), respectively. Tick infestation was the most commonly observed ectoparasitosis (76.9%; 10/13), followed by fleas (23.1%; 3/13). Regarding the severity of infestations, 69.2% (9/13) were severe and 23.1% (3/13) were mild. According to the clinical history, most cases showed no clinical signs prior to death (28%; 21/75). When present, clinical signs mainly included starvation (10.7%; 8/75), trauma (8.0%; 6/75), and sepsis (6.7%; 5/75) (Figure 7).

Table 1 - General epidemiological aspects of Brazilian marsh deer from 1995 to 2015

Category	Subcategories	No.	%
Age group	<i>Adult</i>	48	64.0
	<i>Juvenile</i>	12	16.0
	<i>Fawn</i>	12	16.0
	<i>Unreported</i>	3	4.0
Gender	<i>Female</i>	40	53.3
	<i>Male</i>	31	41.3
	<i>Unreported</i>	4	5.3
Condition	<i>Free ranging</i>	61	81.3
	<i>Captive</i>	7	9.3
	<i>Unreported</i>	7	9.3
Time in captivity	<i>>15 days</i>	32	42.7
	<i>0-24 hours</i>	6	8.0
	<i>24-72 hours</i>	3	4.0
	<i>3-15 days</i>	9	12.0
	<i>Unreported</i>	25	33.3
Body condition	<i>Poor</i>	19	25.3
	<i>Regular</i>	12	16.0
	<i>Good</i>	40	53.3
	<i>Unreported</i>	4	5.3
Trauma	<i>Yes</i>	11	14.7
	<i>No</i>	61	81.3
	<i>Unreported</i>	3	4.0
Ectoparasites	<i>Yes</i>	13	17.3
	<i>No</i>	59	78.7
	<i>Unreported</i>	3	4.0

Figure 7 - Distribution according to reported clinical diagnosis of Brazilian marsh deer from 1995 to 2015

5.1.2. Pathologic analysis

Respiratory system

a) *Macroscopic changes*

Gross pathological findings are recorded in table 2. Edema, congestion, and pneumonia were the main macroscopic findings.

Table 2 - Respiratory system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total
	F	J	A	U	Fe	M	U	G	R	P	U	% (Na/Ne)
Edema	66.7 (8/12)	66.7 (8/12)	55.6 (25/45)	0 (0/1)	56.8 (21/37)	61.3 (19/31)	50 (1/2)	41.7 (5/12)	47.4 (9/19)	71.1 (27/38)	0 (0/1)	58.6 (41/70)
<i>Lung congestion</i>	33.3 (4/12)	33.3 (4/12)	40 (18/45)	0 (0/1)	37.8 (14/37)	38.7 (12/31)	0 (0/2)	16.7 (2/12)	31.6 (6/19)	47.4 (18/38)	0 (0/1)	37.1 (26/70)
<i>Tracheal congestion</i>	0 (0/12)	16.7 (2/12)	8.9 (4/45)	0 (0/1)	10.8 (4/37)	6.5 (2/31)	0 (0/2)	0 (0/12)	0 (0/19)	15.8 (6/38)	0 (0/1)	8.6 (6/70)
Congestion	33.3 (4/12)	41.7 (5/12)	42.2 (19/45)	0 (0/1)	37.8 (14/37)	45.2 (14/31)	0 (0/2)	16.7 (2/12)	31.6 (6/19)	52.6 (20/38)	0 (0/1)	40.0 (28/70)
<i>Aspiration pneumonia</i>	16.7 (2/12)	0 (0/12)	0 (0/45)	0 (0/1)	5.4 (2/37)	0 (0/31)	0 (0/2)	8.3 (1/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	2.9 (2/70)
<i>Bronchopneumonia</i>	0 (0/12)	16.7 (2/12)	13.3 (6/45)	0 (0/1)	13.5 (5/37)	9.7 (3/31)	0 (0/2)	16.7 (2/12)	15.8 (3/19)	7.9 (2/38)	0 (0/1)	11.4 (8/70)
<i>Pleuropneumonia</i>	0 (0/12)	0 (0/12)	8.9 (4/45)	0 (0/1)	2.7 (1/37)	9.7 (3/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	7.9 (2/38)	0 (0/1)	5.7 (4/70)
<i>Non classified pneumonia</i>	8.3 (1/12)	0 (0/12)	20 (9/45)	0 (0/1)	10.8 (4/37)	16.1 (5/31)	50 (1/2)	25 (3/12)	15.8 (3/19)	10.5 (4/38)	0 (0/1)	14.3 (10/70)
Pneumonia	25 (3/12)	16.7 (2/12)	42.2 (19/45)	0 (0/1)	32.4 (12/37)	35.5 (11/31)	50 (1/2)	50 (6/12)	42.1 (8/19)	26.3 (10/38)	0 (0/1)	34.3 (24/70)
<i>Lung</i>	16.7 (2/12)	25 (3/12)	8.9 (4/45)	0 (0/1)	8.1 (3/37)	19.4 (6/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	21.1 (8/38)	0 (0/1)	12.9 (9/70)
<i>Trachea</i>	16.7 (2/12)	8.3 (1/12)	6.7 (3/45)	0 (0/1)	8.1 (3/37)	9.7 (3/31)	0 (0/2)	16.7 (2/12)	10.5 (2/19)	5.3 (2/38)	0 (0/1)	8.6 (6/70)
Hemorrhage	33.3 (4/12)	33.3 (4/12)	13.3 (6/45)	0 (0/1)	13.5 (5/37)	29.0 (9/31)	0 (0/2)	25 (3/12)	10.5 (2/19)	23.7 (9/38)	0 (0/1)	20.0 (14/70)
<i>Emphysema</i>	8.3 (1/12)	8.3 (1/12)	13.3 (6/45)	0 (0/1)	16.2 (6/37)	3.2 (1/31)	50 (1/2)	0 (0/12)	10.5 (2/19)	15.8 (6/38)	0 (0/1)	11.4 (8/70)
<i>Pulmonary calcification</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
<i>Pulmonary lymphangiectasis</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
<i>Trachea; ruminal content</i>	8.3 (1/12)	0 (0/12)	0 (0/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
No macroscopic alterations	8.3 (1/12)	25 (3/12)	8.9 (4/45)	0 (0/1)	13.5 (5/37)	9.7 (3/31)	0 (0/2)	16.7 (2/12)	21.1 (4/19)	5.3 (2/38)	0 (0/1)	11.4 (8/70)
Autolysis	0 (0/12)	0 (0/12)	2.2 (1/45)	100 (1/1)	2.7 (1/37)	0 (0/31)	50 (1/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	100 (1/1)	2.9 (2/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U= not reported.

All circulatory disorders (edema, congestion and hemorrhage) were observed homogeneously distributed in all categories of age group and gender; however, individuals in poor body condition presented higher incidence of edema (71.1%; 27/38) and congestion (52.6%; 20/38). Pneumonia was more frequent in adults than in juvenile and fawns. No pathological findings were observed in 11.4% (8/70) animals.

b) Microscopic findings

Lungs

All histopathological findings are listed in table 4.

I) Hemodynamic Disorders

Pulmonary edema was the most common hemodynamic disorder (95.7%; 57/70). Most cases were moderate with (44.8%; 30/57), the percentage of severe cases was (22.4%; 15/57) (Table 4). All juvenile males with poor body condition displayed this disorder. Pulmonary edema was identified in 71.4% (50/70) of the cases, most frequently moderate (32.0%; 16/50) and severe (28.0%; 14/50) (Table 3). Hemorrhage was diagnosed in (27.1%; 19/70) of the cases. Of those cases, 47.5% (8/19) were classified as moderate. Concomitant congestion and edema were observed in 48 cases. In 9 cases both conditions were considered severe, and in 14 cases hemorrhage was also diagnosed. Thrombosis was observed in three cases (4.3%; 3/70); all three were adults (two males and one female), with two individuals presenting good body condition and one presenting poor body condition.

Table 3 - Severity of major pulmonary hemodynamic disorders in marsh deer

Hemodynamic Disorder Intensity	Congestion (n=67)		Edema (n=50)		Hemorrhage (n=19)	
	N	%	N	%	N	%
<i>Mild</i>	6	9.0	12	24.0	2	10.5
<i>Mild to moderate</i>	11	16.4	1	2.0	4	21.1
<i>Moderate</i>	30	44.8	16	32.0	9	47.4
<i>Moderate to severe</i>	5	7.5	7	14.0	1	5.3
<i>Severe</i>	15	22.4	14	28.0	3	15.8

Table 4 - Lungs: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Congestion</i>	91.7 (11/12)	100 (10/10)	95.6 (43/45)	100 (3/3)	91.9 (34/37)	100 (29/29)	100 (4/4)	94.7 (36/38)	91.7 (11/12)	100 (17/17)	100 (3/3)	95.7 (57/70)
<i>Edema</i>	75 (9/12)	100 (10/10)	66.7 (30/45)	33.3 (1/3)	64.9 (24/37)	86.2 (25/29)	25 (1/4)	81.6 (31/38)	58.3 (7/12)	64.7 (11/17)	33.3 (1/3)	71.4 (50/70)
<i>Pneumonia</i>	75 (9/12)	70 (7/10)	68.9 (32/45)	33.3 (1/3)	64.9 (24/37)	79.3 (23/29)	25 (1/4)	78.9 (30/38)	83.3 (10/12)	41.2 (7/17)	33.3 (1/3)	68.6 (48/70)
<i>Hemorrhage</i>	16.7 (2/12)	0 (0/10)	37.8 (17/45)	0 (0/3)	27 (10/37)	31 (9/29)	0 (0/4)	34.2 (13/38)	41.7 (5/12)	5.9 (1/17)	0 (0/3)	27.1 (19/70)
<i>Emphysema</i>	8.3 (1/12)	10 (1/10)	8.9 (4/45)	0 (0/3)	8.1 (3/37)	10.3 (2/29)	0 (0/4)	5.3 (2/38)	8.3 (1/12)	17.6 (3/17)	0 (0/3)	8.6 (6/70)
<i>Anthracosis</i>	8.3 (1/12)	20 (2/10)	2.2 (1/45)	0 (0/3)	5.4 (2/37)	6.9 (2/29)	0 (0/4)	10.5 (4/38)	0 (0/12)	0 (0/17)	0 (0/3)	5.7 (4/70)
<i>Fibrosis</i>	0 (0/12)	10 (1/10)	4.4 (2/45)	0 (0/3)	0 (0/37)	10.3 (2/29)	0 (0/4)	5.3 (2/38)	0 (0/12)	5.9 (1/17)	0 (0/3)	4.3 (3/70)
<i>Thrombosis</i>	0 (0/12)	0 (0/10)	6.7 (3/45)	0 (0/3)	2.7 (1/37)	6.9 (2/29)	0 (0/4)	5.3 (2/38)	0 (0/12)	5.9 (1/17)	0 (0/3)	4.3 (3/70)
<i>Atelectasia</i>	0 (0/12)	0 (0/10)	4.4 (2/45)	0 (0/3)	2.7 (1/37)	3.4 (1/29)	0 (0/4)	5.3 (2/38)	0 (0/12)	0 (0/17)	0 (0/3)	2.9 (2/70)
<i>TPP</i>	8.3 (1/12)	0 (0/10)	2.2 (1/45)	0 (0/3)	2.7 (1/37)	3.4 (1/29)	0 (0/4)	2.6 (1/38)	8.3 (1/12)	0 (0/17)	0 (0/3)	2.9 (2/70)
<i>NHF</i>	0 (0/12)	0 (0/10)	2.2 (1/45)	0 (0/3)	2.7 (1/37)	0 (0/29)	0 (0/4)	2.6 (1/38)	0 (0/12)	0 (0/17)	0 (0/3)	1.4 (1/70)
<i>Autolysis</i>	8.3 (1/12)	30 (3/10)	6.7 (3/45)	0 (0/3)	2.7 (1/37)	20.7 (6/29)	0 (0/4)	13.2 (5/38)	8.3 (1/12)	5.9 (1/17)	0 (0/3)	10 (7/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U= not reported; TPP= Type II Pneumocyte Proliferation; NHF= No Histopathological Findings.

II) Tissue inflammation and Repair

Inflammatory response was observed in 68.6% (48/70) of the cases. Distribution of inflammatory exudate was categorized into four morphological patterns: luminal (alveolar, bronchiolar and bronchial) 43.8% (21/48), interstitial (septal, bronchovascular and interlobular) 47.9% (23/48), pleural-subpleural 4.2% (2/48), and perivascular 20.8% (10/48). Cellular infiltrates were predominately granulocytic 27.1% (13/48), mononuclear 18.8% (9/48), and 54.2% (26/48) were identified as both granulocytes and mononuclear cells. Multiple nematode larvae were observed in the alveolar lumen of 14 cases, in this cases where observed, congestion, edema, intra-alveolar fibrin deposition, in some cases, infiltrate of histiocytes and multinucleated giant cells phagocytosing the nematodes. Intralesional bacterial colonies were observed in 6 cases, all of these with bronchopneumonia, in 2 cases where observed bacterial embolism (Table 5).

Table 5 - Epidemiological aspects of pneumonia in Brazilian marsh deer from 1995 to 2015

Type of pneumonia	Age group %(Na/Ne)				Gender %(Na/Ne)			Body condition %(Na/Ne)				Total % (Na/Ne)	
	F	J	A	U	Fe	M	U	G	R	P	U	Part.	Gen.
<i>Interstitial pneumonia</i>	44.4 (4/9)	57.1 (4/7)	41.9 (13/31)	100 (1/1)	41.7 (10/24)	47.8 (11/23)	100.0 (1/1)	46.7 (14/30)	40 (4/10)	42.9 (3/7)	100 (1/1)	45.8 (22/48)	31.4 (22/70)
<i>Bronchopneumonia</i>	33.3 (3/9)	28.6 (2/7)	48.4 (15/31)	0 (0/1)	45.8 (11/24)	39.1 (9/23)	0 (0/1)	36.7 (11/30)	50 (5/10)	57.1 (4/7)	0 (0/1)	41.7 (20/48)	28.6 (20/70)
<i>Perivascularitis</i>	22.2 (2/9)	14.3 (1/7)	22.6 (7/31)	0 (0/1)	20.8 (5/24)	21.7 (5/23)	0 (0/1)	20 (6/30)	20 (2/10)	28.6 (2/7)	0 (0/1)	20.8 (10/48)	14.3 (10/70)
<i>Pleuritis</i>	11.1 (1/9)	0 (0/7)	3.2 (1/31)	0 (0/1)	4.2 (1/24)	4.3 (1/23)	0 (0/1)	6.7 (2/30)	0 (0/10)	0 (0/7)	0 (0/1)	4.2 (2/48)	2.9 (2/70)
<i>Bronchitis</i>	0 (0/9)	0 (0/7)	6.5 (2/31)	0 (0/1)	4.2 (1/24)	4.3 (1/23)	0 (0/1)	3.3 (1/30)	0 (0/10)	14.3 (1/7)	0 (0/1)	4.2 (2/48)	2.9 (2/70)
<i>Aspiration pneumonia</i>	0 (0/9)	0 (0/7)	3.2 (1/31)	0 (0/1)	4.2 (1/24)	0 (0/23)	0 (0/1)	3.3 (1/30)	0 (0/10)	0 (0/7)	0 (0/1)	2.1 (1/48)	1.4 (1/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U= not reported; Part= pneumonia cases; Gen= all cases.

Interstitial pneumonia

Interstitial pneumonia was diagnosed in 31.4% (22/70) of the evaluated animals. Five animals presented mainly neutrophilic, three presented lymphocytic and histiocytic and 13 presented mixed infiltrates. The inflammatory response was located mainly in the septal interstitium (22 cases); only two cases showed type II pneumocyte proliferation.

Bronchopneumonia

Bronchopneumonia was present in 28.6% (20/70) of the evaluated cases. It was possible to identify three types of inflammatory infiltrate: suppurative, containing polymorphonuclear leukocytes in alveolar, bronchiolar and bronchial lumen, with occasional leukocytosis and hemodynamic disorders; fibrinosuppurative, containing the same type of polymorphonuclear infiltrate, but with fibrin deposition; and fibrinonecrotic, with poor leukocyte response, fibrin deposition and necrosis of the alveolar wall and associated cellular populations. Intralesional bacterial colonies were observed in six cases, and multiple nematode larvae were diagnosed in four.

Perivasculitis

This inflammatory process was observed in 14.3% (10/70) of the cases. All of them exhibited lymphocytic and plasma cells infiltrates; however, in four cases, granulocytes were observed. On those, concomitant fibrinosuppurative bronchopneumonia was also diagnosed.

Other inflammatory patterns

Pleuritis was observed in two cases (2.8%; 2/70), characterized by pleural and sub-pleural neutrophilic exudate; one with pleural thickening, and fibrin deposition, and the other with proliferated of connective tissue.

Bronchitis was diagnosed in two cases (2.8%; 2/70), in which a secondary inflammatory process was also observed. Aspiration pneumonia was observed in only one case, and it was possible to identify plant material in the alveolar lumen and associated neutrophilic exudate.

Verminous pneumonia was diagnosed in 14 cases (20.0%; 14/70). Histopathological changes were: granulocytic and histiocytic (with giant multinucleated cells) infiltrates, phagocytosis of larvae, marked congestion and edema, and rare emphysema and fibrosis. Bacterial colonies were observed in 6 cases of bronchopneumonia.

III) Miscellaneous processes

Multifocal bronchial and peribronchial anthracosis was observed in four cases. Two were classified as mild and the other two as moderate. Atelectasia was diagnosed in only two cases (adults).

Trachea

Tracheal tissues were evaluated in 16% (9/75) of the cases. Histopathological findings are listed in table 6.

Table 6 - Trachea: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)			Gender % (Na/Ne)		Body Condition % (Na/Ne)		Total % (Na/Ne)
	F	J	A	Fe	M	G	R	
<i>Hemorrhage</i>	0 (0/2)	0 (0/1)	66.7 (4/6)	50 (2/4)	40 (2/5)	33.3 (2/6)	66.7 (2/3)	44.4 (4/9)
<i>Congestion</i>	50 (1/2)	0 (0/1)	16.7 (1/6)	0 (0/4)	40 (2/5)	16.7 (1/6)	33.3 (1/3)	22.2 (2/9)
<i>Tracheitis</i>	0 (0/2)	0 (0/1)	33.3 (2/6)	25 (1/4)	20 (1/5)	16.7 (1/6)	33.3 (1/3)	22.2 (2/9)
<i>NHF</i>	0 (0/2)	100 (1/1)	33.3 (2/6)	50 (2/4)	20 (1/5)	50 (3/6)	0 (0/3)	33.3 (3/9)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular.

I) Hemodynamic disturbances

Hemorrhage was observed in 44.4% (4/9) cases, while congestion in only 22.2% (2/9). Both processes were mainly distributed in the mucosa and submucosa, and were predominantly mild-moderate and moderate.

II) Inflammation and Repair

Tracheitis was identified in two (33.3%; 2/6) adults. A female displayed moderate mixed leukocyte infiltrate (neutrophilic, plasma cells and lymphocytic) associated with hemorrhage, while a male showed a severe neutrophilic exudate with hemorrhage and congestion.

Cardiovascular system

a) Macroscopic changes

Pericardial effusions, hemorrhage and inflammation were the most frequent necropsy findings. Other pathological processes are listed in table 7. Pericardial effusions were reported mainly in fawns and juveniles. Hemorrhage was diagnosed in juveniles with poor body condition and adults with myocarditis. Macroscopic changes were not observed in 34.3% (24/70) of the cases.

Table 7 - Cardiovascular system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Hemopericardium</i>	8.3 (1/12)	8.3 (1/12)	0 (0/45)	0 (0/1)	2.7 (1/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	5.3 (2/38)	0 (0/1)	2.9 (2/70)
<i>Hydropericardium</i>	33.3 (4/12)	33.3 (4/12)	20 (9/45)	0 (0/1)	21.6 (8/37)	29.0 (9/31)	0 (0/2)	33.3 (4/12)	31.6 (6/19)	18.4 (7/38)	0 (0/1)	24.3 (17/70)
Pericardial effusions	41.7 (5/12)	41.7 (5/12)	20 (9/45)	0 (0/1)	24.3 (9/37)	32.3 (10/31)	0 (0/2)	33.3 (4/12)	31.6 (6/19)	23.7 (9/38)	0 (0/1)	27.1 (19/70)
Hemorrhage	8.3 (1/12)	41.7 (5/12)	24.4 (11/45)	0 (0/1)	18.9 (7/37)	32.3 (10/31)	0 (0/2)	25.0 (3/12)	5.3 (1/19)	34.2 (13/38)	0 (0/1)	24.3 (17/70)
<i>Heart, myocarditis</i>	0 (0/12)	0 (0/12)	11.1 (5/45)	0 (0/1)	8.1 (3/37)	6.5 (4/31)	0 (0/2)	8.3 (1/12)	10.5 (2/19)	5.3 (2/38)	0 (0/1)	7.1 (5/70)
<i>Omphaloarteritis / phlebitis</i>	8.3 (1/12)	0 (0/12)	0 (0/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
<i>Pericardium, necrosis</i>	8.3 (1/12)	0 (0/12)	6.7 (3/45)	0 (0/1)	5.4 (1/37)	3.2 (1/31)	50 (1/2)	0 (0/12)	15.8 (3/19)	2.6 (1/38)	0 (0/1)	5.7 (3/70)
Inflammatory response	16.7 (2/12)	0 (0/12)	17.8 (8/45)	0 (0/1)	13.5 (5/37)	12.9 (4/31)	50.0 (1/2)	8.3 (1/12)	26.3 (5/19)	10.5 (4/38)	0 (0/1)	14.3 (10/70)
Cardiomegaly	16.7 (2/12)	0 (0/12)	11.1 (5/45)	0 (0/1)	10.8 (4/37)	9.7 (3/31)	0 (0/2)	16.7 (2/12)	15.8 (3/19)	5.3 (2/38)	0 (0/1)	10 (7/70)
<i>Endocardiosis</i>	16.7 (2/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	6.5 (4/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	5.3 (2/38)	0 (0/1)	4,3 (3/70)
<i>Pericardium, thickening</i>	0 (0/12)	0 (0/12)	4.4 (2/45)	0 (0/1)	5.4 (1/37)	0 (0/31)	0 (0/2)	8.3 (1/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	2.9 (2/70)
<i>Persistent urachus</i>	8.3 (1/12)	0 (0/12)	0 (0/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
Miscellaneous	8.3 (1/12)	0 (0/12)	4.4 (2/45)	0 (0/1)	8.1 (3/37)	0 (0/31)	0 (0/2)	8.3 (1/12)	10.5 (2/19)	0 (0/38)	0 (0/1)	4,3 (3/70)
Autolysis	0 (0/12)	0 (0/12)	2.2 (1/45)	100 (1/1)	2.7 (1/37)	0 (0/31)	50 (1/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	100 (1/1)	2.9 (2/70)
No macroscopic changes	25 (3/12)	33.3 (4/12)	37.8 (17/45)	0 (0/1)	37.8 (14/37)	32.3 (10/31)	0 (0/2)	33.3 (4/12)	36.8 (7/19)	34.2 (13/38)	0 (0/1)	34.3 (24/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported.

b) Microscopic findings

Heart

Of the 41 (54.7%; 41/75) evaluated cases, 68.3% (28/41) did not show any macroscopic changes. Histopathological findings are listed in table 7.

I) Hemodynamic disturbances

Hemorrhage was observed in 19.5% (8/41) of the cases, while congestion was observed in 4.9% (2/41), distributed in the myocardium and pericardium. Lesions were predominantly moderate (hemorrhagic in five cases). Hemorrhage was more frequent in adults than in fawns and juveniles.

II) Cell injury, death

Necrosis of cardiomyocytes was identified in only one case.

Table 8 - Heart: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
Hemorrhage	0 (0/6)	20 (1/5)	17.9 (5/28)	100 (2/2)	10.5 (2/19)	20 (4/20)	100 (2/2)	13 (3/23)	20 (1/5)	18.2 (2/11)	100 (2/2)	19.5 (8/41)
Congestion	0 (0/6)	20 (1/5)	3.6 (1/28)	0 (0/2)	10.5 (2/19)	0 (0/20)	0 (0/2)	4.3 (1/23)	0 (0/5)	9.1 (1/11)	0 (0/2)	4.9 (2/41)
Necrosis	0 (0/6)	0 (0/5)	0 (0/28)	50 (1/2)	0 (0/19)	0 (0/20)	50 (1/2)	0 (0/23)	0 (0/5)	0 (0/11)	50 (1/2)	2.4 (1/41)
NHF	83.3 (5/6)	80 (4/5)	67.9 (19/28)	0 (0/2)	63.2 (12/19)	80 (16/20)	0 (0/2)	82.6 (19/23)	60 (3/5)	54.5 (6/11)	0 (0/2)	68.3 (28/41)
Autolysis	16.7 (1/6)	0 (0/5)	7.1 (2/28)	0 (0/2)	15.8 (3/19)	0 (0/20)	0 (0/2)	4.3 (1/23)	0 (0/5)	18.2 (2/11)	0 (0/2)	7.3 (3/41)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

Alimentary tract, liver and pancreas

a) Macroscopic changes

Inflammation, more predominately enteritis (30.0%; 21/70), congestion and hemorrhage were the most frequent necropsy findings; other pathologic processes are listed in table 9. No macroscopic lesions were observed in 18.6% (13/70) of the cases.

Table 9 - Alimentary system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Oral cavity</i>	0 (0/12)	0 (0/12)	8.9 (4/45)	0 (0/1)	5.4 (2/37)	6.5 (2/31)	0 (0/2)	16.7 (2/12)	5.3 (1/19)	2.6 (1/38)	0 (0/1)	5.7 (4/70)
<i>Forestomachs</i>	0 (0/12)	8.3 (1/12)	4.4 (2/45)	0 (0/1)	8.1 (3/37)	0 (0/31)	0 (0/2)	0 (0/12)	10.5 (2/19)	2.6 (1/38)	0 (0/1)	4.3 (3/70)
<i>Intestine</i>	25 (3/12)	33.3 (4/12)	31.1 (14/45)	0 (0/1)	24.3 (9/37)	35.5 (11/31)	50 (1/2)	25 (3/12)	31.6 (6/19)	31.6 (12/38)	0 (0/1)	30 (21/70)
<i>Liver</i>	0 (0/12)	0 (0/12)	8.9 (4/45)	0 (0/1)	8.1 (3/37)	3.2 (1/31)	0 (0/2)	8.3 (1/12)	5.3 (1/19)	5.3 (2/38)	0 (0/1)	5.7 (4/70)
Necrosis, inflammatory response	25 (3/12)	41.7 (5/12)	42.2 (19/45)	0 (0/1)	37.8 (14/37)	38.7 (12/31)	50 (1/2)	50 (6/12)	47.4 (9/19)	31.6 (12/38)	0 (0/1)	38.5 (27/70)
<i>Forestomachs</i>	0 (0/12)	25 (3/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	9.7 (3/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	7.9 (3/38)	0 (0/1)	5.7 (4/70)
<i>Intestine</i>	8.3 (1/12)	25 (3/12)	8.9 (4/45)	0 (0/1)	10.8 (4/37)	12.9 (4/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	18.4 (7/38)	0 (0/1)	11.4 (8/70)
<i>Liver</i>	16.7 (2/12)	33.3 (4/12)	11.1 (5/45)	0 (0/1)	21.6 (8/37)	9.7 (3/31)	0 (0/2)	8.3 (1/12)	21.1 (4/19)	15.8 (6/38)	0 (0/1)	15.7 (11/70)
Congestion	33.3 (4/12)	66.7 (8/12)	22.2 (10/45)	0 (0/1)	35.1 (13/37)	29.0 (9/31)	0 (0/2)	16.7 (2/12)	31.6 (6/19)	36.8 (14/38)	0 (0/1)	31.4 (22/70)
<i>Forestomach</i>	0 (0/12)	0 (0/12)	4.4 (2/45)	0 (0/1)	5.4 (2/37)	0 (0/31)	0 (0/2)	8.3 (1/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	2.9 (2/70)
<i>Intestine</i>	8.3 (1/12)	8.3 (1/12)	0 (0/45)	0 (0/1)	0 (0/37)	6.5 (2/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	2.9 (2/70)
<i>Liver</i>	25 (3/12)	25.0 (3/12)	13.3 (6/45)	0 (0/1)	16.2 (6/37)	19.4 (6/31)	0 (0/2)	8.3 (1/12)	15.8 (3/19)	21.1 (8/38)	0 (0/1)	17.1 (12/70)
<i>Pancreas</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
Hemorrhage	50 (6/12)	25 (3/12)	15.6 (7/45)	0 (0/1)	24.3 (9/37)	22.6 (7/31)	0 (0/2)	25 (3/12)	15.8 (3/19)	26.3 (10/38)	0 (0/1)	22.9 (16/70)
Hepatomegaly	33.3 (4/12)	8.3 (1/12)	8.9 (4/45)	0 (0/1)	18.9 (7/37)	6.5 (2/31)	0 (0/2)	16.7 (2/12)	10.5 (2/19)	13.2 (5/38)	0 (0/1)	12.9 (9/70)
<i>Lingual cyanosis</i>	16.7 (2/12)	0 (0/12)	4.4 (2/45)	0 (0/1)	2.7 (1/37)	9.7 (3/31)	0 (0/2)	8.3 (1/12)	5.3 (1/19)	5.3 (2/38)	0 (0/1)	5.7 (4/70)
<i>Ruminal bloat</i>	16.7 (2/12)	8.3 (1/12)	0 (0/45)	0 (0/1)	2.7 (1/37)	6.5 (2/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	5.3 (2/38)	0 (0/1)	4.3 (3/70)
<i>Tongue, ulcer</i>	0 (0/12)	16.7 (2/12)	2.2 (1/45)	0 (0/1)	5.4 (2/37)	3.2 (1/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	5.3 (2/38)	0 (0/1)	4.3 (3/70)
<i>Liver, fibrosis</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
<i>Liver, infarct</i>	8.3 (1/12)	0 (0/12)	0 (0/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
<i>Liver, pale</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
<i>Mesentery, edema</i>	8.3 (1/12)	0 (0/12)	0 (0/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
Miscellaneous	33.3 (4/12)	25.0 (3/12)	6.7 (3/45)	0 (0/1)	10.8 (4/37)	19.4 (6/31)	0 (0/2)	16.7 (2/12)	5.3 (1/19)	18.4 (7/38)	0 (0/1)	14.3 (10/70)
No macroscopic alterations	16.7 (2/12)	8.3 (1/12)	22.2 (10/45)	0 (0/1)	21.6 (8/37)	16.1 (5/31)	0 (0/2)	16.7 (2/12)	26.3 (5/19)	15.8 (6/38)	0 (0/1)	18.6 (13/70)
Autolysis	0 (0/12)	0 (0/12)	4.4 (2/45)	100 (1/1)	5.4 (2/37)	0 (0/31)	50.0 (1/2)	0 (0/12)	5.3 (1/19)	2.6 (1/38)	100 (1/1)	4.3 (3/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported.

Hepatitis and stomatitis were diagnosed in 5.7% (4/70) cases, only in adults; while forestomach inflammation was observed in 4.3% (3/70). Congestion of the liver (15.7%; 11/70), intestines (11.4%; 8/70) and forestomach (5.7%; 4/70) was diagnosed only in adults. Hepatic hemorrhage was identified in 17.1% (12/70) cases. Pancreatic, intestinal and forestomach hemorrhage were scarcely reported. Hepatomegaly was observed in 12.9% (9/70) of the cases; four of those were in fawns (33.3%; 4/9).

b) Microscopic findings

Tongue

Histopathological changes were not observed in 73.3% (11/15) of the 15 (20.0%; 15/75) evaluated cases. Histopathological findings are listed in table 10. Histopathological changes were observed only in adults, but homogeneously distributed between gender and body condition, and severity (discrete to moderate). Glossitis was diagnosed in 16.7% (1/15) of the cases, hemorrhage in 6.7% and microvesicles in 6.7% (1/15).

Table 10 - Tongue: histopathological findings, biological and epidemiological aspects in marsh deer

Histopath findings	Age group % (Na/Ne)			Gender % (Na/Ne)		Body Condition % (Na/Ne)			Total % (Na/Ne)
	F	J	A	Fe	M	G	R	P	
<i>Glossitis</i>	0 (0/1)	0 (0/4)	40 (4/10)	37.5 (3/8)	14.3 (1/7)	25 (2/8)	50 (1/2)	20 (1/5)	26.7 (4/15)
<i>Hemorrhage</i>	0 (0/1)	0 (0/4)	10 (1/10)	0 (0/8)	14.3 (1/7)	12.5 (1/8)	0 (0/2)	0 (0/5)	6.7 (1/15)
<i>NHF</i>	100 (1/1)	100 (4/4)	60 (6/10)	62.5 (5/8)	85.7 (6/7)	75 (6/8)	50 (1/2)	80 (4/5)	73.3 (11/15)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported.

Rumen

We evaluated 14 (18.7%; 14/75) cases; histopathological findings were not observed in 64.3% (9/14) of them. Histopathological results are listed in table 11. Edema of the lamina propria and rumenitis were the only findings. Rumenitis was

diagnosed in a male fawn in poor body condition. It was a moderate-severe necrotic process, characterized by diffuse mixed leukocyte exudate, with multifocal intralesional yeasts and pseudo hyphae. PAS and Grocott stains were used to further evaluate fungal structures.

Table 11 - Rumen: histopathological findings, biological and epidemiological aspects in marsh deer

Histopath findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Edema</i>	25 (1/4)	100 (1/1)	0 (0/7)	0 (0/2)	16.7 (1/6)	16.7 (1/6)	0 (0/2)	25 (2/8)	0 (0/3)	0 (0/1)	0 (0/2)	14.3 (2/14)
<i>Rumenitis</i>	25 (1/4)	0 (0/1)	0 (0/7)	0 (0/2)	0 (0/6)	16.7 (1/6)	0 (0/2)	0 (0/8)	33.3 (1/3)	0 (0/1)	0 (0/2)	7.1 (1/14)
<i>NHF</i>	25 (1/4)	0 (0/1)	85.7 (6/7)	100 (2/2)	66.7 (4/6)	50 (3/6)	100 (2/2)	50 (4/8)	66.7 (2/3)	100 (1/1)	100 (2/2)	64.3 (9/14)
<i>Autolysis</i>	25 (1/4)	0 (0/1)	14.3 (1/7)	100 (2/2)	16.7 (1/6)	16.7 (1/6)	100 (2/2)	25 (2/8)	0 (0/3)	0 (0/1)	100 (2/2)	14.3 (2/14)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

Reticulum

Thirteen (17.3%; 13/75) cases were evaluated; histopathological results are listed in table 12. Of those, 69.2% (9/13) shown no histopathological changes. Edema and reticulitis were the only findings.

Table 12 - Reticulum: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Congestion</i>	0 (0/2)	0 (0/2)	12.5 (1/8)	0 (0/1)	0 (0/7)	20 (1/5)	0 (0/1)	14.3 (1/7)	0 (0/2)	0 (0/3)	0 (0/1)	7.7 (1/13)
<i>Edema</i>	50 (1/2)	0 (0/2)	12.5 (1/8)	0 (0/1)	14.3 (1/7)	20 (1/5)	0 (0/1)	28.6 (2/7)	0 (0/2)	0 (0/3)	0 (0/1)	15.4 (2/13)
<i>Reticulitis</i>	0 (0/2)	0 (0/2)	0 (0/8)	100 (1/1)	0 (0/7)	0 (0/5)	100 (1/1)	0 (0/7)	0 (0/2)	0 (0/3)	100 (1/1)	7.7 (1/13)
<i>NHF</i>	50 (1/2)	100 (2/2)	75 (6/8)	0 (0/1)	85.7 (6/7)	60 (3/5)	0 (0/1)	57.1 (4/7)	100 (2/2)	100 (3/3)	0 (0/1)	69.2 (9/13)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

Omasum

Nine 12% (9/75) cases were evaluated; histopathological changes were not observed in 88.9% (8/9) of them.

Abomasum

We evaluated 15 (20.0%; 15/75) cases; 80.0% (12/15) of them showed no histopathological changes. Histopathological findings are listed in table 13. Two females were diagnosed with hemorrhagic abomasitis. Congestion was observed in two animals, and hemorrhage in only one juvenile female.

Table 13 - Abomasum: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Abomasitis</i>	0 (0/5)	33.3 (1/3)	20 (1/5)	0 (0/2)	33.3 (2/6)	0 (0/7)	0 (0/2)	16.7 (1/6)	20 (1/5)	0 (0/2)	0 (0/2)	13.3 (2/15)
<i>Congestion</i>	0 (0/5)	33.3 (1/3)	20 (1/5)	0 (0/2)	16.7 (1/6)	14.3 (1/7)	0 (0/2)	33.3 (2/6)	0 (0/5)	0 (0/2)	0 (0/2)	13.3 (2/15)
<i>Hemorrhage</i>	0 (0/5)	33.3 (1/3)	0 (0/5)	0 (0/2)	16.7 (1/6)	0 (0/7)	0 (0/2)	16.7 (1/6)	0 (0/5)	0 (0/2)	0 (0/2)	6.7 (1/15)
<i>NHF</i>	100 (5/5)	66.7 (2/3)	60 (3/5)	100 (2/2)	66.7 (4/6)	85.7 (6/7)	100 (2/2)	66.7 (4/6)	80 (4/5)	100 (2/2)	100 (2/2)	80 (12/15)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

Small intestines

We evaluated 18 (24.0%; 18/75) cases and found no histopathological changes in 44.4% (8/18) of them. Histopathological results are listed in table 14. Three adults (30%) and one juvenile (33.3%) were diagnosed as severe enteritis. One juvenile female was diagnosed with small intestine congestion.

Table 14 - Small intestine: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)			Total % (Na/Ne)
	F	J	A	U	Fe	Male	U	G	P	U	
<i>Congestion</i>	0 (0/2)	33.3 (1/3)	0 (0/10)	0 (0/3)	20 (1/5)	0 (0/10)	0 (0/3)	9.1 (1/11)	0 (0/4)	0 (0/3)	5.6 (1/18)
<i>Enteritis</i>	0 (0/2)	33.3 (1/3)	30 (3/10)	0 (0/3)	40 (2/5)	20 (2/10)	0 (0/3)	27.3 (3/11)	25 (1/4)	0 (0/3)	22.2 (4/18)
<i>NHF</i>	100 (2/2)	0 (0/3)	30 (3/10)	100 (3/3)	20 (1/5)	40 (4/10)	100 (3/3)	36.4 (4/11)	25 (1/4)	100 (3/3)	44.4 (8/18)
<i>Autolysis</i>	0 (0/2)	33.3 (1/3)	40 (4/10)	0 (0/3)	20 (1/5)	40 (4/10)	0 (0/3)	27.3 (3/11)	50 (2/4)	0 (0/3)	27.8 (5/18)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; U=not reported; NHF=no histopathological findings.

Large intestine

Only three cases 4% (3/75), were evaluated. Histopathological results are listed in table 15. Moderate to severe colitis was present in two females and one male. Hemorrhage was diagnosed in two females, while congestion was observed in a third female.

Table 15 - Large intestine: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)		Gender % (Na/Ne)		Body Condition % (Na/Ne)			Total % (Na/Ne)
	J	A	Fe	M	G	R	P	
<i>Congestion</i>	0 (0/1)	50 (1/2)	50 (1/2)	0 (0/1)	0 (0/1)	100 (1/1)	0 (0/1)	33.3 (1/3)
<i>Hemorrhage</i>	100 (1/1)	50 (1/2)	100 (2/2)	0 (0/1)	0 (0/1)	100 (1/1)	100 (1/1)	66.7 (2/3)
<i>Colitis</i>	100 (1/1)	100 (1/2)	100 (2/2)	100 (1/1)	100 (1/1)	100 (1/1)	100 (1/1)	100 (3/3)

Na=n° affected; Ne=n° examined; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

Liver

A total of 59 (78.7%; 59/75) cases were analyzed. Histopathological changes were not observed in 5.1% (3/59) of them. All histopathological changes are noted in table 16.

The most frequently observed hemodynamic disorders were hemorrhage (39.0%; 23/59) and congestion (17.1%; 16/59), most commonly of moderate intensity and portal, midzonal and centrilobular distribution (table 17). Hepatitis was the main histopathological finding in the liver, homogeneously distributed into all epidemiological categories (age group, gender, and body condition). Distribution of the inflammatory response was classified into four morphological patterns: portal (portal, ductal, periportal), mid zonal, centrilobular, and a combination of the previous three categories. Portal hepatitis was observed in 72.4% (21/29) of the cases, portal-midzonal in 10.3% (3/29), midzonal-centrilobular in 3.4% (1/29), and generalized in 13.8% (2/29). Regarding leukocyte exudates, 65.5% (19/29) were identified as mononuclear, 13.8% (4/29) as granulocytic, and 20.7% (6/29) as both granulocytes and mononuclear. Intensity was classified as follows: 41.4% (12/29) moderate, 31.0% (9/29) mild, 24.1% (7/29) mild-moderate, and 3.4% (1/29) severe.

Table 16 - Liver: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Hepatitis</i>	45.5 (5/11)	62.5 (5/8)	51.3 (19/37)	0 (0/3)	51.5 (17/33)	52.2 (12/23)	0 (0/3)	53.1 (17/32)	50 (5/10)	50 (7/14)	0 (0/3)	49.2 (29/59)
<i>Steatosis</i>	45.5 (5/11)	62.5 (5/8)	37.8 (14/37)	66.7 (2/3)	48.5 (16/33)	34.8 (8/23)	66.7 (2/3)	46.9 (15/32)	30 (3/10)	42.9 (6/14)	66.7 (2/3)	44.1 (26/59)
<i>Hemorrhage</i>	36.6 (4/11)	50 (4/8)	40.5 (15/37)	0 (0/3)	45.5 (15/33)	34.8 (8/23)	0 (0/3)	46.9 (15/32)	30 (3/10)	35.7 (5/14)	0 (0/3)	39 (23/59)
<i>Congestion</i>	18.2 (2/11)	25 (2/8)	27 (10/37)	66.7 (2/3)	21.2 (7/33)	30.4 (7/23)	66.7 (2/3)	25 (8/32)	30 (3/10)	21.4 (3/14)	66.7 (2/3)	27.1 (16/59)
<i>Hepatic necrosis</i>	27.3 (3/11)	62.5 (5/8)	10.8 (4/37)	0 (0/3)	12.1 (4/33)	34.8 (8/23)	0 (0/3)	25 (8/32)	0 (0/10)	28.6 (4/14)	0 (0/3)	20.3 (12/59)
<i>Bile duct hyperplasia</i>	9.1 (1/11)	12.5 (1/8)	21.6 (8/37)	0 (0/3)	21.2 (7/33)	13 (3/23)	0 (0/3)	15.6 (5/32)	20 (2/10)	21.4 (3/14)	0 (0/3)	16.9 (10/59)
<i>Cholestasis</i>	0 (0/11)	12.5 (1/8)	5.4 (2/37)	0 (0/3)	6.1 (2/33)	4.3 (1/23)	0 (0/3)	3.1 (1/32)	0 (0/10)	14.3 (2/14)	0 (0/3)	5.1 (3/59)
<i>Hemosiderosis</i>	9.1 (1/11)	0 (0/8)	2.7 (1/37)	0 (0/3)	3 (1/33)	4.3 (1/23)	0 (0/3)	3.1 (1/32)	0 (0/10)	7.1 (2/14)	0 (0/3)	3.4 (2/59)
<i>NHF</i>	0 (0/11)	0 (0/8)	5.4 (2/37)	33.3 (1/3)	3 (1/33)	4.3 (1/23)	33.3 (1/3)	6.3 (2/32)	0 (0/10)	0 (0/14)	33.3 (1/3)	5.1 (3/59)
<i>Autolysis</i>	27.3 (3/11)	12.5 (1/8)	8.1 (3/37)	0 (0/3)	12.1 (4/33)	13 (3/23)	0 (0/3)	12.5 (4/32)	0 (0/10)	21.4 (3/14)	0 (0/3)	11.9 (7/59)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

Table 17 - Severity and morphological patterns of major hepatic hemodynamic disorders diagnosed in marsh deer

Hemodynamic disturbance	Morphological pattern	Intensity					Total	
		I	II	III	IV	V	N	%
Hemorrhage (n=23)	<i>Portal</i>	0	0	0	0	0	0	0.0
	<i>Midzonal</i>	0	0	2	0	0	2	8.7
	<i>Centrilobular</i>	0	0	0	0	0	0	0.0
	<i>Portal and midzonal</i>	0	1	0	0	1	2	8.7
	<i>Midzonal and centrilobular</i>	1	0	2	0	0	3	13.0
	<i>Generalized</i>	3	2	7	2	2	18	69.6
Congestion (n=16)	<i>Portal</i>	0	0	0	0	0	0	0.0
	<i>Midzonal</i>	1	1	0	0	0	2	12.5
	<i>Centrilobular</i>	0	0	0	0	0	0	0.0
	<i>Portal and midzonal</i>	0	0	1	0	0	1	6.3
	<i>Midzonal and centrilobular</i>	0	0	0	0	0	0	0.0
	<i>Generalized</i>	2	1	8	2	0	13	81.3

I=mild; II=mild-moderate; III=moderate; IV=moderate-severe; V=severe

III) Miscellaneous

Steatosis was the second most commonly diagnosed histopathological finding observed in the liver, homogeneously distributed into all epidemiological categories (age group, gender, and body condition), classified as macrovesicular (58.6%; 17/27), microvesicular (17.2%; 5/27) and a combination of these two conditions (13.8%; 4/27). Steatosis intensity was classified as follows: 44.8% (13/27) moderate, 10.7% (6/27) mild, 10.3% (3/27) mild-moderate, and 6.9% (2/27) moderate-severe and severe. Hepatic necrosis was diagnosed in 20.3% of the cases, and bile duct hyperplasia in 16.9%.

Pancreas

Only 5.3% (4/75) of the cases were analyzed. No histopathological findings were observed.

Urinary system

a) Macroscopic changes

Hemorrhage, necrotic-inflammatory response and nephrosis were the most frequent necropsy findings. Other pathological processes are listed in table 18. Macroscopic changes were not observed in 30.0% of the evaluated cases.

Table 18 - Urogenital system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic alterations	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Kidney</i>	33.3 (4/12)	16.7 (2/12)	15.6 (7/45)	0 (0/1)	10.8 (4/37)	25.8 (8/31)	50 (1/2)	25.0 (3/12)	5.3 (1/19)	23.7 (9/38)	0 (0/1)	18.6 (13/70)
<i>Urinary bladder</i>	8.3 (1/12)	8.3 (1/12)	0 (0/45)	0 (0/1)	0 (0/37)	6.5 (2/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	2.9 (2/70)
<i>Genital</i>	0 (0/12)	8.3 (1/12)	4.4 (2/45)	0 (0/1)	8.1 (3/37)	0 (0/31)	0 (0/2)	0 (0/12)	10.5 (2/19)	2.6 (1/38)	0 (0/1)	4.3 (3/70)
Hemorrhage	41.7 (5/12)	33.3 (4/12)	20 (9/45)	0 (0/1)	18.9 (7/37)	32.3 (10/31)	50 (1/2)	33.3 (4/12)	15.8 (3/19)	28.9 (11/38)	0 (0/1)	25.7 (18/70)
<i>Genital</i>	0 (0/12)	8.3 (1/12)	11.1 (5/45)	0 (0/1)	10.8 (4/37)	6.5 (2/31)	0 (0/2)	0 (0/12)	15.8 (3/19)	7.9 (3/38)	0 (0/1)	8.6 (6/70)
<i>Urinary bladder</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
<i>Kidney</i>	16.7 (2/12)	25 (3/12)	13.3 (6/45)	0 (0/1)	10.8 (4/37)	22.6 (7/31)	0 (0/2)	25 (3/12)	21.1 (4/19)	10.5 (4/38)	0 (0/1)	15.7 (11/70)
Necrosis, inflammatory response	16.7 (2/12)	33.3 (4/12)	24.4 (11/45)	0 (0/1)	21.6 (8/37)	29 (9/31)	0 (0/2)	25.0 (3/12)	36.8 (7/19)	18.4 (7/38)	0 (0/1)	24.3 (17/70)
<i>Nephrosis</i>	50 (6/12)	25 (3/12)	15.6 (7/45)	0 (0/1)	24.3 (9/37)	22.6 (7/31)	0 (0/2)	41.7 (5/12)	26.3 (5/19)	15.8 (6/38)	0 (0/1)	22.9 (16/70)
<i>Kidney</i>	16.7 (2/12)	16.7 (2/12)	11.1 (5/45)	0 (0/1)	16.2 (6/37)	9.7 (3/31)	0 (0/2)	8.3 (1/12)	10.5 (2/19)	15.8 (6/38)	0 (0/1)	12.9 (9/70)
<i>Urinary bladder</i>	8.3 (1/12)	16.7 (2/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	9.7 (3/31)	0 (0/2)	0 (0/12)	0 (0/19)	10.5 (4/38)	0 (0/1)	5.7 (4/70)
Congestion	25 (3/12)	33.3 (4/12)	13.3 (6/45)	0 (0/1)	18.9 (7/37)	19.4 (6/31)	0 (0/2)	8.3 (1/12)	10.5 (2/19)	26.3 (10/38)	0 (0/1)	18.6 (13/70)
<i>Kidney, infarct</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
<i>Urinary bladder, rupture</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
<i>Uterus, dilatation</i>	16.7 (2/12)	0 (0/12)	0 (0/45)	0 (0/1)	5.4 (2/37)	0 (0/31)	0 (0/2)	0 (0/12)	10.5 (2/19)	0 (0/38)	0 (0/1)	2.9 (2/70)
Miscellaneous	16.7 (2/12)	0 (0/12)	4.4 (2/45)	0 (0/1)	5.4 (2/37)	6.5 (2/31)	0 (0/2)	0 (0/12)	15.8 (3/19)	2.6 (1/38)	0 (0/1)	5.7 (4/70)
No macroscopic alterations	25 (3/12)	16.7 (2/12)	35.6 (16/45)	0 (0/1)	29.7 (11/37)	32.3 (10/31)	0 (0/2)	25.0 (3/12)	26.3 (5/19)	34.2 (13/38)	0 (0/1)	30.0 (21/70)
Autolysis	0 (0/12)	0 (0/12)	4.4 (2/45)	100 (1/1)	5.4 (2/37)	0 (0/31)	50 (1/2)	0 (0/12)	5.3 (1/19)	2.6 (1/38)	100 (1/1)	4.3 (3/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

b) Microscopic findings

Kidney

We analyzed 66 (88.0%; 66/75) cases. Histopathological results are listed in table 19.

I) Hemodynamic Disorders

Congestion was classified into 3 categories, according to its distribution: corticomedullary (47.8%; 22/46), medullary (41.3%; 19/46), and cortical (10.9%; 5/46). Hemodynamic disorders were also classified according to intensity: mild (10.9%; 5/46), mild-moderate (17.4%; 8/46), moderate (56.5%; 26/46), moderate-severe (10.9%; 5/46), and severe (4.3%; 2/46). Renal congestion had an incidence higher than 60.0% in all epidemiological categories (12.1%; 8/66).

II) Inflammation and Repair

Interstitial nephritis was the most common morphological pattern of inflammatory response (25.8%). Its occurrence ranged from 8% to 30% in all categories. Leukocyte infiltrates were classified as mononuclear 76.5% (13/17), granulocytic 17.6% (3/17), and mixed 5.9% (1/17). Glomerulonephritis was present in 10.6% (7/66) of the cases. PAS stain was used to diagnose membranous glomerulonephritis.

III) Miscellaneous

Degenerative changes, such as intracytoplasmic vacuoles in the renal tubular epithelium (39.4%), presence of casts (10.6%; 7/66), and tubular calcification (6.1%; 4/66). Acute tubular necrosis was found in 13.6% (9/66) of the evaluated cases.

Urinary bladder

A total of 16 (21.3%; 16/75) cases were evaluated. Congestion was present in four and cystitis in only one case. Histopathological results are listed in table 20.

Table 19 - Kidney: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Congestion</i>	63.6 (7/11)	63.6 (7/11)	71.4 (30/42)	100 (02/2)	68.6 (24/35)	67.9 (19/28)	100 (3/3)	71.4 (25/35)	72.7 (8/11)	61.1 (11/18)	100 (02/2)	69.7 (46/66)
<i>Tubular degeneration</i>	36.4 (4/11)	36.4 (4/11)	42.9 (18/42)	0 (0/2)	37.1 (13/35)	42.9 (12/28)	33.3 (1/3)	34.3 (12/35)	45.5 (5/11)	50 (9/18)	0 (0/2)	39.4 (26/66)
<i>Nephritis</i>	9.1 (1/11)	18.2 (2/11)	33.3 (14/42)	0 (0/2)	28.6 (10/35)	25 (7/28)	0 (0/3)	28.6 (10/35)	18.2 (2/11)	27.8 (5/18)	0 (0/2)	25.8 (17/66)
<i>Acute tubular necrosis</i>	0 (0/11)	18.2 (2/11)	16.7 (7/42)	0 (0/2)	17.1 (6/35)	10.7 (3/28)	0 (0/3)	8.6 (3/35)	18.2 (2/11)	22.2 (4/18)	0 (0/2)	13.6 (9/66)
<i>Hemorrhage</i>	18.2 (2/11)	0 (0/11)	14.3 (6/42)	0 (0/2)	8.6 (3/35)	17.9 (5/28)	0 (0/3)	14.3 (5/35)	9.1 (1/11)	11.1 (2/18)	0 (0/2)	12.1 (8/66)
<i>Cylinders</i>	9.1 (1/11)	0 (0/11)	14.3 (6/42)	0 (0/2)	11.4 (4/35)	10.7 (3/28)	0 (0/3)	8.6 (3/35)	18.2 (2/11)	11.1 (2/18)	0 (0/2)	10.6 (7/66)
<i>Glomerulonephritis</i>	9.1 (1/11)	18.2 (2/11)	9.5 (4/42)	0 (0/2)	17.1 (6/35)	3.6 (1/28)	0 (0/3)	11.4 (4/35)	9.1 (1/11)	11.1 (2/18)	0 (0/2)	10.6 (7/66)
<i>Tubular calcification</i>	0 (0/11)	9.1 (1/11)	7.1 (3/42)	0 (0/2)	8.6 (3/35)	3.6 (1/28)	0 (0/3)	2.9 (1/35)	0 (0/11)	16.7 (3/18)	0 (0/2)	6.1 (4/66)
<i>Leukocytosis</i>	0 (0/11)	0 (0/11)	0 (30/42)	50 (1/2)	0 (0/35)	0 (0/28)	33.3 (1/3)	0 (0/35)	0 (0/11)	0 (0/18)	50 (1/2)	1.5 (1/66)
<i>NHF</i>	0 (0/11)	9.1 (1/11)	4.8 (2/42)	0 (0/2)	8.6 (3/35)	0 (0/28)	0 (0/3)	5.7 (2/35)	0 (0/11)	5.6 (1/18)	0 (0/2)	4.5 (3/66)
<i>Autolysis</i>	45.5 (5/11)	18.2 (2/11)	16.7 (7/42)	0 (0/2)	20 (7/35)	25 (7/28)	0 (0/3)	25.7 (9/35)	18.2 (2/11)	16.7 (3/18)	0 (0/2)	21.2 (14/66)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

Table 20 - Urinary bladder: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	Juvenile	A	U	Fe	M	U	G	R	P	U	
<i>Congestion</i>	50 (1/2)	50 (1/2)	18.2 (2/11)	0 (0/1)	28.6 (2/7)	25 (2/8)	0 (0/1)	33.3 (3/9)	25 (1/3)	0 (0/2)	0 (0/1)	25 (4/16)
<i>Cystitis</i>	0 (0/2)	0 (0/2)	9.1 (1/11)	0 (0/1)	0 (0/7)	12.5 (1/8)	0 (0/1)	11.1 (1/9)	0 (0/3)	0 (0/2)	0 (0/1)	6.3 (1/16)
<i>NHF</i>	50 (1/2)	50 (1/2)	72.7 (8/11)	100 (1/1)	71.4 (5/7)	62.5 (5/8)	100 (1/1)	55.6 (5/9)	75 (2/3)	100 (2/2)	100 (1/1)	68.8 (11/16)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF=no histopathological findings.

Genital system

Pathological processes are listed in table 18. Macroscopic changes were not observed in 30.0% (21/70) of the evaluated cases. No histopathological findings were observed in the evaluated tissues: testicles (6.7%; 5/75), uterus (8.0%; 6/75) and ovaries (2.7%; 2/75).

Endocrine system

a) Macroscopic changes

Megaly, congestion and edema were the most frequent necropsy findings. Other pathological processes are listed in table 21. Macroscopic changes were not observed in 85.7% (60/70) of the cases.

Table 21 - Endocrine system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
Adrenal megaly	8.3 (1/12)	0 (0/12)	6.7 (3/45)	0 (0/1)	5.4 (2/37)	6.5 (2/31)	0 (0/2)	16.7 (2/12)	5.3 (1/19)	2.6 (1/38)	0 (0/1)	5.7 (4/70)
Adrenal congestion	8.3 (1/12)	8.3 (1/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	6.5 (2/31)	0 (0/2)	16.7 (2/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	4.3 (3/70)
Adrenal edema	8.3 (1/12)	0 (0/12)	4.4 (2/45)	0% (0/1)	5.4 (2/37)	3.2 (1/31)	0 (0/2)	16.7 (2/12)	5.3 (1/19)	0 (0/38)	0% (0/1)	4.3 (3/70)
Adrenal hemorrhage	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
Adrenal necrosis	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
No macroscopic alterations	91.7 (11/12)	91.7 (11/12)	84.4 (38/45)	0 (0/1)	89.2 (3/37)	83.9 (26/31)	50 (1/2)	83.3 (10/12)	89.5 (17/19)	86.8 (33/38)	0 (0/1)	85.7 (60/70)
Autolysis	0 (0/12)	0 (0/12)	4.4 (2/45)	100 (1/1)	5.4 (2/37)	0 (0/31)	50 (1/2)	0 (0/12)	5.3 (1/19)	2.6 (1/38)	100 (1/1)	4.3 (3/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor, U= not reported.

b) Microscopic findings

Adrenal glands

We evaluated 15 (20%; 15/75) cases. Histopathological findings are listed in table 22. Six animals presented congestion and hemorrhage, mainly cortical (reticular and fascicular layers).

Table 22 - Adrenal glands: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)			Gender % (Na/Ne)		Body Condition % (Na/Ne)			Total % (Na/Ne)
	F	J	A	Fe	M	G	R	P	
<i>Congestion</i>	0 (0/1)	33.3 (1/3)	45.5 (5/11)	71.4 (4/7)	12.5 (1/8)	28.6 (2/7)	40 (2/5)	66.7 (2/3)	40 (6/15)
<i>Hemorrhage</i>	0 (0/1)	33.3 (1/3)	45.5 (5/11)	28.6 (2/7)	50 (4/8)	57.1 (4/7)	20 (1/5)	33.3 (1/3)	40 (6/15)
<i>Degeneration</i>	100 (1/1)	0 (0/3)	0 (0/11)	0 (0/7)	12.5 (1/8)	0 (0/7)	20 (1/5)	0 (0/3)	6.7 (1/15)
<i>Leukocytosis</i>	0 (0/1)	0 (0/3)	9.1 (1/11)	14.3 (1/7)	0 (0/8)	0 (0/7)	0 (0/5)	33.3 (1/3)	6.7 (1/15)
<i>Adrenatitis</i>	0 (0/1)	0 (0/3)	9.1 (1/11)	0 (0/7)	12.5 (1/8)	14.3 (1/7)	0 (0/5)	0 (0/3)	6.7 (1/15)
<i>NHF</i>	0 (0/1)	33.3 (1/3)	9.1 (1/11)	0 (0/7)	25 (2/8)	14.3 (1/7)	20 (1/5)	0 (0/3)	13.3 (2/15)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; NHF=no histopathological findings.

Thyroid

We evaluated 2.7% (2/75) of the cases and found no histopathological changes.

Hematopoietic system

a) Macroscopic changes

Megaly of lymph nodes and spleen (20.0%; 14/70), congestion of lymph nodes and spleen (11.4%; 8/70) and white pulp hyperplasia (7.1%; 5/70) were the most frequent necropsy findings. Other pathological processes are listed in table 23. Macroscopic changes were not observed in 61.4% (43/70) of the evaluated cases.

Table 23 - Hematopoietic system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
Lymphatic nodes	11.1 (5/45)	41.7 (5/12)	8.3 (1/12)	0 (0/1)	13.5 (5/37)	19.4 (6/31)	0 (0/2)	25 (3/12)	21.1 (4/19)	10.5 (4/38)	0 (0/1)	15.7 (11/70)
Spleen	6.7 (3/45)	0 (0/12)	0 (0/12)	0 (0/1)	5.4 (2/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	7.9 (3/38)	0 (0/1)	4.3 (3/70)
Megalia	17.8 (8/45)	41.7 (5/12)	8.3 (1/12)	0 (0/1)	18.9 (7/37)	22.6 (7/31)	0 (0/2)	25.0 (3/12)	21.1 (4/19)	18.4 (7/38)	0 (0/1)	20 (14/70)
Lymphatic nodes	2.2 (1/45)	0 (0/12)	0 (0/12)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
Spleen	8.9 (4/45)	16.7 (2/12)	8.3 (1/12)	0 (0/1)	10.8 (4/37)	9.7 (3/31)	0 (0/2)	8.3 (1/12)	5.3 (1/19)	13.2 (5/38)	0 (0/1)	10 (7/70)
Congestion	11.1 (5/45)	16.7 (2/12)	8.3 (1/12)	0 (0/1)	10.8 (4/37)	12.9 (4/31)	0 (0/2)	8.3 (1/12)	10.5 (2/19)	13.2 (5/38)	0 (0/1)	11.4 (8/70)
White pulp hyperplasia	8.9 (4/45)	0 (0/12)	8.3 (1/12)	0 (0/1)	8.1 (3/37)	6.5 (2/31)	0 (0/2)	16.7 (2/12)	5.3 (1/19)	5.3 (2/38)	0 (0/1)	7.1 (5/70)
Splenic hemorrhage	2.2 (1/45)	0 (0/12)	16.7 (2/12)	0 (0/1)	0 (0/37)	9.7 (3/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	5.3 (2/38)	0 (0/1)	4.3 (3/70)
Spleen, pale	0 (0/45)	8.3 (1/12)	8.3 (1/12)	0 (0/1)	0 (0/37)	6.5 (2/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	2.6 (1/38)	0 (0/1)	2.9 (2/70)
Caseous lymphadenitis	2.2 (1/45)	0 (0/12)	0 (0/12)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
Miscellaneous	2.2 (1/45)	8.3 (1/12)	8.3 (1/12)	0 (0/1)	0 (0/37)	9.7 (3/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	5.3 (2/38)	0 (0/1)	4.3 (3/70)
No macroscopic alterations	66.7 (30/45)	50 (6/12)	58.3 (7/12)	0 (0/1)	70.3 (26/37)	51.6 (16/31)	50 (1/2)	58.3 (7/12)	68.4 (13/19)	60.5 (23/38)	0 (0/1)	61.4 (43/70)
Autolysis	4.4 (2/45)	0 (0/12)	0 (0/12)	100 (1/1)	5.4 (2/37)	0 (0/31)	50 (1/2)	0 (0/12)	5.3 (1/19)	2.6 (1/38)	100 (1/1)	4.3 (3/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported.

Table 24 - Spleen: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>White pulp depletion</i>	45.5 (5/11)	25 (2/8)	50 (12/24)	100 (1/1)	39.1 (9/23)	50 (10/20)	100 (1/1)	45.5 (10/22)	25 (2/8)	53.8 (7/13)	100 (1/1)	45.5 (20/44)
<i>Hemosiderosis</i>	27.3 (3/11)	25 (2/8)	20.8 (5/24)	0 (0/1)	26.1 (4/23)	20 (4/20)	0 (0/1)	27.3 (6/22)	12.5 (1/8)	23.1 (3/13)	0 (0/1)	22.7 (10/44)
<i>Splenitis</i>	27.3 (3/11)	25 (2/8)	12.5 (3/24)	100 (1/1)	26.1 (4/23)	10 (2/20)	100 (1/1)	13.6 (3/22)	37.5 (3/8)	15.4 (2/13)	100 (1/1)	20.5 (9/44)
<i>Megakaryocytes</i>	9.1 (1/11)	0 (0/8)	4.2 (1/24)	0 (0/1)	4.3 (1/23)	5 (1/20)	0 (0/1)	4.5 (1/22)	0 (0/8)	7.7 (1/13)	0 (0/1)	4.5 (2/44)
<i>Necrosis</i>	0 (0/11)	12.5 (1/8)	0 (0/24)	0 (0/1)	0 (0/23)	5 (1/20)	0 (0/1)	4.5 (1/22)	0 (0/8)	0 (0/13)	0 (0/1)	2.3 (1/44)
<i>NHF</i>	9.1 (1/11)	12.5 (1/8)	25 (6/24)	0 (0/1)	21.7 (5/23)	15 (3/20)	0 (0/1)	13.6 (3/22)	25 (2/8)	23.1 (3/13)	0 (0/1)	18.2 (8/44)
<i>Autolysis</i>	9.1 (1/11)	25 (2/8)	4.2 (1/24)	0 (0/1)	4.3 (1/23)	15 (3/20)	0 (0/1)	13.6 (3/22)	0 (0/8)	7.7 (1/13)	0 (0/1)	9.1 (4/44)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular; P=poor; U=not reported; NHF= no histopathological findings

b) Microscopic findings

Spleen

We evaluated 44 (58.7%; 44/75) cases. Histopathological changes are listed in table 24. White pulp depletion (45.5%; 20/44), hemosiderosis (22.7%; 10/44), and splenitis (20.5%; 9/44) were the three main findings. No macroscopic changes were observed in 18.2% (8/44) of the cases. White pulp depletion was severe in 45.0% (9/20), moderate in 20.0% (4/20), moderate-severe and moderate in 15.0% (3/20), and mild in 5.0% (1/20) of the cases; it was the most common process in males, adults and fawns in poor body condition. All cases of hemosiderosis presented red pulp distribution. Two cases presented eosinophilic leukocytic infiltrates, and seven cases had neutrophilic infiltrates.

Lymph nodes

Thirteen (17.3%; 13/75) cases were evaluated; histopathological results are listed in table 25. Congestion (23.1%; 3/13), hemorrhage (7.7%; 1/13) and lymphadenitis (7.7%; 1/13) were the three main findings. Macroscopic changes were not observed in 53.8% (7/13) of the cases.

Table 25 - Lymphatic node: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)			Gender % (Na/Ne)		Body Condition % (Na/Ne)		Total % (Na/Ne)
	F	J	A	F	M	G	P	
<i>Congestion</i>	0 (0/2)	50 (1/2)	22.2 (2/9)	60 (3/5)	0 (0/8)	12.5 (1/8)	40 (2/5)	23.1 (3/13)
<i>Hemorrhage</i>	0 (0/2)	0 (0/2)	11.1 (1/9)	20 (1/5)	0 (0/8)	0 (0/8)	20 (1/5)	7.7 (1/13)
<i>Lymphoid proliferation</i>	0 (0/2)	0 (0/2)	11.1 (1/9)	0 (0/5)	12.5 (1/8)	12.5 (1/8)	0 (0/5)	7.7 (1/13)
<i>Lymphadenitis</i>	0 (0/2)	50 (1/2)	0 (0/9)	0 (0/5)	12.5 (1/8)	0 (0/8)	20 (1/5)	7.7 (1/13)
<i>NHF</i>	100 (2/2)	0 (0/2)	55.6 (5/9)	20.0 (1/5)	75 (6/8)	75 (6/8)	20 (1/5)	53.8 (7/13)
<i>Autolysis</i>	0 (0/2)	0 (0/2)	11.1 (1/9)	0 (0/5)	12.5 (1/8)	12.5 (1/8)	0 (0/5)	7.7 (1/13)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; NHF=no histopathological findings.

Nervous system

a) Macroscopic changes

Congestion (5.7%; 4/70) was the main necropsy finding. Other pathological processes are listed in table 26. Macroscopic findings were not observed in 88.6% (62/70) of the cases.

Table 26 - Nervous system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic changes	Age group %(Na/Ne)				Gender %(Na/Ne)			Body Condition %(Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
Brain congestion	2.2 (1/45)	8.3 (1/12)	16.7 (2/12)	0 (0/1)	2.7 (1/37)	9.7 (3/31)	0 (0/2)	0 (0/12)	0 (0/19)	10.5 (4/38)	0 (0/1)	5.7 (4/70)
<i>Fibrinous encephalitis</i>	0 (0/45)	0 (0/12)	8.3 (1/12)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
<i>Hematomyelia</i>	2.2 (1/45)	0 (0/12)	0 (0/12)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
<i>Spine compression</i>	0 (0/45)	0 (0/12)	8.3 (1/12)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
Miscellaneous	2.2 (1/45)	0 (0/12)	16.7 (2/12)	0 (0/1)	0 (0/37)	9.7 (3/31)	0 (0/2)	0 (0/12)	0 (0/19)	7.9 (3/38)	0 (0/1)	4.3 (3/70)
No macroscopic alterations	91.1 (41/45)	91.7 (11/12)	83.3 (10/12)	0 (0/1)	91.9 (34/37)	87.1 (27/31)	50 (1/2)	100 (12/12)	94.7 (18/19)	84.2 (32/38)	0 (0/1)	88.6 (62/70)
Autolysis	4.4 (2/45)	0 (0/12)	0 (0/12)	100 (1/1)	5.4 (2/37)	0 (0/31)	50 (1/2)	0 (0/12)	5.3 (1/19)	2.6 (1/38)	100 (1/1)	4.3 (3/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; U=not reported.

b) Microscopic findings

Brain

We evaluated ten (13.3%; 10/75) cases. Histopathological results are listed in table 27. Edema was the most frequent finding (30.0%; 3/10): mild (66.7%; 2/3) and moderate (33.3%; 1/3). Hemorrhage was observed in a female fawn, with moderate intensity, while moderate edema was diagnosed in a male fawn.

Integumentary system

a) Macroscopic changes

Skin lacerations 21.4% (15/70) and ectoparasitosis 15.7% (11/70) were the main gross findings. Other pathological processes are listed in table 28. Tick infestation

was more frequent in fawns 33.3% (4/12), than in juveniles and adults. We only diagnosed two adults with myiasis, and another two with fly infestation.

Table 27 - Brain: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)			Gender % (Na/Ne)		Body Condition % (Na/Ne)		Total % (Na/Ne)
	F	J	A	Fe	M	G	R	
<i>Edema</i>	100 (2/2)	0 (0/1)	20 (1/5)	33.3 (1/3)	40 (2/5)	28.6 (2/7)	100 (1/1)	30 (3/10)
<i>Congestion</i>	50 (1/2)	0 (0/1)	0 (0/5)	0 (0/3)	20 (1/5)	0 (0/7)	100 (1/1)	10 (1/10)
<i>Hemorrhage</i>	50 (1/2)	0 (0/1)	0 (0/5)	33.3 (1/3)	0 (0/5)	14.3 (1/7)	0 (0/1)	10 (1/10)
<i>NHF</i>	0 (0/2)	100 (1/1)	80 (4/5)	66.7 (2/3)	60 (3/5)	71.4 (5/7)	0 (0/1)	50 (5/10)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; R=regular, NHF=no histopathological findings.

b) Microscopic findings

Skin

Only 4% (3/75) of the cases were evaluated. One adult male was diagnosed with multifocal moderate non-suppurative dermatitis. The other two cases showed no macroscopic changes.

Musculoskeletal system

a) Macroscopic changes

Fractures (14.3; 10/70%), rhabdomyolysis (12.9%; 9/70), and hemorrhage (7.1%; 7/70) were the most common gross findings. Other pathological processes are listed in table 29. Macroscopic changes were not observed in 58.5% (41/70) of the cases.

Fractures were diagnosed in the skull 70.0% (7/10), ribs 40.0% (4/10), hip, spine, and forelegs 10.0% (1/10).

Rhabdomyolysis was observed in the cervical area and hindlimbs, and classified as multifocal (33.3%; 3/9). We reported five (71%) luxation cases; three adult females had tibiotarsal luxation, and two adults (one female and one male) were diagnosed with atlantoaxial luxation.

Table 28 - Integumentary system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Ticks</i>	33.3 (4/12)	8.3 (1/12)	8.9 (4/45)	0 (0/1)	10.8 (4/37)	12.9 (4/31)	50 (1/2)	10.5 (4/38)	16.7 (2/12)	15.8 (3/19)	0 (0/1)	12.9 (9/70)
<i>Myiasis</i>	0 (0/12)	0 (0/12)	4.4 (2/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	50 (1/2)	2.6 (1/38)	0 (0/12)	5.3 (1/19)	0 (0/1)	2.9 (2/70)
<i>Flies</i>	8.3 (1/12)	8.3 (1/12)	0.0 (0/45)	0 (0/1)	5.4 (2/37)	0 (0/31)	0 (0/2)	2.6 (1/38)	0 (0/12)	5.3 (1/19)	0 (0/1)	2.9 (2/70)
Ectoparasites	41.7 (5/12)	16.7 (2/12)	8.9 (4/45)	0 (0/1)	16.2 (6/37)	12.9 (4/31)	50 (1/2)	13.2 (5/38)	16.7 (2/12)	21.1 (4/19)	0 (0/1)	15.7 (11/70)
<i>Multifocal</i>	8.3 (1/12)	16.7 (2/12)	2.2 (1/45)	0 (0/1)	5.4 (2/37)	6.5 (2/31)	0 (0/2)	5.3 (2/38)	8.3 (1/12)	5.3 (1/19)	0 (0/1)	5.7 (4/70)
<i>Skull</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	2.6 (1/38)	0 (0/12)	0 (0/19)	0 (0/1)	1.4 (1/70)
<i>Cervical</i>	8.3 (1/12)	0 (0/12)	0 (0/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	2.6 (1/38)	0 (0/12)	0 (0/19)	0 (0/1)	1.4(1/70)
Hematoma	16.7 (2/12)	16.7 (2/12)	4.4 (2/45)	0 (0/1)	8.1 (3/37)	9.7 (3/31)	0 (0/2)	10.5 (4/38)	8.3 (1/12)	5.3 (1/19)	0 (0/1)	8.6 (6/70)
<i>Hindlimbs, forelegs</i>	16.7 (2/12)	0 (0/12)	6.7 (3/45)	0 (0/1)	8.1 (3/37)	6.5 (2/31)	0 (0/2)	10.5 (4/38)	0 (0/12)	5.3 (1/19)	0 (0/1)	7.1 (5/70)
<i>Thorax</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	0 (0/38)	0 (0/12)	5.3 (1/19)	0 (0/1)	1.4 (1/70)
<i>Multifocal</i>	8.3 (1/12)	8.3 (1/12)	15.6 (7/45)	0 (0/1)	13.5 (5/37)	12.9 (4/31)	0 (0/2)	13.2 (5/38)	16.7 (2/12)	10.5 (2/19)	0 (0/1)	12.9 (9/70)
Skin laceration	25.0 (3/12)	8.3 (1/12)	24.4 (11/45)	0 (0/1)	24.3 (9/37)	19.4 (6/31)	0 (0/2)	23.7 (9/38)	16.7 (2/12)	21.1 (4/19)	0 (0/1)	21.4 (15/70)
<i>Cyanosis</i>	0 (0/12)	0 (0/12)	6.7 (3/45)	0 (0/1)	2.7 (1/37)	6.5 (2/31)	0 (0/2)	5.3 (2/38)	8.3 (1/12)	0 (0/19)	0 (0/1)	4.3 (3/70)
<i>Dehydrated</i>	0 (0/12)	8.3 (1/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	3.2 (1/31)	0 (0/2)	0 (0/38)	0 (0/12)	10.5 (2/19)	0 (0/1)	2.9 (2/70)
Miscellaneous	8.3 (1/12)	16.7 (2/12)	24.4 (11/45)	0 (0/1)	16.2 (6/37)	22.6 (7/31)	50 (1/2)	21.1 (8/38)	16.7 (2/12)	21.1 (4/19)	0 (0/1)	20.0 (14/70)
Autolysis	8.3 (1/12)	8.3 (1/12)	4.4 (2/45)	100 (1/1)	8.1 (3/37)	3.2 (1/31)	50 (1/2)	5.3 (2/38)	0 (0/12)	10.5 (2/19)	100 (1/1)	7.1 (5/70)
NMA	8.3 (1/12)	25.0 (3/12)	44.4 (20/45)	0 (0/1)	37.8 (14/37)	32.3 (10/31)	0 (0/2)	34.2 (13/38)	41.7 (5/12)	31.6 (6/19)	0 (0/1)	34.3 (24/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; U=not reported; NHF=no histopathological findings.

b) Microscopic findings

Skeletal muscle

We evaluated 31 (41.3%; 31/75) cases. Histopathological results are listed in table 30. Myositis (29.0%; 9/31) was moderate in 55.6% (5/9) of the cases, moderate-severe in 33.3% (3/1), and severe in 11.1% (1/9). Leukocyte infiltrate was classified as neutrophilic in six cases, and mixed in the other three cases. Necrosis (25.8%; 8/31) was mild in 62.5% (5/8), moderate-severe in 12.5% (1/8), and severe in 25.0% (2/8) of the evaluated cases. *Sarcocystis* sp. were observed in three (9.7%; 3/31) cases. Cysts were not associated with an inflammatory response.

Table 29 - Musculoskeletal system: macroscopic findings, biological and epidemiological aspects in marsh deer

Macroscopic changes		Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
		F	J	A	U	Fe	M	U	G	R	P	U	
	<i>Hip</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
	<i>Ribs</i>	0 (0/12)	0 (0/12)	8.9 (4/45)	0 (0/1)	8.1 (3/37)	3.2 (1/31)	0 (0/2)	8.3 (1/12)	10.5 (2/19)	2.6 (1/38)	0 (0/1)	5.7 (4/70)
	<i>Skull</i>	0 (0/12)	8.3 (1/12)	8.9 (4/45)	0 (0/1)	5.4 (2/37)	9.7 (3/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	10.5 (4/38)	0 (0/1)	7.1 (7/70)
	<i>Spine</i>	0 (0/12)	8.3 (1/12)	0 (0/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
	<i>Forelegs</i>	0 (0/12)	8.3 (1/12)	0 (0/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
Bone fractures		0 (0/12)	8.3 (1/12)	20 (9/45)	0 (0/1)	16.2 (6/37)	12.9 (4/31)	0 (0/2)	16.7 (2/12)	15.8 (3/19)	13.2 (5/38)	0 (0/1)	14.3 (10/70)
	<i>Cervical</i>	8.3 (1/12)	0 (0/12)	4.4 (2/45)	0 (0/1)	5.4 (2/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	7.9 (3/38)	0 (0/1)	4.3 (3/70)
	<i>Hindlimbs</i>	0 (0/12)	0 (0/12)	6.7 (3/45)	0 (0/1)	8.1 (3/37)	0 (0/31)	0 (0/2)	0 (0/12)	0 (0/19)	7.9 (3/38)	0 (0/1)	4.3 (3/70)
	<i>Multifocal</i>	0 (0/12)	8.3 (1/12)	4.4 (2/45)	0 (0/1)	5.4 (2/37)	0 (0/31)	50 (1/2)	0 (0/12)	5.3 (1/19)	5.3 (2/38)	0 (0/1)	4.3 (3/70)
Rhabdomyolysis		8.3 (1/12)	8.3 (1/12)	15.6 (7/45)	0 (0/1)	18.9 (7/37)	3.2 (1/31)	50 (1/2)	0 (0/12)	5.3 (1/19)	21.1 (8/38)	0 (0/1)	12.9 (9/70)
Hemorrhage		16.7 (2/12)	8.3 (1/12)	4.4 (2/45)	0 (0/1)	5.4 (2/37)	6.5 (2/31)	50 (1/2)	8.3 (1/12)	5.3 (1/19)	7.9 (3/38)	0 (0/1)	7.1 (7/70)
	<i>Atlantoaxial joint</i>	0 (0/12)	0 (0/12)	4.4 (2/45)	0 (0/1)	2.7 (1/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	2.6 (1/38)	0 (0/1)	2.9 (2/70)
	<i>Tibiotarsal joint</i>	0 (0/12)	0 (0/12)	6.7 (3/45)	0 (0/1)	8.1 (3/37)	0 (0/31)	0 (0/2)	0 (0/12)	0 (0/19)	7.9 (3/38)	0 (0/1)	4.3 (3/70)
Luxation		0 (0/12)	0 (0/12)	11.1 (5/45)	0 (0/1)	10.8 (4/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	5.3 (1/19)	10.5 (4/38)	0 (0/1)	7.1 (5/70)
	<i>Forelegs, abscess</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	2.7 (1/37)	0 (0/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
	<i>Hindlimbs, abscess</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
	<i>Muscle, hyperemia</i>	83 (1/12)	0 (0/12)	0 (0/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	8.3 (1/12)	0 (0/19)	0 (0/38)	0 (0/1)	1.4 (1/70)
	<i>Skull, caseous osteitis</i>	0 (0/12)	0 (0/12)	2.2 (1/45)	0 (0/1)	0 (0/37)	3.2 (1/31)	0 (0/2)	0 (0/12)	0 (0/19)	2.6 (1/38)	0 (0/1)	1.4 (1/70)
Miscellaneous		8.3 (1/12)	0 (0/12)	6.7 (3/45)	0 (0/1)	2.7 (1/37)	9.7 (3/31)	0 (0/2)	16.7 (2/12)	0 (0/19)	5.3 (2/38)	0 (0/1)	5.7 (4/70)
No macroscopic alterations		66.7 (8/12)	75 (9/12)	53.3 (24/45)	0 (0/1)	51.4 (19/37)	71 (22/31)	0 (0/2)	66.7 (8/12)	73.7 (14/19)	50 (19/38)	0 (0/1)	58.6 (41/70)
Autolysis		0.0 (0/12)	0 (0/12)	4.4 (2/45)	100 (1/1)	5.4 (2/37)	0 (0/31)	50 (1/2)	0 (0/12)	5.3 (1/19)	2.6 (1/38)	100 (1/1)	4.3 (3/70)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; U=not reported.

Table 30 - Skeletal muscle: histopathological findings, biological and epidemiological aspects in marsh deer

Histopathological findings	Age group % (Na/Ne)				Genre % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Myositis</i>	50 (1/2)	25 (1/4)	29.2 (7/24)	0 (0/1)	25 (4/16)	38.5 (5/13)	0 (0/2)	23.5 (4/17)	50 (2/4)	33.3 (3/9)	0 (0/1)	29 (9/31)
<i>Necrosis</i>	50 (1/2)	25 (1/4)	25 (6/24)	0 (0/1)	25 (4/16)	30.8 (4/13)	0 (0/2)	11.8 (2/17)	50 (2/4)	44.4 (4/9)	0 (0/1)	25.8 (8/31)
<i>Rhabdomyolysis</i>	0 (0/2)	25 (1/4)	29.2 (7/24)	0 (0/1)	31.3 (5/16)	23.1 (3/13)	0 (0/2)	17.6 (3/17)	75 (3/4)	22.2 (2/9)	0 (0/1)	25.8 (8/31)
<i>Hemorrhage</i>	0 (0/2)	25 (1/4)	25 (6/24)	0 (0/1)	25 (4/16)	23.1 (3/13)	0 (0/2)	23.5 (4/17)	25 (1/4)	22.2 (2/9)	0 (0/1)	22.6 (7/31)
<i>Myofiber degeneration</i>	0 (0/2)	50 (2/4)	12.5 (3/24)	0 (0/1)	25 (4/16)	7.7 (1/13)	0 (0/2)	5.9 (1/17)	0 (0/4)	44.4 (4/9)	0 (0/1)	16.1 (5/31)
<i>Sarcocystis cyst</i>	0 (0/2)	0 (0/4)	12.5 (3/24)	0 (0/1)	6.3 (1/16)	15.4 (2/13)	0 (0/2)	5.9 (1/17)	50 (2/4)	0 (0/9)	0 (0/1)	9.7 (3/31)
<i>Congestion</i>	0 (0/2)	0 (0/4)	8.3 (2/24)	0 (0/1)	6.3 (1/16)	7.7 (1/13)	0 (0/2)	5.9 (1/17)	0 (0/4)	11.1 (1/9)	0 (0/1)	6.5 (2/31)
<i>Edema</i>	0 (0/2)	25 (1/4)	0 (0/24)	0 (0/1)	0 (0/16)	7.7 (1/13)	0 (0/2)	5.9 (1/17)	0 (0/4)	0 (0/9)	0 (0/1)	3.2 (1/31)
<i>NHF</i>	0 (1/2)	0 (0/4)	33.3 (8/24)	100 (1/1)	37.5 (6/16)	15.4 (2/13)	100 (2/2)	41.2 (7/17)	0 (0/4)	22.2 (2/9)	100 (1/1)	32.3 (10/31)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; U=not reported; NHF=no histopathological findings.

5.3 BROWN BROCKET DEER (*MAZAMA GOUAZOUBIRA*)

5.3.1 Biological and epidemiological aspects

One hundred and thirty-one cases were recorded between 1995 and 2015. Monthly and annual distribution, weather and general epidemiological aspects are shown in figures 8-10.

Figure 8 - Annual distribution of Brazilian brown brocket deer submissions between 1995-2015

Figure 9 - Monthly distribution of Brazilian brown brocket deer cases between 1995-2015

Figure 10 - Seasonality in mortality of Brazilian brown brocket deer cases between 1995-2015, and its relationship to annual distribution

Increased case load was observed in May and July. The year presenting the highest number of cases was 1996, with 30 records. Table 31 shows the epidemiological distribution of the evaluated population; the highest frequency of adult animals corresponds to free-ranging deer (71.8%; 94/131). 52.7% (69/131) of the population was in good body condition. 49.6% (6/131) represented the trauma cases, the two major causes of trauma were dog attacks (23.1%; 15/65) and vehicular trauma (20.0%; 13/65) (table 32).

Table 31 - Brown brocket deer: general epidemiological aspects

Category	Subcategory	Nº	%
Age group	Adult	60	45.8
	Fawn	16	12.2
	Juvenile	27	20.6
	Unreported	28	21.4
Gender	Female	54	41.2
	Male	59	45.0
	Unreported	18	13.7
Condition	Captive	23	17.6
	Free-ranging	94	71.8
	Unreported	14	10.7
Body condition	Poor	13	9.9
	Good	69	52.7
	Regular	14	10.7
	Unreported	35	26.7
Trauma	Not	39	29.8
	Trauma	65	49.6
	Unreported	27	20.6
Ectoparasites	Not	60	45.8
	Ectoparasites	39	29.8
	Unreported	32	24.4

Even though most cases were not clinically diagnosed, trauma (21.4%; 28/131) was the most important diagnosis, followed by dog attacks (9.9%; 13/131), and vehicle collision (8.4%; 11/131). It is important to notice that these three findings correspond to trauma, diagnosed in 39.7% (52/131) of the cases (figure 11).

Table 32 - Brown brocket deer: general aspects of trauma.

Causes of trauma	Nº	%
Dog attack	15	23.1
Intraspecific	3	4.6
Self-induced	1	1.5
Unknown	3	4.6
Vehicle-collision	13	20.0

Figure 11 - Distribution according to the reported clinical diagnosis.

5.3.2 Pathologic analysis

Respiratory system

a) Macroscopic changes

Edema, congestion and hemorrhage were the main necropsy findings. Other pathological findings are listed in table 33.

Table 33 - Respiratory system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
Edema	46.7 (7/15)	57.7 (15/26)	52.2 (24/46)	66.7 (2/3)	50 (22/44)	57.8 (26/45)	0 (0/1)	30.8 (4/13)	69.2 (9/13)	55.6 (35/63)	0 (0/1)	53.3 (48/90)
<i>Lung</i>	66.7 (10/15)	50 (13/26)	34.8 (16/46)	66.7 (2/3)	45.5 (20/44)	44.4 (20/45)	100 (1/1)	30.8 (4/13)	46.2 (6/13)	47.6 (30/63)	100 (1/1)	45.6 (41/90)
<i>Trachea</i>	0 (0/15)	11.5 (3/26)	8.7 (4/46)	0 (0/3)	6.8 (3/44)	8.9 (4/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	9.5 (6/63)	0 (0/1)	7.8 (7/90)
Congestion	66.7 (10/15)	53.8 (14/26)	39.1 (18/46)	66.7 (2/3)	47.7 (21/44)	48.9 (22/45)	100 (1/1)	38.5 (5/13)	46.2 (6/13)	50.8 (32/63)	100 (1/1)	48.9 (44/90)
<i>Lung</i>	6.7 (1/15)	15.4 (4/26)	10.9 (5/46)	0 (0/3)	15.9 (7/44)	6.7 (3/45)	0 (0/1)	7.7 (1/13)	15.4 (2/13)	11.1 (35/63)	0 (0/1)	11.1 (10/90)
<i>Trachea</i>	6.7 (1/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	4.8 (3/63)	0 (0/1)	3.3 (3/90)
Hemorrhage	13.3 (2/15)	19.2 (5/26)	13 (6/46)	0 (0/3)	20.5 (9/44)	8.9 (4/45)	0 (0/1)	7.7 (1/13)	15.4 (2/13)	15.9 (10/63)	0 (0/1)	14.4 (13/90)
<i>Aspiration pneumonia</i>	0 (0/15)	7.7 (2/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	2.2 (1/45)	0 (0/1)	7.7 (1/13)	7.7 (1/13)	1.6 (1/63)	0 (0/1)	3.3 (3/90)
<i>Bronchopneumonia</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Non classified pneumonia</i>	0 (0/15)	7.7 (2/26)	13 (6/46)	0 (0/3)	9.1 (4/44)	8.9 (4/45)	0 (0/1)	7.7 (1/13)	7.7 (1/13)	9.5 (6/63)	0 (0/1)	8.9 (8/90)
Pneumonia	0 (0/15)	19.2 (5/26)	15.2 (7/46)	0 (0/3)	15.9 (7/44)	11.1 (5/45)	0 (0/1)	15.4 (2/13)	15.4 (2/13)	12.7 (8/63)	0 (0/1)	13.3 (12/90)
Emphysema	6.7 (1/15)	7.7 (2/26)	4.3 (2/46)	0 (0/3)	11.4 (5/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	6.3 (4/63)	0 (0/1)	5.6 (5/90)
Atelectasis	6.7 (1/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	2.3 (1/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	3.2 (2/63)	0 (0/1)	3.3 (3/90)
<i>Glottis; edema</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Lung; perforation</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Pleura; parasitic cysts</i>	0 (0/15)	0 (0/26)	4.3 (2/46)	0 (0/3)	2.3 (1/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	3.2 (2/63)	0 (0/1)	2.2 (2/90)
<i>Trachea; foreign material</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	2.2 (2/90)
<i>Trachea; perforation</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Miscellaneous	6.7 (1/15)	7.7 (2/26)	10.9 (5/46)	0 (0/3)	11.4 (5/44)	6.7 (3/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	11.1 (35/63)	0 (0/1)	8.9 (8/90)
NPA	20.0 (3/15)	19.2 (5/26)	19.2 (5/26)	0 (0/3)	18.2 (8/44)	20 (9/45)	0 (0/1)	30.8 (4/13)	15.4 (2/13)	17.5 (11/63)	0 (0/1)	18.9 (17/90)
Autolysis	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	0.0 (0/45)	0.0 (0/1)	7.7 (1/13)	0.0 (0/13)	1.6 (1/63)	0 (0/1)	2.2 (2/90)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; U=not reported.

Edema and congestion were homogeneously distributed among all epidemiological categories. However, hemorrhage was present in 21.4% (12/56) of the traumatized animals and in 2.9% (1/34) of non-traumatized animals. Emphysema was not observed in males. Macroscopic changes were not observed in 18.9% (17/90) of the cases.

b) Microscopic findings

Lungs

All histopathological processes are shown in table 35.

I) Hemodynamic Disorders

The most common hemodynamic disorder was pulmonary congestion (82.1%; 78/95), primarily moderate (50.0%; 39/78) and mild-moderate (12.8%; 10/78), especially in juveniles (95.0%; 19/20). Pulmonary edema was identified in 57.9% (55/78) of the cases; moderate (36.4%; 20/55), moderate-severe (18.2%; 10/55), and severe (18.2%; 10/55). Hemorrhage was diagnosed in 30.5% (26/78) of the cases, 51.7% (15/26) of moderate intensity. Concomitant congestion and edema was observed in 52 cases, and associated to hemorrhage in other 19 cases. Thrombosis was observed in one case (table 34).

Table 34 - Severity of major pulmonary hemodynamic disorders in brown brocket deer.

Hemodynamic disorder	Congestion		Edema		Hemorrhage	
	N	%	N	%	N	%
<i>Mild</i>	5	6.4	9	16.4	3	10.3
<i>Mild to moderate</i>	10	12.8	6	10.9	4	13.8
<i>Moderate</i>	39	50.0	20	36.4	15	51.7
<i>Moderate to severe</i>	16	20.5	10	18.2	4	13.8
<i>Severe</i>	8	10.3	10	18.2	3	10.3

Table 35 - Lungs: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Congestion</i>	72.7 (8/11)	95 (19/20)	75 (30/40)	87.5 (21/24)	74.4 (29/39)	85.4 (35/41)	93.3 (14/15)	89.8 (44/49)	50.0 (4/8)	77.8 (7/9)	79.3 (23/29)	82.1 (78/95)
<i>Edema</i>	45.5 (5/11)	60 (12/20)	52.5 (21/40)	70.8 (17/24)	53.8 (21/39)	56.1 (23/41)	73.3 (11/15)	69.4 (34/49)	0 (0/8)	33.3 (3/9)	62.1 (18/29)	57.9 (55/95)
<i>Pneumonia</i>	63.6 (7/11)	55 (11/20)	50 (20/40)	58.3 (14/24)	51.3 (20/39)	53.7 (22/41)	66.7 (10/15)	53.1 (26/49)	62.5 (5/8)	44.4 (4/9)	58.6 (17/29)	54.7 (52/95)
<i>Hemorrhage</i>	9.1 (1/11)	30 (6/20)	32.5 (13/40)	37.5 (9/24)	23.1 (9/39)	34.1 (14/41)	40 (6/15)	30.6 (15/49)	12.5 (1/8)	11.1 (1/9)	41.4 (12/29)	30.5 (29/95)
<i>Atelectasis</i>	9.1 (1/11)	0 (0/20)	20 (8/40)	12.5 (3/24)	15.4 (6/39)	7.3 (3/41)	20 (3/15)	16.3 (8/49)	12.5 (1/8)	0 (0/9)	10.3 (3/29)	12.6 (12/95)
<i>TNP</i>	18.2 (2/11)	10 (2/20)	10 (4/40)	12.5 (3/24)	10.2 (4/39)	12.2 (5/41)	13.3 (2/15)	14.3 (7/49)	0 (0/8)	11.1 (1/9)	10.3 (3/29)	11.6 (11/95)
<i>Emphysema</i>	9.1 (1/11)	10 (2/20)	10 (4/40)	8.3 (2/24)	7.7 (3/39)	12.2 (5/41)	6.7 (1/15)	14.3 (7/49)	0 (0/8)	0 (0/9)	6.9 (2/29)	9.5 (9/95)
<i>Anthracosis</i>	0 (0/11)	5 (1/20)	12.5 (5/40)	0 (0/24)	10.2 (4/39)	4.9 (2/41)	0 (0/15)	10.2 (5/49)	0 (0/8)	0 (0/9)	3.4 (1/29)	6.3 (6/95)
<i>Hemosiderosis</i>	0 (0/11)	0 (0/20)	7.5 (3/40)	12.5 (3/24)	5.1 (2/39)	4.9 (2/41)	13.3 (2/15)	8.2 (4/49)	0 (0/8)	0 (0/9)	6.9 (2/29)	6.3 (6/95)
<i>Fibrosis</i>	0 (0/11)	0 (0/20)	7.5 (3/40)	4.2 (1/24)	5.1 (2/39)	2.4 (1/41)	6.7 (1/15)	4.1 (2/49)	12.5 (1/8)	0 (0/9)	3.4 (1/29)	4.2 (4/95)
<i>BD</i>	0 (0/11)	0(0/20)	5 (2/40)	0 (0/24)	2.6 (1/39)	2.4 (1/41)	0 (0/15)	4.1 (2/49)	0 (0/8)	0 (0/9)	0 (0/29)	2.1 (2/95)
<i>PT</i>	0 (0/11)	0 (0/20)	5 (2/40)	0 (0/24)	5.1 (2/39)	0 (0/41)	0 (0/15)	4.1 (2/49)	0 (0/8)	0 (0/9)	0 (0/29)	2.1 (2/95)
<i>Thrombosis</i>	0 (0/11)	0 (0/20)	0 (0/40)	4.2 (1/24)	0 (0/39)	0 (0/41)	6.7 (1/15)	0 (0/49)	0 (0/8)	0 (0/9)	3.4 (1/29)	1.1 (1/95)
<i>NHF</i>	9.1 (1/11)	0 (0/20)	7.5 (3/40)	8.3 (2/24)	10.2 (4/39)	4.9 (2/41)	0 (0/15)	2 (1/49)	25 (2/8)	0 (0/9)	10.3 (3/29)	6.3 (6/95)
<i>Autolysis</i>	9.1 (1/11)	15 (3/20)	50 (20/40)	4.2 (1/24)	20.5 (8/39)	12.2 (5/41)	0 (0/15)	16.3 (8/49)	0 (0/8)	22.2 (2/9)	10.3 (3/29)	13.7 (13/95)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; U=not reported; TNP= type II pneumocytes proliferation; BD=bronchial epithelium desquamation; PT=pleural thickening; NHF=no histopathological findings.

II) Inflammation and Repair

Inflammatory response was observed in 54.7% of the evaluated cases. Distribution of inflammatory response was categorized into four morphological patterns: lumen (alveolar, bronchiolar and bronchial), interstitium (septal, broncovascular and interlobular), pleura-subpleural, and perivascular. Interstitial pattern was observed in 53.8% (28/52), luminal in 32.7% (17/52), and interstitial and luminal patterns were concomitantly observed in seven cases. Regarding leukocyte infiltrates, in 23.1% (12/52) cases granulocytes predominated, in 17.3% (9/52) mononuclear cells, and in 59.6% (31/52) of both granulocytes and mononuclear cells. Larvae of helminth parasites (Cestoda) were observed in two cases. Intralesional bacterial colonies were diagnosed in three cases of bronchopneumonia (table 36).

Interstitial pneumonia was diagnosed in 34.7% (33/95) of the cases, with either mixed (66.7%; 22/33) or mononuclear (33.3%; 11/33) infiltrates. The intensities varied as follows: moderate (36.4%; 12/33), mild (30.3%; 10/33), moderate-severe (18.2%; 6/33), mild-moderate (9.1%; 3/33), and severe (6.1%; 2/33). Septal-interstitial pattern was observed in all cases of inflammatory response; type II pneumocyte proliferation was present in 11.6% (11/95) of them.

Bronchopneumonia was diagnosed in 34.6% (28/52) of the cases. It was possible to identify two types of inflammatory infiltrate: suppurative, with polymorphonuclear leukocytes in alveolar, bronchiolar and bronchial lumen, occasionally associated with leukocytosis and hemodynamic disorders, and secondly, fibrinosuppurative, with polymorphonuclear infiltrate and fibrin deposition.

Mononuclear perivascularitis was observed in 3.2% (3/95) of the cases. All cases exhibited infiltrate containing lymphocytes, plasma cells and histiocytic cells. Aspiration pneumonia, embolic pneumonia, eosinophilic pneumonia and pleuritis had a prevalence of 1.1% (1/95). Multiple plant fibers were observed in the alveolar lumen with associated neutrophilic exudate in cases of aspiration pneumonia. Pleuritis was characterized by pleural and sub-pleural neutrophilic exudate, pleural thickening and fibrin deposition. Bronchitis was diagnosed in 2.1% (2/52) of the cases, always associated with other inflammatory processes.

Table 36 - Epidemiological aspects of brown brocket deer with pneumonia

Type of pneumonia	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Interstitial pneumonia</i>	71.4 (5/7)	63.6 (7/11)	65 (13/20)	57.1 (8/14)	70 (14/20)	63.6 (14/22)	50 (5/10)	69.2 (18/26)	80 (4/5)	50 (2/4)	52.9 (9/17)	63.5 (33/52)
<i>Bronchopneumonia</i>	28.6 (2/7)	45.5 (5/11)	30 (6/20)	35.7 (5/14)	35 (7/20)	31.8 (7/22)	40 (4/10)	26.9 (7/26)	40 (2/5)	50 (2/4)	41.2 (7/17)	34.6 (28/52)
<i>Aspiration pneumonia</i>	0 (0/7)	0 (0/11)	5 (1/20)	0 (0/14)	5 (1/20)	0 (0/22)	0 (0/10)	0 (0/26)	0 (0/5)	0 (0/4)	5.9 (1/17)	1.9 (1/52)
<i>Embolic pneumonia</i>	0 (0/7)	0 (0/11)	0 (0/20)	7.1 (1/14)	0 (0/20)	0 (0/22)	10 (1/10)	0 (0/26)	0 (0/5)	0 (0/4)	5.9 (1/17)	1.9 (1/52)
<i>Mononuclear perivascularitis</i>	0 (0/7)	9.1 (1/11)	5 (1/20)	7.1 (1/14)	0 (0/20)	13.6 (3/22)	0 (0/10)	3.8 (1/26)	20 (1/5)	0 (0/4)	5.9 (1/17)	5.8 (3/52)
<i>Eosinophilic pneumonia</i>	0 (0/7)	0 (0/11)	5 (1/20)	0 (0/14)	5 (1/20)	0 (0/22)	0 (0/10)	3.8 (1/26)	0 (0/5)	0 (0/4)	0 (0/17)	1.9 (1/52)
<i>Bronchitis</i>	0 (0/7)	0 (0/11)	10 (2/20)	0 (0/14)	5 (1/20)	4.5 (1/22)	0 (0/10)	7.7 (2/26)	0 (0/5)	0 (0/4)	0 (0/17)	3.8 (2/52)
<i>Pleuritis</i>	14.3 (1/7)	0 (0/11)	0 (0/20)	0 (0/14)	5 (1/20)	0 (0/22)	0 (0/10)	3.8 (1/26)	0 (0/5)	0 (0/4)	0 (0/17)	1.9 (1/52)

Na=n° affected; Ne=n° examined; F=fawn; J=juvenile; A=adult; Fe=female; M=male; G=good; P=poor; U=not reported.

III) Miscellaneous processes

We diagnosed six cases of anthracosis, presenting multifocal bronchial and peribronchial alveolar macrophages, and variable intensities: mild (83.7% 5/6) and mild-moderate (16.7%; 1/6). Atelectasis was observed in 12.6% (6/95) of the cases.

Trachea

We evaluated 14 (10.7%; 14/131) cases. Histopathological results are listed in table 37. Hemorrhage was observed in 21.4% (3/14) of the cases, while congestion was observed in 7.1% (1/14); both pathologies presented mainly mucosal and submucosal distribution, and were predominantly moderate or moderate-severe.

Table 37 - Trachea: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopath. findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Hemorrhage</i>	0 (0/3)	50 (2/4)	0 (0/5)	50 (1/2)	25 (2/8)	20 (1/5)	0 (0/1)	20 (2/10)	0 (0/1)	0 (0/1)	50 (1/2)	21.4% (3/14)
<i>Congestion</i>	0 (0/3)	25 (1/4)	0 (0/5)	0 (0/2)	0 (0/8)	20 (1/5)	0 (0/1)	10 (1/10)	0 (0/1)	0 (0/1)	0 (0/2)	7.1% (1/14)
<i>NHF</i>	100 (3/3)	50 (2/4)	80 (4/5)	50 (1/2)	62.5 (5/8)	80 (4/5)	100 (1/1)	70 (7/10)	100 (1/1)	100 (1/1)	50 (1/2)	71.4% (10/14)
<i>Autolysis</i>	0 (0/3)	0 (0/4)	20 (1/5)	0 (0/2)	12.5 (1/8)	0 (0/5)	0 (0/1)	10 (1/10)	0 (0/1)	0 (0/1)	0 (0/2)	7.1% (1/14)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Cardiovascular system

a) Macroscopic changes

Hemorrhage, pericardial effusions and endocardiosis were the most frequent gross findings. Others pathological findings are listed in table 38. Macroscopic changes were not observed in 65.5% (59/90) of the cases.

b) Microscopic findings

Heart

We evaluated 79 (60.3%; 79/131) cases. Histopathological findings are listed in table 39. No macroscopic changes were observed in 65.8% (52/79) of the cases.

I) Hemodynamic disturbances (HD)

Hemorrhage and congestion were diagnosed respectively in 13.9% (11/79) and 7.6% (6/79) of the cases. Both disturbances were observed in the myocardium and pericardium. Intensity was predominantly moderate (5/11 hemorrhage; 3/6 congestion). These pathologies were not observed in fawns, or animals with regular or poor body condition. Thrombosis was diagnosed in one case with fungal myocarditis.

Cysts of *Sarcocystis* sp. were observed in 8.9% of the cases, all in adult deer. One (3.8%) of the myocarditis cases was diagnosed as severe fungal myocarditis, supported by (PAS and Grocott-positive). In this case we found: intravascular, transmural and intramyocardial multifocal thin septate with acute-angle hyphae and conidia measuring approximately 10µm.

Alimentary tract, liver and pancreas

a) Macroscopic changes

Congestion, inflammatory response and hepatomegaly were the most frequent findings. Other pathological findings are listed in table 40. Hepatic congestion was diagnosed in 26.7% (24/90), enteritis in 12.2% (11/90), hepatomegaly in 17.8% (16/90), and hepatic degeneration in 14.4% (13/90) of the cases, homogeneously distributed among all epidemiological categories.

Table 38 - Cardiovascular system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Hemorrhage</i>	0 (0/15)	11.5 (3/26)	19.6 (9/46)	33.3 (1/3)	15.9 (7/44)	13.3 (6/45)	0 (0/1)	7.7 (1/13)	17.5 (11/63)	7.7 (1/13)	0 (0/1)	14.4 (13/90)
<i>Hydro pericardium</i>	6.7 (1/15)	11.5 (3/26)	8.7 (4/46)	0 (0/3)	9.1 (4/44)	8.9 (4/45)	0 (0/1)	15.4 (2/13)	9.5 (6/63)	0 (0/13)	0 (0/1)	8.9 (8/90)
<i>Endocardiosis</i>	6.7 (1/15)	7.7 (2/26)	6.5 (3/46)	0 (0/3)	4.5 (2/44)	8.9 (4/45)	0 (0/1)	0 (0/13)	9.5 (6/63)	0 (0/13)	0 (0/1)	6.7 (6/90)
<i>Cardiomegaly</i>	0 (0/15)	11.5 (3/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	3.2 (2/63)	15.4 (2/13)	0 (0/1)	4.4 (4/90)
<i>Inflammatory response</i>	0 (0/15)	7.7 (2/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	2.2 (1/45)	0 (0/1)	7.7 (1/13)	1.6 (1/63)	7.7 (1/13)	0 (0/1)	3.3 (3/90)
<i>Heart; thick white lines</i>	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	3.2 (2/63)	0 (0/13)	0 (0/1)	2.2 (2/90)
<i>Pericardium; thickening</i>	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	2.3 (1/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	16 (1/63)	7.7 (1/13)	0 (0/1)	2.2 (2/90)
<i>No macroscopic alterations</i>	93.3 (14/15)	61.5 (16/26)	58.7 (27/46)	66.7 (2/3)	63.6 (28/44)	66.7 (30/45)	100 (1/1)	61.5 (8/13)	63.5 (40/63)	76.9 (10/13)	100 (1/1)	65.6 (59/90)
<i>Autolysis</i>	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	6.8 (3/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	3.2 (2/63)	0 (0/13)	0 (0/1)	3.3 (3/90)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported.

Table 39 - Heart: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Hemorrhage</i>	0 (0/6)	17.6 (3/17)	11.4 (4/35)	19.0 (4/21)	9.4 (3/32)	11.8 (4/34)	30.8 (4/13)	17.9 (7/39)	0 (0/8)	0 (0/6)	15.4 (4/26)	13.9 (11/79)
<i>Sarcocystis cyst</i>	0 (0/6)	0 (0/17)	14.3 (5/35)	9.5 (2/21)	6.3 (2/32)	11.8 (4/34)	7.7 (1/13)	12.8 (5/39)	0 (0/8)	0 (0/6)	7.7 (2/26)	8.9 (7/79)
<i>Congestion</i>	0 (0/6)	11.8 (2/17)	2.9 (1/35)	14.3 (3/21)	12.5 (4/32)	2.9 (1/34)	7.7 (1/13)	7.7 (3/39)	0 (0/8)	0 (0/6)	7.7 (2/26)	7.6 (6/79)
<i>Myocarditis</i>	0 (0/6)	0 (0/17)	0 (0/35)	14.3 (3/21)	0 (0/32)	2.9 (1/34)	15.4 (2/13)	0 (0/39)	0 (0/8)	0 (0/6)	11.5 (3/26)	3.8 (3/79)
<i>Myocardial necrosis</i>	0 (0/6)	5.9 (1/17)	0 (0/35)	4.8 (1/21)	3.1 (1/32)	0 (0/34)	7.7 (1/13)	0 (0/39)	12.5 (1/8)	0 (0/6)	3.8 (1/26)	2.5 (2/79)
<i>Fatty infiltration</i>	0 (0/6)	0 (0/17)	2.9 (1/35)	0 (0/21)	0 (0/32)	2.9 (1/34)	0 (0/13)	2.6 (1/39)	0 (0/8)	0 (0/6)	0 (0/26)	1.3 (1/79)
<i>Edema</i>	0 (0/6)	0 (0/17)	2.9 (1/35)	0 (0/21)	0 (0/32)	2.9 (1/34)	0 (0/13)	2.6 (1/39)	0 (0/8)	0 (0/6)	0 (0/26)	1.3 (1/79)
<i>Leukocytosis</i>	0 (0/6)	0 (0/17)	0 (0/35)	4.8 (1/21)	0 (0/32)	0 (0/34)	7.7 (1/13)	0 (0/39)	0 (0/8)	0 (0/6)	3.8 (1/26)	1.3 (1/79)
<i>Thrombosis</i>	0 (0/6)	0 (0/17)	0 (0/35)	4.8 (1/21)	0 (0/32)	0 (0/34)	7.7 (1/13)	0 (0/39)	0 (0/8)	0 (0/6)	3.8 (1/26)	1.3 (1/79)
<i>NHF</i>	100 (6/6)	76.5 (13/17)	65.7 (23/35)	47.6 (10/21)	65.6 (21/32)	70.6 (24/34)	53.8 (7/13)	64.1 (25/39)	75.0 (6/8)	100 (6/6)	57.7 (15/26)	65.8 (52/79)
<i>Autolysis</i>	0 (0/6)	0 (0/17)	0 (0/35)	4.8 (1/21)	0 (0/32)	2.9 (1/34)	0 (0/13)	0 (0/39)	0 (0/8)	0 (0/6)	3.8 (1/26)	1.3 (1/79)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Table 40 - Alimentary system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Forestomachs</i>	0 (0/15)	7.7 (2/26)	0 (0/46)	0 (0/3)	0 (0/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	3.2 (2/63)	0 (0/13)	0 (0/1)	2.2 (2/90)
<i>Small intestine</i>	6.7 (1/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	2.3 (1/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	4.8 (3/63)	0 (0/13)	0 (0/1)	3.3 (3/90)
<i>Liver</i>	33.3 (5/15)	19.2 (5/26)	26.1 (12/46)	66.7 (2/3)	25 (11/44)	26.7 (12/45)	100 (1/1)	15.4 (2/13)	30.2 (19/63)	15.4 (2/13)	100 (1/1)	26.7 (24/90)
Congestion	33.3 (5/15)	19.2 (5/26)	28.3 (13/46)	6.7 (2/3)	25 (11/44)	28.9 (13/45)	100 (1/1)	15.4 (2/13)	31.7 (20/63)	15.4 (2/13)	100 (1/1)	27.8 (25/90)
<i>Forestomach</i>	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	3.2 (2/63)	0 (0/13)	0 (0/1)	2.2 (2/90)
<i>Small intestine</i>	13.3 (2/15)	3.8 (1/26)	17.4 (8/46)	0 (0/3)	15.9 (7/44)	8.9 (4/45)	0 (0/1)	7.7 (1/13)	15.9 (10/63)	0 (0/13)	0 (0/1)	12.2 (11/90)
<i>Large intestine</i>	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	2.3 (1/44)	4.4 (2/45)	0 (0/1)	7.7 (1/13)	3.2 (2/63)	0 (0/13)	0 (0/1)	3.3 (3/90)
<i>Liver</i>	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	4.5 (2/44)	2.2 (1/45)	0 (0/1)	7.7 (1/13)	3.2 (2/63)	0 (0/13)	0 (0/1)	3.3 (3/90)
Inflammatory	13.3 (2/15)	15.4 (4/26)	26.1 (12/46)	0 (0/3)	22.7 (10/44)	17.8 (8/45)	0 (0/1)	23.1 (3/13)	23.8 (15/63)	0 (0/13)	0 (0/1)	20 (18/90)
Hepatomegaly	20.0 (3/15)	11.5 (3/26)	21.7 (10/46)	0 (0/3)	18.2 (8/44)	17.8 (8/45)	0 (0/1)	7.7 (1/13)	19.0 (12/63)	23.1 (3/13)	0 (0/1)	17.8 (16/90)
<i>Forestomach</i>	0 (0/15)	3.8 (1/26)	8.7 (4/46)	0 (0/3)	6.8 (3/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	6.3 (4/63)	7.7 (1/13)	0 (0/1)	5.6 (5/90)
<i>Small intestine</i>	0 (0/15)	3.8 (1/26)	6.5 (3/46)	0 (0/3)	6.8 (3/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	6.3 (4/63)	0 (0/13)	0 (0/1)	4.4 (4/90)
<i>Liver</i>	13.3 (2/15)	0 (0/26)	4.3 (2/46)	0 (0/3)	6.8 (3/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	6.3 (4/63)	0 (0/13)	0 (0/1)	4.4 (4/90)
Hemorrhage	13.3 (2/15)	7.7 (2/26)	15.2 (7/46)	0 (0/3)	15.9 (7/44)	8.9 (4/45)	0 (0/1)	0 (0/13)	15.9 (10/63)	7.7 (1/13)	0 (0/1)	12.2 (11/90)
<i>Forestomach, parasitic cyst</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	0 (0/13)	1.6 (1/63)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Liver, parasitic cyst</i>	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	1.6 (1/63)	7.7 (1/13)	0 (0/1)	2.2 (2/90)
<i>Liver, cholestasis</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	0 (0/63)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Liver, parasitic cyst</i>	6.7 (1/15)	0 (0/26)	0 (0/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	0 (0/13)	1.6 (1/63)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Liver, perforation</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	1.6 (1/63)	0 (0/13)	0 (0/1)	1.1 (1/90)
Miscellaneous	6.7 (1/15)	11.5 (3/26)	6.5 (3/46)	0 (0/3)	9.1 (4/44)	6.7 (3/45)	0 (0/1)	7.7 (1/13)	7.9 (5/63)	7.7 (1/13)	0 (0/1)	7.8 (7/90)
No macroscopic alterations	46.7 (7/15)	46.2 (12/26)	32.6 (15/46)	33.3 (1/3)	31.8 (14/44)	46.7 (21/45)	0 (0/1)	46.2 (6/13)	31.7 (20/63)	69.2 (9/13)	0 (0/1)	38.9 (35/90)
Autolysis	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	6.8 (3/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	3.2 (2/63)	0 (0/13)	0 (0/1)	3.3 (3/90)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported.

b) Microscopic findings

Tongue

Twenty-nine (22.1%) cases were evaluated. Histopathological results are listed in table 41. In the latter cases, 75.9% (22/29) presented no histopathological findings. Glossitis was observed in .8% (4/29) of the cases; two of them with marked leukocyte infiltrate, epithelial ulceration and diffuse hemorrhage, with occasional/scattered ballooning degeneration and microvesicles.

Table 41 - Tongue: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Glossitis</i>	0.0 (0/4)	0.0 (0/7)	18.2 (2/11)	28.6 (2/7)	0.0 (0/14)	25.0 (3/12)	33.3 (1/3)	10.0 (2/20)	0.0 (0/1)	0.0 (0/1)	28.6 (2/7)	13.8 (4/29)
<i>Hemorrhage</i>	0.0 (0/4)	0.0 (0/7)	9.1 (1/11)	42.9 (3/7)	7.1 (1/14)	8.3 (1/12)	66.7 (2/3)	5.0 (1/20)	0.0 (0/1)	0.0 (0/1)	42.9 (3/7)	13.8 (4/29)
<i>Microvesicles</i>	0.0 (0/4)	0.0 (0/7)	9.1 (1/11)	14.3 (1/7)	0.0 (0/14)	8.3 (1/12)	33.3 (1/3)	5.0 (1/20)	0.0 (0/1)	0.0 (0/1)	14.3 (1/7)	6.9 (2/29)
<i>Ulceration</i>	0.0 (0/4)	0.0 (0/7)	0.0 (0/11)	28.6 (2/7)	0.0 (0/14)	8.3 (1/12)	33.3 (1/3)	0.0 (0/20)	0.0 (0/1)	0.0 (0/1)	28.6 (2/7)	6.9 (2/29)
<i>Ballooning degeneration</i>	0.0 (0/4)	0.0 (0/7)	9.1 (1/11)	0.0 (0/7)	0.0 (0/14)	8.3 (1/12)	0.0 (0/3)	5.0 (1/20)	0.0 (0/1)	0.0 (0/1)	0.0 (0/7)	3.4 (1/29)
<i>Congestion</i>	0.0 (0/4)	0.0 (0/7)	0.0 (0/11)	14.3 (1/7)	0.0 (0/14)	0.0 (0/12)	33.3 (1/3)	0.0 (0/20)	0.0 (0/1)	0.0 (0/1)	14.3 (1/7)	3.4 (1/29)
<i>Sarcocystis Cyst</i>	0.0 (0/4)	0.0 (0/7)	9.1 (1/11)	0.0 (0/7)	7.1 (1/14)	0.0 (0/12)	0.0 (0/3)	5.0 (1/20)	0.0 (0/1)	0.0 (0/1)	0.0 (0/7)	3.4 (1/29)
<i>NHF</i>	100.0 (4/4)	100.0 (7/7)	63.6 (7/11)	57.1 (4/7)	85.7 (12/14)	75.0 (9/12)	33.3 (1/3)	80.0 (16/20)	100.0 (1/1)	100.0 (1/1)	57.1 (4/7)	75.9 (22/29)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Esophagus

Fourteen (10.7%) cases were evaluated; histopathological results are listed in table 42. Cysts of *Sarcocystis* sp. were observed in 14.3% (10/14) of the cases. Hemodynamic disturbances and inflammatory response was diagnosed in two cases. Histopathologic changes were not observed in 71.4% (10/14) of the cases.

Rumen

Fifty-two (39.7%) cases were evaluated; histopathological results are listed in table 43. Edema of the lamina propria (5.8%; 3/52) and rumenitis (3.8%; 2/52) were the most commonly observed findings. Edema was diagnosed in two adults and one fawn. Rumenitis was diagnosed in a juvenile and in an infant in good body condition. Hemorrhage was diagnosed in one female fawn in good body condition that had evidences of trauma. No histopathological findings were observed in 84.6% (44/52) of the cases.

Table 42 - Esophagus: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)			Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	U	
<i>Sarcocystis cyst</i>	0 (0/2)	0 (0/2)	28.6 (2/7)	0 (0/3)	0 (0/6)	40 (2/5)	0 (0/3)	22.2 (2/3)	0 (0/2)	0 (0/3)	14.3 (2/14)
<i>Edema</i>	0 (0/2)	0 (0/2)	14.3 (1/7)	0 (0/3)	16.7 (1/6)	0 (0/5)	0 (0/3)	11.1 (1/3)	0 (0/2)	0 (0/3)	7.1 (1/14)
<i>Esophagitis</i>	0 (0/2)	0 (0/2)	0 (0/7)	33.3 (1/3)	0 (0/6)	0 (0/5)	33.3 (1/3)	0 (0/3)	0 (0/2)	33.3 (1/3)	7.1 (1/14)
<i>Hemorrhage</i>	0 (0/2)	0 (0/2)	0 (0/7)	33.3 (1/3)	0 (0/6)	0 (0/5)	33.3 (1/3)	0 (0/3)	0 (0/2)	33.3 (1/3)	7.1 (1/14)
<i>Ulceration</i>	0 (0/2)	0 (0/2)	0 (0/7)	33.3 (1/3)	0 (0/6)	0 (0/5)	33.3 (1/3)	0 (0/3)	0 (0/2)	33.3 (1/3)	7.1 (1/14)
<i>NHF</i>	100 (2/2)	100 (2/2)	57.1 (4/7)	66.7 (2/3)	83.3 (5/6)	60 (3/5)	66.7 (2/3)	66.7 (2/3)	100 (2/2)	66.7 (2/3)	71.4 (10/14)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Table 43 - Rumen: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopath findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Edema</i>	25 (1/4)	0 (0/13)	8 (2/25)	0 (0/10)	5 (1/20)	8 (2/25)	0 (0/7)	6.9 (2/29)	0 (0/5)	20 (1/5)	0 (0/13)	5.8 (3/52)
<i>Rumenitis</i>	25 (1/4)	7.7 (1/13)	0 (0/25)	0 (0/10)	5 (1/20)	4 (1/25)	0 (0/7)	6.9 (2/29)	0 (0/5)	0 (0/5)	0 (0/13)	3.8 (2/52)
<i>Ballooning degeneration</i>	25 (1/4)	0 (0/13)	0 (0/25)	0 (0/10)	5 (1/20)	0 (0/25)	0 (0/7)	3.4 (1/29)	0 (0/5)	0 (0/5)	0 (0/13)	1.9 (1/52)
<i>Hemorrhage</i>	25 (1/4)	0 (0/13)	0 (0/25)	0 (0/10)	5 (1/20)	0 (0/25)	0 (0/7)	3.4 (1/29)	0 (0/5)	0 (0/5)	0 (0/13)	1.9 (1/52)
<i>NHF</i>	50 (2/4)	92.3 (12/13)	84 (21/25)	90 (9/10)	85 (17/20)	84 (21/25)	85.7 (6/7)	79.3 (23/29)	100 (5/5)	80 (4/5)	92.3 (12/13)	84.6 (44/52)
<i>Autolysis</i>	0 (0/4)	0 (0/13)	8 (2/25)	10 (1/10)	5 (1/20)	4 (1/25)	14.3 (1/7)	6.9 (2/29)	0 (0/5)	0 (0/5)	7.7 (1/13)	5.8 (3/52)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Reticulum

Thirty-three (25.2%) cases were evaluated; histopathological results are listed in table 44. Of those, 93.9% (31/33) presented no histopathological changes. Only three histological findings were observed: ballooning degeneration, edema and reticulitis.

Table 44 - Reticulum: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Ballooning degeneration</i>	0 (0/4)	0 (0/5)	0 (0/16)	12.5 (1/8)	0 (0/15)	0 (0/12)	16.7 (1/6)	0 (0/18)	0 (0/4)	0 (0/2)	11.1 (1/9)	3 (1/33)
<i>Edema</i>	0 (0/4)	0 (0/5)	6.3 (1/16)	0 (0/8)	6.7 (1/15)	0 (0/12)	0 (0/6)	5.6 (1/18)	0 (0/4)	0 (0/2)	0 (0/9)	3 (1/33)
<i>Reticulitis</i>	0 (0/4)	0 (0/5)	0 (0/16)	12.5 (1/8)	0 (0/15)	0 (0/12)	16.7 (1/6)	0 (0/18)	0 (0/4)	0 (0/2)	11.1 (1/9)	3 (1/33)
<i>NHF</i>	100 (4/4)	100 (5/5)	93.8 (15/16)	87.5 (7/8)	93.3 (14/15)	100 (12/12)	83.3 (5/6)	94.4 (17/18)	100 (4/4)	100 (2/2)	88.9 (8/9)	93.9 (31/33)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings

Omasum

Twenty-six (19.8%) cases were evaluated. Edema was observed in only one adult female (table 45). 96.2% (25/26) of the cases did not present any al histopathologic changes.

Table 45 - Omasum: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopath findings	Age group % (Na/Ne)				Genre % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Edema</i>	0.0 (0/3)	0.0 (0/6)	8.3 (1/12)	0.0 (0/5)	9.1 (1/11)	0.0 (0/11)	0.0 (0/4)	6.3 (1/16)	0.0 (0/2)	0.0 (0/1)	0.0 (0/7)	3.8 (1/26)
<i>NHF</i>	100.0 (3/3)	100.0 (6/6)	91.7 (11/12)	100.0 (5/5)	90.9 (10/11)	100.0 (11/11)	100.0 (4/4)	93.8 (15/16)	100.0 (2/2)	100.0 (1/1)	100.0 (7/7)	96.2 (25/26)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=unreported; NHF=no histopathological findings

Abomasum

Forty-two (32.1%) cases were evaluated; histopathological results are listed in table 46. 83.3% (35/42) of the cases did not present any histopathological changes. Two adults (male and female) presented hemorrhage. One female fawn had signs of congestion, and one adult female presented edema.

Table 46 - Abomasum: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopath. findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Congestion</i>	14.3 (1/7)	0 (0/8)	0 (0/17)	0 (0/10)	7.7 (1/13)	0 (0/22)	0 (0/7)	0 (0/20)	16.7 (1/6)	0 (0/3)	0 (0/13)	2.4 (1/42)
<i>Hemorrhage</i>	0 (0/7)	0 (0/8)	11.8 (2/17)	0 (0/10)	7.7 (1/13)	4.5 (1/22)	0 (0/7)	10 (2/20)	0 (0/6)	0 (0/3)	0 (0/13)	4.8 (2/42)
<i>Edema</i>	0 (0/7)	0 (0/8)	5.9 (1/17)	0 (0/10)	7.7 (1/13)	0 (0/22)	0 (0/7)	5 (1/20)	0 (0/6)	0 (0/3)	0 (0/13)	2.4 (1/42)
<i>Autolysis</i>	0 (0/7)	0 (0/8)	5.9 (1/17)	30 (3/10)	0 (0/13)	9.1 (2/22)	28.6 (2/7)	5 (1/20)	0 (0/6)	0 (0/3)	23.1 (3/13)	9.5 (4/42)
<i>NHF</i>	85.7 (6/7)	100 (8/8)	82.4 (14/17)	70 (7/10)	84.6 (11/13)	86.4 (19/22)	71.4 (5/7)	85 (17/20)	83.3 (5/6)	100 (3/3)	76.9 (10/13)	83.3 (35/42)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings

Small intestine

Forty-six (35.1%) cases were evaluated; histopathological results are listed in table 47. 32.6% (15/46) of the cases did not present any histopathological changes. Enteritis (13.0%; 6/46), congestion (13.0%; 6/46) and hemorrhage (6.5%; 3/46) were the three main histopathological findings. Enteritis was more frequent in females than in males, characterized by mononuclear leukocyte infiltrates in four cases and mixed in two other cases. Hemorrhagic enteritis was diagnosed in one case. Severity ranged between moderate (three cases), moderate-severe (one), mild (one), and mild-moderate (one).

Table 47 - Small intestine: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Enteritis</i>	33.3 (2/6)	28.6 (2/7)	5.3 (1/19)	7.1 (1/14)	27.8 (5/18)	0 (0/18)	10 (1/10)	13.6 (3/22)	40 (2/5)	0 (0/4)	6.7 (1/15)	13 (6/46)
<i>Congestion</i>	33.3 (2/6)	14.3 (1/7)	5.3 (1/19)	14.3 (2/14)	22.2 (4/18)	0 (0/18)	20 (2/10)	9.1 (2/22)	40 (2/5)	0 (0/4)	13.3 (2/15)	13 (6/46)
<i>Hemorrhage</i>	0 (0/6)	0 (0/7)	15.8 (3/19)	0 (0/14)	5.6 (1/18)	11.1 (2/18)	0 (0/10)	13.6 (3/22)	0 (0/5)	0 (0/4)	0 (0/15)	6.5 (3/46)
<i>Edema</i>	0 (0/6)	0 (0/7)	5.3 (1/19)	0 (0/14)	5.6 (1/18)	0 (0/18)	0 (0/10)	4.5 (1/22)	0 (0/5)	0 (0/4)	0 (0/15)	2.2 (1/46)
<i>Serosal helminth cyst</i>	0 (0/6)	0 (0/7)	5.3 (1/19)	0 (0/14)	0 (0/18)	5.6 (1/18)	0 (0/10)	4.5 (1/22)	0 (0/5)	0 (0/4)	0 (0/15)	2.2 (1/46)
<i>NHF</i>	50 (3/6)	42.8 (3/7)	26.3 (5/19)	28.6 (4/14)	44.4 (8/18)	22.2 (4/18)	30 (3/10)	31.8 (7/22)	20 (1/5)	75 (3/4)	26.7 (4/15)	32.6 (15/46)
<i>Autolysis</i>	16.7 (1/6)	28.6 (2/7)	52.6 (10/19)	57.1 (8/14)	27.8 (5/18)	61.1 (11/18)	50 (5/10)	40.9 (9/22)	40 (2/5)	25 (1/4)	60 (9/15)	45.7 (21/46)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings

Large intestine

Seventeen (13%) cases were evaluated; histopathological results are listed in table 48. 35.3% (6/17) of the cases did not present any histopathological changes.

Three pathologic findings were observed: colitis (29.4%; 5/17), hemorrhage (17.6%; 3/17), and necrosis (17.6%; 3/17). The severity of the colitis cases ranged from moderate and mild (two cases each (40.0%; 2/5)), to mild-moderate (one case (20.0%; 1/5)). Four cases presented mononuclear (three) and mixed (one) leukocyte infiltrate. Necrotizing enterocolitis was diagnosed in three cases, while hemorrhagic colitis was diagnosed in only one case.

Table 48 - Large intestine: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group %(Na/Ne)				Genre %(Na/Ne)			Body Condition %(Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Colitis</i>	0 (0/1)	50 (2/4)	14.3 (1/7)	40 (2/5)	16.7 (1/6)	37.5 (3/8)	33.3 (1/3)	36.4 (4/11)	0 (0/1)	0 (0/1)	25 (1/4)	29.4 (5/17)
<i>Hemorrhage</i>	0 (0/1)	50 (2/4)	14.3 (1/7)	0 (0/5)	0 (0/6)	37.5 (3/8)	0 (0/3)	27.3 (3/11)	0 (0/1)	0 (0/1)	0 (0/4)	17.6 (3/17)
<i>Necrosis</i>	0 (0/1)	25 (1/4)	14.3 (1/7)	20 (1/5)	0 (0/6)	25.0 (2/8)	33.3 (1/3)	18.2 (2/11)	0 (0/1)	0 (0/1)	25 (1/4)	17.6 (3/17)
<i>NHF</i>	100 (1/1)	25 (1/4)	57.1 (4/7)	0 (0/5)	50 (3/6)	37.5 (3/8)	0 (0/3)	36.4 (4/11)	100 (1/1)	100 (1/1)	0 (0/4)	35.3 (6/17)
<i>Autolysis</i>	0 (0/1)	25 (1/4)	28.6 (2/7)	60 (3/5)	33.3 (2/6)	25 (2/8)	66.7 (2/3)	27.3 (3/11)	0 (0/1)	0 (0/1)	75 (3/4)	35.3 (6/17)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Liver

Ninety-two (70.2%) cases were analyzed. 10.9% (10/92) showed no histopathological changes. All histopathological processes are listed in table 49.

I) Hemodynamic Disorders

The most commonly diagnosed hemodynamic disorders were congestion (44.6%; 41/92) and hemorrhage (21.7%; 20/92). Congestion followed a histopathological pattern: generalized 39.0% (16/41), midzonal-centrilobular 34.1% (14/41), midzonal 19.5% (8/41), portal-midzonal 4.9% (2/41) and centrilobular 2.4% (1/41). Moderate congestion was diagnosed in 80.5% (33/41) of the cases, ranging between mild-moderate 9.8% (4/41), moderate-severe 7.3% (3/41), and severe 2.4% (1/41). Midzonal hemorrhage was observed in 35% (7/20) of the cases: generalized 25% (5/20), midzonal-centrilobular 20% (4/20), centrilobular 10% (2/20), and portal-midzonal 10% (2/20). Moderate hemorrhage was observed in 45% (9/20) of the cases.

Table 49 - Liver: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Steatosis</i>	60.0 (6/10)	42.9 (9/21)	55.3 (21/38)	60.9 (14/23)	45.9 (17/37)	56.1 (23/41)	71.4 (10/14)	50.0 (24/48)	55.6 (5/9)	57.1 (4/7)	60.7 (17/28)	54.3 (50/92)
<i>Congestion</i>	30 (3/10)	52.4 (11/21)	36.8 (14/38)	56.5 (13/23)	37.8 (14/37)	41.5 (17/41)	71.4 (10/14)	39.6 (19/48)	44.4 (4/9)	42.9 (3/7)	53.6 (15/28)	44.6 (41/92)
<i>Hepatitis</i>	30 (3/10)	38.1 (8/21)	42.1 (16/38)	43.5 (10/23)	32.4 (12/37)	41.5 (17/41)	57.1 (8/14)	41.7 (20/48)	22.2 (2/9)	28.6 (2/7)	46.4 (13/28)	40.2 (37/92)
<i>Hemorrhage</i>	10 (1/10)	19.0 (4/21)	23.7 (9/38)	26.1 (6/23)	10.8 (4/37)	34.1 (14/41)	14.3 (2/14)	25.0 (12/48)	11.1 (1/9)	0.0 (0/7)	25.0 (7/28)	21.7 (20/92)
<i>Bile duct hyperplasia</i>	20.0 (2/10)	19.0 (4/21)	21.1 (8/38)	13.0 (3/23)	24.3 (9/37)	17.1 (7/41)	7.1 (1/14)	20.8 (10/48)	11.1 (1/9)	14.3 (1/7)	17.9 (5/28)	18.5 (17/92)
<i>Hepatic necrosis</i>	10 (1/10)	9.5 (2/21)	21.1 (8/38)	26.1 (6/23)	18.9 (7/37)	9.8 (4/41)	42.9 (6/14)	14.6 (7/48)	11.1 (1/9)	0.0 (0/7)	32.1 (9/28)	18.5 (17/92)
<i>Leukocytosis</i>	10 (1/10)	19.0 (4/21)	7.9 (3/38)	8.7 (2/23)	10.8 (4/37)	12.2 (5/41)	7.1 (1/14)	14.6 (7/48)	0.0 (0/9)	0.0 (0/7)	10.7 (13/28)	10.9 (10/92)
<i>Hepatocellular binucleation</i>	0.0 (0/10)	4.8 (1/21)	7.9 (3/38)	0.0 (0/23)	8.1 (3/37)	2.4 (1/41)	0.0 (0/14)	6.3 (3/48)	0.0 (0/9)	0.0 (0/7)	3.6 (1/28)	4.3 (4/92)
<i>Cholestasis</i>	0.0 (0/10)	4.8 (1/21)	2.6 (1/38)	4.3 (1/23)	0.0 (0/37)	4.9 (2/41)	7.1 (1/14)	2.1 (1/48)	0.0 (0/9)	14.3 (1/7)	3.6 (1/28)	3.3 (3/92)
<i>Hemosiderosis</i>	0.0 (0/10)	0.0 (0/21)	0.0 (0/38)	4.3 (1/23)	0.0 (0/37)	0.0 (0/41)	7.1 (1/14)	0.0 (0/48)	0.0 (0/9)	0.0 (0/7)	3.6 (1/28)	1.1 (1/92)
<i>Megakaryocytes</i>	10 (1/10)	0.0 (0/21)	0.0 (0/38)	0.0 (0/23)	2.7 (1/37)	0.0 (0/41)	0.0 (0/14)	2.1 (1/48)	0.0 (0/9)	0.0 (0/7)	0.0 (0/28)	1.1 (1/92)
<i>Neoplasia</i>	0.0 (0/10)	0.0 (0/21)	2.6 (1/38)	0.0 (0/23)	0.0 (0/37)	2.4 (1/41)	0.0 (0/14)	2.1 (1/48)	0.0 (0/9)	0.0 (0/7)	0.0 (0/28)	1.1 (1/92)
<i>Thrombosis</i>	0.0 (0/10)	0.0 (0/21)	0.0 (0/38)	4.3 (1/23)	0.0 (0/37)	0.0 (0/41)	7.1 (1/14)	0.0 (0/48)	0.0 (0/9)	0.0 (0/7)	3.6 (1/28)	1.1 (1/92)
<i>NHF</i>	20.0 (2/10)	23.8 (5/21)	5.3 (2/38)	4.3 (1/23)	13.5 (5/37)	12.2 (5/41)	0.0 (0/14)	14.6 (7/48)	11.1 (1/9)	14.3 (1/7)	3.6 (1/28)	10.9 (10/92)
<i>Autolysis</i>	10 (1/10)	9.5 (2/21)	18.4 (7/38)	26.1 (6/23)	24.3 (9/37)	12.2 (5/41)	14.3 (2/14)	16.7 (8/48)	0.0 (0/9)	28.6 (2/7)	21.4 (6/28)	17.4 (16/92)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

II) Inflammation and Repair

Hepatitis was the third most common finding, most frequently observed in animals in good body condition. Leukocyte exudate characteristics were variable: mononuclear 45.9% (17/37), granulocytic 10.8% (4/37), and mixed 43.2% (16/37). Moderate hepatitis was presented in 45.9% (17/37) of the cases, ranging from mild-moderate 27.0% (10/37), to mild 24.3% (9/37), and moderate-severe 2.7% (1/37). Portal hepatitis was diagnosed in 59.5% (22/37).

III) Miscellaneous

Steatosis was the most commonly diagnosed histopathological process. Over 60% of the steatosis cases occurred in fawns and those animals that did not present trauma lesions. Occurrence in other epidemiological categories ranged from 40% to 56%. Steatosis was classified as macrovesicular (100.0%; 50/50) and microvesicular (20.0%; 10/50). Mild steatosis was observed in 30.0% (15/50) of the cases, moderate in 30.0% (15/50), mild-moderate in 20.0% (10/50), moderate-severe in 14.0% (7/50), and severe in 6.0% (3/50). Hepatocellular necrosis was observed in 18.5% of the cases, with severity ranging among moderate (41.2% (7/17)), mild-moderate (35.3% (6/17)), mild (17.6% (3/17)), and severe 5.9% (1/17). Steatosis was classified into midzonal 47.1% (8/17), midzonal-centrilobular 23.5% (4/17), generalized 17.6% (3/17), and portal-midzonal 11.8% (2/17).

Pancreas

Nine (6.9%) cases were evaluated. One adult female was diagnosed with multifocal intraductal trematode larvae without inflammatory response. The eight remaining cases did not present any histopathological findings.

Urinary system

a) Macroscopic changes

Interstitial nephritis (23.3%; 21/90), congestion (15.6%; 14/90) and hemorrhage (12.2%; 11/90) were the most common necropsy findings; other pathological processes are listed in table 50. No macroscopic changes were observed in 52.2% (47/90) of the cases. Among the inflammatory and necrotic processes, interstitial nephritis was the most frequently diagnosed (23.3%), present in 30.4% (14/46) of the

adults. Renal congestion (14.4%; 13/90) and urinary bladder hemorrhage (6.7%; 6/90) were the most common hemodynamic disturbances.

b) Microscopic findings

Kidney

Eighty-eight (67.2%) of the evaluated cases presented histopathological changes (listed in table 51): congestion (70.5%; 62/88), glomerulonephritis (21.6%; 19/88), and tubular degeneration (19.3%; 17/88) were the most frequent findings. 2.3% (2/88) of the evaluated cases presented no macroscopic changes.

I) Hemodynamic Disorders

Congestion was classified in three morphological patterns: cortical (9.7%; 6/62), corticomedullary (56.5%; 35/62) and medullary (33.9%; 21/62); moderate congestion was observed in 50.0% (21/62), mild moderate in 21.0% (13/62), mild in 19.4% (12/62), moderate severe in 6.5% (4/62), and severe in 3.2% (2/62). Renal congestion had an occurrence rate higher than 78% in adults and juveniles. Renal hemorrhage was observed in 9.1% (8/88) of the evaluated cases.

II) Inflammation and Repair

Glomerulonephritis (21.6%; 19/88) was the most common morphological pattern of inflammatory response. Its occurrence ranged from 15% to 30% in all categories. Proliferative glomerulonephritis corresponds to 89.5% (17/19), and membranous to 10.5% (2/19) of the evaluated cases. Moderate glomerulonephritis was observed in 47.4% (9/19), mild in 31.6% (6/19), and mild-moderate in 21.1% (4/19) of the cases.

Interstitial nephritis was observed in 10.2% (9/88) of the evaluated cases. Nephritis was most frequently classified as mild (66.7%; 6/9). Leukocyte exudate was observed in ten cases, and classified as mononuclear (eight) and mixed (two). Only one case of pyelonephritis was diagnosed. The diagnosis of membranous glomerulonephritis was performed with the aid of PAS staining.

Table 50 - Urogenital system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Interstitial nephritis</i>	20 (3/15)	15.4 (4/26)	30.4 (14/46)	0 (0/3)	22.7 (10/44)	24.4 (12/45)	0 (0/1)	23.1 (3/13)	23.1 (3/13)	23.8 (15/63)	0 (0/1)	23.3 (21/90)
<i>Glomerulonephritis</i>	6.7 (1/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	4.5 (2/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	2.2 (2/90)
<i>Urinary bladder, necrosis</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Uterus, endometritis</i>	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	0 (0/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	1.6 (1/63)	0 (0/1)	2.2 (2/90)
Inflammatory	26.7 (4/15)	26.9 (7/26)	32.6 (15/46)	0 (0/3)	31.8 (14/44)	26.7 (12/45)	0 (0/1)	30.8 (4/13)	30.8 (4/13)	28.6 (18/63)	0 (0/1)	28.9 (26/90)
<i>Kidney</i>	6.7 (1/15)	19.2 (5/26)	15.2 (7/46)	0 (0/3)	18.2 (8/44)	11.1 (5/45)	0 (0/1)	15.4 (2/13)	15.4 (2/13)	14.3 (9/63)	0 (0/1)	14.4 (13/90)
<i>Urinary bladder</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Congestion	6.7 (1/15)	23.1 (6/26)	15.2 (7/46)	0 (0/3)	18.2 (8/44)	13.3 (11/45)	0 (0/1)	15.4 (2/13)	15.4 (2/13)	15.9 (10/63)	0 (0/1)	15.6 (14/90)
<i>Kidney</i>	0 (0/15)	7.7 (2/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	4.8 (3/63)	0 (0/1)	3.3 (3/90)
<i>Urinary bladder</i>	6.7 (1/15)	11.5 (3/26)	4.3 (2/46)	0 (0/3)	2.3 (2/44)	11.1 (5/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	7.9 (5/63)	0 (0/1)	6.7 (6/90)
<i>Testicle</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (2/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Uterus</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	2.3 (2/44)	0 (0/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	0 (0/63)	0 (0/1)	1.1 (1/90)
Hemorrhage	6.7 (1/15)	23.1 (6/26)	8.7 (7/46)	0 (0/3)	9.1 (4/44)	15.6 (7/45)	0 (0/1)	7.7 (1/13)	7.7 (1/13)	14.3 (9/63)	0 (0/1)	12.2 (11/90)
<i>Kidney, atrophy</i>	6.7 (1/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	0 (0/45)	0 (0/1)	0 (0/13)	0 (0/13)	3.2 (2/63)	0 (0/1)	2.2 (2/90)
<i>Kidney, infarct</i>	6.7 (1/15)	0 (0/26)	0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	0 (0/63)	0 (0/1)	1.1 (1/90)
<i>Kidney, tubular crystals</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Urinary bladder, mucosa, thickening</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Urinary bladder, rupture</i>	6.7 (1/15)	0 (0/26)	0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	0 (0/63)	0 (0/1)	1.1 (1/90)
Miscellaneous	20 (3/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	4.5 (2/44)	8.9 (4/45)	0 (0/1)	0 (0/13)	15.4 (2/13)	6.3 (4/63)	0 (0/1)	6.7 (6/90)
Autolysis	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	6.8 (3/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	3.2 (2/63)	0 (0/1)	3.3 (3/90)
No macroscopic alterations	46.7 (7/15)	57.7 (15/26)	47.8 (22/46)	100 (3/3)	47.7 (21/44)	55.6 (25/45)	100 (1/1)	46.2 (6/13)	53.8 (7/13)	52.4 (33/63)	100 (1/1)	52.2 (47/90)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

III) Miscellaneous

Degenerative processes in the tubular epithelium were observed in 19.3% (17/88) of the evaluated cases, while cylinders 12.5% (11/88) and tubular calcification 1.1% (1/88) were less frequently observed. Acute tubular necrosis was observed in 2.3% (2/88) of the cases: one juvenile and one specimen of unknown (unreported) age.

Table 51 - Kidney: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Congestion</i>	33.3 (3/9)	80 (16/20)	78.4 (29/37)	63.6 (14/22)	71.9 (23/32)	66.7 (28/42)	78.6 (11/14)	77.3 (34/44)	100 (8/8)	25 (2/8)	64.3 (18/28)	70.5 (62/88)
<i>Glomerulonephritis</i>	33.3 (3/9)	30 (6/20)	18.9 (7/37)	13.6 (3/22)	34.4 (11/32)	14.3 (6/42)	14.3 (2/14)	20.5 (9/44)	37.5 (3/8)	12.5 (1/8)	21.4 (6/28)	21.6 (19/88)
<i>Tubular degeneration</i>	22.2 (2/9)	10 (2/20)	29.7 (11/37)	9.1 (2/22)	18.8 (6/32)	21.4 (9/42)	14.3 (2/14)	25 (11/44)	12.5 (1/8)	12.5 (1/8)	14.3 (4/28)	19.3 (17/88)
<i>Cylinders</i>	11.1 (1/9)	10 (2/20)	13.5 (5/37)	13.6 (3/22)	15.6 (5/32)	9.5 (4/42)	14.3 (2/14)	9.1 (4/44)	25 (2/8)	12.5 (1/8)	14.3 (4/28)	12.5 (11/88)
<i>Interstitial nephritis</i>	11.1 (1/9)	20.5 (4/20)	5.4 (2/37)	9.1 (2/22)	18.8 (11/32)	2.4 (1/42)	14.3 (2/14)	9.1 (4/44)	0 (0/8)	12.5 (1/8)	14.3 (4/28)	10.2 (9/88)
<i>Hemorrhage</i>	11.1 (1/9)	10 (2/20)	13.5 (5/37)	0 (0/22)	12.5 (4/32)	9.5 (4/42)	0 (0/14)	18.2 (8/44)	0 (0/8)	0 (0/8)	0 (0/28)	9.1 (8/88)
<i>Acute tubular necrosis</i>	0 (0/9)	5 (1/20)	0 (0/37)	4.5 (1/22)	0 (0/32)	2.4 (1/42)	7.1 (1/14)	0 (0/44)	12.5 (1/8)	0 (0/8)	3.6 (1/28)	2.3 (2/88)
<i>Tubular calcification</i>	0 (0/9)	0 (0/20)	2.7 (1/37)	0 (0/22)	3.1 (1/32)	0 (0/42)	0 (0/14)	2.3 (1/44)	0 (0/8)	0 (0/8)	0 (0/28)	1.1 (1/88)
<i>NHF</i>	22.2 (2/9)	0 (0/20)	0 (0/37)	0 (0/22)	6.3 (2/32)	0 (0/42)	0 (0/14)	2.3 (1/44)	0 (0/8)	12.5 (1/8)	0 (0/28)	2.3 (2/88)
<i>Autolysis</i>	22.2 (2/9)	35 (7/20)	32.4 (12/37)	54.5 (12/22)	31.3 (10/32)	40.5 (17/42)	42.9 (6/14)	38.6 (17/44)	0 (0/8)	25 (2/8)	50 (14/28)	37.5 (33/88)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Urinary bladder

Nineteen (14.5%) cases were evaluated; histopathological results are listed in table 52. No histopathological changes were observed in 84.2% (16/19) of the cases. Hemorrhage was observed in two cases, while cystitis was diagnosed in only one case. Both cases had no general or epidemiological data available.

Genital system

a) Macroscopic changes

Pathologic processes are listed in Table 50. Macroscopic changes were not observed in 52.2% of the evaluated cases.

Table 52 - Urinary bladder: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Genre % (Na/Ne)			Body Condition % (Na/Ne)			Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	P	U	
<i>Hemorrhage</i>	33.3 (1/3)	0 (0/7)	0 (0/7)	50 (1/2)	0 (0/7)	9.1 (1/11)	100 (1/1)	0 (0/13)	33.3 (1/3)	33.3 (1/3)	10.5 (2/19)
<i>Cystitis</i>	0 (0/3)	0 (0/7)	0 (0/7)	50 (1/2)	0 (0/7)	0 (0/11)	100 (1/1)	0 (0/13)	0 (0/3)	33.3 (1/3)	5.3 (1/19)
<i>Congestion</i>	0 (0/3)	0 (0/7)	14.3 (1/7)	0 (0/2)	0 (0/7)	9.1 (1/11)	0 (0/1)	7.7 (1/13)	0 (0/3)	0 (0/3)	5.3 (1/19)
<i>Necrosis</i>	0 (0/3)	0 (0/7)	0 (0/7)	50 (1/2)	0 (0/7)	0 (0/11)	100 (1/1)	0 (0/13)	0 (0/3)	33.3 (1/3)	5.3 (1/19)
<i>Ulceration</i>	0 (0/3)	0 (0/7)	0 (0/7)	50 (1/2)	0 (0/7)	0 (0/11)	100 (1/1)	0 (0/13)	0 (0/3)	33.3 (1/3)	5.3 (1/19)
<i>NHF</i>	66.7 (2/3)	100 (7/7)	85.7 (6/7)	50 (1/2)	100 (7/7)	81.8 (9/11)	0 (0/1)	92.3 (12/13)	66.7 (2/3)	66.7 (2/3)	84.2 (16/19)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; P=poor; U=not reported; NHF=no histopathological findings.

b) Microscopic findings

Testicular tissue from thirteen cases were evaluated; one presented moderate congestion. Moderate-severe hemorrhagic mixed balanopostitis with necrosis and congestion was diagnosed in penile tissues from two animals. No pathological processes were observed in the seven evaluated cases with of ovarian and uterine tissues available.

Endocrine system

a) Macroscopic changes

Adrenal glands were the only organ evaluated with pathological processes at necropsy. Congestion (2.2%; 2/90), megaly (1.1%; 1/90) and hemorrhage (1.1%; 1/90) were the only necropsy findings (table 53) observed. No macroscopic changes were diagnosed in 85.7% of the evaluated cases. Congestion was observed in adults only, one female and one male. Adrenomegaly was found in one female and hemorrhage in one male. All gross findings were reported in traumatized animals.

b) Microscopic findings

Adrenal glands

Thirty-two (24.4%) cases were evaluated; histopathological findings are listed in table 54. Hemorrhage (56.3%; 18/32), congestion (31.3%; 10/32) and adrenalitis (9.4%; 3/32) were the three main pathological findings. No histopathological changes were diagnosed in 28.1% of the evaluated cases.

Table 53 - Endocrine system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
Adrenal congestion	0 (0/15)	0 (0/26)	4.3 (2/46)	0 (0/3)	2.3 (1/44)	2.2 (1/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	2.2 (2/90)
Adrenomegaly	0 (0/15)	0 (0/26)	0 (0/46)	33.3 (1/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Adrenal hemorrhage	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Autolysis	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	6.8 (3/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	3.2 (2/63)	0 (0/1)	3.3 (3/90)
No macroscopic alterations	100 (15/15)	96.2 (25/26)	87.0 (40/46)	66.7 (2/3)	86.4 (38/44)	95.6 (43/45)	100 (1/1)	84.6 (11/13)	92.3 (12/13)	92.1 (58/63)	100 (1/1)	91.1 (82/90)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported.

I) Hemodynamic disturbances

Hemorrhage was classified into the following morphological patterns: cortico-glomerular layer (5.6%; 1/18), cortico-reticular layer (38.9%; 7/18), cortical reticular-fascicular layers (33.3%; 6/18), and cortical-all layers (22.2%; 4/18). Mild hemorrhage was observed in 44.4% (8/18), moderate 33.3% (6/18), mild-moderate 11.1% (2/18), and moderate-severe 11.1% (2/18). Adrenal congestion ranged from moderate (50%; 5/10), to mild-moderate (30%; 3/10), to mild (20%; 2/10).

II) Inflammation

Adrenalitis was observed in three (9.4%) cases, presenting the following histopathological findings: mixed (lymphocytic and neutrophilic) (two), and neutrophilic leukocyte exudate (one), all presenting a cortical morphological pattern; mild-moderate (two) and moderate (one) adrenalitis.

Thyroid

Nine (6.9%; 9/131) cases were evaluated, with no histopathological findings.

Hematopoietic

a) Macroscopic changes

Megaly (13.3%; 12/90), congestion (8.9%; 8/90), and white pulp hyperplasia (8.9%; 8/90) were the three most frequently observed gross findings; other pathological processes are listed in table 55. No macroscopic changes were observed in 70.0% (63/90) of the evaluated cases.

Table 54 - Adrenal glands: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)			Total
	F	J	A	U	Fe	M	U	G	R	U	
<i>Hemorrhage</i>	0 (0/3)	42.9 (3/7)	62.5 (10/16)	83.3 (5/6)	52.9 (9/17)	53.8 (7/13)	100 (2/2)	57.9 (11/19)	20 (1/5)	75 (6/8)	56.3 (18/32)
<i>Congestion</i>	33.3 (1/3)	14.3 (1/7)	37.5 (6/16)	33.3 (2/6)	41.2 (7/17)	15.4 (2/13)	50 (1/2)	15.8 (3/19)	40 (2/5)	62.5 (5/8)	31.3 (10/32)
<i>Adrenatitis</i>	0 (0/3)	14.3 (1/7)	6.3 (1/16)	16.7 (1/6)	11.8 (2/17)	7.7 (1/13)	0 (0/2)	5.3 (1/19)	0 (0/5)	25 (2/8)	9.4 (3/32)
<i>Necrosis</i>	0 (0/3)	14.3 (1/7)	6.3 (1/16)	0 (0/6)	11.8 (2/17)	0 (0/13)	0 (0/2)	5.3 (1/19)	0 (0/5)	12.5 (1/8)	6.3 (2/32)
<i>Degeneration</i>	0 (0/3)	0 (0/7)	6.3 (1/16)	0 (0/6)	5.9 (1/17)	0 (0/13)	0 (0/2)	0 (0/19)	0 (0/5)	12.5 (1/8)	3.1 (1/32)
<i>NHF</i>	66.7 (2/3)	28.6 (2/7)	25.0 (4/16)	16.7 (1/6)	29.4 (5/17)	30.8 (4/13)	0 (0/2)	31.6 (6/19)	40 (2/5)	12.5 (1/8)	28.1 (9/32)
<i>Autolysis</i>	0 (0/3)	14.3 (1/7)	18.8 (3/16)	0 (0/6)	5.9 (1/17)	23.1 (3/13)	0 (0/2)	21.1 (4/19)	0 (0/5)	0 (0/8)	12.5 (4/32)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; U=not reported; NHF=no histopathological findings.

b) Microscopic findings

Spleen

Eighty-three (63.4%) cases were evaluated; histopathological findings are listed in table 56. White pulp depletion (32.5%; 27/83) and splenitis (20.5%; 17/83) were the three main findings. No macroscopic changes were observed in 24.1% of the evaluated cases.

White pulp depletion was mild in 3.7% (1/27), moderate in 55.6% (15/27), moderate-severe in 25.9% (7/27), and severe in 14.8% (4/27) of the cases. Age group (juvenile and adult), body condition (good) and trauma (traumatized) occurred in more than 40%.

Table 55 - Hematopoietic system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Lymphatic node, congestion</i>	6.7 (1/15)	7.7 (2/26)	6.5 (3/46)	0 (0/3)	6.8 (3/44)	6.7 (3/45)	0 (0/1)	15.4 (2/13)	7.7 (1/13)	4.8 (3/63)	0 (0/1)	6.7 (6/90)
<i>Spleen</i>	6.7 (1/15)	3.8 (1/26)	8.7 (4/46)	33.3 (1/3)	4.5 (2/44)	8.9 (4/45)	100 (1/1)	0 (0/13)	7.7 (1/13)	7.9 (5/63)	100 (1/1)	7.8 (7/90)
Megaly	13.3 (2/15)	7.7 (2/26)	15.2 (7/46)	33.3 (1/3)	11.4 (5/44)	13.3 (11/45)	100 (1/1)	15.4 (2/13)	15.4 (2/13)	11.1 (7/63)	100 (1/1)	13.3 (12/90)
<i>Lymphatic node, congestion</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Spleen</i>	0 (0/15)	15.4 (4/26)	6.5 (3/46)	0 (0/3)	6.8 (3/44)	8.9 (4/45)	0 (0/1)	7.7 (1/13)	7.7 (1/13)	7.9 (5/63)	0 (0/1)	7.8 (7/90)
Congestion	0 (0/15)	15.4 (4/26)	8.7 (4/46)	0 (0/3)	6.8 (3/44)	11.1 (5/45)	0 (0/1)	7.7 (1/13)	7.7 (1/13)	9.5 (6/63)	0 (0/1)	8.9 (8/90)
White pulp hyperplasia	6.7 (1/15)	15.4 (4/26)	6.5 (3/46)	0 (0/3)	6.8 (3/44)	11.1 (5/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	11.1 (7/63)	0 (0/1)	8.9 (8/90)
lymphadenitis	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Spleen, hemorrhage	6.7 (1/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	6.8 (3/44)	0 (0/45)	0 (0/1)	0 (0/13)	0 (0/13)	4.8 (3/63)	0 (0/1)	3.3 (3/90)
Spleen, pale	0 (0/15)	3.8 (1/26)	0.0 (0/46)	0 (0/3)	2.3 (2/44)	0 (0/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	0 (0/63)	0 (0/1)	1.1 (1/90)
No macroscopic alterations	80 (12/15)	61.5 (16/26)	71.7 (33/46)	66.7 (2/3)	68.2 (30/44)	73.3 (33/45)	0 (0/1)	69.2 (9/13)	76.9 (10/13)	69.8 (44/63)	0 (0/1)	70.0 (63/90)
Autolysis	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	2.2 (2/90)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; U=not reported.

Splenitis was most frequently observed in juveniles, males and animals in good body condition. Leukocyte infiltrates were characterized as neutrophilic (52.9% ;9/17), eosinophilic (29.4%; 5/17) and unidentified (17.6%; 3/17); this latter infiltrate was comprised of granulocytes not possible to identify. Splenitis was observed in two morphological patterns: red (17.6%; 3/17) and white pulp (41.2%; 7/17), and in both red and white pulps in seven cases. Hemosiderosis was observed in seven cases; two presenting megakaryocytes.

Lymphatic node

Nineteen (14.5%) cases were evaluated; histopathological findings are listed in table 57. Hemorrhage (26.3%; 5/19), congestion (15.8%; 3/19), and lymphatic proliferation (15.8%; 3/19), were the most common findings. No macroscopic changes were observed in 52.6% of the evaluated cases. Hemorrhage was moderate in four cases and moderate-severe in one case. All congestion cases were moderate. Hemosiderosis was observed in two cases, while anthracosis was observed in one case.

Thymus

Only 2.3% (3/131) of the cases were evaluated, none of these had histopathologic alterations.

Nervous system

a) Macroscopic changes

Congestion (11.1%; 10/90) and hemorrhage (4.4%; 4/90) were the main gross findings; other pathological processes are listed in table 58. No macroscopic changes were observed in 78.9% of the evaluated cases. Cerebrum (3.3%; 3/90) and subdural (1.1%; 1/90) hemorrhage were observed. A brain abscess was diagnosed in one juvenile male.

Table 56 Spleen: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>White pulp depletion</i>	0 (0/8)	42.1 (8/19)	42.4 (14/33)	21.7 (5/23)	35.5 (11/31)	35.1 (13/37)	20 (3/15)	42.9 (18/42)	28.6 (2/7)	0 (0/5)	24.1 (7/29)	32.5 (27/83)
<i>Mononuclear proliferation</i>	62.5 (5/8)	47.4 (9/19)	27.3 (9/33)	34.8 (8/23)	45.2 (14/31)	32.4 (12/37)	33.3 (5/15)	33.3 (14/42)	28.6 (2/7)	60 (3/5)	41.4 (12/29)	37.3 (31/83)
<i>Splenitis</i>	0 (0/8)	26.3 (5/19)	18.2 (6/33)	26.1 (6/23)	9.7 (3/31)	24.3 (9/37)	33.3 (5/15)	21.4 (9/42)	14.3 (1/7)	20 (1/5)	20.7 (6/29)	20.5 (17/83)
<i>Hemorrhage</i>	0 (0/8)	15.8 (3/19)	12.1 (4/33)	4.3 (1/23)	16.1 (5/31)	5.4 (2/37)	6.7 (1/15)	11.9 (5/42)	0 (0/7)	0 (0/5)	10.3 (3/29)	9.6 (8/83)
<i>Hemosiderosis</i>	0 (0/8)	10.5 (2/19)	12.1 (4/33)	4.3 (1/23)	9.7 (3/31)	8.1 (3/37)	6.7 (1/15)	11.9 (5/42)	0 (0/7)	20 (1/5)	3.4 (1/29)	8.4 (7/83)
<i>Necrosis</i>	0 (0/8)	5.3 (1/19)	9.1 (3/33)	13 (3/23)	9.7 (3/31)	8.1 (3/37)	6.7 (1/15)	9.5 (4/42)	0 (0/7)	0 (0/5)	10.3 (3/29)	8.4 (7/83)
<i>Megakaryocytes</i>	12.5 (1/8)	0 (0/19)	3 (1/33)	0 (0/23)	6.5 (2/31)	0 (0/37)	0 (0/15)	4.8 (2/42)	0 (0/7)	0 (0/5)	0 (0/29)	2.4 (2/83)
<i>NHF</i>	37.5 (3/8)	10.5 (2/19)	33.3 (11/33)	17.4 (4/23)	19.4 (6/31)	29.7 (11/37)	20 (3/15)	28.6 (12/42)	42.9 (3/7)	20 (1/5)	13.8 (4/29)	24.1 (20/83)
<i>Autolysis</i>	0 (0/8)	5.3 (1/19)	0 (0/33)	13 (3/23)	6.5 (2/31)	2.7 (1/37)	6.7 (1/15)	2.4 (1/42)	0 (0/7)	0 (0/5)	10.3 (3/29)	4.8 (4/83)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=Poor; U=not reported; NHF=no histopathological findings.

Table 57 - Lymphatic node: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Hemorrhage</i>	0 (0/3)	40 (2/5)	16.7 (1/6)	40 (2/5)	25 (2/8)	25 (2/8)	33.3 (1/3)	25 (3/12)	0 (0/2)	50 (2/4)	0 (0/1)	26.3 (5/19)
<i>Congestion</i>	0 (0/3)	20 (1/5)	0 (0/6)	40 (2/5)	12.5 (1/8)	12.5 (1/8)	33.3 (1/3)	8.3 (1/12)	0 (0/2)	50 (2/4)	0 (0/1)	15.8 (3/19)
<i>Lymphatic proliferation</i>	33.3 (1/3)	20 (1/5)	0 (0/6)	20 (1/5)	25 (2/8)	12.5 (1/8)	0 (0/3)	8.3 (1/12)	50 (1/2)	25 (1/4)	0 (0/1)	15.8 (3/19)
<i>Hemosiderosis</i>	0 (0/3)	0 (0/5)	16.7 (1/6)	20 (1/5)	12.5 (1/8)	0 (0/8)	33.3 (1/3)	8.3 (1/12)	0 (0/2)	25 (1/4)	0 (0/1)	10.5 (2/19)
<i>Anthraxis</i>	0 (0/3)	0 (0/5)	16.7 (1/6)	0 (0/5)	0 (0/8)	12.5 (1/8)	0 (0/3)	8.3 (1/12)	0 (0/2)	0 (0/4)	0 (0/1)	5.3 (1/19)
<i>Lymphadenitis</i>	0 (0/3)	20 (1/5)	0 (0/6)	0 (0/5)	12.5 (1/8)	0 (0/8)	0 (0/3)	8.3 (1/12)	0 (0/2)	0 (0/4)	0 (0/1)	5.3 (1/19)
<i>Lymphatic depletion</i>	0 (0/3)	0 (0/5)	0 (0/6)	20 (1/5)	0 (0/8)	0 (0/8)	33.3 (1/3)	0 (0/12)	0 (0/2)	25 (1/4)	0 (0/1)	5.3 (1/19)
<i>NHF</i>	66.7 (2/3)	40 (2/5)	50 (3/6)	60 (3/5)	62.5 (5/8)	37.5 (3/8)	66.7 (2/3)	50 (6/12)	50 (1/2)	50 (2/4)	100 (1/1)	52.6 (10/19)
<i>Autolysis</i>	0 (0/3)	0 (0/5)	16.7 (1/6)	0 (0/5)	0 (0/8)	12.5 (1/8)	0 (0/3)	8.3 (1/12)	0 (0/2)	0 (0/4)	0 (0/1)	5.3 (1/19)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=Poor; U=not reported; NHF=no histopathological findings.

Table 58 - Nervous system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Macroscopic changes	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
Brain; congestion	6.7 (1/15)	7.7 (2/26)	15.2 (7/46)	0 (0/3)	13.6 (6/44)	8.9 (4/45)	0 (0/1)	9.5 (6/63)	23.1 (3/13)	7.7 (1/13)	0 (0/1)	11.1 (10/90)
<i>Brain</i>	0 (0/15)	7.7 (2/26)	2.2 (1/46)	0 (0/3)	6.8 (3/44)	0 (0/45)	0 (0/1)	3.2 (2/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	3.3 (3/90)
<i>Subdural</i>	6.7 (1/15)	0 (0/26)	0 (0/46)	0 (0/3)	2.3 (2/44)	0 (0/45)	0 (0/1)	0 (0/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
Hemorrhage	6.7 (1/15)	7.7 (2/26)	2.2 (1/46)	0 (0/3)	9.1 (4/44)	0 (0/45)	0 (0/1)	3.2 (2/63)	15.4 (2/13)	0 (0/13)	0 (0/1)	4.4 (4/90)
Brain; hematoma	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	4.4 (2/45)	0 (0/1)	3.2 (2/63)	0 (0/13)	0 (0/13)	0 (0/1)	2.2 (2/90)
Meninges; thickening	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	2.3 (2/44)	0 (0/45)	0 (0/1)	0 (0/63)	0 (0/13)	7.7 (1/13)	0 (0/1)	1.1 (1/90)
Brain; abscess	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
Autolysis	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	6.8 (3/44)	0 (0/45)	0 (0/1)	3.2 (2/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	3.3 (3/90)
No macroscopic alterations	86.7 (13/15)	69.2 (18/26)	80.4 (37/46)	100 (3/3)	70.5 (31/44)	86.7 (39/45)	100 (1/1)	84.1 (53/63)	46.2 (6/13)	84.6 (11/13)	100 (1/1)	78.9 (71/90)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported.

b) Microscopic findings

Brain

Fifty-five (42%) cases were evaluated; histopathological findings are listed in table 59. Congestion was the most frequent finding (29.1%; 16/55). Moderate congestion was observed in 81.3% (13/16) of the cases, while 6.3% (1/16) presented mild, mild-moderate, moderate-severe congestion. Edema was observed in five cases, while hemorrhage was diagnosed in four cases: moderate (three) and severe (one). Nonsuppurative meningitis was diagnosed in one juvenile male presented with an antler fracture at the rehabilitation center. The animal developed progressive neurologic signs during an eight-weeks stay. Upon necropsy, purulent discharge expanded the meninges and the base of the antler. The following histological findings were observed: diffuse severe meningeal and sub-meningeal lymphocytic, histiocytic and rare neutrophil infiltrate, fibroplasia, necrosis, focally expansive white matter necrosis, gliosis (marked proliferation of astrocytes), and mild multifocal perivascular cuffs of histiocytes. Immunohistochemistry was performed to better evaluate the histological changes. GFAP immunolabeling revealed an extensive glial scar.

Table 59 - Brain: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopath. findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Congestion</i>	37.5 (3/8)	18.2 (2/11)	40 (10/25)	9.1 (1/11)	31.8 (7/22)	29.6 (8/27)	16.7 (1/6)	35.7 (10/28)	50 (4/8)	20 (1/5)	7.1 (1/14)	29.1 (16/55)
<i>Edema</i>	0 (0/8)	9.1 (1/11)	12 (3/25)	9.1 (1/11)	9.1 (2/22)	11.1 (3/27)	0 (0/6)	10.7 (3/28)	12.5 (1/8)	0 (0/5)	7.1 (1/14)	9.1 (5/55)
<i>Hemorrhage</i>	0 (0/8)	18.2 (2/11)	8 (2/25)	0 (0/11)	9.1 (2/22)	7.4 (2/27)	0 (0/6)	14.3 (4/28)	0 (0/8)	0 (0/5)	0.0 (0/14)	7.3 (4/55)
<i>Encephalitis</i>	25 (2/8)	0 (0/11)	0 (0/25)	9.1 (1/11)	4.5 (1/22)	7.4 (2/27)	0 (0/6)	0 (0/28)	12.5 (1/8)	20 (1/5)	7.1 (1/14)	5.5 (3/55)
<i>NHF</i>	37.5 (3/8)	45.5 (5/11)	52 (13/25)	54.5 (6/11)	45.5 (10/22)	44.4 (12/27)	83.3 (5/6)	50 (14/28)	50 (4/8)	40 (2/5)	50 (7/14)	49.1 (27/55)
<i>Autolysis</i>	12.5 (1/8)	9.1 (1/11)	8 (2/25)	36.4 (4/11)	18.2 (4/22)	14.8 (2/27)	0 (0/6)	3.6 (1/28)	0 (0/8)	20 (1/5)	42.9 (6/14)	14.5 (8/55)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Integumentary

a) Macroscopic changes

Skin laceration (34.4%; 31/90), hematoma (30.0%; 27/90) and ectoparasitosis (25.6%; 23/90) were the main gross findings; other pathological processes are listed in table 60. Macroscopic changes were not observed in 20.0% (18/90) of the cases.

Table 60 - Integumentary system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Flea</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	0.0 (0/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Myiasis</i>	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	4.4 (2/45)	0 (0/1)	1.6 (1/63)	0 (0/13)	7.7 (1/13)	0 (0/1)	2.2 (2/90)
<i>Ticks</i>	13.3 (2/15)	11.5 (3/26)	32.6 (15/46)	0 (0/3)	20.5 (9/44)	24.4 (11/45)	0 (0/1)	23.8 (15/63)	30.8 (4/13)	7.7 (1/13)	0 (0/1)	22.2 (20/90)
<i>Lice</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	1.6 (1/63)	0 (0/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Ectoparasitosis</i>	13.3 (2/15)	15.4 (4/26)	37 (17/46)	0 (0/3)	22.7 (10/44)	28.9 (10/45)	0 (0/1)	25.4 (16/63)	38.5 (5/13)	15.4 (2/13)	0 (0/1)	25.6 (23/90)
<i>Multifocal</i>	20 (3/15)	15.4 (4/26)	41.3 (19/46)	33.3 (1/3)	34.1 (15/44)	26.7 (12/45)	0 (0/1)	34.9 (22/63)	38.5 (5/13)	0 (0/13)	0 (0/1)	30.0 (27/90)
<i>Head</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	1.6 (1/63)	0 (0/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Hindlimbs</i>	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	4.4 (2/45)	0 (0/1)	1.6 (1/63)	0 (0/13)	7.7 (1/13)	0 (0/1)	2.2 (2/90)
<i>Skin laceration</i>	20 (3/15)	23.1 (6/26)	45.6 (21/46)	33.3 (1/3)	34.1 (15/44)	35.6 (16/45)	0 (0/1)	38.1 (24/63)	38.5 (5/13)	15.4 (2/13)	0 (0/1)	34.4 (31/90)
<i>Multifocal</i>	6.7 (1/15)	7.7 (2/26)	30.4 (14/46)	33.3 (1/3)	13.6 (6/44)	26.7 (12/45)	0 (0/1)	25.4 (16/63)	7.7 (1/13)	7.7 (1/13)	0 (0/1)	20 (18/90)
<i>Cervical</i>	0 (0/15)	0 (0/26)	4.3 (2/46)	0 (0/3)	4.5 (2/44)	0 (0/45)	0 (0/1)	1.6 (1/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	2.2 (2/90)
<i>Forelegs</i>	0 (0/15)	0 (0/26)	4.3 (2/46)	0 (0/3)	2.3 (1/44)	2.2 (1/45)	0 (0/1)	1.6 (1/63)	0 (0/13)	7.7 (1/13)	0 (0/1)	2.2 (2/90)
<i>Thorax</i>	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	2.3 (1/44)	2.2 (1/45)	0 (0/1)	1.6 (1/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	2.2 (2/90)
<i>Abdomen</i>	6.7 (1/15)	0 (0/26)	0.0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	1.6 (1/63)	0 (0/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Hindlimbs</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	1.6 (1/63)	0 (0/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Mandibular</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	1.6 (1/63)	0 (0/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Hematoma</i>	13.3 (2/15)	15.4 (4/26)	43.5 (20/46)	33.3 (1/3)	25.0 (11/44)	35.6 (16/45)	0 (0/1)	34.9 (22/63)	23.1 (3/13)	15.4 (2/13)	0 (0/1)	30 (27/90)
<i>Cyanosis</i>	6.7 (1/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	2.3 (1/44)	4.4 (2/45)	0 (0/1)	3.2 (2/63)	0 (0/13)	7.7 (1/13)	0 (0/1)	3.3 (3/90)
<i>Hindlimbs, edema</i>	0 (0/15)	3.8 (1/26)	0 (0/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	0.0 (0/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>Metatarsal fistula</i>	6.7 (1/15)	0 (0/26)	0 (0/46)	0 (0/3)	2.3 (1/44)	0 (0/45)	0 (0/1)	0.0 (0/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	1.1 (1/90)
<i>NHF</i>	33.3 (5/15)	26.9 (7/26)	10.9 (5/46)	33.3 (1/3)	20.5 (9/44)	17.8 (8/45)	100 (1/1)	23.8 (15/63)	0 (0/13)	15.4 (2/13)	100 (1/1)	20 (18/90)
<i>Autolysis</i>	0 (0/15)	3.8 (1/26)	6.5 (3/46)	0 (0/3)	6.8 (3/44)	2.2 (1/45)	0 (0/1)	4.8 (3/63)	7.7 (1/13)	0 (0/13)	0 (0/1)	4.4 (4/90)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

b) Microscopic findings

Skin

Nineteen (14.5%) cases were evaluated; histopathological results are listed in table 61. The most frequent findings were: dermatitis (31.6%; 6/19), necrosis of dermis and epidermis (31.6%; 6/19) and hyperkeratosis (15.8%; 3/19).

Table 61 - Skin: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Dermatitis</i>	50 (1/2)	66.7 (2/3)	25 (2/8)	16.7 (1/6)	25 (2/8)	44.4 (4/9)	0 (0/2)	28.6 (2/7)	33.3 (1/3)	50 (1/2)	28.6 (2/7)	31.6 (6/19)
<i>Necrosis</i>	50 (1/2)	66.7 (2/3)	25 (2/8)	16.7 (1/6)	25 (2/8)	44.4 (4/9)	0 (0/2)	28.6 (2/7)	33.3 (1/3)	50 (1/2)	28.6 (2/7)	31.6 (6/19)
<i>Hyperkeratosis</i>	0 (0/2)	66.7 (2/3)	12.5 (1/8)	0 (0/6)	0 (0/8)	33.3 (3/9)	0 (0/2)	14.3 (1/7)	0 (0/3)	50 (1/2)	14.3 (1/7)	15.8 (3/19)
<i>Ulceration</i>	50 (1/2)	33.3 (1/3)	12.5 (1/8)	0 (0/6)	12.5 (1/8)	22.2 (2/9)	0 (0/2)	14.3 (1/7)	33.3 (1/3)	0 (0/2)	14.3 (1/7)	15.8 (3/19)
<i>Acari</i>	0 (0/2)	0 (0/3)	12.5 (1/8)	0 (0/6)	0 (0/8)	11.1 (1/9)	0 (0/2)	14.3 (1/7)	0 (0/3)	0 (0/2)	0 (0/7)	5.3 (1/19)
<i>Fibrosis</i>	0 (0/2)	0 (0/3)	0 (0/8)	16.7 (1/6)	0 (0/8)	11.1 (1/9)	0 (0/2)	0 (0/7)	0 (0/3)	0 (0/2)	14.3 (1/7)	5.3 (1/19)
<i>Hemorrhage</i>	50 (1/2)	0 (0/3)	0 (0/8)	0 (0/6)	12.5 (1/8)	0 (0/9)	0 (0/2)	0 (0/7)	33.3 (1/3)	0 (0/2)	0 (0/7)	5.3 (1/19)
<i>NHF</i>	50 (1/2)	33.3 (1/3)	75 (6/8)	83.3 (5/6)	75 (6/8)	55.6 (5/9)	100 (2/2)	71.4 (5/7)	66.7 (2/3)	50 (1/2)	71.4 (5/7)	68.4 (13/19)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings.

Musculoskeletal system

a) Macroscopic changes

Bone fracture (28.9%; 26/90), rhabdomyolysis (17.8%; 16/90) and hemorrhage (8.9%; 8/90) were the most common gross findings. Other pathological processes are listed in table 62. No macroscopic changes were observed in 45.6% (41/90) of the cases. Hemorrhage was diagnosed in the cervical neck area, hind limbs (37.5%; 3/8), and forelegs. All other organs or structures presenting less frequent hemorrhagic changes were collectively classified as multifocal (12.5%; 1/8).

b) Microscopic findings

Skeletal muscle

Forty-four (33.6%) cases were evaluated; histopathological results are listed in table 63. Rhabdomyolysis (36.4%) was characterized as moderate (43.8%; 7/16), mild (37.5%; 6/16), mild-moderate, moderate-severe, severe (6.3%; 1/16). Only neutrophilic myositis (20.5%) was diagnosed. Cysts of *Sarcocystis* sp. were observed in seven cases, without any signs of inflammatory response.

Table 62 - Musculoskeletal system: macroscopic findings, biological and epidemiological aspects in brown brocket deer

Macroscopic alterations	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	F	J	A	U	Fe	M	U	G	R	P	U	
<i>Forelegs</i>	0 (0/15)	3.8 (1/26)	6.5 (3/46)	0 (0/3)	4.5 (2/44)	4.4 (2/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	4.8 (3/63)	0 (0/1)	4.4 (4/90)
<i>Hind limbs</i>	13.3 (2/15)	0 (0/26)	15.2 (7/46)	0 (0/3)	6.8 (3/44)	13.3 (11/45)	0 (0/1)	15.4 (2/13)	0 (0/13)	11.1 (7/63)	0 (0/1)	10 (10/90)
<i>Hip</i>	0 (0/15)	7.7 (2/26)	6.5 (3/46)	0 (0/3)	9.1 (4/44)	2.2 (1/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	6.3 (4/63)	0 (0/1)	5.6 (5/90)
<i>Spine</i>	0 (0/15)	11.5 (3/26)	0 (0/46)	0 (0/3)	2.3 (2/44)	4.4 (2/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	3.2 (2/63)	0 (0/1)	3.3 (3/90)
<i>Ribs</i>	6.7 (1/15)	0 (0/26)	6.5 (3/46)	0 (0/3)	6.8 (3/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	6.3 (4/63)	0 (0/1)	4.4 (4/90)
<i>Skull</i>	0 (0/15)	3.8 (1/26)	8.7 (4/46)	0 (0/3)	9.1 (4/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	6.3 (4/63)	0 (0/1)	5.6 (5/90)
Bone fracture	13.3 (2/15)	23.1 (6/26)	39.1 (18/46)	0 (0/3)	29.5 (13/44)	28.9 (13/45)	0 (0/1)	23.1 (3/13)	15.4 (2/13)	33.3 (21/63)	0 (0/1)	28.9 (26/90)
Rhabdomyolysis	26.7 (4/15)	15.4 (4/26)	15.2 (7/46)	33.3 (1/3)	25 (11/44)	11.1 (5/45)	0 (0/1)	15.4 (2/13)	23.1 (3/13)	17.5 (11/63)	0 (0/1)	17.8 (16/90)
<i>Cervical</i>	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	6.8 (3/44)	0 (0/45)	0 (0/1)	0 (0/13)	0 (0/13)	4.8 (3/63)	0 (0/1)	3.3 (3/90)
<i>Forelegs</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	2.3 (2/44)	0 (0/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
<i>Hindlimbs</i>	0 (0/15)	3.8 (1/26)	4.3 (2/46)	0 (0/3)	4.5 (2/44)	2.2 (1/45)	0 (0/1)	15.4 (2/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	3.3 (3/90)
<i>Multifocal</i>	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	2.3 (2/44)	0 (0/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Hemorrhage	0 (0/15)	7.7 (2/26)	13.0 (6/46)	0 (0/3)	15.9 (7/44)	2.2 (1/45)	0 (0/1)	15.4 (2/13)	0 (0/13)	9.5 (6/63)	0 (0/1)	8.9 (8/90)
Carpometacarpal joint, arthritis	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Knee, luxation	6.7 (1/15)	0 (0/26)	0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Muscle, atrophy	6.7 (1/15)	0 (0/26)	0 (0/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	7.7 (1/13)	0 (0/63)	0 (0/1)	1.1 (1/90)
Muscle, hindlimbs, edema	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Muscle, hip, phlegmon	0 (0/15)	0 (0/26)	2.2 (1/46)	0 (0/3)	0 (0/44)	2.2 (1/45)	0 (0/1)	0 (0/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	1.1 (1/90)
Autolysis	0 (0/15)	3.8 (1/26)	2.2 (1/46)	0 (0/3)	4.5 (2/44)	0 (0/45)	0 (0/1)	7.7 (1/13)	0 (0/13)	1.6 (1/63)	0 (0/1)	2.2 (2/90)
No macroscopic alterations	53.3 (8/15)	61.5 (16/26)	32.6 (15/46)	66.7 (2/3)	34.1 (15/44)	55.6 (25/45)	100 (1/1)	44.4 (28/63)	38.5 (5/13)	53.8 (7/13)	100 (1/1)	45.6 (41/90)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported.

Table 63 - Skeletal muscle: histopathological findings, biological and epidemiological aspects in brown brocket deer

Histopathological findings	Age group % (Na/Ne)				Gender % (Na/Ne)			Body Condition % (Na/Ne)				Total % (Na/Ne)
	Fawn	Juv	Adult	U	Fem	Male	U	Good	Reg	Poor	U	
Rhabdomyolysis	33.3 (1/3)	44.4 (4/9)	34.8 (8/23)	33.3 (3/9)	44.4 (8/18)	35 (7/20)	16.7 (1/6)	40.7 (11/27)	66.7 (2/3)	25 (1/4)	20 (2/10)	36.4 (16/44)
Hemorrhage	0 (0/3)	22.2 (2/9)	34.8 (8/23)	11.1 (1/9)	38.9 (7/18)	20 (4/20)	0 (0/6)	29.6 (8/27)	33.3 (1/3)	25 (1/4)	10 (1/10)	25.0 (11/44)
Myositis	0 (0/3)	22.2 (2/9)	21.7 (5/23)	22.2 (2/9)	27.8 (5/18)	10 (2/20)	33.3 (2/6)	14.8 (4/27)	33.3 (1/3)	50 (2/4)	20 (2/10)	20.5 (9/44)
Sarcocystis cyst	0 (0/3)	0 (0/9)	17.4 (4/23)	33.3 (3/9)	11.1 (2/18)	15 (3/20)	33.3 (2/6)	11.1 (3/27)	33.3 (1/3)	0 (0/4)	30 (3/10)	15.9 (7/44)
Myofiber degeneration	0 (0/3)	0 (0/9)	13 (3/23)	0 (0/9)	56 (1/18)	10 (2/20)	0 (0/6)	7.4 (2/27)	0 (0/3)	0 (0/4)	10 (1/10)	6.8 (3/44)
Congestion	0 (0/3)	11.1 (1/9)	0 (0/23)	0 (0/9)	5.6 (1/18)	0 (0/20)	0 (0/6)	3.7 (1/27)	0 (0/3)	0 (0/4)	0 (0/10)	2.3 (1/44)
NHF	66.7 (2/3)	44.4 (4/9)	30.4 (7/23)	22.2 (2/9)	33.3 (6/18)	35 (7/20)	33.3 (2/6)	33.3 (9/27)	33.3 (1/3)	50 (2/4)	30 (3/10)	34.1 (15/44)

Na=n° affected; Ne=n° examined; A=adult; F=fawn; J=juvenile; Fe=female; M=male; G=good; Reg.=regular; P=poor; U=not reported; NHF=no histopathological findings

5.4 CAUSES OF DEATH (CD)

Causes of death are listed in table 64.

Table 64 - Causes of death in Brazilian South American deer from 1995 to 2015

<i>Cause of death</i>	Marsh Deer	Brown Brocket Deer
<i>Respiratory</i>	53.3% (40/75)	25.2% (33/131)
<i>Euthanasia</i>	4.0% (3/75)	12.2% (16/131)
<i>Trauma</i>	4.0% (3/75)	9.2% (12/131)
<i>Nutritional</i>	4.0% (3/75)	5.3% (7/131)
<i>Alimentary</i>	4.0% (3/75)	0.8% (1/131)
<i>Cardiovascular, infectious</i>	0.0% (0/75)	2.3% (3/131)
<i>Stress, capture myopathy</i>	0.0% (0/75)	2.3% (3/131)
<i>Urinary</i>	2.7% (2/75)	0.8% (1/131)
<i>Parasitism</i>	1.3% (1/75)	0.0% (0/131)
<i>Nervous</i>	1.3% (1/75)	0.8% (1/131)
<i>Hematopoietic</i>	0.0% (0/75)	0.8% (1/131)
<i>Not Evident</i>	24.0% (18/75)	25.2% (33/131)
<i>Undetermined, autolysis</i>	1.3% (1/75)	16.0% (21/131)

5.4.1 CD in marsh deer.

The major causes of death in MD were, respiratory (53.3% 40/75), alimentary (4.0%; 3/75), nutritional (4.0%; 3/75), trauma (4.0%; 3/75), and euthanasia (4.0%; 3/75). Urinary, parasitism and nervous had lower occurrences. The CD could not be determined in 24.0% (18/75). Lastly, in 1.3% (1/75) the CD was undetermined due to autolysis.

The most common causes of death in fawns involved the **respiratory tract** (41.7%; 5/12) (named “respiratory”) and included: pulmonary edema (80.0%; 4/5), and pulmonary hemorrhage (20.0%; 1/5). **alimentary tract** involvement followed (25.0%; 3/12) (named “alimentary”), including: mycotic rumenitis (33.3%; 1/3), massive hepatocelular necrosis (33.3%; 1/3), and hemorrhagic hepatitis (33.3%; 1/3). **Parasite infestation** (8.3%; 1/12) (“parasite”), was considered the CD in one fawn. In 25.0% (3/12), the CD was not evident (table 65).

Table 65 - Causes of death, biological and epidemiological aspects of marsh deer

Causes of death	Age group % (Na/Ne)			Gender % (Na/Ne)		Total
	Fawn	Juvenile	Adult	Female	Male	
<i>Respiratory</i>	41.7 (5/12)	75 (9/12)	54.2 (26/48)	52.5 (21/40)	61.3 (19/31)	53.3 (40/75)
<i>Alimentary</i>	25 (3/12)	0 (0/12)	0 (0/48)	0 (0/40)	9.7 (3/31)	4 (3/75)
<i>Euthanasia</i>	0 (0/12)	0 (0/12)	6.3 (3/48)	7.5 (3/40)	0 (0/31)	4 (3/75)
<i>Nutritional</i>	0 (0/12)	8.3 (1/12)	4.2 (2/48)	7.5 (3/40)	0 (0/31)	4 (3/75)
<i>Traumatism</i>	0 (0/12)	0 (0/12)	6.3 (3/48)	5 (2/40)	3.2 (1/31)	4 (3/75)
<i>Urinary</i>	0 (0/12)	0 (0/12)	4.2 (2/48)	2.5 (1/40)	3.2 (1/31)	2.7 (2/75)
<i>Nervous</i>	0 (0/12)	0 (0/12)	2.1 (1/48)	0 (0/40)	3.2 (1/31)	1.3 (1/75)
<i>Parasitism</i>	8.3 (1/12)	0 (0/12)	0 (0/48)	2.5 (1/40)	0 (0/31)	1.3 (1/75)
<i>Not Evident</i>	25 (3/12)	16.7 (2/12)	20.8 (10/48)	20 (8/40)	19.4 (6/31)	24 (18/75)
<i>Undetermined</i>	0 (0/12)	0 (0/12)	2.1 (1/48)	2.5 (1/40)	0 (0/31)	1.3 (1/75)

Na=n° affected; Ne=n° examined;

The main CD for juveniles was **respiratory** (75.0%). In this category pulmonary edema (7/7) was the unique process. Secondly, nutritional (8.3%) corresponded to severe starvation. In 16.7% (2/12), the CD was not evident (table 65).

The main CD for adults was **respiratory** (54.2%). In this category, the pathological processes that lead to death in adults were: pulmonary edema (61.5%; 16/26), pneumonia (30.8%; 8/26), and pulmonary infarct (7.7%; 2/26). Secondly, multiple trauma (33.3%; 1/3), white pulp depletion (33.3%; 1/3) and pulmonary edema (33.3%; 1/3) were the associated diagnosis in **trauma** category (6.3%; 3/48). Multiple trauma was the sole cause of **euthanasia** (6.3%; 3/48). Severe starvation was the nutritional (4.2%; 2/48) CD in two adults. Suppurative pyelonephritis (1/2) and nephrosis (1/2) were the **urinary** (4.2%; 2/48) CD in two adults. **Nervous** (2.1%; 1/48) was represented by one case of spine cord compression. In 20.8% (10/48), the CD was not evident. One case was undetermined due to autolysis (Table 65).

The main CD for females was **respiratory** (52.5%; 21/40). The pathological processes that lead to death in females were: pulmonary edema (66.7%; 14/21), pneumonia (23.8%; 5/21), pulmonary hemorrhage (4.8%; 1/26) and infarct (4.8%; 1/26). Multiple trauma was the only cause of **euthanasia** (7.5%; 3/40). Severe starvation was the **nutritional** (7.5%; 3/40) CD in three females. **Trauma** (5.0%; 2/40) was the third CD; In this case severe hepatic steatosis was seen concurrently. Severe tick infestation was the **parasite** (2.5%; 1/40) CD. Diffuse suppurative pyelonephritis was the pathological processes that leading to death in **urinary** category (2.5%; 1/40). In 20.0% (8/40); the CD was not evident. One case was undetermined due to autolysis (table 65).

The main CD for males was **respiratory** (61.3%; 19/31). The pathological processes that lead to death in this category were: pulmonary edema (78.9%; 15/19), pneumonia (15.8%; 3/19), and pulmonary infarct (5.3%; 1/19). In fawns, mycotic rumenitis, hemorrhagic hepatitis and hepatic necrosis were the pathological processes that leading to death in the **alimentary** category (9.7%; 3/31). Nephrosis was the only CD of **urinary** (3.2%; 1/31), spinal cord compression of **nervous** (3.2%; 1/31), and trauma with concurrent interstitial pneumonia of **nervous** (3.2%; 1/31). In 19.4% (6/31) the CD was not evident (table 65).

5.4.2 CD in brown brocket deer.

The major causes of death in BBD were: Respiratory (25.2% 33/131), euthanasia (12.2%; 16/131), trauma (9.2%; 12/131). Infectious (2.3%; 3/131), and nervous (0.8%; 1/131) had lower occurrences. Death was ruled as undetermined in 25.2% (33/131). After a thorough investigation, clinical, pathological-anatomical and histopathological analysis, did not revealed a conclusive manner of death (table 64).

Respiratory (25.0%; 4/16) was the most common CD in fawns. The pathological processes that lead to death in fawns were: Pulmonary edema (75.0%; 3/4), and pneumonia (25.0%; 1/4). Severe starvation was the **nutritional** (18.8%; 3/16) CD in three cases. In 43.8% (7/16), the CD was not evident. Two cases were undetermined (table 66).

The main CD for juveniles was **respiratory** (22.2%; 6/27). The pathological processes that lead to death were, pulmonary edema (83.3%; 5/6), and pneumonia (16.7%; 1/6) were the pathological processes that leading to death. Multiple trauma was the only cause of **euthanasia** (14.8%; 4/27). **Trauma** (11.1%; 3/27) was the third CD, in this instance severe hepatic steatosis was seen concurrently. Severe starvation was the **nutritional** (3.7%; 1/27) CD in one juvenile. Hemorrhagic colitis was the only CD in the category **alimentary** (3.7%; 1/27). And severe chronic meningeal abscess was the only CD in the category **nervous** (3.7%; 1/27). In 22.2% (6/27), the CD was not evident. Four cases were undetermined due to autolysis (table 66).

The main CD for adults was **respiratory** (23.3%; 14/60). The pathological processes leading to death in adults were, pulmonary edema (57.1%; 8/14), and pneumonia (42.9%; 6/14). Secondly, multiple trauma was the only cause of **euthanasia** (18.3%; 11/60). **Trauma** (15.0%; 9/60) was the third CD in this instance severe hepatic steatosis and pulmonary edema were seen concurrently. Severe starvation was the CD in the category **nutritional** (5.0%; 3/60) with three adults.

Capture myopathy was the **stress** CD in two adults (3.3%; 2/60). Nephrosis was the only CD in the category urinary (1.7%; 1/60). Systemic hemorrhagic disease was the only CD in the category circulatory (1.7%; 1/60). In 13.3% (8/60), the CD was not evident. Eleven cases were undetermined due to autolysis (table 65).

Table 66 - Causes of death, biological and epidemiological aspects of brown brocket deer

Causes of death	Age group % (Na/Ne)			Gender % (Na/Ne)		Total
	Fawn	Juvenile	Adult	Female	Male	
Respiratory	25 (4/16)	22.2 (6/27)	23.3 (14/60)	18.5 (10/54)	28.8 (17/59)	25.2 (33/131)
Euthanasia, traumatism	0 (0/16)	14.8 (4/27)	6.7 (4/60)	7.4 (4/54)	6.8 (4/59)	6.1 (8/131)
Euthanasia, vehicle-collision	0 (0/16)	0 (0/27)	11.7 (7/60)	5.6 (3/54)	6.8 (4/59)	5.3 (7/131)
Euthanasia	0 (0/16)	14.8 (4/27)	18.3 (11/60)	13 (7/54)	13.6 (8/59)	11.5 (15/131)
Traumatism, intraenclosure	0 (0/16)	3.7 (1/27)	1.7 (1/60)	1.9 (1/54)	1.7 (1/59)	1.5 (2/131)
Traumatism, predator	0 (0/16)	0 (0/27)	1.7 (1/60)	0 (0/54)	1.7 (1/59)	0.8 (1/131)
Traumatism, undetermined	0 (0/16)	3.7 (1/27)	5 (3/60)	1.9 (1/54)	5.1 (3/59)	3.1 (4/131)
Traumatism, vehicle-collision	0 (0/16)	3.7 (1/27)	6.7 (4/60)	5.6 (3/54)	3.4 (2/59)	3.8 (5/131)
Trauma	0 (0/16)	11.1 (3/27)	15 (9/60)	9.3 (5/54)	11.9 (7/59)	9.2 (12/131)
Nutritional	18.8 (3/16)	3.7 (1/27)	5 (3/60)	5.6 (3/54)	6.8 (4/59)	5.3 (7/131)
Cardiovascular, infectious	0 (0/16)	0 (0/27)	1.7 (1/60)	1.9 (1/54)	1.7 (1/59)	2.3 (3/131)
Stress, capture myopathy	0 (0/16)	3.7 (1/27)	3.3 (2/60)	3.7 (2/54)	1.7 (1/59)	2.3 (3/131)
Digestive	0 (0/16)	3.7 (1/27)	0 (0/60)	0 (0/54)	1.7 (1/59)	0.8 (1/131)
Hematopoietic	0 (0/16)	0 (0/27)	0 (0/60)	0 (0/54)	0 (0/59)	0.8 (1/131)
Nervous	0 (0/16)	3.7 (1/27)	0 (0/60)	0 (0/54)	1.7 (1/59)	0.8 (1/131)
Urinary	0 (0/16)	0 (0/27)	1.7 (1/60)	0 (0/54)	1.7 (1/59)	0.8 (1/131)
<i>Not evident</i>	43.8 (7/16)	22.2 (6/27)	13.3 (8/60)	29.6 (16/54)	15.3 (9/59)	25.2 (33/131)
<i>Undetermined, autolysis</i>	12.5 (2/16)	14.8 (4/27)	18.3 (11/60)	18.5 (10/54)	15.3 (9/59)	16 (21/131)

Na=n° affected; Ne=n° examined.

The main CD for females was **respiratory** (18.5%; 10/54). The pathological processes leading to death in females were: Pulmonary edema (50.0%; 5/10), pneumonia (40.0%; 4/10), and pulmonary infarct (10.0%; 1/10) were the pathological processes leading to death in females. Multiple trauma was the only cause of **euthanasia** (13.0%; 7/54). **Trauma** (9.3%; 5/54) was the third CD. Severe starvation was the **nutritional** (5.6%; 3/54) CD in three females. Capture myopathy was the only CD in the category **stress** (3.7%; 2/54) with two cases. Systemic hemorrhagic disease was the only CD in the category circulatory (1.9%; 1/54). In 29.6% (16/54), the CD was not evident. Ten cases were undetermined due to autolysis (table 66).

The main CD for males was **respiratory** (28.8%; 1/59). The pathological processes leading to death in females were: Pulmonary edema (70.6%; 12/17), pneumonia (23.5%; 4/17), pulmonary hemorrhage (5.9%; 1/17). Multiple trauma was the only cause of **euthanasia** (13.6%; 8/59). **Trauma** (11.9%; 7/59) was the third CD. Severe starvation was the only CD in the category **nutritional** (6.8%; 4/59) in four males. Capture myopathy was the only CD in the category the **stress** (1.7%; 1/59). Systemic hemorrhagic disease was the only CD in the category **circulatory** (1.7%; 1/59). Severe chronic meningeal abscess was the CD in category **nervous** (1.7%; 1/59). In 15.3% (9/59) the CD was not evident. Nine cases were undetermined due to autolysis (table 66).

6 DISCUSSION

In Brazil, cattle breeding represent the second largest in the world, with about 200 million head. In 2015 the gross domestic product (GDP) of agriculture represented 21.46%, indicating its importance in the Brazilian economy. The extensive grazing system represents 93% in all regions of the country (FERRAZ; DE FELÍCIO, 2010; CEAEA, 2016).

This method of grazing favors the direct and indirect contact between cattle and wild ruminants. The epidemiology of human and livestock emerging infectious diseases shows the significant implication of wildlife. It has been estimated that 77% of mammalian livestock pathogens are generalist and can infect multiple species. One example of infectious disease with major economic implications is FMD. Outbreaks of this disease in free countries cause losses of >US\$1.5 billion a year. This disease has been widely studied in deer, showing infection, virulence and viral elimination. While FMD has been rarely reported in South American cattle, to the best of our knowledge, no reports of FMD in South American deer have been reported (DASZAK, CUNNINGHAM; HYATT, 2000; CLEAVELAND, LAURENSEN; TAYLOR, 2001; KNIGHT-JONES; RUSHTON, 2013). A potential spillover from cattle to deer populations in Brazil would have major detrimental socioeconomic consequences, the dimensions of which, are largely unknown. To provide scientific knowledge on baseline pathologic conditions that can be used by the scientific community, political and decision makers, we sought to find histopathological alterations of shared diseases between cattle and deer. The following paragraphs discuss the main findings and their possible relationship with bovine and human health.

RESPIRATORY

According to Radostits et al. (2007) pulmonary congestion is caused by an increase in the amount of blood in the lungs, may be a consequence of increased hydrostatic pressure, decreased oncotic pressure, increased capillary permeability and reduced lymphatic drainage. A great number of infectious, e.g., early stages of pneumonia, sepsis) and non-infectious (e.g., inhalation of smoke and fumes,

anaphylactic reactions, hypostasis, plant or drug intoxication, trauma) etiologies can produce these disturbances. Its diagnostic importance sometimes is reduced due to its lack of specificity (CASWELL; WILLIAMS, 2015). In deer, pulmonary congestion and edema are commonly associated with bluetongue, adenovirus, epizootic hemorrhagic disease and malignant catarrhal fever infection (PRESTWOOD et al., 1974; WOODS et al., 1996; WOODS et al., 1999; SORDEN et al., 2000; LEHMKUHL et al., 2001; KEEL et al., 2003). Severe tick infestation (4MD), severe starvation (6MD, 2BBD), trauma (9MD, 35BBD), and pneumonia (18MD, 10BBD) were observed in cases with pulmonary edema and congestion superior to moderate intensity. These pathologic processes are possibly correlated to pulmonary edema and congestion.

In domestic animals and livestock, pulmonary hemorrhage is frequently associated with thrombocytopenia, anticoagulant rodenticide toxicity, sepsis, disseminated intravascular coagulation, severe congestion, pulmonary hypertension, infarction, ruptured aneurysms, trauma and migrating foreign bodies (CASWELL; WILLIAMS, 2015). However, in cattle, it is uncommon, and associated with embolic pneumonia (RADOSTITS et al., 2007). In North American, European and South American deer infected by BTV, EHDV, and adenovirus, hemorrhages are a consistent featured due to endothelial damage (WOODS et al., 1996; WOODS et al., 1997; SORDEN; WOODS; LEHMKUHL 2000; LEHMKUHL; HOBBS; WOODS, 2001; NOON et al., 2002; FAVERO et al., 2013). In our research, pulmonary hemorrhage occurred in 27.1% MD and 30.5% BBD). This finding was found concomitantly with marked congestion, edema and in some cases with pneumonia. In two cases of MD with thrombosis we found hemorrhage as well.

Pneumonia is frequently associated with viruses, bacteria, or a combination, as well as fungi, metazoan parasites and physical and chemical agents. In cattle, most pneumonic processes are bronchogenic but some have a hematogenous origin (RADOSTITS et al., 2007). Pneumonia is one of the most relevant infectious processes in routine cervid pathology (WOBESER et al., 1975; ORR, 1991; RHYAN; SAARI, 1995; TURNQUIST; FALES, 1998; LUCIOLI et al., 2008; DA COSTA et al., 2008). In our study, we observed evidences of pneumonia in more than 50% research, representing up to 68.8% in MD. Mononuclear and mononuclear/granulocytic infiltrates were observed in interstitial pneumonia (occurrence= 47.9% MD; 53.8% BBD).

Distribution of infiltrate was mainly septal interstitium however, broncovascular and interlobar areas were also affected. Type II pneumocyte proliferation was an uncommon feature (2.9% MD; 11.6% BBD). Some cases of interstitial pneumonia in MD had nematode larvae, marked congestion, and minimal granulocytic infiltrate.

Bacterial pneumonia, typically manifested as bronchopneumonia, in cattle is commonly associated with *Manheimia hemolitica*, *Pasteurella multocida*, *Histophilus somnus*, *Mycoplasma bovis*, *Actinomyces pyogenes*, *Streptococcus* spp., *Actinobacillus actinoides*, and *Klebsiella* spp. in calves. In deer, major bacteria isolated in pneumonias are *Actinomyces pyogenes*, *Arcanobacterium pyogenes*, *Escherichia coli*, *Fusobacterium necrophorum*, *Klebsiella pneumoniae*, *M. haemolytica*, *Mycobacterium* sp., and *Streptococcus gallolyticus* (WOBESER et al., 1975; ORR, 1991; RHYAN; SAARI, 1995; TURNQUIST; FALES, 1998; LUCIOLI et al., 2008; DA COSTA et al., 2008). Bronchopneumonia was detected in 28.6% (20/70) of MD and 34.6% (28/52) of BBD. Three types of inflammatory infiltrate were identified: suppurative (BBD, MD), fibrinosuppurative (BBD, MD) and fibrinonecrotic (MD). In this study, we found intralesional cocci and coccobacillary bacterial colonies associated with bronchopneumonia (occurrence= 3.8% MD; 32.7% BBD). However, microbiological cultures were not performed. Based on morphological characteristics, gram staining properties and previous literature, we speculate *Manheimia* sp., and *Streptococcus* sp., could be involved in these cases.

Parasitic pneumonia by lungworms is relatively common in growing cattle, and is mostly caused by *Dictyocaulus viviparus* (SCOTT; PENNY; MACRAE, 2011). Clinical presentations include dyspnea, coughing, occasionally mild toxemia and inconsistent fever. The course may last several days (RADOSTITS et al., 2007). Most common lungworms in deer include *Dictyocaulus* spp. and *Protostrongylus* spp. In this study, parasitic pneumonias evidenced variable numbers of embryonated and non-embryonated nematode eggs, and nematode larvae in the alveolar lumens. Inflammatory infiltrates associated included macrophages and multinucleated giant cells, accompanied by type II pneumocyte hyperplasia, edema, and emphysema in fatal cases. Proliferation of first stage larvae causes diffuse interstitial pneumonia. Multiple granulomas in the parenchyma are also a common feature of verminous pneumonia (PRESTWOOD et al., 1971; SUTHERLAND, 1976; CHARLESTON, 1980;

RAHKO et al., 1992; MASON, 1994; HOSKIN et al., 2000; PANADERO et al., 2001; MAWHINNEY et al., 2010; CHARLESTON, 2011). In the present study, verminous pneumonia was diagnosed in 14 MD. Histopathological changes consisted of granulocytic and, in some cases, histiocytic (with giant multinucleate cells) inflammatory infiltrates, with or without larval phagocytosis, hemodynamic disturbances [mainly congestion, classified as moderate-severe and severe in seven cases (50%), edema (moderate-severe and severe in six cases (42.9%)), and little emphysema and fibrosis. Contemporaneous bacterial colonies were observed in 6 animals. Morphological characteristics of nematode eggs were compatible with Strongylida Order, presumably *Protostrongylus* or *Dictyocaulus* genera.

According to existing literature, mononuclear perivascularitis has been reported as a common histopathological lesion of MCF. In OvHV-II infection, perivascularitis is characterized by lymphocytic and plasmacytic inflammatory infiltrates in adventitial and sometimes subintimal areas in small to medium sized blood vessels in multiple organs (BROWN; BLOSS, 1992; TOMKINS et al., 1997; PALMER et al., 2013). Similarly, in CpHV-II infection, reports show marked infiltration of lymphocytes and minimal plasma cells on adventitia of small to medium sized blood vessels, with common skin lesions (KEEL et al., 2003; LI et al., 2003). In our study, mononuclear perivascularitis was identified in 20.8% MD. This inflammatory pattern is compatible with MCF; however, definitive etiological determination requires molecular analysis.

Anthracosis is considered an incidental finding in animals inhabiting nearby urban centers (OZKAN; BEYTUT, 2001). It is associated with poor air quality and may act as a risk factor for respiratory disease (SOUZA et al., 1998). Multifocal bronchial and peribronchial anthracosis together with hemosiderin-laden, was observed in four MD and in 6 BBD. These cases included animals that were traumatized in peripheral suburban areas, lending support to a possible relation to habitat quality.

Tracheitis frequently accompanies bronchitis and bronchopneumonia, as well as, hemodynamic disturbances (CASWELL; WILLIAMS, 2015). In this study, tracheal hemorrhage was the first pathologic condition (occurrence= MD 44.4%; BBD 21.4%). Tracheitis was observed in two MD and was associated with inflammatory and hemodynamic injuries in the lungs.

CARDIOVASCULAR

Myocardial hemorrhage occurs uncommonly in domestic ruminant pathology Robinson and Robinson (2015). Hemorrhage of the myocardium and epicardium is a common finding in BTV and EHDV infection. Petechial to ecchymotic hemorrhaging in the base of the aorta, just next to semilunar valves and pulmonary serosa, is a classical finding in orbiviral infection (LÓPEZ-OLVERA ET al., 2010; FALCONI et al., 2012; DROLET et al., 2013). We found hemorrhage of the heart to be the most common pathologic finding in the cardiovascular system, encompassing 19.5% in MD and 13.9% in BBD. Although we did not see hemorrhage in the base of the aorta, systemic hemorrhage and hemorrhage in the myocardium and epicardium plus clinical signs, rendered these animals suspect of orbiviral disease. Molecular analyses are needed to further identify the etiological agent in these cases.

Myocarditis is an uncommon condition in cattle. It can be caused by viruses and bacteria often associated with mastitis, endocarditis and respiratory disease; in large animal medicine, fungal myocarditis is an uncommon condition (SCOTT; PENNY; MACRAE, 2011). In humans, *Aspergillus* is the second most common cause of fungal myocarditis. Immunosuppression and angioinvasion are two common characteristics of *Aspergillus* infections (NOSANCHUK, 2002). *Aspergillus* sp. is often associated with respiratory disease in deer (JENSEN et al., 1989; LAAKKONEN, 1998). According to the scientific literature, white tailed deer and red deer with *Sarcocystis* infestation, frequently show signs of myocarditis (CALERO-BERNAL et al., 2015; REISSIG et al., 2016). Myocarditis was diagnosed in two BBD. *Sarcocystis* spp. cysts were found in the myocardium of 8.9% BBD; however, an associated inflammatory response was not evident. One myocarditis case (3.8% BBD), was ascribed to severe fungal infection. Fungal elements were highlighted by PAS and Grocott stains (GUARNER; BRANDT, 2011). Major histologic findings in this case consisted of marked necrosis of the myocardium, thrombosis and hemorrhage. Based on hyphae morphology, *Aspergillus* sp. was considered the most likely etiology. In the present case, the deer also diffuse embolic pneumonia, necrotizing esophagitis, neutrophilic splenitis and rhabdomyolysis. Fungal elements were observed only in heart tissue sections.

ALIMENTARY

Petechial to ecchymotic hemorrhage of gastrointestinal tract may be associated with BTV, EHDV and MCF infection, and is characterized by Hemorrhagic enterocolitis has been associated with MCF. (BROWN; BLOSS, 1992; TOMKINS et al., 1997; LÓPEZ-OLVERA et al., 2010; FALCONI et al., 2012; DROLET et al., 2013; PALMER et al., 2013). Other reported causes of gastrointestinal hemorrhaging in deer have included: abomasal ulceration concurrent with bacterial pneumonia, enterocolitis, chronic diarrhea and capture myopathy (PALMER; WATERS; WHIPPLE, 2001). In this research, hemorrhage was observed in the tongue (MD 6.7%; BBD 13.8%), rumen (BBD 1.9%), abomasum (MD 6.7%; BBD 4.8%), small intestine (BBD 6.5%) and large intestine (MD 66.7%; BBD 17.6%). Histopathological injuries were compatible with hemorrhagic disease, but due to the absence of molecular diagnosis, it is not possible to confirm an infection by either virus. In some cases of hemorrhage, we found lymphoplasmacytic perivascularitis in the lungs, so it is not possible to rule out MCF.

Actinobacillosis is one the most common opportunistic diseases of the oral cavity in ruminant medicine *Actinobacillus* spp., are considered normal flora of the oral cavity. (UZAL; PLATTNER; HOSTTTER, 2015). In deer, necrotizing glossitis may be observed in BTV cases (PRESTWOOD et al., 1974; STAIR; ROBINSON; JONES, 1968). There have been reports of purulent and necrotizing glossitis by *Mannheimia* spp. (KUPCA et al., 2014). In this research, inflammatory response was observed in tongue (MD 16.7%; BBD 13.8%), rumen (MD 7.1%; BBD 3.8%), reticulum (MD 7.7%; BBD 3.0%), abomasum (MD 13.3%), small intestine (MD 22.2%; BBD 13.0%) and large intestine (MD 100%; 29.4%). We observed necrotizing and hemorrhagic glossitis in cases with multi systemic hemorrhages, which is compatible with hemorrhagic disease. Although it was not possible to confirm the etiology, the presence of orbivirus (EHDV, BTV) cannot be rule out.

According to the literature, ruminal mycosis is a sequel of acute disease, sudden increase in the ruminal load and excessive accumulation of lactic acid (THOMSON, 1967; CORDES; SHORTRIDGE, 1968). Candidiasis in rumenitis has been recorded in wildebeest (PALING; KARSTAD; GROOTENHUIS, 1978). Ruminal mycosis was reported in white tailed deer and American bison (WOBESER; RUNGE, 1975; DYER;

NEWELL, 2002). In this study, one case of rumenitis was of fungal etiology. Morphologic features in this case were similar to previous reports, likely associated with *Candida* spp. (WOBESER; RUNGE, 1975; DYER; NEWELL, 2002).

In the pancreas, Dicrocoelid flukes of the genus *Eurytrema* are the most common in large animal medicine. In ruminant *Eurytrema pancreaticum* is the most important specie (JUBB; STENT, 2015). In Brazil, *Dicrocoellium coelomaticum* has been found in cattle (ILHA; LORETTI; REIS, 2005). In the present study, we found multiple intraductal trematode larvae, which were not associated with parenchymal injury. To our knowledge, there are no reports of pancreatic trematodiasis in South American deer.

Hepatocelular steatosis is common in injured hepatocytes, because the normal high throughput of fatty acids and triglycerides can be readily impeded at various points in the complex pathway of hepatic lipid metabolism and secretion of very low- density lipoproteins (VLDLs). In ruminants, the associations between hepatic steatosis, ketosis, and displacement of abomasum have been widely studied (CULLEN; STALKER 2015). In lambs, steatosis may be secondary to vitamin E deficiency (MENZIES et al., 2004). Despite the fact that this is a frequent finding in routine veterinary pathology, only were observed accounts of frequency of this condition in studies of general histopathologic diagnosis (HAIGH; BEREZOWSKI; WOODBURY, 2005; KRZYSIAK et al., 2014; REISSIG et al., 2016). In MD, steatosis was the second most common histopathological finding observed in the liver, homogeneously distributed between epidemiological categories (age group, gender, and body condition), classified as macrovesicular (58.6%), microvesicular (17.2%) and a combination of these two conditions (13.8%). Steatosis was severe in 6.9%. In BBD, it was the most commonly diagnosed histopathological process. Over 60% of the steatosis cases occurred in fawns and in animals with no trauma lesions. Occurrence in other epidemiological categories ranged from 40% to 56%. Steatosis was classified as macrovesicular (100.0%) and microvesicular (20.0%).

In bovine medicine, hepatic diseases include two groups: hepatitis and hepatosis. In cattle, hepatitis can be classified by etiology as follows: toxic (e.g., inorganic-organic poisons, plants, fungi, algae, insects), infectious (e.g., Rift Valley

fever virus, *Clostridium piliformis*, *Deltaproteobacterium*, flukes, migrating larvae of *Ascaris* sp.), and nutritional (selenium and vitamin E deficiency) (RADOSTITS et al., 2007). Hepatic necrosis is not considered a frequent cause of death in deer (HATTEL et al., 2004). Experimental infections with Bluetongue Virus caused hepatic hemodynamic disturbances in white tailed deer (KARSTAD; TRAINER 1967). Hepatic chronic degenerative processes (e.g., cirrhosis) caused by giant liver fluke (*Fascioloides magna*) has been described in some European species (NOVOBILSKÝ et al., 2007; KARAMON et al., 2015). Hepatitis E virus has been identified in some European and Asian deer, and successfully transmitted to humans through meat consumption (TEI et al., 2003; BOADELLA et al., 2010; RUTJES et al., 2010). However, deer do not display clinical disease. In MD, hepatitis was the main histopathological finding in the liver, homogeneously distributed into all epidemiological categories (age group, gender, and body condition). Portal hepatitis was observed in 72.4% (21/29). In BBD hepatitis was the third most common finding, most frequently observed in animals in good body condition. Leukocyte exudate characteristics were variable: mononuclear 45.9% (17/37), granulocytic 10.8% (4/37), and mixed 43.2% (16/37). The etiologies in these cases remain unknown.

URINARY

According to existing literature, active hyperemia is restricted to medulla and observed in acute septicemia and toxemia. Congestion is more often distributed to the cortico-medullary junction zone. Renal hemorrhages are restricted to the cortex in cases of bacteremia-viremia and are sometimes observed in healthy slaughtered animals (CIANCIOLO; MOHR, 2015). In MD, congestion was classified into 3 categories, according to its distribution: corticomedullary (47.8%; 22/46), medullary (41.3%; 19/46), and cortical (10.9%; 5/46). Renal congestion had an incidence of over 60.0% in all epidemiological categories. In BBD, congestion was classified in three morphological patterns: cortical (9.7%; 6/62), corticomedullary (56.5%; 35/62) and medullary (33.9%; 21/62). Renal congestion had an occurrence rate higher than 78% in adults and juveniles.

In veterinary pathology, glomerulonephritis is diagnosed frequently, and appears to be common in ruminant (CIANCIOLO; MOHR, 2015). Glomerulonephritis

was reported in 1960 by Christian, Flyger and Davis in a Sika deer. In MD, glomerulonephritis was observed in 10.6% (7/66) of the cases, aided by PAS, particularly in membranous glomerulonephritis cases. In BBD, glomerulonephritis (21.6%; 19/88) was the most common morphological pattern of renal inflammatory response. Its occurrence ranged from 15% to 30% in all categories. Proliferative glomerulonephritis corresponds to 89.5% (17/19), and membranous to 10.5% (2/19) of the evaluated cases. Moderate glomerulonephritis was observed in 47.4% (9/19), mild in 31.6% (6/19), and mild-moderate in 21.1% (4/19) of the cases.

Interstitial nephritis is rarely recognized as a cause of clinical disease in farm animals although it is a frequent postmortem finding in some species. In calves' nephritis is a common incidental finding at necropsy but does not present as a clinical urinary tract disease (RADOSTITS et al., 2007). Interstitial nephritis was observed in 25.8% of MD and 10.2% of BBD. The causes of Interstitial nephritis are seldom known and probably not specific (CIANCIOLO; MOHR, 2015). In MD, interstitial nephritis was the most common morphological pattern of inflammatory response (25.8%). Its occurrence ranged from 8% to 30% in all categories. In BBD, interstitial nephritis was observed in 10.2% (9/88) of the evaluated cases. Nephritis was most frequently classified as mild (66.7%; 6/9). Leukocyte exudate was observed in ten cases, and classified as mononuclear (eight) and mixed (two). Only one case of pyelonephritis was diagnosed. In our research, interstitial nephritis was considered a nonspecific finding.

Most common histological findings in deer with capture myopathy include: marked tubular dilatation and necrosis with protein (myoglobin) casts and acute rhabdomyolysis (WOBESER et al., 1976; WALLACE; BUSH; MONTALI, 1885; BERINGER et al., 1996; PATERSON, 2007; CATÃO-DIAS; CAMARGO, 2010; DECHEN QUINN et al., 2014; NUVOLI et al., 2014). In MD, acute tubular necrosis was observed in 13.6% (9/66) of the evaluated cases. In BBD, acute tubular necrosis was observed in 2.3% (2/88) of the cases: one juvenile and one specimen of unknown (unreported) age. In cases of sudden death necropsy examination often revealed muscular injury further supported by histological changes compatible with rhabdomyolysis. Hemoglobin (IHC positive) and necrotic casts were found in all cases.

Hemodynamic disturbances, such as hemorrhage in urinary bladder have been widely linked to infections by MCFV and adenovirus (SANFORD; LITTLE; RAPLEY, 1977; DENHOLM; WESTBURY, 1982; WOODS et al., 1999; SCHULTHEISS et al., 2007). In MD, congestion was present in four and cystitis in only one case. In BBD, Hemorrhage was observed in two cases, while cystitis was diagnosed in only one case. In this study, most cases with urinary bladder hemorrhage presented other lesions suggestive of MCF infection.

ENDOCRINE

The adrenal glands were enlarged as a result of swelling of the cortex. Gram-negative bacteria, especially coliforms, produce suppurative inflammation with necrosis as part of septicemia in many species (THOMAS; GRÖNE, 2015). In MD, six animals presented congestion and hemorrhage, mainly cortical (reticular and fascicular layers). Both hemodynamic disorders were more frequently observed in adults. In BBD, hemorrhage (56.3%; 18/32), congestion (31.3%; 10/32) and adrenalitis (9.4%; 3/32) were the three main pathological findings.

NERVOUS

Intracranial abscesses are common in North American deer, and is typically associated with secondary bacterial infection after trauma or antler fracture, being more frequent in males. Main bacteria isolated from intracranial abscess include *Trueperella pyogenes* and *Actinomyces pyogenes* and are considered an important cause of death in North American deer (DAVIDSON et al., 1990; BAUMANN, 2001; KARNS et al., 2009). Bacterial meningoencephalitis is also found in European with an apparent higher incidence of *Listeria monocytogenes* (ERIKSEN et al., 1998). In our research, encephalitis was observed in five BBD. A meningeal abscess was diagnosed in one juvenile male presented with an antler fracture at the rehabilitation center. The animal developed progressive neurologic signs during an eight-week stay. Upon necropsy, purulent discharge was present in the skull (meninges) and at the base of the antler. Histological findings were those of meningeal abscesses and IHC analysis based on anti-GFAP antibody revealed an extensive glial scar. Although the infectious agent was not evident, this lesion is presumed to be of bacterial origin.

TEGUMENTARY

Mite dermatitis has been commonly observed in North American species of deer, being trombidiosis and demodicosis most commonly reported. In trombidiosis, skinlesions are characterized by chronic diffuse plasma cell dermatitis with intralesional larvae of trombiculin mites. In demodicosis, infestation is found in hair follicles and sebaceous glands dilated and filled with large numbers of mites without associated inflammatory response (LITTLE; CARMICHAEL; RAKICH, 1997; GENTES; PROCTOR; WOBESER, 2007). Dermatitis was observed in 31.6% of BBD. In one case we observed a structure compatible with a mite.

MORTALITY

Despite comprehensive studies of mortality in North American and European deer have been published; To our knowledge, long term studies on mortality of South American species are very limited (OROZCO et al., 2013). In Argentinian MD, causes of death identified included: high parasite burdens, unusual adverse climatic conditions and apparent reduction in the availability of pastures (OROZCO et al., 2013). The present study revealed that in MD and BBD the most important causes of death involved respiratory disturbances. Our findings are in agreement with observations of respiratory disease as a major cause of death in free-ranging and captive North American deer populations (HATTEL et al., 2004; HATTEL et al., 2007).

Trauma was the second major cause of death in BBD (euthanasia 16 cases + trauma 12 cases), supporting previous that identified trauma as one of the leading causes of death in captive and free ranging deer (AUDIGÉ, WILSON; MORRIS, 2001; AGUIRRE; BROJER; MORNER, 1999; HATTEL et al., 2004; HAIGH; BEREZOWSKI; WOODBURY, 2005). In MD, most bone fractures were diagnosed in the skull 70.0% (7/10), ribs 40.0% (4/10), hip, spine, and forelegs 10.0% (1/10)., while in BBD, involved the hindlimbs (38.0%; 10/26), hip (19.2%; 6/26), skull (19.2%; 6/26), ribs (15.4%; 4/26), forelegs (15.4%; 4/26), and spine (11.5%; 3/26).

Predation is considered the most common source of mortality in free-ranging deer (BLEICH; TAYLOR, 1998; SMITH-FLUECK; FLUECK, 2001; VREELAND; DIEFENBACH; WALLINGFORD, 2004; KAMLER; JEDRZEJEWSKI; JEDRZEJEWSKA, 2007). Feral dog predation is a serious problem for biodiversity

conservation (CAUSEY; CUDE, 1980; BUTLER; DU TOIT; BINGHAM, 2004). In this study, fourteen BBD were predated by feral dogs, most often involving females and animals in good body condition. Most cases occurred in cold season (71.4%).

Vehicular trauma is one of the most important trauma-specific mortality in deer (NETTLES et al., 2002; VREELAND; DIEFENBACH; WALLINGFORD, 2004). According to Bager et al. (2016), more than 470 million animals die due to vehicle collision in Brazil annually. In our research, we observed 13 cases of vehicular trauma in BBD, mostly involving adult animals in good body condition. Most fatal collisions occurred in cold season. Contemporaneous pathological findings in these animals included: lymphoid depletion and pneumonia. There are three recognized group of risk factors in animal vehicle collision (AVC): animal (individual behavior and species ecology), traffic (vehicle, driver, traffic), and environmental (road corridor and landscape). Although uncertain in our cases, concomitant disease processes might have affected behavioral changes and predisposed to trauma, as previously recognized (DAVENPORT; DAVENPORT, 2006; WOBESER, 2006). If disease related behavioral change can lead to AVC, then pathological evaluation of AVC animals could be used as an indicator of population health.

Health surveillance of wild deer populations suggests that starvation is a basic factor predisposing to death in European and North American deer. Starvation is a direct result of forage-poor habitats. Fawns and older deer are most susceptible to starvation (CHEATUM, 1951; AGUIRRE; BROJER; MORNER, 1999; BENDER et al., 2004; FARMER; PERSON; BOWYER, 2006; WEBB; HEWITT; HELICKSON, 2007; MCDONALD et al., 2011). In our research, starvation was not a frequent cause of death in MD and BBD. Additionally, enterocolitis, gastrointestinal parasitism, myopathy and nephritis were identified as CD in few cases (AGUIRRE, BROJER; MORNER, 1999; AUDIGÉ; WILSON; MORRIS, 2001; NETTLES et al., 2002; HATTEL et al., 2004; HATTEL et al., 2007).

In fawns, infectious diseases (necrobacillosis) and starvation are the most important causes of morbidity and mortality (HAIGH; BEREZOWSKI; WOODBURY, 2005; HATTEL et al., 2007; CARSTENSEN et al., 2009).

7 FINAL COMMENTS

The aim of the present study was to identify and characterize the macroscopic and microscopic features of pathologic processes and possible etiological agents in two species of South American deer, in order to determine causes of death.

The most causes of death for MD were respiratory, alimentary, nutritional and trauma, plus euthanasia. In BBD, most causes of death were respiratory, euthanasia and trauma. Respiratory diseases were often defined by pulmonary edema and pneumonia. We have provided evidence that respiratory disease, mainly pneumonia, is a critical pathological process in deer. Although no etiologies were identified, there are evidences of both bacterial and viral etiology. We suggest that respiratory disease should be prioritized in South American deer health research.

Our results suggest trauma, mainly anthropogenic-trauma (Animal-Vehicle-Collision and feral dog predation), is a common ailment in BBD. We believe anthropogenic-trauma may become a primary threat for populations of BBD; however, further research is needed to confirm this assessment.

In contrast to the current scientific literature, starvation was not an important cause of death in MD and BBD. More than 3/4 of the population we studied showed good body condition. In our opinion, this fact is a good indicator of the ecosystem quality during the last 20 years. Contrary to what we expected, capture myopathy had a low occurrence. This suggests there have been improvements in the management of the animals; however, more research is needed.

In the present work, we identified, described, characterized and tabulated pathological processes in function to biological and epidemiological aspects. One of the most valuable outcomes of this work is to provide information about occurrence of each macroscopic and microscopic process. We hope this study fills in a gap of knowledge on baseline pathology in South American deer. We have documented the first reports of ruminal and myocardial mycosis in MD and BBD, respectively; first report

of pancreatic trematodiasis in BBD; and first histopathological findings suggestive of MCF in MD and BBD.

It is important to emphasize the importance of adequate macroscopic examinations, sampling, and data collection. Retrospective studies can only be performed if these are of adequate quality. In our opinion, pathologic findings in retrospective studies do not necessarily represent the current health status of wildlife populations; instead, they do provide an overview of the main pathological processes.

REFERENCES

- AALBÆK, B.; ERIKSEN, L.; RIMLER, R. B.; LEIFSSON, P. S.; BASSE, A.; CHRISTIANSEN, T.; ERIKSEN, E. Typing of *Pasteurella multocida* from haemorrhagic septicaemia in Danish fallow deer (*Dama dama*). **APMIS**, v. 107, n. 7-12, p. 913-920, 1999.
- ADETUNJI, S. A.; GOMEZ, G.; DE LA CONCHA-BERMEJILLO, A.; OLIVEIRA, F.; ARENAS-GAMBOA, A. M. Reticulo-ruminal milk accumulation (ruminal drinking) in five pre-ruminant White-Tailed Deer (*Odocoileus virginianus*) in Texas. **Brazilian Journal of Veterinary Pathology**, v. 9, n. 2, p. 47-54 2016.
- AGUIRRE, A. A.; BRÖJER, C.; MÖRNER, T. Descriptive epidemiology of roe deer mortality in Sweden. **Journal of Wildlife Diseases**, v. 35, n. 4, p. 753-762, 1999.
- ARADAIB, I. E.; AKITA, G. Y.; PEARSON, J. E.; OSBURN, B. I. Comparison of polymerase chain reaction and virus isolation for detection of Epizootic Hemorrhagic Disease Virus in clinical samples from naturally infected deer. **Journal of Veterinary Diagnostic Investigation**, v. 7, n. 2, p. 196-200, 1995.
- ARAUJO, J. P., NOGUEIRA, N. F., FUKUTA, T., HAIGH, J. C. Viral Diseases. In **Neotropical Cervidology: Biology and Medicine of Neotropical Deer** (DUARTE J. M. N.; GONZALEZ, S. eds.). Funep, 2010.
- ARAÚJO JR, J. P.; NOGUEIRA, M. F.; DUARTE, J. M. B. Survey for foot-and-mouth disease in the endangered marsh deer (*Blastocerus dichotomus*) from marshlands of the Parana River Basin, Brazil. **Journal of Wildlife Diseases**, v. 46, n. 3, p. 939-943, 2010.
- AUDIGÉ, L.; WILSON, R.; MORRIS, R. S. Disease and mortality on red deer farms in New Zealand. **The Veterinary Record**, v. 148, p. 334-340, 2001.
- BAGER, A.; DA SILVA LUCAS, P.; BOURSCHEIT, A.; KUCZACH, A.; MAIA, B. Os caminhos da conservação da biodiversidade brasileira frente aos impactos da infraestrutura viária. **Biodiversidade Brasileira**, n. 1, p. 75-86, 2016.
- BALSEIRO, A.; MARÍN, J. G.; SOLANO, P.; GARRIDO, J. M.; PRIETO, J. M. Histopathological classification of lesions observed in natural cases of paratuberculosis in free-ranging fallow deer (*Dama dama*). **Journal of Comparative Pathology**, v. 138, n. 4, p. 180-188, 2008.
- BAUGHMAN, B.; ZHANG, S.; JIN, L.; PACE, L. W.; COOLEY, J.; YAN, L.; ZHANG, M. Z. Diagnosis of Deerpox virus infection in a white-tailed deer (*Odocoileus virginianus*) fawn. **Journal of Veterinary Diagnostic Investigation**, v. 23, n. 5, p. 965-970, 2011.

BARRETT, M. W.; CHALMERS, G. A. Congenital anomalies in a neonatal White-Tailed Deer in Alberta. **Journal of Wildlife Diseases**, v. 11, n. 4, p. 497-501, 1975.

BAUMANN, C. D.; DAVIDSON, W. R.; ROSCOE, D. E.; BEHELER-AMASS, K. Intracranial abscessation in white-tailed deer of North America. **Journal of Wildlife Diseases**, v. 37, n. 4, p. 661-670, 2001.

BEAUDOIN, R. L.; SAMUEL, W. M.; STROME, C. P. A. A Comparative study of the parasites in two populations of white-tailed deer. **Journal of Wildlife Diseases**, v. 6, n. 1, p. 56-63, 1970.

BEIGLBÖCK, C.; STEINECK, T.; TATARUCH, F.; RUF, T. Environmental cadmium induces histopathological changes in kidneys of roe deer. **Environmental Toxicology and Chemistry**, v. 21, n. 9, p. 1811-1816, 2002.

BELAY, E. D.; GAMBETTI, P.; SCHONBERGER, L. B.; PARCHI, P.; LYON, D. R.; CAPELLARI, S.; MCQUISTON, J. H.; BRADLEY, K.; DOWDLE, G.; CRUTCHER, J. M.; NICHOLS, C. R. Creutzfeldt-Jakob disease in unusually young patients who consumed venison. **Archives of Neurology**, v. 58, n. 10, p. 1673-1678, 2001.

BENDER, L. C.; SCHIRATO, G. A.; SPENCER, R. D.; MCALLISTER, K. R.; MURPHIE, B. L. Survival, cause-specific mortality, and harvesting of male black-tailed deer in Washington. **Journal of Wildlife Management**, v. 68, n. 4, p. 870-878, 2004.

BERGOTTINI, R.; MATTIELLO, S.; CRIPPA, L.; SCANZIANI, E. Cilia-associated respiratory (CAR) bacillus infection in adult red deer, chamois, and roe deer. **Journal of wildlife diseases**, v. 41, n. 2, p. 459-462, 2005.

BERINGER, J.; HANSEN, L. P.; WILDING, W.; FISCHER, J.; SHERIFF, S. L. Factors affecting capture myopathy in White-Tailed Deer. **The Journal of wildlife management**, p. 373-380, 1996.

BLACK, H.; MITCHELL, P. J. The lesions of heat stroke in deer, and other respiratory evaporative-cooling animals. **New Zealand Veterinary Journal**, v. 57, n. 1, p. 70-70, 2009.

BLACK-DÉCIMA, P.; ROSSI, R. V.; VOGLIOTTI, A.; CARTES, J. L.; MAFFEI, L.; DUARTE, J. M. B.; JULIÁ, J. P. Brown brocket deer *Mazama gouazoubira* (Fischer 1814). In: GONZÁLEZ, S.; DUARTE, J. M. B. (Ed.). **Neotropical cervidology: biology and medicine of latin american deer**. Jaboticabal: UNESP, 2010. p. 190-201.

BLEICH, V. C.; TAYLOR, T. J. Survivorship and cause-specific mortality in five populations of mule deer. **The Great Basin Naturalist**, p. 265-272, 1998.

BLUTKE, A.; MÄRZ, K.; MATENAERS, C.; OSWALD, K.; HERMANN, W.; WANKE, R. Polycystic kidney disease in a European Roe Deer (*Capreolus capreolus*). **Journal of Zoo and Wildlife Medicine**, v. 44, n. 2, p. 487-490, 2013.

BOADELLA, M.; CASAS, M.; MARTÍN, M.; VICENTE, J.; SEGALÉS, J.; DE LA FUENTE, J.; GORTÁZAR, C. Increasing contact with hepatitis E virus in red deer, Spain. **Emerging Infectious Diseases**, v. 16, n. 12, p. 1994-1996, 2010.

BOJESSEN, A. M.; LARSEN, J.; PEDERSEN, A. G.; MÖRNER, T.; MATTSON, R.; BISGAARD, M. Identification of a novel *Mannheimia granulomatis* lineage from lesions in roe deer (*Capreolus capreolus*). **Journal of Wildlife Diseases**, v. 43, n. 3, p. 345-352, 2007.

BOYCE, W. M.; WOODS, L. W.; KEEL, M. K.; MACLACHLAN, N. J.; PORTER, C. O.; LEHMKUHL, H. D. An epizootic of adenovirus-induced hemorrhagic disease in captive black-tailed deer (*Odocoileus hemionus*). **Journal of Zoo and Wildlife Medicine**, v. 31, n. 3, p. 370-373, 2000.

BROWN, C. C.; BLOSS, L. L. An epizootic of malignant catarrhal fever in a large captive herd of white-tailed deer (*Odocoileus virginianus*). **Journal of Wildlife Diseases**, v. 28, n. 2, p. 301-305, 1992.

BUTLER, J. R. A.; DU TOIT, J. T.; BINGHAM, J. Free-ranging domestic dogs (*Canis familiaris*) as predators and prey in rural Zimbabwe: threats of competition and disease to large wild carnivores. **Biological Conservation**, v. 115, n. 3, p. 369-378, 2004.

BYERLY, C. S.; WILSON, R. B.; HOLSCHEIY, M. A. Pleural mesothelioma in a European spotted fallow deer (*Cervus dama*). **Journal of Wildlife Diseases**, v. 25, n. 4, p. 597-598, 1989.

CALERO-BERNAL, R.; HABELA, M. Á. First report of *Cephenemyia stimulator* (Diptera, Oestridae) parasitizing roe deer (*Capreolus capreolus*) in Extremadura (Spain). **Galemys**, v. 25, p. 29-34, 2013.

CALERO-BERNAL, R.; VERMA, S. K.; CERQUEIRA-CÉZAR, C. K.; SCHAFER, L. M.; VAN WILPE, E.; DUBEY, J. P. *Sarcocystis mehlhorni*, n. sp. (Apicomplexa: Sarcocystidae) from the black-tailed deer (*Odocoileus hemionus columbianus*). **Parasitology Research**, v. 114, n. 12, p. 4397-4403, 2015.

CARSTENSEN, M.; DELGIUDICE, G. D.; SAMPSON, B. A.; KUEHN, D. W. Survival, Birth Characteristics, and Cause-Specific Mortality of White-Tailed Deer Neonates. **The Journal of Wildlife Management**, v. 73, n. 2, p. 175-183, 2009.

CASWELL, J.L.; WILLIAMS, K. J. Respiratory system. In: MAXIE, G. (Ed.) **Jubb, Kennedy & Palmer's pathology of domestic animals**. e-Book. Oxford: Elsevier Health Sciences, 2015.

CATÃO-DIAS, J. L.; CAMARGO, C. M. S. Capture Myopathy. In: DUARTE, J. M. B.; GONZALEZ, S. (Org.). **Neotropical cervidology**: biology and medicine of latin american deer. Jaboticabal: FUNEP/IUCN, 2010. v. 1, p. 239-244.

CAULKETT, N. A.; CRIBB, P. H.; HAIGH, J. C. Comparative cardiopulmonary effects of carfentanil-xylazine and medetomidine-ketamine used for immobilization of Mule Deer and Mule Deer/White-Tailed Deer hybrids. **Canadian Journal of Veterinary Research**, v. 64, n. 1, p. 64, 2000.

CAUSEY, M. K.; CUDE, C. A. Feral dog and white-tailed deer interactions in Alabama. **The Journal of Wildlife Management**, v. 44, n. 2, p. 481-484, 1980.

CEAEA. PIB do agronegócio brasileiro. Centro de estudos avançados em economia aplicada - ESALQ/USP. 2016. Disponível em: <<http://www.cepea.esalq.usp.br/br/pib-do-agronegocio-brasileiro.aspx>>. Acesso em: 2016.

CHARLESTON, W. A. G. Lungworm and lice of the red deer (*Cervus elaphus*) and the fallow deer (*Dama dama*)—a review. **New Zealand Veterinary Journal**, v. 28, n. 8, p. 150-152, 1980.

CHASE, C. C.; BRAUN, L. J.; LESLIE-STEEN, P.; GRAHAM, T.; MISKIMINS, D.; RIDPATH, J. F. Bovine Viral Diarrhea Virus multiorgan infection in two White-Tailed Deer in southeastern South Dakota. **Journal of Wildlife Diseases**, v. 44, n. 3, p. 753-759, 2008.

CHEATUM, E. L. Disease in relation to winter mortality of deer in New York. **The Journal of Wildlife Management**, v. 15, n. 2, p. 216-220, 1951.

CHRISTIAN, J. J.; FLYGER, V.; DAVIS, D. E. Factors in the mass mortality of a herd of sika deer, *Cervus nippon*. **Chesapeake Science**, v. 1, n. 2, p. 79-95, 1960.

CIANCIOLO, R. E.; MOHR, F. C. Urinary System. In, MAXIE, G. (Ed.). **Jubb, Kennedy & Palmer's pathology of domestic animals**. e-Book. Oxford: Elsevier Health Sciences, 2015.

CLARK, K. A.; ROBINSON, R. M.; WEISHUHN, L. L.; MCCONNELL, S. T. E. W. A. R. T. Further observations on malignant catarrhal fever in Texas deer. **Journal of wildlife diseases**, v. 8, n. 1, p. 72-74, 1972.

CLEAVELAND, S.; LAURENSEN, M. K.; TAYLOR, L. H. Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. **Philosophical Transactions of the Royal Society of London B: biological sciences**, v. 356, n. 1411, p. 991-999, 2001.

CORDES, D. O.; SHORTRIDGE, E. H. Systemic phycomycosis and aspergillosis of cattle. **New Zealand Veterinary Journal**, v. 16, n. 5, p. 65-80, 1968.

CORTI, P.; SAUCEDO, C.; HERRERA, P. Evidence of Bovine Viral Diarrhea, but absence of Infectious Bovine Rhinotracheitis and bovine brucellosis in the endangered Huemul Deer (*Hippocamelus bisulcus*) in Chilean Patagonia. **Journal of Wildlife Diseases**, v. 49, n. 3, p. 744-746, 2013.

COUVILLION, C. E.; NETTLES, V. F.; RAWLINGS, C. A.; JOYNER, R. L. Elaeophorosis in white-tailed deer: pathology of the natural disease and its relation to oral food impactions. **Journal of wildlife diseases**, v. 22, n. 2, p. 214-223, 1986.

CULLER, J. M.; STALKER, M. L. Liver and Biliary System. In, MAXIE, G. (ed.) **Jubb, Kennedy & Palmer's pathology of domestic animals**. e-Book. Oxford: Elsevier Health Sciences, 2015.

DARDIRI, A. H.; LOGAN, L. L.; MEBUS, C. A. Susceptibility of white-tailed deer to experimental heartwater infections. **Journal of Wildlife Diseases**, v. 23, n. 2, p. 215-219, 1987.

DAS NEVES, C. G.; ROTH, S.; RIMSTAD, E.; THIRY, E.; TRYLAND, M. Cervid herpesvirus 2 infection in reindeer: a review. **Veterinary microbiology**, v. 143, n. 1, p. 70-80, 2010.

DAVENPORT, J.; DAVENPORT, J. L. **The Ecology of transportation**: managing mobility for the environment. [S.l.]: Springer, 2006.

DAVIDSON, W. R.; NETTLES, V. F.; HAYES, L. E.; HOWERTH, E. W.; COUVILLION, C. E. Epidemiologic features of an intracranial abscessation/suppurative meningoencephalitis complex in white-tailed deer. **Journal of Wildlife Diseases**, v. 26, n. 4, p. 460-467, 1990.

DASZAK, P.; CUNNINGHAM, A. A.; HYATT, A. D. Emerging infectious diseases of wildlife--threats to biodiversity and human health. **Science**, v. 287, n. 5452, p. 443-449, 2000.

DA COSTA, M. M.; DE ARAÚJO, M. M.; DE MORAES PEIXOTO, R.; DA COSTA KREWER, C.; RIET-CORREA, F.; DE VARGAS, A. C. Isolamento e caracterização de *Klebsiella pneumoniae* em veado catingueiro (*Mazama* spp)-relato de caso. **Veterinária Notícias**, v. 14, n. 2, p. 75-78, 2008.

DE JONG, C. B.; VAN WIEREN, S. E.; GILL, R. M. A.; MUNRO, R. Relationship between diet and liver carcinomas in roe deer in Kielder Forest and Galloway Forest. **Veterinary Record**, v. 155, n. 7, p. 197-200, 2004.

DEBBIE, J. G.; FRIEND, M. Lymphosarcoma in a white-tailed deer. **Bulletin of the Wildlife Disease Association**, v. 3, n. 1, p. 38-39, 1967.

DEEM, S. L.; NOSS, A. J.; VILLARROEL, R.; UHART, M. M.; KARESH, W. B. Disease survey of free-ranging grey brocket deer (*Mazama gouazoubira*) in the Gran Chaco, Bolivia. **Journal of Wildlife Diseases**, v. 40, n. 1, p. 92-98, 2004.

DENHOLM, L. J.; WESTBURY, H. A. Malignant catarrhal fever in farmed rusa deer (*Cervus Timorensis*): 1. clinico-pathological observations. **Australian Veterinary Journal**, v. 58, n. 3, p. 81-87, 1982.

DIAZ, R.; STÉEN, M.; FABER, W. E. Studies of a disease with ulcerative and necrotizing lesions in Swedish Roe Deer (*Capreolus capreolus*). **Journal of Zoo and Wildlife Medicine**, v. 27, n. 1, p. 71-75, 1996.

DINKINES, W. C.; LOCHMILLER, R. L.; BARTUSH, W. S.; DEYOUNG, C. A.; QUAILS JR, C. W.; FULTON, R. W. Cause-specific mortality of White-Tailed Deer as influenced by military training activities in southwestern Oklahoma. **Journal of Wildlife Diseases**, v. 28, n. 3, p. 391-399, 1992.

DONG, W.; PAN, Y.; LIU, J. The earliest Muntiacus (Artiodactyla, Mammalia) from the Late Miocene of Yuanmou, southwestern China. **Comptes Rendus Palevol**, v. 3, n. 5, p. 379-386, 2004.

DRIEMEIER, D.; BRITO, M. F.; TRAVERSO, S. D.; CATTANI, C.; CRUZ, C. E. F. Outbreak of malignant catarrhal fever in brown brocket deer (*Mazama gouazoubira*) in Brazil. **Veterinary Record**, v. 151, n. 9, p. 271-271, 2002.

DROLET, R.; LAROCHELLE, D.; GEBHART, C. J. Proliferative enteritis associated with *Lawsonia intracellularis* (ileal symbiont intracellularis) in White-Tailed Deer. **Journal of Veterinary Diagnostic Investigation**, v. 8, n. 2, p. 250-253, 1996.

DUARTE, J. M. B.; GONZÁLEZ, S.; MALDONADO, J. E. The surprising evolutionary history of South American deer. **Molecular Phylogenetics and Evolution**, v. 49, n. 1, p. 17-22, 2008.

DUARTE, J. M. B.; REIS, M. L. (Org.). **Plano de ação nacional para a conservação dos cervídeos ameaçados de Extinção**. Brasília: Instituto Chico Mendes de Conservação da Biodiversidade, (ICMBIO), 2012. (Série Espécies Ameaçadas, 22).

DUBEY, J. P.; RIGOULET, J.; LAGOURETTE, P.; GEORGE, C.; LONGEART, L.; LENET, J. L. Fatal transplacental neosporosis in a deer (*Cervus eldi siamensis*). **The Journal of Parasitology**, v. 82, n. 2, p. 338, 1996.

DUBEY, J. P.; SREEKUMAR, C.; ROSENTHAL, B. M.; VIANNA, M. C. B.; NYLUND, M.; NIKANDER, S.; OKSANEN, A. Redescription of *Besnoitia tarandi* (Protozoa: Apicomplexa) from the reindeer (*Rangifer tarandus*). **International Journal for Parasitology**, v. 34, n. 11, p. 1273-1287, 2004.

DUNCAN, C.; RIDPATH, J.; PALMER, M. V.; DRISKELL, E.; SPRAKER, T. Histopathologic and immunohistochemical findings in two white-tailed deer fawns persistently infected with bovine viral diarrhea virus. **Journal of Veterinary Diagnostic Investigation**, v. 20, n. 3, p. 289-296, 2008.

DYER, N. W.; NEWELL, T. K. Mycotic rumenitis in American bison (*Bison bison*). **Journal of Veterinary Diagnostic Investigation**, v. 14, n. 5, p. 414-416, 2002.

DYER, N. W.; KROGH, D. F.; SCHAAN, L. P. Pulmonary mycoplasmosis in farmed white-tailed deer (*Odocoileus virginianus*). **Journal of Wildlife Diseases**, v. 40, n. 2, p. 366-370, 2004.

ELAZHARY, M. A. S. Y.; FRECHETTE, J. L.; SILIM, A.; ROY, R. S. Serological evidence of some bovine viruses in the caribou (*Rangifer tarandus caribou*) in Quebec. **Journal of Wildlife Diseases**, v. 17, n. 4, p. 609-612, 1981.

ERIKSEN, L.; AALBÆK, B.; LEIFSSON, P. S.; BASSE, A.; CHRISTIANSEN, T.; ERIKSEN, E.; RIMLER, R. B. Hemorrhagic septicemia in fallow deer (*Dama dama*) caused by *Pasteurella multocida multocida*. **Journal of Zoo and Wildlife Medicine**, v. 30, n. 2, p. 285-292, 1999.

ERIKSEN, L.; LARSEN, H. E.; CHRISTIANSEN, T.; JENSEN, M. M.; ERIKSEN, E. An outbreak of meningo-encephalitis in fallow deer caused by *Listeria monocytogenes*. **The Veterinary Record**, v. 122, n. 12, p. 274-276, 1988.

ERDÉLYI, K.; DENCŐ, L.; LEHOCZKI, R.; HELTAI, M.; SONKOLY, K.; CSÁNYI, S.; SOLYMOSI, N. Endemic papillomavirus infection of roe deer (*Capreolus capreolus*). **Veterinary microbiology**, v. 138, n. 1, p. 20-26, 2009.

EVE, J. H.; KELLOGG, F. E. Management implications of abomasal parasites in Southeastern white-tailed deer. **The Journal of Wildlife Management**, v. 41, n. 2, p. 169-177, 1977.

FALCONI, C.; LÓPEZ-OLVERA, J. R.; GORTÁZAR, C. BTV infection in wild ruminants, with emphasis on red deer: a review. **Veterinary microbiology**, v. 151, n. 3, p. 209-219, 2011.

FARAJOLLAHI, A.; GATES, R.; CRANS, W.; KOMAR, N. Serologic evidence of West Nile virus and St. Louis encephalitis virus infections in white-tailed deer (*Odocoileus virginianus*) from New Jersey, 2001. **Vector-Borne & Zoonotic Diseases**, v. 4, n. 4, p. 379-383, 2004.

FARMER, C. J.; PERSON, D. K.; BOWYER, R. T. Risk factors and mortality of black-tailed deer in a managed forest landscape. **Journal of Wildlife Management**, v. 70, n. 5, p. 1403-1415, 2006.

FAVERO, C. M.; MATOS, A. C. D.; CAMPOS, F. S.; CÂNDIDO, M. V.; COSTA, É. A.; HEINEMANN, M. B.; STANCIOLI, E. F. B.; LOBATO, Z. I. P. Epizootic hemorrhagic disease in brocket deer, Brazil. **Emerging Infectious Diseases**, v. 19, n. 2, p. 346, 2013.

FERRAZ, J. B. S.; DE FELÍCIO, P. E. Production systems—An example from Brazil. **Meat science**, v. 84, n. 2, p. 238-243, 2010.

FINNEY, D. **Statistical method in biological assay**. London, UK: Griffin, 1952.

FLÅØYEN, A.; HANDELAND, K.; STUVE, G.; RYENG, K. A.; REFSUM, T. Experimental *Nartheicum ossifragum* nephrotoxicity in cervids from Norway. **Journal of Wildlife Diseases**, v. 35, n. 1, p. 24-30, 1999.

FLETCHER, W. O.; STALLKNECHT, D. E.; KEARNEY, M. T.; EERNISSE, K. A. Epizootic vesicular stomatitis in Colorado, 1982: some observations on the possible role of wildlife populations in an enzootic maintenance cycle. **Journal of Wildlife Diseases**, v. 23, n. 2, p. 192-198, 1987.

FLETCHER, W. O.; STALLKNECHT, D. E.; KEARNEY, M. T.; EERNISSE, K. A. Antibodies to vesicular stomatitis New Jersey type virus in White-Tailed Deer on Ossabaw Island, Georgia, 1985 to 1989. **Journal of Wildlife Diseases**, v. 27, n. 4, p. 675-680, 1991.

FOREYT, W. J.; LEATHERS, C. W. Traumatic reticulopericarditis in a Mule Deer. **Journal of Wildlife Diseases**, v. 22, n. 3, p. 446-446, 1986.

FOREYT, W. J. Experimental *Fascioloides magna* infections of Mule Deer (*Odocoileus hemionus hemionus*). **Journal of Wildlife Diseases**, v. 28, n. 2, p. 183-187, 1992.

GEIST, V. **Deer of the world: their evolution, behaviour, and ecology**. [S.l.]: Stackpole books, 1998.

GENNARI, S. M., MAYUMI, S., SOARES, R. M., MACHADO, R. Z. Protozoan diseases. In **Neotropical Cervidology: Biology and Medicine of Neotropical Deer** (DUARTE J. M. N.; GONZALEZ, S. eds.). Funep, 2010.

GENTES, M. L.; PROCTOR, H.; WOBESER, G. Demodicosis in a mule deer (*Odocoileus hemionus hemionus*) from Saskatchewan, Canada. **Journal of wildlife diseases**, v. 43, n. 4, p. 758-761, 2007.

GENTRY, A. W. The Miocene differentiation of old world Pecora (Mammalia). **Historical Biology**, v. 7, n. 2, p. 115-158, 1994.

GILBERT, C.; ROPIQUET, A.; HASSANIN, A. Mitochondrial and nuclear phylogenies of Cervidae (Mammalia, Ruminantia): systematics, morphology, and biogeography. **Molecular Phylogenetics and Evolution**, v. 40, n. 1, p. 101-117, 2006.

GIOVANNINI, A.; CANCELLOTTI, F. M.; TURILLI, C.; RANDI, E. Serological investigations for some bacterial and viral pathogens in fallow deer (*Cervus dama*) and wild boar (*Sus scrofa*) of the San Rossore Preserve, Tuscany, Italy. **Journal of Wildlife Diseases**, v. 24, n. 1, p. 127-132, 1988.

GOBLE, F. C. Tissue changes in white-tailed deer (*Odocoileus virginianus borealis*) accompanying natural infections of lungworms (genera *Protostrongylus* and *Dictyocaulus*). **The Journal of Wildlife Management**, v. 5, n. 2, p. 141-158, 1941.

GONZALEZ, S.; KITCHENER, A. C.; LISTER, A. M. Survival of the Irish elk into the Holocene. **Nature**, v. 405, n. 6788, p. 753-754, 2000.

GONZÁLEZ-ACUÑA, D.; NEIRA-RAMIREZ, V.; MORENO-SALAS, L.; QUEZADA, M. First report of paratuberculosis in Southern Pudu deer (Artiodactyla: Cervidae). **Arquivo Brasileiro de Medicina Veterinária e Zootecnia**, v. 63, n. 4, p. 1025-1027, 2011.

GORDON, M. A.; SALKIN, I. F.; STONE, W. B. *Dermatophilus dermatitis* enzootic in deer in New York State and vicinity. **Journal of Wildlife Diseases**, v. 13, n. 2, p. 184-190, 1977.

GUARNER, J.; BRANDT, M. E. Histopathologic diagnosis of fungal infections in the 21st century. **Clinical Microbiology Reviews**, v. 24, n. 2, p. 247-280, 2011.

HAIGH, J.; BEREZOWSKI, J.; WOODBURY, M. R. A cross-sectional study of the causes of morbidity and mortality in farmed white-tailed deer. **Canadian Veterinary Journal**, v. 46, n. 6, p. 507-512, 2005.

HATTEL, A. L.; SHAW, D. P.; LOVE, B. C.; WAGNER, D. C.; DRAKE, T. R.; BROOKS, J. W. A retrospective study of mortality in Pennsylvania captive white-tailed deer (*Odocoileus virginianus*): 2000–2003. **Journal of Veterinary Diagnostic Investigation**, v. 16, n. 6, p. 515-521, 2004.

HATTEL, A. L.; SHAW, D. P.; FISHER, J. S.; BROOKS, J. W.; LOVE, B. C.; DRAKE, T. R.; WAGNER, D. C. Mortality in Pennsylvania captive elk (*Cervus elaphus*): 1998–2006. **Journal of Veterinary Diagnostic Investigation**, v. 19, n. 3, p. 334-337, 2007.

HEADLEY, S. A.; SOUSA, I. K.; MINERVINO, A. H.; BARROS, I. O.; BARRÊTO JÚNIOR, R. A.; ALFIERI, A. F.; ALFIERI, A. A. Molecular confirmation of ovine herpesvirus 2-induced malignant catarrhal fever lesions in cattle from Rio Grande do Norte, Brazil. **Pesquisa Veterinária Brasileira**, v. 32, n. 12, p. 1213-1218, 2012.

HERRON, A. J.; GARMAN, R. H.; BAITCHMAN, R.; KRAUS, A. L. Clostridial myositis in a reindeer (*Rangifer tarandus*): a case report. **The Journal of Zoo Animal Medicine**, v. 10, n. 1, p. 31-34, 1979.

HOSKIN, S. O.; WILSON, P. R.; CHARLESTON, W. A. G.; BARRY, T. N. A model for study of lungworm (*Dictyocaulus* sp.) and gastrointestinal nematode infection in young red deer (*Cervus elaphus*). **Veterinary parasitology**, v. 88, n. 3, p. 199-217, 2000.

HOY, J. A.; HAAS, G. T.; HOY, R. D.; HALLOCK, P. Observations of brachygnathia superior (underbite) in wild ruminates in Western Montana, USA. **Wildlife Biology in Practice**, v. 7, n. 2, p. 15-29, 2011.

ILHA, M. R. S.; LORETTI, A. P.; REIS, A. C. F. Wasting and mortality in beef cattle parasitized by *Eurytrema coelomaticum* in the State of Paraná, southern Brazil. **Veterinary Parasitology**, v. 133, n. 1, p. 49-60, 2005.

INGEBRIGTSEN, D. K.; LUDWIG, J. R.; MCCLURKIN, A. W. Occurrence of antibodies to the etiologic agents of Infectious Bovine Rhinotracheitis, Parainfluenza 3, Leptospirosis, and Brucellosis in White-Tailed Deer in Minnesota. **Journal of Wildlife Diseases**, v. 22, n. 1, p. 83-86, 1986.

JENSEN, H. E.; JØRGENSEN, J. B.; SCHØNHEYDER, H. Pulmonary mycosis in farmed deer: allergic zygomycosis and invasive aspergillosis. **Journal of Medical and Veterinary Mycology**, v. 27, n. 5, p. 329-334, 1989.

JORI, F.; CARON, A.; THOMPSON, P. N.; DWARKA, R.; FOGGIN, C.; DE GARINE-WICHATITSKY, M.; HOFMEYR, M.; VAN HEERDEN, J.; HEATH, L. Characteristics of foot-and-mouth disease viral strains circulating at the wildlife/livestock interface of the great limpopo transfrontier conservation area. **Transboundary and Emerging Diseases**, v. 63, n. 1, p. e58-e70, 2016.

JUBB, K. V. F.; STENT, A. W. Pancreas. In, MAXIE, G. (ed.) **Jubb, Kennedy & Palmer's pathology of domestic animals. e-Book**. Oxford: Elsevier Health Sciences, 2015.

KAMLER, J. F.; JEDRZEJEWSKI, W.; JEDRZEJEWSKA, B. Survival and cause-specific mortality of red deer *Cervus Elaphus* in Białowieża National Park, Poland. **Wildlife Biology**, v. 13, n. 1, p. 48-52, 2007.

KARAMON, J.; LARSKA, M.; JASIK, A.; SELL, B. First report of the giant liver fluke (*Fascioloides magna*) infection in farmed fallow deer (*Dama dama*) in Poland—pathomorphological changes and molecular identification. **Bulletin of the Veterinary Institute in Pulawy**, v. 59, n. 3, p. 339-344, 2015.

KARNS, G. R.; LANCIA, R. A.; DEPERNO, C. S.; CONNER, M. C.; STOSKOPF, M. K. Intracranial abscessation as a natural mortality factor for adult male white-tailed

deer (*Odocoileus virginianus*) in Kent County, Maryland, USA. **Journal of Wildlife Diseases**, v. 45, n. 1, p. 196-200, 2009.

KARSTAD, L.; TRAINER, D. O. Histopathology of experimental bluetongue disease of white-tailed deer. **The Canadian Veterinary Journal**, v. 8, n. 11, p. 247, 1967.

KARSTAD, L. A. R. S.; HANSON, R. P. Vesicular stomatitis in deer. **American Journal of Veterinary Research**, v. 18, n. 66, p. 162-166, 1957.

KEEL, M. K.; GAGE, P. J.; NOON, T. H.; BRADLEY, G. A.; COLLINS, J. K. Caprine herpesvirus-2 in association with naturally occurring malignant catarrhal fever in captive sika deer (*Cervus nippon*). **Journal of Veterinary Diagnostic Investigation**, v. 15, n. 2, p. 179-183, 2003.

KNIGHT-JONES, T. J. D.; RUSHTON, J. The economic impacts of foot and mouth disease—What are they, how big are they and where do they occur?. **Preventive Veterinary Medicine**, v. 112, n. 3, p. 161-173, 2013.

KOLLER, L. D.; OLSON, C. Pulmonary fibroblastomas in a deer with cutaneous fibromatosis. **Cancer research**, v. 31, n. 10, p. 1373-1375, 1971.

KRZYSIAK, M. K.; DACKIEWICZ, J.; KĘSIK-MALISZEWSKA, J.; LARSKA, M. Post-mortem evaluation of pathological lesions in European bison (*Bison bonasus*) in the Białowieża Primeval Forest between 2008 and 2013. **Bulletin of the Veterinary Institute in Pulawy**, v. 58, p. 421-431, 2014.

KUPCA, A. M.; RETTINGER, A.; ZIMMERMANN, P.; HOERMANSDORFER, S.; KONRAD, R.; HAFNER-MARX, A. Severe purulent and necrotizing glossitis in a fallow deer (*Dama dama*) due to an infection with the involvement of *Mannheimia granulomatis*. **Berliner und Munchener Tierarztliche Wochenschrift**, v. 128, n. 7-8, p. 285-288, 2014.

LAACKONEN, Juha. Pneumocystis carinii in wildlife. **International journal for parasitology**, v. 28, n. 2, p. 241-252, 1998.

LAMARQUE, F.; BARRAT, J.; HATIER, C.; ARTOIS, M. Causes of mortality in roe deer (*Capreolus capreolus*) diagnosed by an epidemiological surveillance network in France. **Gibier Faune Sauvage**, v. 16, n. 2, p. 101-122, 1999.

LAMONTAGNE, L.; SADI, L.; JOYAL, R. Serological evidence of Bovine Herpesvirus 1-related virus infection in the White-Tailed Deer population on Anticosti Island, Quebec. **Journal of Wildlife Diseases**, v. 25, n. 2, p. 202-205, 1989.

LANCE, W. R.; HIBLER, C. P.; DEMARTINI, J. Experimental contagious ecthyma in Mule Deer, White-Tailed Deer, pronghorn and wapiti. **Journal of Wildlife Diseases**, v. 19, n. 3, p. 165-169, 1983.

LAWRENCE, P. K.; BEY, R. F.; WIENER, B.; KITTIHOTIRAT, W.; BUMGARNER, R. E. Genome sequence of a presumptive *Mannheimia haemolytica* strain with an A1/A6-cross-reactive serotype from a white-tailed deer (*Odocoileus virginianus*). **Genome Announcements**, v. 2, n. 2, p. e00114-14, 2014.

LEHMKUHL, H. D.; HOBBS, L. A.; WOODS, L. W. Characterization of a new adenovirus isolated from black-tailed deer in California. **Archives of virology**, v. 146, n. 6, p. 1187-1196, 2001.

LI, H., DYER, N., KELLER, J., CRAWFORD, T. B. Newly recognized herpesvirus causing malignant catarrhal fever in white-tailed deer (*Odocoileus virginianus*). **Journal of Clinical Microbiology**, v. 38, n. 4, p. 1313-1318, 2000.

LI, H.; WUNSCHMANN, A.; KELLER, J.; HALL, D. G.; CRAWFORD, T. B. Caprine Herpesvirus-2-associated Malignant Catarrhal Fever in white-tailed deer (*Odocoileus virginianus*). **Journal of Veterinary Diagnostic Investigation**, v. 15, n. 1, p. 46-49, 2003.

LIEBLER-TENORIO, E. M.; RIDPATH, J. F.; NEILL, J. D. Distribution of viral antigen and development of lesions after experimental infection with highly virulent Bovine Viral Diarrhea virus type 2 in calves. **American Journal of Veterinary Research**, v. 63, n. 11, p. 1575-1584, 2002.

LITTLE, S. E.; CARMICHAEL, K. P.; RAKICH, P. M. Trombidiosis-induced dermatitis in white-tailed deer (*Odocoileus virginianus*). **Veterinary Pathology**, v. 34, n. 4, p. 350-352, 1997.

LUCIOLI, J.; FURLAN, F. H.; SPRICIGO, D. A.; FERRAZ, S. M.; TRAVERSO, S. D. Bronchopneumonia caused by *Arcanobacterium pyogenes* in Pampas Deer (*Ozoteceros bezearticus*). **Acta Scientiae Veterinariae**, v. 36, n. 1, p. 51-53, 2008.

LUDET, C. J.; SCHROEDER, W.; ROTTMANN, O.; KUEHN, R. Mitochondrial DNA phylogeography of Red Deer (*Cervus elaphus*). **Molecular Phylogenetics and Evolution**, v. 31, n. 3, p. 1064-1083, 2004.

LUGTON, I. W.; WILSON, P. R.; MORRIS, R. S.; NUGENT, G. Epidemiology and pathogenesis of *Mycobacterium bovis* infection of red deer (*Cervus elaphus*) in New Zealand. **New Zealand Veterinary Journal**, v. 46, n. 4, p. 147-156, 1998.

MACKINTOSH, C.; HAIGH, J. C.; GRIFFIN, F. Bacterial diseases of farmed deer and bison. **Revue Scientifique et Technique-Office International des Épizooties**, v. 21, n. 1, p. 249-264, 2002.

MACLACHLAN, N. J.; DREW, C. P.; DARPEL, K. E.; WORWA, G. The pathology and pathogenesis of bluetongue. **Journal of comparative pathology**, v. 141, n. 1, p. 1-16, 2009.

MADSON, D. M.; OPRIESSNIG, T. Multicentric T-cell lymphosarcoma in a white-tailed deer (*Odocoileus virginianus*). **Journal of wildlife diseases**, v. 45, n. 3, p. 791-794, 2009.

MALMSTEN, J.; JAKUBEK, E.B.; BJÖRKMAN, C. Prevalence of antibodies against *Toxoplasma gondii* and *Neospora caninum* in moose (*Alces alces*) and roe deer (*Capreolus capreolus*) in Sweden. **Veterinary Parasitology**, v. 177, n. 3, p. 275-280, 2011.

MARTÍN-HERNANDO, M. P.; TORRES, M. J.; AZNAR, J.; NEGRO, J. J.; GANDÍA, A.; GORTÁZAR, C. Distribution of lesions in red and fallow deer naturally infected with *Mycobacterium bovis*. **Journal of comparative pathology**, v. 142, n. 1, p. 43-50, 2010.

MASON, P. Parasites of deer in New Zealand. **New Zealand Journal of Zoology**, v. 21, n. 1, p. 39-47, 1994.

MASTERS, FLACH. (2013), Cervidae and Tragulidae. In (MILLER, R. E.; FOWLER, M. E. Eds). **Fowler's Zoo and Wild Animal Medicine Current Therapy**. Elsevier Health Sciences, Volume 8. 2011.

MAWHINNEY, I.; WOODGER, N.; KNUDSEN, S. atypical interstitial pneumonia in grazing adult red deer (*Cervus elaphus*). **Journal of Comparative Pathology**, v. 143, n. 2, p. 209-212, 2010.

MAZZULLO, G.; LAZZARO, C.; MACRÌ, B. Palatoschisis in a fallow deer fawn. **Large Animals Review**, v. 11, n. 4, p. 15-19, 2005.

MCDONALD, J. E.; DESTEFANO, S.; GAUGHAN, C.; MAYER, M.; WOYTEK, W. A.; CHRISTENSEN, S.; FULLER, T. K. Survival and harvest-related mortality of white-tailed deer in massachusetts. **Wildlife Society Bulletin**, v. 35, n. 3, p. 209-219, 2011.

MCVICAR, J. W.; SUTMOLLER, P.; FERRIS, D. H.; CAMPBELL, C. H. Foot-and-mouth disease in white-tailed deer: clinical signs and transmission in the laboratory. **Proceedings, Annual Meeting of the United States Animal Health Association**, n. 78, p. 169-180, 1973.

MENZIES, P.; LANGS, L.; BOERMANS, H.; MARTIN, J.; MCNALLY, J. Myopathy and hepatic lipidosis in weaned lambs due to vitamin E deficiency. **The Canadian Veterinary Journal**, v. 45, n. 3, p. 244, 2004.

MIDURA, T. F.; NYGAARD, G. S.; WOOD, R. M.; BODILY, H. L. Clostridium botulinum type F: isolation from venison jerky. **Applied Microbiology**, v. 24, n. 2, p. 165-167, 1972.

MONTANÉ, J.; MARCO, I.; MANTECA, X.; LÓPEZ, J.; LAVIN, S. Delayed acute capture myopathy in three roe deer. **Journal of Veterinary Medicine Series A**, v. 49, n. 2, p. 93-98, 2002.

MONIWA, M.; EMBURY-HYATT, C.; ZHANG, Z.; HOLE, K.; CLAVIJO, A.; COPPS, J.; ALEXANDERSEN, S. Experimental foot-and-mouth disease virus infection in white tailed deer. **Journal of Comparative Pathology**, v. 147, n. 2, p. 330-342, 2012.

NETTLES, V. F.; PRESTWOOD, A. K.; NICHOLS, R. G.; WHITEHEAD, C. J. Meningeal worm-induced neurologic disease in black-tailed deer. **Journal of Wildlife Diseases**, v. 13, n. 2, p. 137-143, 1977.

NETTLES, V. F.; QUIST, C. F.; LOPEZ, R. R.; WILMERS, T. J.; FRANK, P.; ROBERTS, W.; CHITWOOD, S.; DAVIDSON, W. R. Morbidity and mortality factors in Key Deer (*Odocoileus virginianus clavium*). **Journal of Wildlife Diseases**, v. 38, n. 4, p. 685-692, 2002.

NOON, T. H.; WESCHE, S. L.; HEFFELFINGER, J.; FULLER, A.; BRADLEY, G. A.; REGGIARDO, C. Hemorrhagic disease in deer in Arizona. **Journal of Wildlife Diseases**, v. 38, n. 1, p. 177-181, 2002.

NOSANCHUK, J. D. Fungal myocarditis. **Frontiers in Bioscience**, v. 7, p. d1423-38, 2002.

NOVOBILSKÝ, A.; HORÁČKOVÁ, E.; HIRTOVÁ, L.; MODRÝ, D.; KOUDELA, B. The giant liver fluke *Fascioloides magna* (Bassi 1875) in cervids in the Czech Republic and potential of its spreading to Germany. **Parasitology Research**, v. 100, n. 3, p. 549-553, 2007.

NUVOLI, S.; BURRAI, G. P.; SECCI, F.; COLUMBANO, N.; CAREDDU, G. M.; MANDAS, L.; PIRINO, S.; ANTUOFERMO, E. Capture myopathy in a corsican red deer *Cervus elaphus corsicanus* (Ungulata: Cervidae). **Italian Journal of Zoology**, v. 81, n. 3, p. 457-462, 2014.

O'BRIEN, D. J.; SCHMITT, S. M.; FIERKE, J. S.; HOGLE, S. A.; WINTERSTEIN, S. R.; COOLEY, T. M.; KANEENE, J. B. Epidemiology of *Mycobacterium bovis* in free-ranging white-tailed deer, Michigan, USA, 1995–2000. **Preventive veterinary medicine**, v. 54, n. 1, p. 47-63, 2002.

OROZCO, M. M.; MARULL, C.; JIMÉNEZ PÉREZ, I.; GÜRTLER, R. E. Mortalidad invernal de ciervo de los pantanos (*Blastocerus dichotomus*) en humedales del noreste de Argentina. **Mastozoología Neotropical**, v. 20, n. 1, p. 163-170, 2013.

ORR, M. A review of respiratory disease in New Zealand deer. **Surveillance**, v. 18, n. 2, p. 17-18, 1991.

O'TOOLE, D.; LI, H.; SOURK, C.; MONTGOMERY, D. L.; CRAWFORD, T. B. Malignant Catarrhal Fever in a bison (*Bison bison*) feedlot, 1993–2000. **Journal of Veterinary Diagnostic Investigation**, v. 14, n. 3, p. 183-193, 2002.

OZCAN, K.; BEYTUT, E. Pathological investigations on anthracosis in cattle. **Veterinary Record**, v. 149, p. 90-92, 2001.

PALING, R. W.; KARSTAD, L.; GROOTENHUIS, J. G. Rumenitis with candidiasis in a wildebeest (*Connochaetes taurinus*) calf. **The Journal of Zoo Animal Medicine**, v. 9, n. 4, p. 137-141, 1978.

PALMER, M. V.; WHIPPLE, D. L. *Arcanobacterium pyogenes* as a cause of fatal pleuropneumonia after capture and transport of white-tailed deer (*Odocoileus virginianus*). **Journal of Veterinary Diagnostic Investigation**, v. 11, n. 5, p. 468-471, 1999.

PALMER, M. V.; WATERS, W. R.; WHIPPLE, D. L. Abomasal ulcers in captive White-Tailed Deer (*Odocoileus virginianus*). **Journal of Comparative Pathology**, v. 125, n. 2, p. 224-227, 2001.

PALMER, M. V.; CARPENTER, J. G. Congenital polycystic kidney in a White-Tailed Deer (*Odocoileus virginianus*). **Journal of Veterinary Diagnostic Investigation**, v. 16, n. 5, p. 475-477, 2004.

PALMER, M. V.; THACKER, T. C.; MADISON, R. J.; KOSTER, L. G.; SWENSON, S. L.; LI, H. Active and latent Ovine Herpesvirus-2 (OvHV-2) infection in a herd of captive white-tailed deer (*Odocoileus virginianus*). **Journal of Comparative Pathology**, v. 149, n. 2, p. 162-166, 2013.

PAN, S. X.; MEN, X. X.; FENG, J. C.; ZHOU, Y. J.; XU, H. F. A review of studies on habitat selection by small and solitary forest ruminants. **Journal of Zhejiang Forestry College**, v. 3, p. 019, 2007.

PANADERO, R.; CARRILLO, E. B.; LÓPEZ, C.; DÍEZ-BAÑOS, N.; DÍEZ-BAÑOS, P.; MORRONDO, M. P. Bronchopulmonary helminths of roe deer (*Capreolus capreolus*) in the northwest of Spain. **Veterinary parasitology**, v. 99, n. 3, p. 221-229, 2001.

PASSLER, T.; WALZ, H. L.; DITCHKOFF, S. S.; VAN SANTEN, E.; BROCK, K. V.; WALZ, P. H. Distribution of Bovine Viral Diarrhea virus antigen in persistently infected White-Tailed Deer (*Odocoileus virginianus*). **Journal of Comparative Pathology**, v. 147, n. 4, p. 533-541, 2012.

PATERSON, J. Capture myopathy. In: WEST, G.; HEARD, D.; CAULKETT, N. (Ed.). **Zoo animal and wildlife immobilization and anesthesia**. Oxford, UK: Blackwell Publishing Ltd., 2007. p. 115-122.

PICONE, J., WILLIAMS, J. F., LEID, R. W., FAY, L. D. Polycystic liver in four White-Tailed Deer. **Journal of wildlife diseases**, v. 17, n. 3, p. 395-400, 1981.

PIOVEZAN, U.; TIEPOLO, L. M.; TOMAS, W. M.; DUARTE, J. B.; VARELA, D.; MARINHO-FILHO, J. S. Marsh deer *Blastocerus dichotomus* (Illiger, 1815). In: GONZALES S.; DUARTE, J. M. B. (Ed.). **Neotropical cervidology**: biology and medicine of latin Imerican deer. Jaboticabal, FUNEP/IUCN, 2010. p. 66-76.

POWER, S. B.; HAAGSMA, J.; SMYTH, D. P. Paratuberculosis in farmed red deer (*Cervus elaphus*) in Ireland. **The Veterinary Record**, v. 132, n. 9, p. 213-216, 1993.

PRESTWOOD, A. K.; SMITH, J. F.; BROWN, J. Lungworms in white-tailed deer of the Southeastern United States. **Journal of Wildlife Diseases**, v. 7, n. 3, p. 149-154, 1971.

PRESTWOOD, A. K.; KISTNER, T. P.; KELLOGG, F. E.; HAYES, F. A. The 1971 Outbreak of hemorrhagic disease among white-tailed deer of the Southeastern United States. **Journal of Wildlife Diseases**, v. 10, n. 3, p. 217-224, 1974.

PROPHET, E. B. **Laboratory methods in histotechnology**. Amer Registry of Pathology, 1992.

PROTHERO, D. R. Mammalian evolution. In: PROTHERO, D. R.; SCHOCH, R. M. (Ed.). **Major features of vertebrate evolution**: short courses in paleontology 7. Tennessee: Paleontological Society and University of Tennessee Press, 1994. p. 238-270.

PYBUS, M. J.; SAMUEL, W. M.; WELCH, D. A.; SMITS, J.; HAIGH, J. C. Mortality of fallow deer (*Dama dama*) experimentally-infected with meningeal worm, *Parelaphostrongylus tenuis*. **Journal of Wildlife Diseases**, v. 28, n. 1, p. 95-101, 1992.

QUINN, A. C. D.; WILLIAMS, D. M.; PORTER, W. F.; FITZGERALD, S. D.; HYNES, K. Effects of capture-related injury on postcapture movement of White-Tailed Deer. **Journal of Wildlife Diseases**, v. 50, n. 2, p. 250-258, 2014.

QUIST, C. F.; HOWERTH, E. W.; FISCHER, J. R.; WYATT, R. D.; MILLER, D. M.; NETTLES, V. F. Evaluation of low-level aflatoxin in the diet of White-Tailed Deer. **Journal of Wildlife Diseases**, v. 33, n. 1, p. 112-121, 1997.

RADOSTITS, O. M.; GAY, C. C.; HINCHCLIFF, K. W.; CONSTABLE, P. D. A. **Textbook of the diseases of cattle, horses, sheep, pigs and goats**. 10. ed. Oxford, UK: Saunders Elsevier, 2007.

RAHKO, T.; SAARI, S.; NIKANDER, S. Histopathological lesions in spontaneous dictyocaulotic pneumonia of the reindeer (*Rangifer tarandus tarandus* L.). **Rangifer**, v. 12, n. 2, p. 115-122, 1992.

RAJAN, A.; SREEKUMARAN, T.; SULOCHANA, S. Carcinoma of the Mucosa of the Ethmoid in the Deer (*Axis axis*). **Veterinary Pathology Online**, v. 19, n. 4, p. 458-460, 1982.

RAIZMAN, E. A.; POGRANICHNIY, R.; LEVY, M.; NEGRON, M.; LANGOHR, I.; ALSTINE, W. V. Experimental infection of White-Tailed Deer fawns (*Odocoileus virginianus*) with Bovine Viral Diarrhea Virus type-1 isolated from free-ranging white-tailed deer. **Journal of Wildlife Diseases**, v. 45, n. 3, p. 653-660, 2009.

REISSIG, E. C.; MORÉ, G.; MASSONE, A.; UZAL, F. A. Sarcocystosis in wild red deer (*Cervus elaphus*) in Patagonia, Argentina. **Parasitology Research**, v. 115, n. 5, p. 1773-1778, 2016.

RHYAN, J. C.; SAARI, D. A. A comparative study of the histopathologic features of bovine tuberculosis in cattle, fallow deer (*Dama dama*), sika deer (*Cervus nippon*), and red deer and elk (*Cervus elaphus*). **Veterinary Pathology**, v. 32, n. 3, p. 215-220, 1995.

ROBINSON, W. F.; ROBINSON, N. Cardiovascular system. In, MAXIE, G. (Ed.). **Jubb, Kennedy & Palmer's pathology of domestic animals**. e-Book. Oxford, UK: Elsevier Health Sciences, 2015.

ROHER, D. P.; NIELSEN, S. W.; STONE, W. B. Carcinoma in the urinary bladder of a white-tailed deer (*Odocoileus virginianus*). **Journal of Wildlife Diseases**, v. 18, n. 3, p. 361-363, 1982.

ROLLOR, E. A. Syndactylism and brachygnathia in a White-Tailed Deer. **Journal of Wildlife Diseases**, v. 29, n. 4, p. 618-619, 1993.

ROSE, K. D. Skeleton of Diacodexis, oldest known artiodactyl. **Science**, v. 216, n. 4546, p. 621-623, 1982.

ROSOL, T. J.; GRONE, A. Endocrine glands. In: MAXIE, G. (Ed.). **Jubb, Kennedy & Palmer's pathology of domestic animals**. e-Book. Oxford, UK: Elsevier Health Sciences, 2015.

RUTJES, S. A.; LODDER-VERSCHOOR, F.; LODDER, W. J.; VAN DER GIESSEN, J.; REESINK, H.; BOUWKNEGT, M.; DE RODA HUSMAN, A. M. Seroprevalence and molecular detection of hepatitis E virus in wild boar and red deer in The Netherlands. **Journal of Virological Methods**, v. 168, n. 1, p. 197-206, 2010.

SACKS, J. J.; DELGADO, D. G.; LOBEL, H. O.; PARKER, R. L. Toxoplasmosis infection associated with eating undercooked venison. **American Journal of Epidemiology**, v. 118, n. 6, p. 832-838, 1983.

SADI, L.; JOYAL, R.; ST-GEORGES, M.; LAMONTAGNE, L. Serologic survey of White-Tailed Deer on Anticosti Island, Quebec for Bovine Herpesvirus 1, Bovine Viral

Diarrhea, and Parainfluenza 3. **Journal of Wildlife Diseases**, v. 27, n. 4, p. 569-577, 1991.

SAMUEL, W. M.; BEAUDOIN, R. L. Evaluation of two survey methods for detection of helminth infections in White-Tailed Deer (*Odocoileus virginianus*). **Bulletin of the Wildlife Disease Association**, v. 2, n. 4, p. 100-107, 1966.

SANFORD, S. E.; LITTLE, P. B.; RAPLEY, W. A. The gross and histopathologic lesions of malignant catarrhal fever in three captive sika deer (*Cervus nippon*) in southern Ontario. **Journal of Wildlife Diseases**, v. 13, n. 1, p. 29-32, 1977.

SATO, Y.; KOBAYASHI, C.; ICHIKAWA, K.; KUWAMOTO, R.; MATSUURA, S.; KOYAMA, T. An occurrence of Salmonella typhimurium infection in sika deer (*Cervus nippon*). **Journal of Veterinary Medical Science**, v. 62, n. 3, p. 313-315, 2000.

SCALA, C.; ORTIZ, K.; CATINAUD, J.; LEMBERGER, K. Hematuria and urinary bladder lesions compatible with bracken fern (*Pteridium aquilinum*) intoxication in captive fallow deer (*Dama dama*). **Journal of Zoo and Wildlife Medicine**, v. 45, n. 2, p. 380-385, 2014.

SCAGLIARINI, A.; GALLINA, L.; BATTILANI, M.; TURRINI, F.; SAVINI, F.; LAVAZZA, A.; ALBERTI, A. *Cervus elaphus* papillomavirus (CePV1): New insights on viral evolution in deer. **Veterinary microbiology**, v. 165, n. 3, p. 252-259, 2013.

SCHMITT, S. M.; FITZGERALD, S. D.; COOLEY, T. M.; BRUNING-FANN, C. S.; SULLIVAN, L.; BERRY, D.; CARLSON, T.; MINNIS, R. B.; PAYEUR, J. B.; SIKARSKIE, J. Bovine tuberculosis in free-ranging white-tailed deer from Michigan. **Journal of Wildlife Diseases**, v. 33, n. 4, p. 749-758, 1997.

SCOTT, P. R.; PENNY, C. D.; MACRAE, A. **Cattle medicine**. [S.l.]: CRC Press, 2011.

SMITH-FLUECK, J. A. M.; FLUECK, W. T. Natural mortality patterns in a population of southern Argentina huemul (*Hippocamelus bisulcus*), an endangered Andean cervid. **Zeitschrift für Jagdwissenschaft**, v. 47, n. 3, p. 178-188, 2001.

SCHULTHEISS, P. C.; VAN CAMPEN, H.; SPRAKER, T. R.; BISHOP, C.; WOLFE, L.; PODELL, B. Malignant Catarrhal Fever associated with Ovine Herpesvirus-2 in free-ranging Mule Deer in Colorado. **Journal of Wildlife Diseases**, v. 43, n. 3, p. 533-537, 2007.

SHOPE, R. E.; MACNAMARA, L. G.; MANGOLD, R. A virus-induced epizootic hemorrhagic disease of the Virginia white-tailed deer (*Odocoileus virginianus*). **Journal of Experimental Medicine**, v. 111, n. 2, p. 155-170, 1960.

SOLDATI, S.; KIUPEL, M.; WISE, A.; MAES, R.; BOTTERON, C.; ROBERT, N. Meningoencephalomyelitis caused by *Neospora caninum* in a juvenile fallow deer (*Dama dama*). **Journal of Veterinary Medicine Series A**, v. 51, n. 6, p. 280-283, 2004.

SORDEN, S. D.; WOODS, L. W.; LEHMKUHL, H. D. Fatal pulmonary edema in white-tailed deer (*Odocoileus virginianus*) associated with adenovirus infection. **Journal of veterinary diagnostic investigation**, v. 12, n. 4, p. 378-380, 2000.

SOUZA, M. B.; SALDIVA, P. H.; POPE, C. A.; CAPELOZZI, V. L. Respiratory changes due to long-term exposure to urban levels of air pollution: a histopathologic study in humans. **CHEST Journal**, v. 113, n. 5, p. 1312-1318, 1998.

STAIR, E. L.; ROBINSON, R. M.; JONES, L. P. Spontaneous bluetongue in Texas white-tailed deer. **Pathologia Veterinaria Online**, v. 5, n. 2, p. 164-173, 1968.

STERN, A. W.; RITCHEY, J. W.; HALL, B.; KETZ-RILEY, C. J.; GENOVA, S. G. Nonsteroidal Anti-Inflammatory Drug—Associated Renal Papillary Necrosis in a White-Tailed Deer (*Odocoileus Virginianus*). **Journal of Veterinary Diagnostic Investigation**, v. 22, n. 3, p. 476-478, 2010.

STUEN, S.; ENGWALL, E. O.; VAN DE POLL, I.; SCHOULS, M. Granulocytic ehrlichiosis in a roe deer calf in Norway. **Journal of Wildlife Diseases**, v. 37, n. 3, p. 614-617, 2001.

SUTHERLAND, R. J. *Elaphostrongylus cervi* in Cervids in New Zealand: 1. The gross and histological lesions in red deer (*Cervus elaphus*). **New Zealand veterinary journal**, v. 24, n. 11, p. 263-266, 1976.

SZABÓ, M. P. J.; MATUSHIMA, E. R.; PEREIRA, M. D. C.; WERTHER, K.; BARBANTI DUARTE, J. M. Cat flea (*Ctenocephalides felis*) infestation in quarantined Marsh Deer (*Blastocerus dichotomus*) populations. **Journal of Zoo and Wildlife Medicine**, v. 31, n. 4, p. 576-577, 2000.

TATE, C. M.; HOWERTH, E. W.; STALLKNECHT, D. E.; ALLISON, A. B.; FISCHER, J. R.; MEAD, D. G. Eastern equine encephalitis in a free-ranging white-tailed deer (*Odocoileus virginianus*). **Journal of Wildlife Diseases**, v. 41, n. 1, p. 241-245, 2005.

TEI, S.; KITAJIMA, N.; TAKAHASHI, K.; MISHIRO, S. Zoonotic transmission of hepatitis E virus from deer to human beings. **The Lancet**, v. 362, n. 9381, p. 371-373, 2003.

THOMSON, R. G. Rumenitis in cattle. **The Canadian Veterinary Journal**, v. 8, n. 8, p. 189, 1967.

THOMSON, G. R.; VOSLOO, W.; BASTOS, A. D. S. Foot and mouth disease in wildlife. **Virus Research**, v. 91, n. 1, p. 145-161, 2003.

TOMKINS, N. W.; JONSSON, N. N.; YOUNG, M. P.; GORDON, A. N.; MCCOLL, K. A. An outbreak of malignant catarrhal fever in young rusa deer (*Cervus timorensis*). **Australian Veterinary Journal**, v. 75, n. 10, p. 722-723, 1997.

TURNQUIST, S. E.; FALES, W. H. Disseminated *Actinomyces pyogenes* infection in a free-ranging white-tailed deer. **Journal of Veterinary Diagnostic Investigation**, v. 10, n. 1, p. 86-89, 1998.

UHART, M. M.; VILA, A. R.; BEADE, M. S.; BALCARCE, A.; KARESH, W. B. Health evaluation of pampas deer (*Ozotoceros bezoarticus celer*) at Campos del Tuyu Wildlife Reserve, Argentina. **Journal of Wildlife Diseases**, v. 39, n. 4, p. 887-893, 2003.

UHART, M., MANGINI, P. R., SAUCEDO GALVEZ, C. E., CORTI, P., MILANO, F. A., JORGE, M. C., SILVA GIRIO, R. L., MATHIAS, L. A., SCHETTINO, A. M., CATENA, M. C., TERRAGNO, R., APRILE, G. Bacterial Diseases. In **Neotropical Cervidology: Biology and Medicine of Neotropical Deer** (DUARTE J. M. N.; GONZALEZ, S. eds.). Funep, 2010.

UZAL, F. A.; PLATTNER, B. L.; HOSTETTER, J. M. Alimentary system. In, MAXIE, G. (ed.) **Jubb, Kennedy & Palmer's pathology of domestic animals**. e-Book. Oxford: Elsevier Health Sciences, 2015.

VAN CAMPEN, H.; RIDPATH, J.; WILLIAMS, E.; CAVENDER, J.; EDWARDS, J.; SMITH, S.; SAWYER, H. Isolation of bovine viral diarrhea virus from a free-ranging mule deer in Wyoming. **Journal of Wildlife Diseases**, v. 37, n. 2, p. 306-311, 2001.

VEATCH, J. K.; CARPENTER, J. W. Metastatic adenocarcinoma of the mammary gland in a Père David's deer. **Journal of Veterinary Diagnostic Investigation**, v. 5, n. 4, p. 639-640, 1993.

VOS, A. **Deer farming**: guidelines on practical aspects. Roma: FAO, 1982. (FAO Animal Production and Health Paper 27). Disponível em: <www.fao.org/DOCREP/004/X6529E/X6529E00.htm>. Acces: 10 fev. 2015.

VREELAND, J. K.; DIEFENBACH, D. R.; WALLINGFORD, B. D. Survival rates, mortality causes, and habitats of Pennsylvania White-Tailed Deer fawns. **Wildlife Society Bulletin**, v. 32, n. 2, p. 542-553, 2004.

WALLACE, R. S.; BUSH, M.; MONTALI, R. J. Deaths from exertional myopathy at the National Zoological Park from 1975 to 1985. **Journal of Wildlife Diseases**, v. 23, n. 3, p. 454-462, 1987.

WEBB, S. L.; HEWITT, D. G.; HELICKSON, M. W. Survival and Cause-Specific Mortality of Mature Male White-Tailed Deer. **The Journal of Wildlife Management**, v. 71, n. 2, p. 555-558, 2007.

WELCH, P. J.; HAVILL, P. F.; JULIAN, A. F.; POOLE, W. S. H.; CORRIN, K. C.; GLADDEN, N. R. Experimental tuberculosis in red deer (*Cervus elaphus*). **New Zealand Veterinary Journal**, v. 31, n. 12, p. 213-216, 1983.

WILLIAMS, E. S.; BECERRA, V. M.; THOME, E. T.; GRAHAM, T. J.; OWENS, M. J.; NUNAMAKER, C. E. Spontaneous poxviral dermatitis and keratoconjunctivitis in free-ranging mule deer (*Odocoileus hemionus*) in Wyoming. **Journal of Wildlife Diseases**, v. 21, n. 4, p. 430-433, 1985.

WILSON, J. E. Occurrence of rabies in deer (*Odocoileus virginianus*). **Journal of mammalogy**, v. 30, n. 2, p. 203-203, 1949.

WOBESER, G.; RUNGE, W. Multiple anomalies in a white-tailed deer fetus. **Journal of Wildlife Diseases**, v. 9, n. 4, p. 356-358, 1973.

WOBESER, G.; RUNGE, W. Rumen overload and rumenitis in white-tailed deer. **The Journal of Wildlife Management**, p. 596-600, 1975.

WOBESER, G.; BELLAMY, J. E.; BOYSEN, B. G.; MACWILLIAMS, P. S.; RUNGE, W. Myopathy and myoglobinuria in a wild White-Tailed Deer. **Journal of the American Veterinary Medical Association**, v. 169, n. 9, p. 971-974, 1976.

WOBESER, G. A. **Essentials of disease in wild animals**. Canada: John Wiley & Sons, 2013

WOOLF, A.; KRADEL, D. Occurrence of rumenitis in a supplementary fed White-Tailed Deer herd. **Journal of Wildlife Diseases**, v. 13, n. 3, p. 281-285, 1977.

WOOLF, A.; KRADEL, D.; ROTHENBACHER, H. Prevalence of renal urolithiasis in a large, captive White-Tailed Deer herd. **Journal of Wildlife Diseases**, v. 12, n. 3, p. 306-309, 1976.

WOODS, L. W.; ANDERSON, M. L.; SWIFT, P. K.; SVERLOW, K. W. Systemic neosporosis in a California black-tailed deer (*Odocoileus hemionus columbianus*). **Journal of Veterinary Diagnostic Investigation**, v. 6, n. 4, p. 508-510, 1994.

WOODS, L. W.; SWIFT, P. K.; BARR, B. C.; HORZINEK, M. C.; NORDHAUSEN, R. W.; STILLIAN, M. H.; PATTON, J. F.; OLIVER, M. N.; JONES, K. R.; MACLACHLAN, N. J. Systemic adenovirus infection associated with high mortality in mule deer (*Odocoileus hemionus*) in California. **Veterinary Pathology Online**, v. 33, n. 2, p. 125-132, 1996.

WOODS, L. W.; HANLEY, R. S.; CHIU, P. H.; BURD, M.; NORDHAUSEN, R. W.; STILLIAN, M. H.; SWIFT, P. K. Experimental adenovirus hemorrhagic disease in yearling black-tailed deer. **Journal of Wildlife Diseases**, v. 33, n. 4, p. 801-811, 1997.

WOODS, L. W.; HANLEY, R. S.; CHIU, P. H.; LEHMKUHL, H. D.; NORDHAUSEN, R. W.; STILLIAN, M. H.; SWIFT, P. K. Lesions and transmission of experimental adenovirus hemorrhagic disease in Black-Tailed Deer fawns. **Veterinary Pathology**, v. 36, n. 2, p. 100-110, 1999.

WOODS, L. W.; LEHMKUHL, H. D.; SWIFT, P. K.; CHIU, P. H.; HANLEY, R. S.; NORDHAUSEN, R. W.; STILLIAN, M. H.; DREW, M. L. Experimental adenovirus hemorrhagic disease in White-Tailed Deer fawns. **Journal of Wildlife Diseases**, v. 37, n. 1, p. 153-158, 2001.

WYAND, D. S.; LANGHEINRICH, K.; HELMBOLDT, C. F. Aspergillosis and renal oxalosis in a white-tailed deer. **Journal of Wildlife Diseases**, v. 7, n. 1, p. 52-56, 1971.

YOON, B. I., KWEON, O. K., KWON, S. W., SHIN, N. S., SEO, I. B., KIM, D. Y. Concurrent multicentric hemangiosarcoma and ovarian teratoma in an aged Père David's deer (*Elaphurus davidianus*). **Journal of Zoo and Wildlife Medicine**, p. 456-458, 1999.

ZIMPEL, C. K.; GRAZZIOTIN, A. L.; BARROS FILHO, I. R. D.; GUIMARAES, A. M. D. S.; SANTOS, L. C. D.; MORAES, W. D.; CUBAS, Z. S.; OLIVEIRA, M. J.; PITUCO, E. M.; LARA, M. C. C. S. H.; VILLALOBOS, E. M. C.; SILVA, L. M. P.; CUNHA, E. M. S.; CASTRO, V.; BIONDO, A. W. Occurrence of antibodies anti-*Toxoplasma gondii*, *Neospora caninum* and *Leptospira interrogans* in a captive deer herd in Southern Brazil. **Revista brasileira de parasitologia veterinária= Brazilian journal of veterinary parasitology: Orgao Oficial do Colegio Brasileiro de Parasitologia Veterinaria**, v. 24, n. 4, p. 482-487, 2015.

APPENDIX

APPENDIX A. Summary of the gross lesions.

LEGENDS.

Photo 1) BBD, adult, male (MG-131). Carcass. Focal open fracture on the tibia of right hindlimb.

Photo 2) BBD, adult, male (MG-090). Hindlimbs. Multifocal cutaneous lacerations and abrasions with hair loss/alopecia.

Photo 3) BBD, adult, male (MG-131). Head. Multifocal cutaneous lacerations and hemorrhage (white arrowhead) around the lips, eyes and pinna.

Photo 4) BBD, adult, male (MG-095). Head. Multiple ticks on the skin of the zygomatic and buccinator region (white arrowhead).

Photo 5) BBD, adult, male (MG-090). Hindlimb. Multifocal locally extensive intramuscular hemorrhage of semimembranosus and semitendinosus muscles.

Photo 6) BBD, adult, female (MG-136). Rumen. Severe, diffuse distention of the ventral and dorsal ruminal sacs (ruminal bloat).

Photo 1 - BBD. Open fracture on the right hind limb

Photo 2 - BBD. Hindlimbs, lacerations and alopecia

Photo 3 - BBD. Note lacerations and hemorrhage (white arrowhead) around the eyes and lips

Photo 4 - BBD. Multiple ticks on the skin of the zygomatic region (white arrowhead)

Photo 5 - BBD. Hemorrhage of the semimembranosus and semitendinosus muscles

Photo 6 - BBD. Ruminal bloat

LEGENDS.

Photo 7) BBD, adult, male (MG-131). Carcass, ventral abdomen. Inguinal hernia.

Photo 8) BBD, adult, male (MG-131). Abdomen, hernial sac. Intestinal loops with hemorrhage and mesentery.

Photo 9) BBD, adult, male (MG-131). Distal hind limb. Comminuted fracture of the metatarsal bones

Photo 10) BBD, adult, male (MG-128). Rumen. The ventral sac contains two phytobezoars in in the lumen.

Photo 11) BBD, adult, female (MG-136). Distal intrathoracic trachea. Abundant white-red foamy fluid indicative of severe pulmonary edema.

Photo 12) BBD, adult, male (MG-043) Left lung. Severe fibrinonecrotizing and hemorrhagic bronchopneumonia with pleuritis.

Photo 7 - BBD. Inguinal hernia

Photo 8 - BBD. Hernial sac with hemorrhagic intestinal loops and mesentery

Photo 9 - BBD. Comminuted fracture of the metatarsal bones

Photo 10 - BBD. Phytobezoars in rumen

Photo 11 - BBD. Respiratory system. Severe pulmonary edema

Photo 12 - BBD. Respiratory system. Bronchopneumonia

LEGENDS.

Photo 13 - MD, adult, female (BD-11). Trachea. Mucosal and submucosal edema and hemorrhage, with necrosis and ulcer of the epithelial lining, dilatation of blood and lymphatic vessels and neutrophilic-lymphocytic infiltrate. **Hemorrhagic tracheitis**. H&E. Scale bar= 200µm.

Photo 14 - MD, adult, female (BD-12). Trachea. Mucosal and submucosal edema and hemorrhage, distended blood vessels, necrosis and ulcer of the epithelial lining and mononuclear inflammatory infiltrate. **Mild moderate mononuclear and hemorrhagic tracheitis**. H&E. Scale bar= 100µm.

Photo 15 - MD, adult, male (BD-41). Lung. Note endothelial damage, organized fibrin occlusion of the vascular lumen, and mononuclear infiltration within the interstitium and septal area. **Pulmonary thrombosis**. H&E. 100µm

Photo 16 - BBD, adult, male (MG-076). Lung. The alveolar spaces are diffusely occupied by a large number of erythrocytes. **Severe pulmonary hemorrhage**. H&E. Scale bar= 100µm.

Photo 17 - BBD, adult, female (MG-081). Lung. The lamina propria and submucosa are infiltrated by a small number of granulocytes. **Mild moderate granulocytic bronchiolitis**. H&E. 100 µm.

Photo 18 - MD, adult, male (BD-42). Lung. Focal intraalveolar embryonated and larvated nematode egg (50x70 µm). These elements are associated with minimal histiocytic infiltrate. **Lungworm eggs compatible with Strongylida Order** (Presumably *Protostrongylus* or *Dictyocaulus* genres). H&E. Scale bar= 50µm.

Photo 13 - MD. Trachea. Hemorrhagic tracheitis. H&E. Scale bar= 200µm

Photo 14 - MD. Trachea. Mild moderate mononuclear and hemorrhagic tracheitis. H&E. Scale bar= 100µm.

Photo 15 - MD. Lung. Pulmonary thrombosis. H&E. 100µm

Photo 16 - BBD. Lung. Severe pulmonary hemorrhage, compatible with infarct. H&E. Scale bar= 100µm

Photo 17 - BBD. Lung. Mild moderate granulocytic bronchiolitis. H&E. 100µm

Photo 18 - MD. Lung. Lungworm eggs. H&E. 50µm

LEGENDS.

Photo 19 - MD, adult, male (BD-42). Lung. Alveolar lumen: nematode larva of approximately 250-350 μm long x 40-50 μm wide, composed by cuticle/tegument, hypodermis, pseudocoelum and numerous nuclei. ***Lungworm eggs compatible with Strongylida Order*** (Presumably *Protostrongylus* or *Dictyocaulus* genres). H&E. Scale bar= 50 μm .

Photo 20 - MD, adult, male (BD-42). Lung. Strongylida egg in the alveolar lumen, surrounded by minimal histiocytic infiltrate. ***Early histiocytic infiltration of lungworm on alveolar lumen.*** H&E. Scale bar= 50 μm .

Photo 21 - MD, adult, male (BD-42). Lung. L1 Strongylida larvae surrounded by early granulomatous infiltrate containing multinucleated giant cells (foreign body type) and few granulocytes. ***Parasitic granuloma.*** H&E. Scale bar= 50 μm .

Photo 22 - MD, adult, male (BD-42). Lung. Focal microgranuloma containing few granulocytes with no evident parasitic structure. ***Pulmonary granuloma.*** H&E. Scale bar= 50 μm .

Photo 23 - MD, adult, female (BD-45). Lung. The alveoli are filled with large numbers of neutrophils (viable and degenerate), reactive and foamy macrophages and rare multinucleated giant cells, mixed with erythrocytes (hemorrhage) and fibrin. ***Fibrinosuppurative bronchopneumonia.*** H&E. Scale bar= 50 μm .

Photo 24 - MD, juvenile, female (BD-40). Lung. Marked neutrophilic infiltration and few alveolar macrophages in the alveolar lumen. ***Early stage of suppurative bronchopneumonia.*** H&E. 100 μm .

Photo 19 - MD. Lung. Nematode Larvae compatible with Strongylida Order. H&E. 50µm

Photo 20 - MD. Lung. Early histiocytic infiltration of lung-worm on alveolar lumen. H&E. 50µm

Photo 21 - MD. Lung. Parasitic granuloma. H&E. 50µm

Photo 22 - MD. Lung. Pulmonary granuloma. H&E. 50µm.

Photo 23 - MD. Lung. Fibrinosuppurative bronchopneumonia. H&E. 50µm

Photo 24 - MD. Lung. Early stage of suppurative bronchopneumonia. H&E. 100µm

LEGENDS.

Photo 25 - MD, adult, female (BD-45). Lung. Presence of marked, predominantly fibrinous and neutrophilic exudate in the alveolar and bronchiolar lumen. This exudate contains degenerate neutrophils, alveolar macrophages, cellular debris, few erythrocytes, and fibrin, lamina propria and peribronchial inflammatory infiltrates and bronchiolar epithelial hyperplasia. ***Fibrinosuppurative bronchopneumonia***. H&E. Scale bar= 200µm.

Photo 26 - MD, adult, male (BD-34). Lung. The normal alveolar structure has been replaced by fibrinous exudate along with few macrophages and neutrophils, hemorrhage. There is necrosis of the alveolar wall. ***Fibrinonecrotizing bronchopneumonia***. H&E. 100µm.

Photo 27 - BBD, adult, female (MG-027). Lung. Note extensive fibrin deposition in the alveolar lumen, presence of exudate containing few alveolar macrophages and neutrophils. And alveolar wall congestion, hemorrhage, necrosis and multiple bacterial colonies. ***Fibrinous bronchopneumonia***. H&E. 100µm.

Photo 28 - MD, adult, female (BD-11). Lung. Thickened alveolar septum by distended capillaries (hyperemia, congestion), and few extravasated lymphocytes. Mild acute interstitial pneumonia. H&E. Scale bar= 50µm.

Photo 29 - MD, adult, male (BD-22). Lung. Foci of type II pneumocyte proliferation/hyperplasia (arrow), mononuclear infiltration with few granulocytes in the septal interstitium, and thickening of the alveolar wall. ***Multifocal type II pneumocyte hyperplasia***. H&E. 100 µm.

Photo 30 - BBD, undetermined (MG-042). Lung. Note mild considerable thickening of the alveolar wall. Diffuse mononuclear infiltration of the septal interstitium containing few granulocytes, congestion of alveolar capillaries, and minimal hemorrhage. ***Mild diffuse mononuclear interstitial pneumonia***. H&E. Scale bar= 50µm.

Photo 25 - MD. Lung. Fibrinosuppurative bronchopneumonia. H&E. 200µm

Photo 26 - MD. Lung. Fibrinonecrotic bronchopneumonia. H&E. 100µm

Photo 27 - BBD. Lung. Fibrinous bronchopneumonia. H&E. 100µm

Photo 28 - MD. Lung. Mild acute interstitial pneumonia. H&E. 50µm

Photo 29 - MD, adult, male (BD-22). Lung. Multifocal type II pneumocyte hyperplasia. H&E. 100 µm

Photo 30 - BBD. Lung. Mild diffuse mononuclear interstitial pneumonia. H&E. 50µm

LEGENDS.

Photo 31 - MD, fawn, male (BD-07). Lung. Venule: the adventitial collagen is circumferentially expanded by clear space (edema) and small numbers of lymphocytes, plasma cells and few neutrophils. There is also type II pneumocyte hyperplasia. Focal lymphoplasmacytic **perivascularitis**. H&E. Scale bar= 50µm.

Photo 32 - MD, fawn, male (BD-07). Lung. Septal interstitium: small-size blood vessel with histiocytic infiltration containing few lymphocytes. **Pulmonary mononuclear perivascularitis**. H&E. Scale bar= 50µm.

Photo 33 - MD, adult, male (BD-40). Lung. Venule: adventitial collagen fibers are disrupted and distended by edema and marked infiltration of lymphocytes and plasma cells with few neutrophils. Presence of neutrophilic infiltrate in the alveolar lumen. **Mixed pulmonary perivascularitis associated with bronchopneumonia**. H&E. 100µm.

Photo 34 - MD, undetermined (BD-16). Heart. Area of extensive focal hemorrhage in the epicardium. **Myocardial hemorrhage**. H&E. Scale bar= 200µm.

Photo 35 - BBD, adult, male (MG-049). Heart. The myocardium has been focally replaced by fibro-fatty tissue (fibrosis). **Fibrofatty infiltration**. H&E. Scale bar= 200µm.

Photo 36 - BBD, undetermined (MG-014). Heart, blood vessel. Mycotic thrombosis associated necrotizing arteritis with occlusive thrombosis and medium-sized hyphae angioinvasion (arrow). Mycotic thrombosis and angioinvasion. PAS. Scale bar= 100µm.

Photo 31 - MD. Lung. Pulmonary mononuclear perivascularitis. H&E. 50µm

Photo 32 - MD. Lung. Pulmonary mononuclear perivascularitis. H&E. 50µm

Photo 33 - MD. Lung. Mixed pulmonary perivascularitis associated with bronchopneumonia. H&E. 100µm

Photo 34 - MD. Heart. Myocardial hemorrhage. H&E. 200µm

Photo 35 - BBD. Heart. Fibrofatty infiltration. H&E. 200µm

Photo 36 - BBD. Heart. Mycotic thrombosis and angioinvasion. PAS. 100µm

LEGENDS.

Photo 37 - BBD, undetermined (MG-014). Heart. Note multiple irregularly branched, thin parallel-walled and septated hyphae of approximately 5-10µm in width, in the myocardium. ***Mycotic myocarditis***. PAS. Scale bar= 50µm.

Photo 38 - BBD, undetermined (MG-018). Tongue. Severe muscle fiber necrosis, diffuse leukocytic infiltrate and mild hemorrhage. ***Severe necrosuppurative glossitis***. H&E. 200µm.

Photo 39 - BBD, male (MG-013). Rumen. Mucosal ulceration and severe hemorrhage, and mucosal and submucosal diffuse leukocytic infiltrate. ***Severe hemorrhagic, ulcerative and necrotizing rumenitis***. H&E. 200µm.

Photo 40 - MD, fawn, male (BD-10). Rumen. Marked papillary necrosis with ballooning degeneration, hyperplasia, cellular debris and marked leukocytic infiltrate. ***Necroulcerative rumenitis***. H&E. 100µm.

Photo 41 - MD, fawn, male (BD-10). Rumen. Neutrophilic inflammatory exudate with few histiocytes and lymphocytes, associated with necrosis and intralesional hyphae compatible with *Candida* sp. ***Mycotic necrotizing rumenitis***. H&E. 100µm.

Photo 42 - MD, fawn, male (BD-10). Rumen. Note multiple, dense mats of PAS-stained hyphae-like structures disseminated in the mucosa and submucosa. ***Mycotic necrotizing rumenitis***. H&E. 100µm.

Photo 37 - BBD. Heart. Mycotic myocarditis. PAS. 50µm

Photo 38 - BBD. Tongue. Severe necrosuppurative glossitis. H&E. 200µm

Photo 39 - BBD. Tongue. Severe hemorrhagic ulcerative and necrotizing rumenitis. H&E. 200µm

Photo 40 - MD. Rumen. Necroulcerative rumenitis. H&E. 100µm

Photo 41 - MD. Rumen. Mycotic necrotizing rumenitis. H&E. 100µm

Photo 42 - MD. Rumen. Mycotic necrotizing rumenitis. H&E. 100µm

LEGENDS.

Photo 43 - MD, fawn, male (BD-10). Rumen. Presence of yeasts, hyphae and pseudohyphae compatible with *Candida* sp. ***Mycotic necrotizing rumenitis***. H&E. 100µm.

Photo 44 - BBD, undetermined (MG-029). Reticulum. Marked papillary necrosis and diffuse leukocyte infiltration. ***Necrotizing reticulitis***. H&E. 200µm.

Photo 45 - MD, adult, female (BD-12). Colon. Note extensive hemorrhage, congestion, necrosis and leukocyte infiltration in all tissue layers. ***Hemorrhagic necrotizing colitis***. H&E. 200µm.

Photo 46 - MD, adult, female (BD-43). Liver. Note marked hepatocellular intracytoplasmic macrovesicular change with nuclei marginalization and minimal Kupffer cell hyperplasia. ***Macrovesicular hepatic steatosis***. H&E. 50µm.

Photo 47 - MD, juvenile, male (BD-03). Liver. Note marked hepatocellular intracytoplasmic microvacuolar degeneration. ***Microvesicular steatosis***. H&E. 50µm.

Photo 48 - MD, juvenile, male (BD-03). Liver. Marked multifocal coalescing hemorrhage (asterisk) associated with hepatocyte necrosis (upper right, zone III) and minimal leukocytic infiltrate. ***Hepatic necrosis with multifocal coalescing hemorrhage***. H&E. 200µm.

Photo 43 - MD. Rumen. Mycotic necrotizing rumenitis. H&E. 100µm

Photo 44 - BBD. Reticulum. Necrotizing reticulitis. H&E. 200µm

Photo 45 - MD. Colon. Hemorrhagic necrotizing colitis. H&E. 200µm

Photo 46 - MD. Liver. Macrovesicular steatosis. H&E. 50µm

Photo 47 - MD. Liver. Microvesicular steatosis. H&E. 50µm

Photo 48 - MD. Liver. Hepatic necrosis with multifocal coalescing hemorrhage. H&E. 200µm

LEGENDS.

Photo 49 - BBD, undetermined (MG-008). Liver. Thrombus in a centrilobular vein associated with hepatocellular necrosis in corresponding zone III. ***Thrombosis and centrilobular necrosis***. H&E. 100µm.

Photo 50 - MD, fawn, male (BD-07). Liver. Note severe and diffuse hepatocyte loss and relative increase of bile ducts (arrow). ***Massive hepatocellular necrosis***. H&E. 200µm.

Photo 51 - BBD, juvenile, male (MG-013). Liver. Lymphohistiocytic infiltrate in periductal and portal spaces, with minimal hemorrhage. ***Periportal/pericholangitis lymphohistiocytic hepatitis***. H&E. 50µm.

Photo 52 - BBD, juvenile, male (MG-050). Liver. Infiltrate containing lymphocytes, histiocytes and few granulocytes in the portal space abutting on the limiting plate. ***Focal Periportal lymphohistiocytic hepatitis***. H&E. 50µm.

Photo 53 - BBD, undetermined (MG-042). Liver. The portal triad is markedly disrupted and expanded by... Note severe leukocyte infiltration (lymphohistiocytic). ***Portal lymphohistiocytic cholangiohepatitis***. H&E. 50µm.

Photo 54 - BBD, fawn, male (MG-036). Liver. Note hepatocellular necrosis, associated with leukocytic infiltrate (lymphohistiocytic, with few granulocytes) in zone II. ***Lymphohistiocytic hepatitis***. H&E. 50µm.

Photo 49 - BBD. Liver. Thrombosis and centrolobular necrosis. H&E. 100µm

Photo 50 - MD. Liver. Massive hepatocellular necrosis. H&E. 200µm

Photo 51 - BBD. Liver. Periportal lymphohistiocytic hepatitis. H&E. 50µm

Photo 52 - BBD. Liver. Focal Periportal lymphohistiocytic hepatitis. H&E. 50µm

Photo 53 - BBD. Liver. Portal lymphohistiocytic cholangiohepatitis. H&E. 50µm

Photo 54 - BBD. Liver. Lymphohistiocytic hepatitis. H&E. 50µm

LEGENDS.

Photo 55 - BBD, juvenile, female (MG-032). Kidney. Marked infiltration of lymphocytes, plasma cells and histiocytes in the tunica externa/adventitial collagen of a small-sized cortical artery. **Focal mild-moderate lymphocytic-histiocytic perivascularitis**. H&E. 50µm.

Photo 56 - BBD, undetermined (MG-042). Kidney. Focal mixed leukocytic infiltrate (granulocytes, histiocytes and lymphocytes) in interstitium of the cortex. **Focal mixed interstitial nephritis**. H&E. 50µm.

Photo 57 - MD, adult, female (BD-43). Kidney. Cellular debris and granulocytic cylinders in the lumen of dilated cortical tubules with minimal interstitial infiltrate of lymphocytes, histiocytes and granulocytes. **Diffuse mixed tubulointerstitial nephritis and leukocytic tubular casts**. H&E. 50µm.

Photo 58 - BBD, adult, male (MG-048). Kidney. Glomeruli: proliferation of parietal and mesangial cells and histiocytes. **Focal proliferative glomerulonephritis**. H&E. 50µm.

Photo 59 - BBD, adult, male (MG-048). Kidney. Glomeruli: thickened parietal membrane. **Focal mild membranous glomerulonephritis**. PAS. 50µm

Photo 60 - BBD, adult, female (MG-085). Kidney. Presence of necrotic casts in the lumen of cortical tubules, tubular degeneration and proteinosis. **Necrotic cylinders**. H&E. 50µm.

Photo 55 - BBD. Kidney. Focal mild-moderate lymphocytic-histiocytic perivascularitis. H&E. 50µm

Photo 56 - BBD. Kidney. Focal mixed interstitial nephritis. H&E. 50µm

Photo 57 - MD. Kidney. Diffuse mixed tubulointerstitial nephritis and leukocytic tubular casts. H&E. 50µm

Photo 58 - BBD. Kidney. Focal moderate proliferative glomerulonephritis. H&E. 50µm

Photo 59 - BBD. Kidney. Focal mild membranous glomerulonephritis. PAS. 50µm

Photo 60 - BBD. Kidney. Necrotic cylinders. H&E. 50µm

LEGENDS.

Photo 61 - BBD, fawn, male (MG-053). Skeletal muscle. Acute, monophasic segmental myofiber hyaline degeneration, necrosis and loss. Diffuse hemorrhage and rhabdomyolysis. ***Rhabdomyolysis***. H&E. 100µm.

Photo 62 - BBD, adult, female (MG-073). Skeletal muscle. Degenerated neutrophil- and cellular debris-containing thrombus in the lumen of a medium-sized artery. ***Thrombosis***. H&E. 50µm.

Photo 63 - MD, adult, female (BD-43). Skeletal muscle. Note marked neutrophil infiltration in the endomysium and perymysium, myofiber necrosis and hemorrhage. Presence of neutrophil infiltration and phagocytosis in some muscular fibers. ***Suppurative myositis***. H&E. 50µm.

Photo 64 - BBD, adult, female (MG-073). Skeletal muscle. Marked lymphohistiocytic infiltrate in the epineurium, and leukocyte infiltration of muscle fibers. ***Nonsuppurative perineuritis***. H&E. 100µm.

Photo 65 - BBD, adult, female (MG-027). Skeletal muscle. Muscle fiber protozoal cyst of approximately 150-200 µm, containing oval, thin walled and multiple elongated bradyzoites of approximately 6-8 µm, compatible with *Sarcocystis* sp. There is no evident inflammatory response. ***Focal intrasarcoplasmic Sarcocystid cyst***. H&E. 50µm.

Photo 66 - BBD, juvenile, female (MG-021). Adrenal gland. Mild diffuse congestion of the cortical fascicular and reticular layers. ***Cortical congestion of adrenal gland***. H&E. 200µm.

Photo 61 - BBD. Skeletal muscle.
Rhabdomyolysis. H&E. 100µm

Photo 62 - BBD. Skeletal muscle.
Thrombosis. H&E. 50µm

Photo 63 - MD. Skeletal muscle.
Suppurative myositis. H&E. 50µm

Photo 64 - BBD. Skeletal muscle.
Nonsuppurative perineuritis. H&E. 100µm

Photo 65 - BBD. Skeletal muscle. Focal
Sarcocystid cyst. H&E. 50µm

Photo 66 - BBD. Adrenal gland. Cortical
congestion of adrenal gland. H&E. 200µm

LEGENDS.

Photo 67 - MD, adult, female (BD-21). Adrenal gland. Marked leukocytic infiltrate and necrosis of the cortical-reticular layer. **Adrenalitis**. H&E. 50µm.

Photo 68 - MD, adult, female (BD-21). Adrenal gland. Marked lymphocytic infiltrate and reticular cell necrosis in the cortical-reticular layer. **Focal nonsuppurative adrenalitis**. H&E. 50µm.

Photo 69 - BBD, juvenile, male (MG-039). Brain. Meningeal thickening with marked leukocytic infiltrate and fibroplasia. **Chronic meningitis**. H&E. 200µm.

Photo 70 - BBD, juvenile, male (MG-039). Brain. Note collagen fibers in the sub-meningeal space (blue). **Focally extensive severe meningeal fibroplasia**. Masson. 200µm.

Photo 71 - BBD, juvenile, male (MG-039). Brain. Marked connective tissue-associated lymphohistiocytic exudate containing few granulocytes. **Focal mixed chronic meningitis**. H&E. 50µm.

Photo 72 - BBD, juvenile, male (MG-039). Brain. Staining of glial fibrillary acidic protein on white matter - glial scarring. **Focally extensive severe meningeal scar**. IHQ - GFAP. 200µm.

Photo 67 - MD. Adrenal gland. Adrenalitis. H&E. 50µm

Photo 68 - MD. Adrenal gland. Focal nonsuppurative adrenalitis. H&E. 50µm

Photo 69 - BBD. Brain. Chronic meningitis. H&E. 200µm

Photo 70 - BBD. Brain. Focally extensive severe meningeal fibroplasia. Masson. 200µm

Photo 71 - BBD. Brain. Focal mixed chronic meningitis. H&E. 50µm

Photo 72 - BBD. Brain. Focally extensive severe meningeal scar. IHQ - GFAP. 200µm

LEGENDS.

Photo 73 - BBD, adult, female (MG-017). Spleen. Note brownish intracytoplasmic pigment (hemosiderin) in macrophages, and single megakaryocytes. ***Hemosiderosis and extramedullary hematopoiesis***. H&E. Scale bar= 50µm.

Photo 74 - BBD, fawn, female (MG-019). Skin. Marked dermal and subdermal necrosis, with the presence of cellular debris and neutrophilic infiltrate with degenerated neutrophils, hemorrhage, and fibrinous vasculitis. ***Focally expansive moderate-severe pyoulcerative dermatitis***. H&E. Scale bar= 100µm.

Photo 75 - BBD, juvenile, male (MG-033). Skin. Dermal ulceration, hyperkeratosis, ballooning degeneration and extensive necrosis. ***Focally expansive moderate ulcerative dermatitis***. H&E. Scale bar= 200µm.

Photo 73 - BBD. Spleen. Hemosiderosis and extramedullar hematopoiesis. H&E. 50µm

Photo 74 - BBD. Skin. Focally expansive moderate-severe pyoulcerative dermatitis. H&E. 100µm

Photo 75 - BBD. Skin. Focally expansive moderate ulcerative dermatitis. H&E. 200µm