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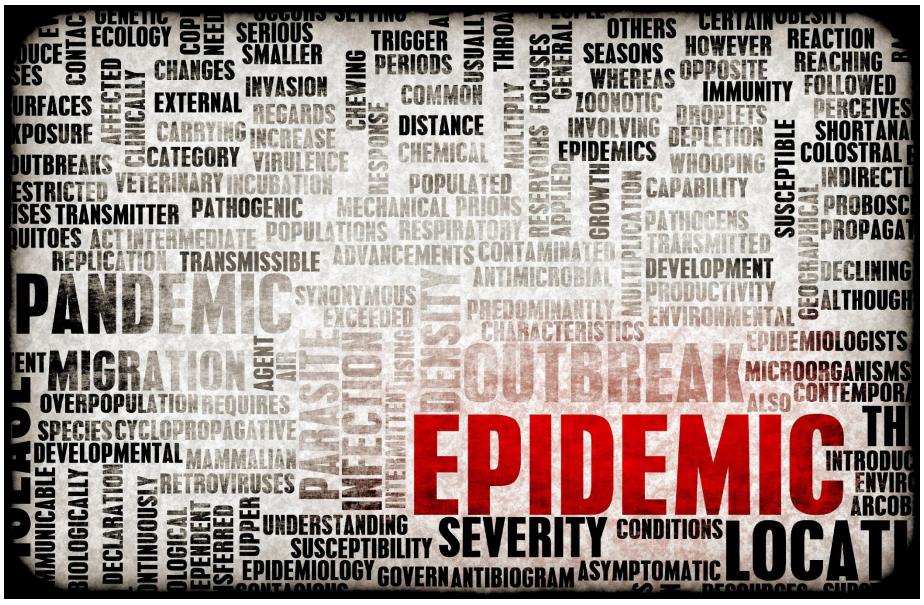
I have the following disclosures related to my presentation:

- Funding Sources - None
- Financial Interests - None
- Other Interests - None

I have the following additional disclosures related to my presentation:

- I am a member of the WSAVA Vaccination Guidelines Group, an independent group of academics, whose work is sponsored by MSD Animal Health.
- For attendance at several previous scientific meetings, the cost of my travel, food and accommodation has been covered by Intervet and Boehringer Ingelheim
- I have in the past undertaken paid teaching work for Merial, Intervet and MSD Animal Health.
- I have undertaken clinical research sponsored by Intervet and Pfizer.

Spoilt for choice…



http://www.phgfoundation.org/blog/16301

AUSTRALIAN VETERINARY JOURNAL
AUSTRALIA'S PREMIER VETERINARY SCIENCE TEXT
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SMALL ANIMALS

Experimental Hendra virus infection of dogs: virus replication, shedding and potential for transmission

DJ Middleton ✉, S Riddell, R Klein, R Arkinstall, J Haining, L Frazer, C Mottley, R Evans, D Johnson, J Pallister

First published: 26 January 2017 Full publication history
DOI: 10.1111/avj.12552 View/save citation
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View issue TOC
Volume 95, Issue 1-2
January/February 2017
Pages 10–18

Abstract

Objective

Characterisation of experimental Hendra virus (HeV) infection in dogs and assessment of associated transmission risk.

Methods

Beagle dogs were exposed oronasally to Hendra virus/Australia/Horse/2008/Redlands or to blood collected from HeV-infected ferrets. Ferrets were exposed to oral fluids collected from dogs after canine exposure to HeV. Observations made and samples tested post-exposure were used to assess the clinical course and replication sites of HeV in dogs, the infectivity for ferrets of canine oral fluids and features of HeV infection in dogs following contact with infective blood.

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CASE REPORTS AND CLINICAL REVIEW

Clinical management of *Brucella suis* infection in dogs and implications for public health

DR James, G Golovsky, JM Thornton, L Goodchild, M Havlicek, P Martin, MB Krockenberger, DJE Marriott, V Ahuja, R Malik ✉, SM Mor ✉

First published: 26 January 2017 Full publication history
DOI: 10.1111/avj.12550 View/save citation
Cited by (CrossRef): 0 articles Check for updates Citation tools ▾

View issue TOC
Volume 95, Issue 1-2
January/February 2017
Pages 19–25

Abstract

Background

Brucellosis caused by *Brucella suis* is a notifiable disease that has recently emerged in dogs in New South Wales (NSW). Given the potential for zoonotic transmission, euthanasia of affected dogs is recommended, but this action is not mandatory. We report the clinical management of three dogs that underwent treatment at their owners' request.

Case reports

A 14-month-old spayed female crossbreed originally obtained from an urban animal shelter underwent extensive investigations in 2011–12 for lameness and back pain, culminating in decompressive laminectomy. Diagnosis of multifocal discospondylitis and spinal empyema was made, with *B. suis* cultured from surgical biopsy specimens. The dog responded to long-term

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Outline of this talk

- Canine leptospirosis – including some unusual clinical presentations
- Parvovirus(es) update, including infections of cats
- An update on feline immunodeficiency and the FIV vaccine
- Update on global companion animal vaccination recommendations

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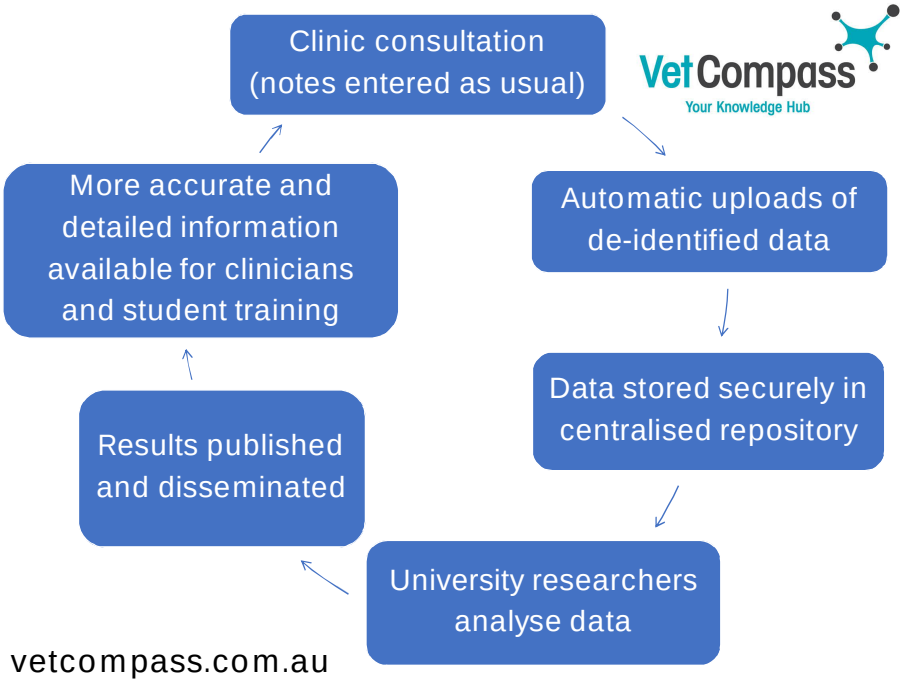

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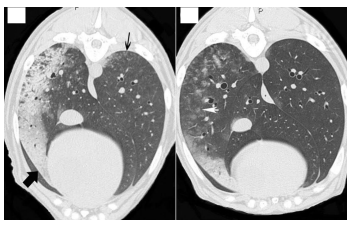
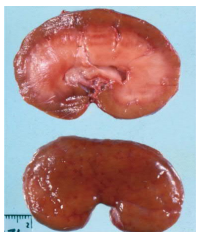
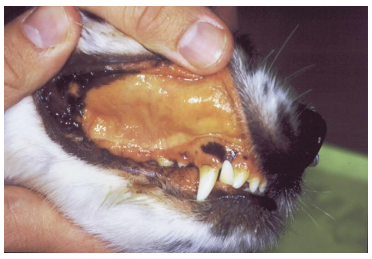
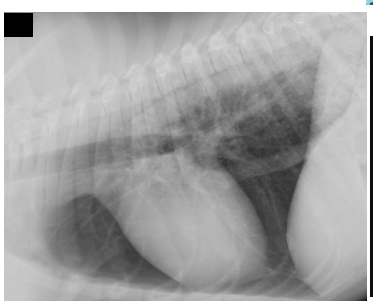
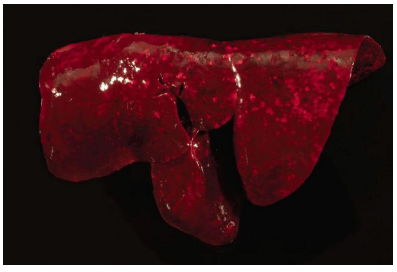
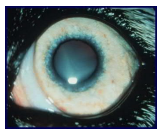
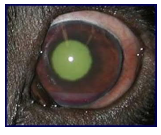
Outline of this talk

- Canine leptospirosis – including some unusual clinical presentations
- Parvovirus update, including infections of cats
- An update on feline immunodeficiency and the FIV vaccine
- Update on global companion animal vaccination recommendations

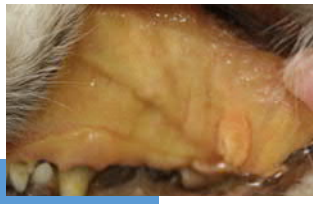
Canine leptospirosis – A diversity of clinical presentations

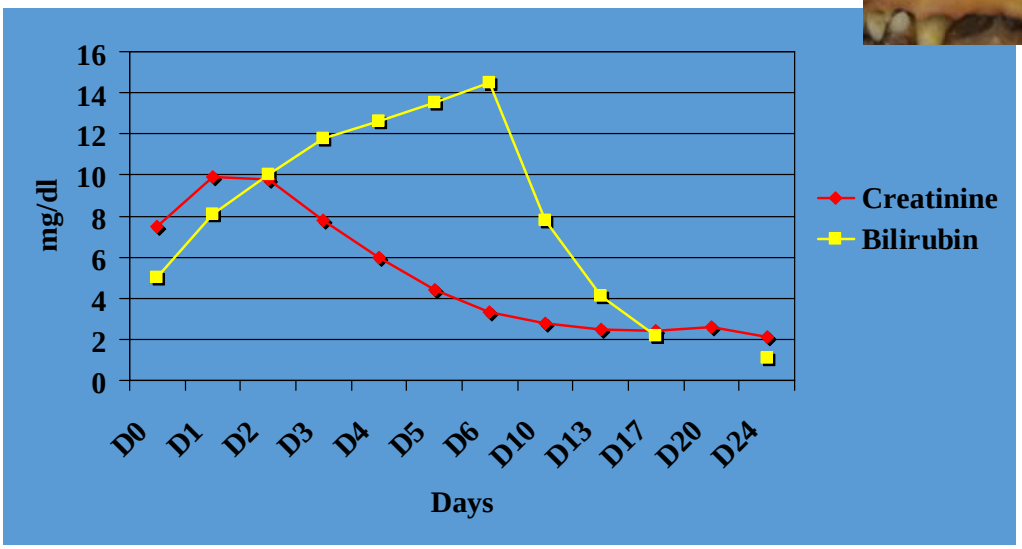








Andy – An Old Classic

- Andy, a 10-year-old male neutered Golden Retriever; lives on a farm
- Referred for icterus and azotaemia
- Presented in late Spring (after rain)
- Oliguric acute to subacute renal failure (< 2ml/kg/hour urine output)
- Developing jaundice
- Initial leptospirosis titres were all < 1:100

Andy: Creatinine and Bilirubin





Case Example: Thor
No jaundice

- 43kg, male, previously healthy German shepherd dog went into oliguric renal failure during a cold winter over the course of 1 – 2 weeks. "Sub-acute renal failure".
- No jaundice
- Suburban dog, no known access to toxins
- Slight neutrophilia, mild fever

Thor: MAT Results from Day 3

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Serogroup	Titre
Hardjo	< 1:100
Icterohaemorrhagiae	< 1:100
Canicola	< 1:100
Grippotyphosa	1:3200
Pomona	1:800

J Am Vet Med Assoc. 1996 Oct 1;209(7):1265-7.

Leptospira interrogans serovar grippotyphosa infection in dogs.

Brown CA[1], Roberts AW, Miller MA, Davis DA, Brown SA, Bolin CA, Jarecki-Black J, Greene CE, Miller-Liebl D.

Author information

[1]Athens Diagnostic Laboratory, College of Veterinary Medicine, University of Georgia, Athens 30602, USA.

Abstract

Leptospirosis attributed to infection with serovar grippotyphosa was diagnosed in 11 dogs. In naturally and experimentally infected dogs, a stereotypic serologic response to infection with Leptospira serovar grippotyphosa was detected. Although the highest serum antibody titers developed against serovar grippotyphosa, most dogs also had lower titers against serovars bratislava and pomona. Acute renal failure was evident in 10 dogs. One dog died prior to initiation of treatment; the remaining 10 dogs were treated with antibiotics and fluids. Two dogs were euthanized, 2 dogs recovered without clinical or biochemical evidence of residual renal dysfunction, and 6 dogs recovered but had varying degrees of renal insufficiency. Hepatic involvement appeared to be a minor component of the disease in these dogs. Our results indicate that Leptospira serovar grippotyphosa infection is an important problem in dogs and should be considered when evaluating a dog with renal failure.

PMID: 8837647

[PubMed - indexed for MEDLINE]

"Hepatic involvement appeared to be a minor component of the disease in these dogs."

Table 2. Summary of 18 leptospirosis-seropositive dogs sampled at dog shelters in mainland Australia during 2004

Case no.	Shelter	Origin	Age (years)	Sex	Titre	Serovar
New South Wales						
1	Central Coast	Urban	1	F	1:50	Canicola
2	Central Coast	Urban	1	F	1:50	Canicola
3	Orange	Rural	1	M	1:100	Copenhageni
4	Newcastle	Urban	3	M	1:200	Arborea
5	Yagoona	Urban	0.8	Fe	1:100	Ballum
6	Yagoona	Urban	3	M	1:100 1:50	Medanensis Panama
7	Yagoona	Urban	2	M	1:100	Copenhageni
8	Yagoona	Urban	0.5	F	1:50	Copenhageni
9	Yagoona	Urban	4	F	1:100	Copenhageni
10	Yagoona	Urban	1.3	F	1:50 1:100 1:800 1:400 1:800	Copenhageni Zanoni Robinsoni Javanica Arborea
Western Australia						
1	Perth	Urban	1	M	1:100	Ballum
Northern Territory						
1	Darwin	Rural	2	M	1:50 1:50	Ballum Arborea
Victoria						
1	Melbourne	Urban	4	F	1:400	Pomona
2	Melbourne	Urban	1	F	1:200	Arborea
3	Melbourne	Urban	2	F	1:200	Pomona
Queensland						
1	Cairns	Urban	0.6	M	1:200	Australis
2	Brisbane	Urban	1.3	F	1:200 1:1600	Ballum Arborea
3	Brisbane	Urban	2	F	1:50 1:400	Ballum Arborea

Clinical and epidemiological features of canine leptospirosis in North Queensland

RI MILLER,[a] SP ROSS,[b] ND SULLIVAN[a] and NR PERKINS[c]

Aust Vet J 2007;85:13–19 doi: 10.1111/j.1751-0813.2006.00089.x

Could there be temporal as well as geographic variation in seroprevalence of the different serogroups / serotypes?

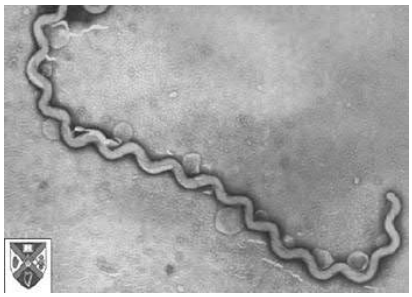
Some fatal leptospirosis cases in very young dogs

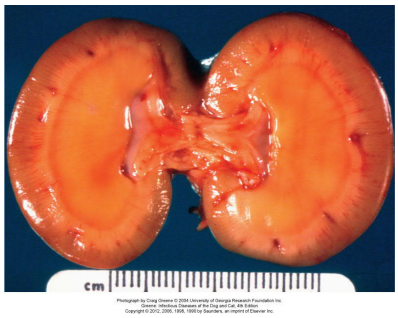
Fatal leptospirosis cases in very young dogs

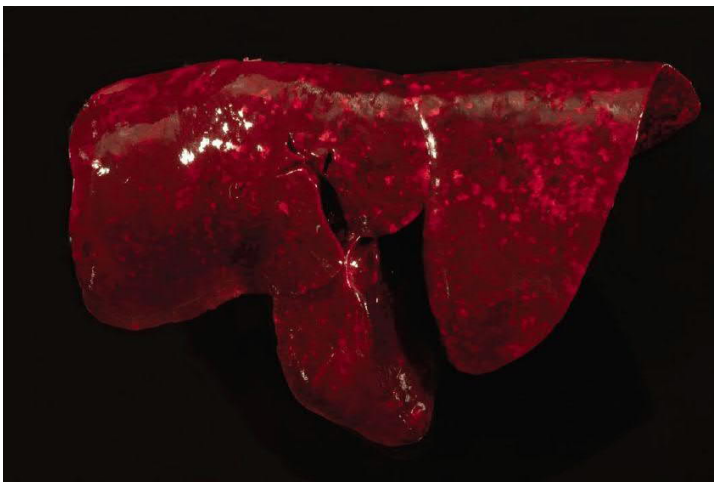
- Young dogs, < 6 months, with a severe hepatic and or renal syndrome
- Despite clinical severity, subtle renal changes on necropsy and histopathology
- Some with and some without jaundice
- Azotaemia more consistently present than jaundice
- Many with pulmonary oedema...

Journal of Veterinary Diagnostic Investigation
2014, Vol. 26(6) 799–804

Lepto? A respiratory pathogen in dogs???

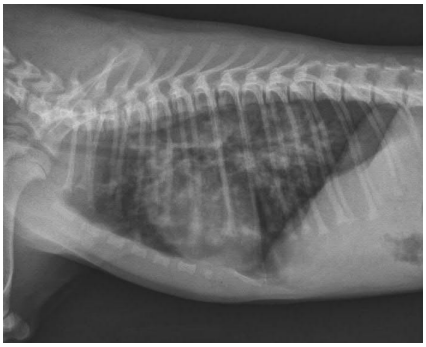


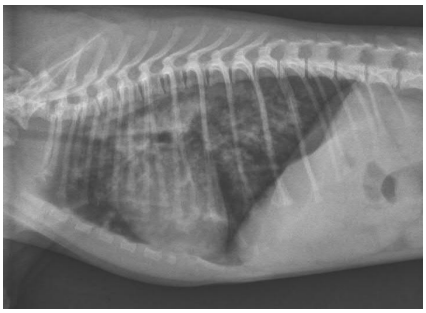


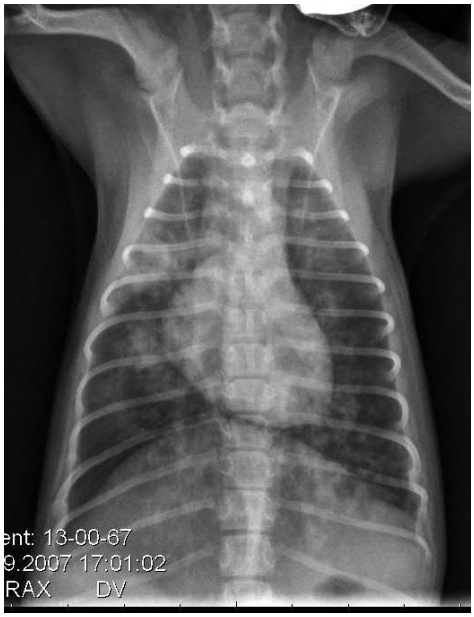


Pulmonary haemorrhage and oedema

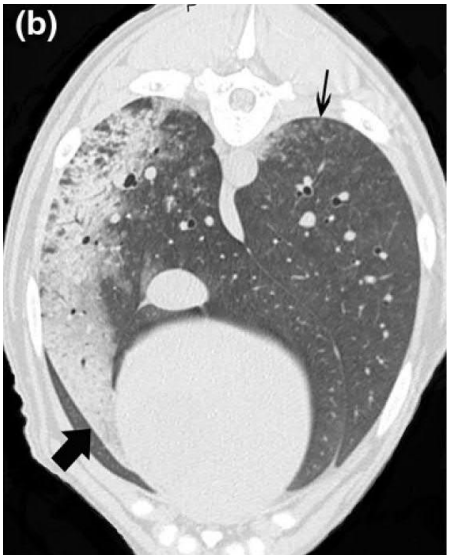
SAGE-Hindawi Access to Research
Veterinary Medicine International
Volume 2010, Article ID 928541, 7 pages
doi:10.4061/2010/928541







If this were not leptospirosis, what could it be?



Serial CT features of pulmonary leptospirosis in 10 dogs.
K. Gendron, A. Christe, S. Walter, A. Schweighauser, T. Francey, M. G. Doherr, J. Lang.
Veterinary Record February 15, 2014.

Veterinary Medicine International
Volume 2010, Article ID 928541, 7 pages
doi:10.4061/2010/928541

Case Report

An Emerging Pulmonary Haemorrhagic Syndrome in Dogs: Similar to the Human Leptospiral Pulmonary Haemorrhagic Syndrome?

R. Klopfleisch,[1] B. Kohn,[2] S. Plog,[1] C. Weingart,[2] K. Nöckler,[3] A. Mayer-Scholl,[3] and A. D. Gruber[1]

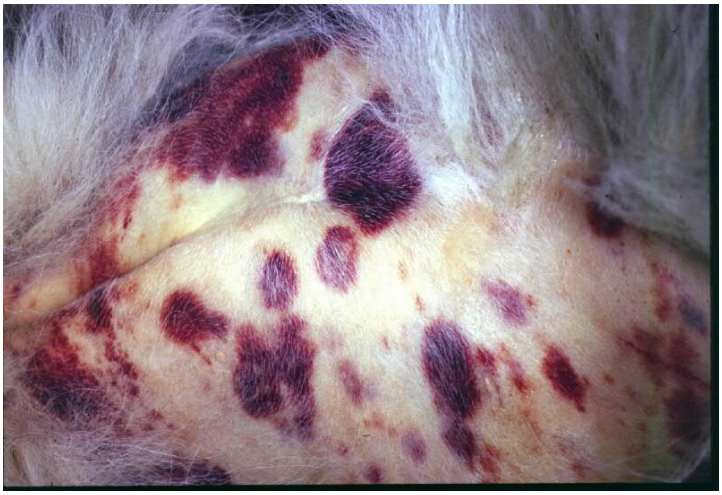
[1] *Institute of Veterinary Pathology, Department of Veterinary Medicine, Freie Universität Berlin, Robert-von-Ostertag-Straße 15, 14163 Berlin, Germany*
[2] *Small Animal Clinic, Department of Veterinary Medicine, Freie Universität Berlin, Oertzenweg 19 b, 14163 Berlin, Germany*
[3] *Department of Molecular Diagnostics, Federal Institute for Risk Assessment, Diedersdorfer Weg 1, 14191 Berlin, Germany*

Correspondence should be addressed to R. Klopfleisch, klopfleisch.robert@vetmed.fu-berlin.de

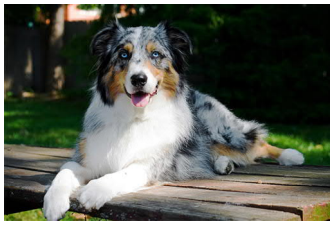
Received 24 November 2010; Accepted 17 December 2010

A large majority of these dogs with serious respiratory disease also have renal involvement (azotaemia)

Haemostatic dysfunction in leptospirosis



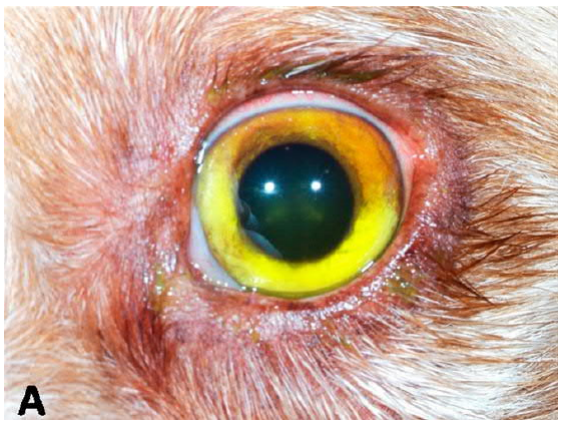
The dog whose eye changed colour…



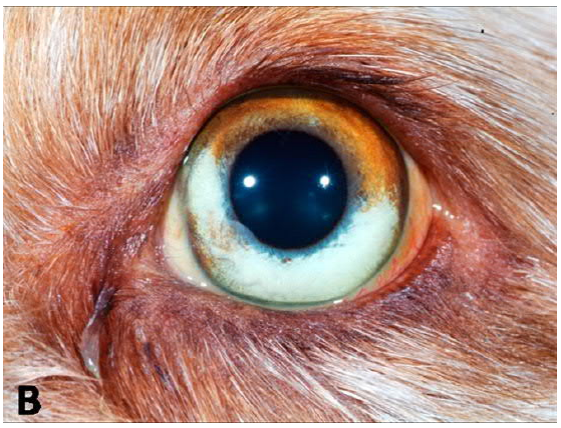
- 7-year-old male castrated Australian shepherd dog. Lives on a farm.
- Sudden change of eye colour, plus lethargy, anorexia.

http://dogtime.com/dog-breeds/australian-shepherd#/slide/7

Initial Presentation



After standard treatment for leptospirosis



(*J Am Anim Hosp Assoc* 2011; 47:e162–e167. DOI 10.5326/JAAHA-MS-5590)

(*J Am Anim Hosp Assoc* 2011; 47:e162–e167. DOI 10.5326/JAAHA-MS-5590)

CASE REPORTS

Leptospirosis in a Dog with Uveitis and Presumed Cholecystitis

Alexander Gallagher, DVM, MS, DACVIM*

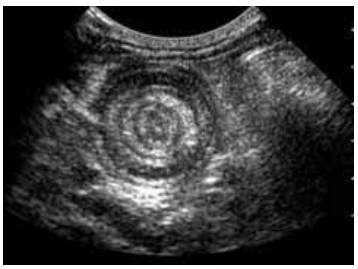
ABSTRACT

A 7 yr old castrated male Australian shepherd dog was examined for acute change in iris color, lethargy, and anorexia. Uveitis, acute renal failure, and presumed cholecystitis were diagnosed. Based on clinical findings, leptosporosis was suspected, and the dog was treated with antibiotics and supportive care. The dog made a complete recovery, and leptospirosis was confirmed on convalescent titers. Due to the zoonotic potential, leptospirosis should be considered in cases of uveitis, as well as possible cholecystitis. (*J Am Anim Hosp Assoc* 2011; 47:e162–e167. DOI 10.5326/JAAHA-MS-5590)

Again, the severe azotaemia was diagnostically crucial

Case Series

Journal of Veterinary Emergency and Critical Care **19**(4) 2009, pp 363–368
doi: 10.1111/j.1476-4431.2009.00432.x



Small intestinal intussusception in five dogs with acute renal failure and suspected leptospirosis (*L. australis*)

Ariane Schweighauser, Dr med vet, DACVIM; Iwan A. Burgener, Dr med vet, DACVIM, DECVIM; Frédéric Gaschen, Dr med vet, DACVIM, DECVIM; Nicole Luckschander, Dr med vet, DACVIM, DECVIM; Andreas Hasler, Dr med vet, DACVIM; Johann Lang, Dr med vet, DECVDI and Thierry Francey, Dr med vet, DACVIM*

Abstract

Objectives – This case series describes 5 dogs with small intestinal intussusception and acute kidney injury due to infection with *Leptospira interrogans* serovar Australis.

Case Series Summary – Small intestinal intussusception was observed in 4 dogs diagnosed with acute kidney injury due to leptospirosis presented between 1997 and 2005. Intussusception was diagnosed at initial presentation or later during hospitalization. An additional dog fulfilling our inclusion criteria was presented to a small animal specialty clinic nearby and was included. Upon admission, all dogs were severely azotemic and thrombocytopenic. All 5 dogs showed the strongest microscopic agglutination test serology reaction to *L. interrogans* serovar Australis. Two dogs survived with no apparent residual renal damage, 1 survived with subsequent mild chronic kidney disease, and 2 dogs were euthanized at the owners' request due to a guarded prognosis.

New or Unique Information Provided – Intussusception can occur or may be seen in dogs with leptospirosis due to *L. interrogans* serovar Australis and patients should be monitored closely for this potential complication. As all 5 dogs described in this case series showed the highest titer for *L. interrogans* serovar Australis, these precautions may be especially applied in geographic areas where this particular serovar is seen.

(J Vet Emerg Crit Care 2009; 19(4): 363–368) doi: 10.1111/j.1476-4431.2009.00432.x

Keywords: gastrointestinal motility disorder, kidney

"Upon admission, all dogs were severely azotaemic." – A very important clue

CASE REPORTS

Clinical Leptospirosis in Three Cats (2001–2009)

Josianne Arbour, DVM, Marie-Claude Blais, DMV, DACVIM, Lisa Carioto, DVM, DVSc, DACVIM, Doris Sylvestre, DMV, MSc

ABSTRACT

Based on previous research, cats were thought to have been resistant to the development of clinical signs following infection with *Leptospira* spp. This case report presents three confirmed, naturally infected clinical cases of feline leptospirosis. The cases presented were all indoor/outdoor cats that were known to hunt. They were also all presented at different stages of renal insufficiency; however, they did not show any liver involvement. The authors suggest that there may be a longer incubation period in cats than dogs and recommend further research in the form of a large, clinical study. (*J Am Anim Hosp Assoc* 2012; 48:256–260. DOI 10.5326/JAAHA-MS-5748)

JAAHA 48:256-260, 2012

Cat 1: Hyposthenuria (1.005), marked neutrophilia, azotaemia. Pomona 1:12,800. Complete response to ampicillin & doxycycline.

Cat 2: PU/PD, haematuria, RBC casts, uveitis, forelimb lameness, azotaemia. 1:1600 Pomona & Bratislava. Improved but persistent uveitis.

Cat 3: Collapsed, severe azotaemia, thrombocytopenia, large irregular kidneys, CNS signs, dyspnoea, death. Severe tubulointerstitial nephritis. Bratislava & Autumnalis 1:1600, Pomona & Icterohaemorrhagiae 1:3200

Research update • February 2017

IDEXX

More information on the clinical performance of the SNAP® Lepto Test is now available

IDEXX, as a leader in pet health-care innovation, developed an enzyme-linked immunosorbent assay (ELISA) for *Leptospira*-specific antibodies that can be performed as a point-of-care SNAP® test or as an IDEXX Reference Laboratories test. The SNAP® Lepto Test and the Canine *Leptospira* spp. Antibody by ELISA provide fast results at a low cost to assist veterinarians in diagnosing this potentially life-threatening infection. Summaries of two new papers based on research sponsored by IDEXX and published in the (peer-reviewed) *International Journal of Applied Research in Veterinary Medicine* on the performance of the ELISA for *Leptospira*-specific antibodies are provided below.

Performance of a recombinant LipL32-based rapid in-clinic ELISA (SNAP Lepto) for the detection of antibodies against *Leptospira* in dogs[1]

A broad population of canine samples was tested to evaluate the overall agreement of the SNAP Lepto Test with the microscopic agglutination test (MAT).

Purpose

The purpose of this study was to compare the LipL32-based SNAP Lepto Test to the MAT for detection of anti-*Leptospira* spp. antibodies.

Study design

The canine serum samples included in this study were: 460 samples submitted for MAT testing, 150 MAT-negative samples from healthy dogs residing in Alaska, 52 samples positive for anti-*Borrelia burgdorferi* antibodies, and samples from 28 dogs following *Leptospira* vaccination.

Peak MAT titer	Number of samples	Number of SNAP Lepto Test positive	Percent SNAP Lepto Test positive
100	8	5	62.5%
200	20	11	55.0%
400	29	21	72.4%
800	53	37	69.8%
1600	34	25	73.5%
3200	13	10	76.9%
6400	19	16	84.2%
12800	32	29	90.6%
25600	14	14	100.0%
51200	18	18	100.0%
102400	19	19	100.0%
Total	**259**	**205**	**79.2%**

Table 1. SNAP Lepto Test performance with MAT-positive samples by peak titer

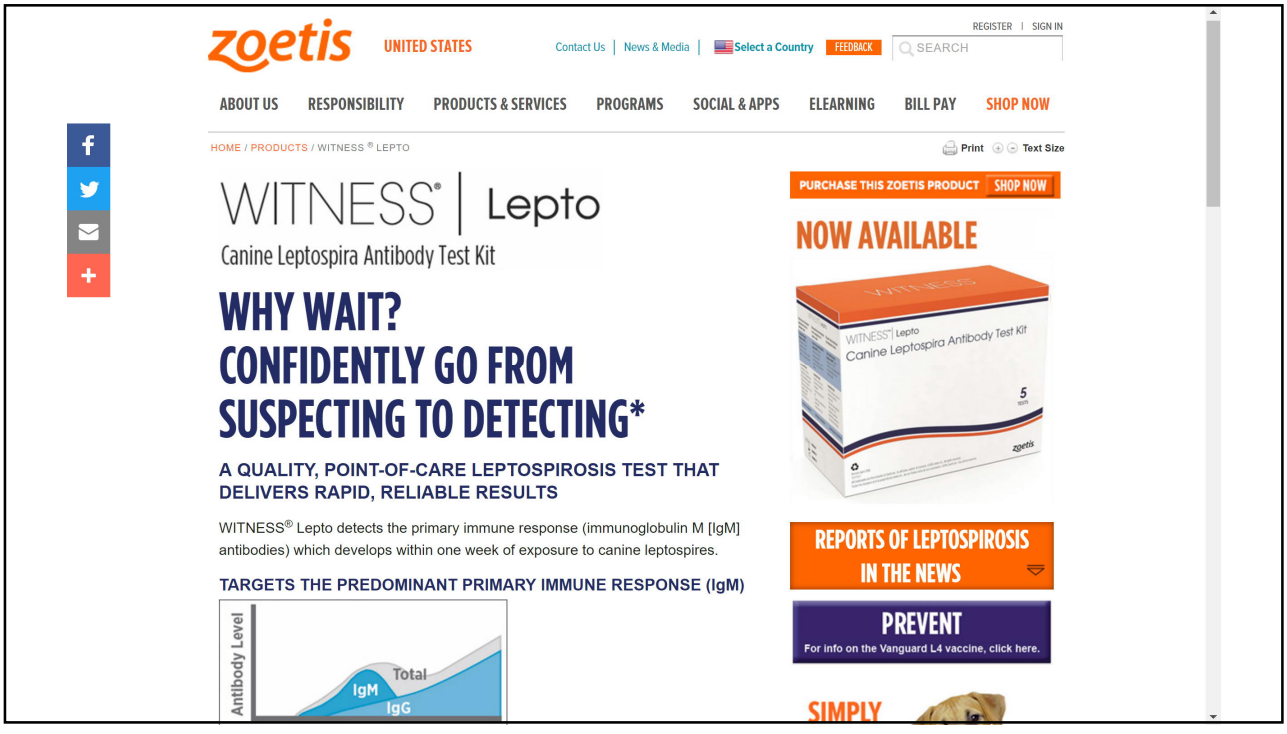
Performance of the new Idexx in-practice SNAP test

Criteria for diagnosis	Number of confirmed leptospirosis cases	Number testing positive on SNAP Lepto Test
Leptospira spp. RealPCR Test positive only (MAT negative)	4	1*
MAT ≥1:800 on initial testing with no history of *Leptospira* vaccination	8	7
MAT titer of ≥1:3200 on initial testing with a previous history of *Leptospira* vaccination or an unknown vaccination history	4	4
4-fold increase in MAT titer between acute and convalescent samples	6	6

*Only known *Leptospira* vaccinate in this confirmed leptospirosis category

Table 3. Criteria used to classify the clinical canine population having a differential diagnosis of leptospirosis

Cases may test negative early in the clinical course, seroconverting a little later



Last week

Paper

OPEN ACCESS

Evaluation of a rapid IgM detection test for diagnosis of acute leptospirosis in dogs

J. Lizer, M. Grahlmann, H. Hapke, S. Velineni, D. Lin, B. Kohn

Recently, a lateral flow assay (LFA) for detection of *Leptospira*-specific IgM in canine sera became commercially available in Europe. The present study aims to evaluate the diagnostic performance of this assay using canine sera from a collection of diagnostic accessions. Diagnostic sensitivity was assessed by testing 37 acute-phase and 9 corresponding convalescent-phase sera from dogs with a confirmed diagnosis of leptospirosis. Specificity was determined by testing sera from sick dogs with non-leptospiral infections (n=15) and healthy dogs with incomplete history of vaccination (n=45). During acute phase of illness, LFA scored positive for 28/37 sera with a sensitivity of 75.7 per cent while only 9/37 (24.3 per cent) samples were positive on microscopic agglutination test. The specificity of the LFA was 98.3 per cent (59/60). This test showed 89.7 and 100 per cent overall agreements with clinical diagnosis for acute-phase and convalescent-phase sera, respectively. The impact of vaccination on the LFA was also determined and vaccine-stimulated IgM responses were negative in 19/25 (76 per cent) dogs at 12 weeks post vaccination. In conclusion, the LFA is a rapid and reliable test for early detection of *Leptospira*-specific IgM during acute phase of canine leptospirosis. However, interpretation of a positive result must be made in the context of clinical signs and vaccination history.

NEWS: Reports Show UK Dogs Are Dying From Lepto Vaccine

Leptospirosis In Dogs / By Dana Scott

4.7k

UK dog owners are learning what US dog owners have suspected all along …

…the lepto vaccine is much more dangerous than we're led to believe.

The Nobivac L4 lepto vaccine, which was rolled out in the UK by Merck's UK subsidiary, MSD Animal Health, is reportedly causing adverse effects in the dogs receiving it, including epilepsy, swollen glands, blindness and death.

Recent Posts


Cancer In Dogs: How To Fight Back With These 3 Herbs


Is Your Pet A Hot Dog Or Cool Cat?


The Natural First Aid Kit For Hiking With Your Dog

http://www.dogsnaturallymagazine.com/report-lepto-vaccine-uk-dogs-dying/

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BMC Vet Res. 2017; 13: 138.
Published online 2017 May 25. doi: 10.1186/s12917-017-1056-x
PMCID: PMC5445508

Clinical, serological and echocardiographic examination of healthy field dogs before and after vaccination with a commercial tetravalent leptospirosis vaccine

Andrea M. Spiri,[1,2] Sabrina Rodriguez-Campos,[4] José M. Matos,[3] Tony M. Glaus,[3] Barbara Riond,[1] Claudia E. Reusch,[3] Regina Hofmann-Lehmann,[1,2] and Barbara Willi[1,3]

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Abstract

Go to:

Background

Leptospirosis is a re-emerging bacterial zoonosis caused by spirochetes of the genus *Leptospira*. Severe disease has been reported in dogs in Europe despite vaccination with bivalent *Leptospira* vaccines. Recently, a tetravalent canine *Leptospira* vaccine (Nobivac® L4) was licenced in Europe. The goal of this study was to investigate clinical signs, microscopic agglutination test (MAT) titres, haematology, blood biochemistry, cardiac (c) Troponin I levels and echocardiography before and after vaccination with this tetravalent vaccine. Forty-eight healthy dogs were prospectively enrolled and vaccinated twice, 3–4 weeks apart (T0 and T1). Before vaccination (T0) and 16–31 days after the second vaccination (T2), MAT ($n = 48$), haematology ($n = 48$), blood biochemistry ($n = 36$) and cTroponin I measurements ($n = 29$) were performed, and MAT was repeated 347–413 days after the second vaccination (T3, $n = 44$). Echocardiography was performed before the first and second vaccination (T0 and T1, $n = 24$).

International Journal of *Molecular Sciences*

MDPI

Review

Reverse Vaccinology: An Approach for Identifying Leptospiral Vaccine Candidates

Odir A. Dellagostin [1,*], André A. Grassmann [1], Caroline Rizzi [1], Rodrigo A. Schuch [1], Sérgio Jorge [1], Thais L. Oliveira [1], Alan J. A. McBride [1] and Daiane D. Hartwig [2]

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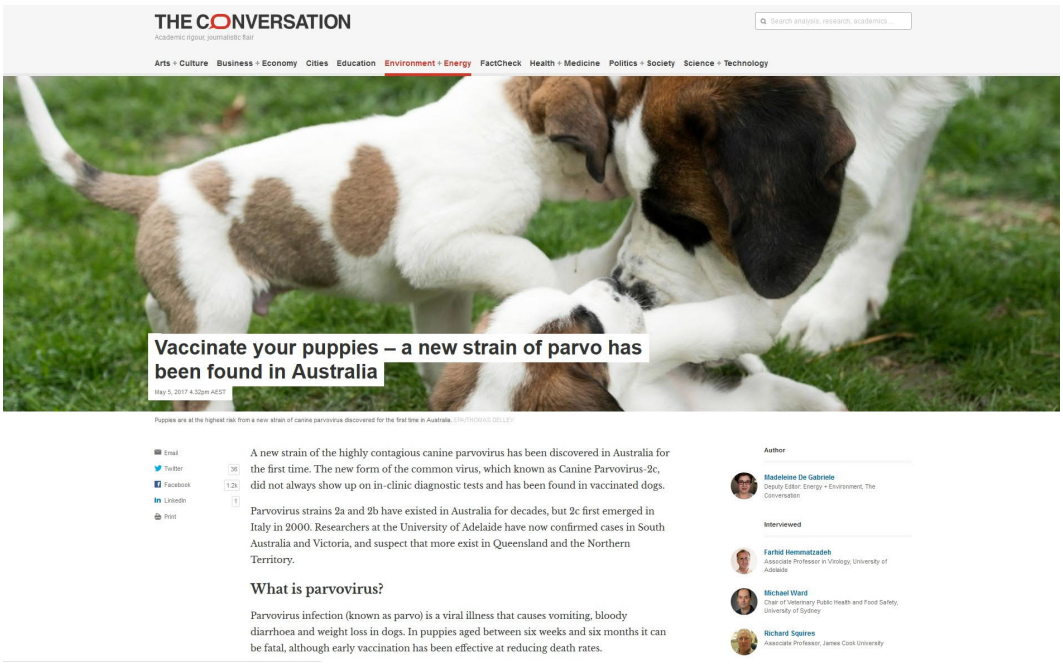
Academic Editor: Christopher Woelk
Received: 27 October 2016; Accepted: 6 January 2017; Published: 14 January 2017

Abstract: Leptospirosis is a major public health problem with an incidence of over one million human cases each year. It is a globally distributed, zoonotic disease and is associated with significant economic losses in farm animals. Leptospirosis is caused by pathogenic *Leptospira* spp. that can infect a wide range of domestic and wild animals. Given the inability to control the cycle of transmission among animals

Outline of this talk

- Canine leptospirosis – including some unusual clinical presentations
- Parvovirus update, including infections of cats
- An update on feline immunodeficiency and the FIV vaccine
- Update on global companion animal vaccination recommendations



THE CONVERSATION

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Vaccinate your puppies – a new strain of parvo has been found in Australia

May 5, 2017 4.32pm AEST

Puppies are at the highest risk from a new strain of canine parvovirus discovered for the first time in Australia. ERIK THOMAS SELLEY

A new strain of the highly contagious canine parvovirus has been discovered in Australia for the first time. The new form of the common virus, which known as Canine Parvovirus-2c, did not always show up on in-clinic diagnostic tests and has been found in vaccinated dogs.

Parvovirus strains 2a and 2b have existed in Australia for decades, but 2c first emerged in Italy in 2000. Researchers at the University of Adelaide have now confirmed cases in South Australia and Victoria, and suspect that more exist in Queensland and the Northern Territory.

What is parvovirus?

Parvovirus infection (known as parvo) is a viral illness that causes vomiting, bloody diarrhoea and weight loss in dogs. In puppies aged between six weeks and six months it can be fatal, although early vaccination has been effective at reducing death rates.

Author

Madeleine De Gabriele
Deputy Editor: Energy + Environment, The Conversation

Interviewed

Farhid Hemmatzadeh
Associate Professor in Virology, University of Adelaide

Michael Ward
Chair of Veterinary Public Health and Food Safety, University of Sydney

Richard Squires
Associate Professor, James Cook University

VIRAL IMMUNOLOGY
Volume 30, Number 5, 2017

Pp. 1–6
DOI: 10.1089/vim.2017.0019

Original Article

JAMES COOK UNIVERSITY AUSTRALIA

Detection of the Canine Parvovirus 2c Subtype in Australian Dogs

Lucy Woolford,[1] Paul Crocker,[1] Hannah Bobrowski,[1] Trevor Baker,[2] and Farhid Hemmatzadeh[1]

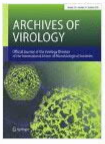
Abstract

Canine parvovirus (CPV-2) is an important cause of hemorrhagic enteritis in dogs. In Australia the disease has been associated with CPV-2a and CPV-2b variants. A third more recently emerged variant overseas, CPV-2c, has not been detected in surveys of the Australian dog population. In this study, we report three cases of canine parvoviral enteritis associated with CPV-2c infection; case 1 occurred in an 8-week-old puppy that died following acute hemorrhagic enteritis. Cases 2 and 3 were an 11-month-old female entire Saint Bernard and a 9-month-old male entire Siberian husky, respectively, both which had completed vaccination schedules and presented with vomiting or mild diarrhea only. Full genomic sequencing of parvoviral DNA from cases 1, 2, and 3 revealed greater than 99% homology to known CPV-2c variants and predicted protein sequences from the VP2 region of viral DNA from all three cases identified; glutamic acid residues at the 426 amino acid residue, characteristic of the CPV-2c variant. Veterinary professionals should be aware that CPV-2c is now present in Australia, detected in a puppy and vaccinated young adult dogs in this study. Further characterization of CPV-2c-associated disease and its prevalence in Australian dogs requires additional research.

Keywords: canine parvovirus 2c, vaccine failure, full genome sequencing

Last year, October…

SpringerLink



Archives of Virology
October 2016, Volume 161, Issue 10, pp 2825–2828

Genetic characterization of feline bocavirus detected in cats in Japan

Authors | Authors and affiliations

Tomomi Takano, Yoshihiro Takadate, Tomoyoshi Doki, Tsutomu Hohdatsu

Brief Report
First Online: 07 July 2016
DOI: 10.1007/s00705-016-2972-y

Cite this article as:
Takano, T., Takadate, Y., Doki, T. et al. Arch Virol (2016) 161: 2825. doi:10.1007/s00705-016-2972-y

103 Downloads

Too early to comment on disease associations; may be relatively benign

Kitten mortality in the United Kingdom: a retrospective analysis of 274 histopathological examinations (1986 to 2000)

T. A. Cave, H. Thompson, S. W. J. Reid, D. R. Hodgson, D. D. Addie

The **Veterinary Record**, October 26, 2002

"The major cause of death of the kittens was FPV, which accounted for 25%. This is surprising given the good uptake of FPV vaccination in the UK, especially by the cat breeding community, and 56 per cent of the kittens were pedigree."

Who is infecting who?

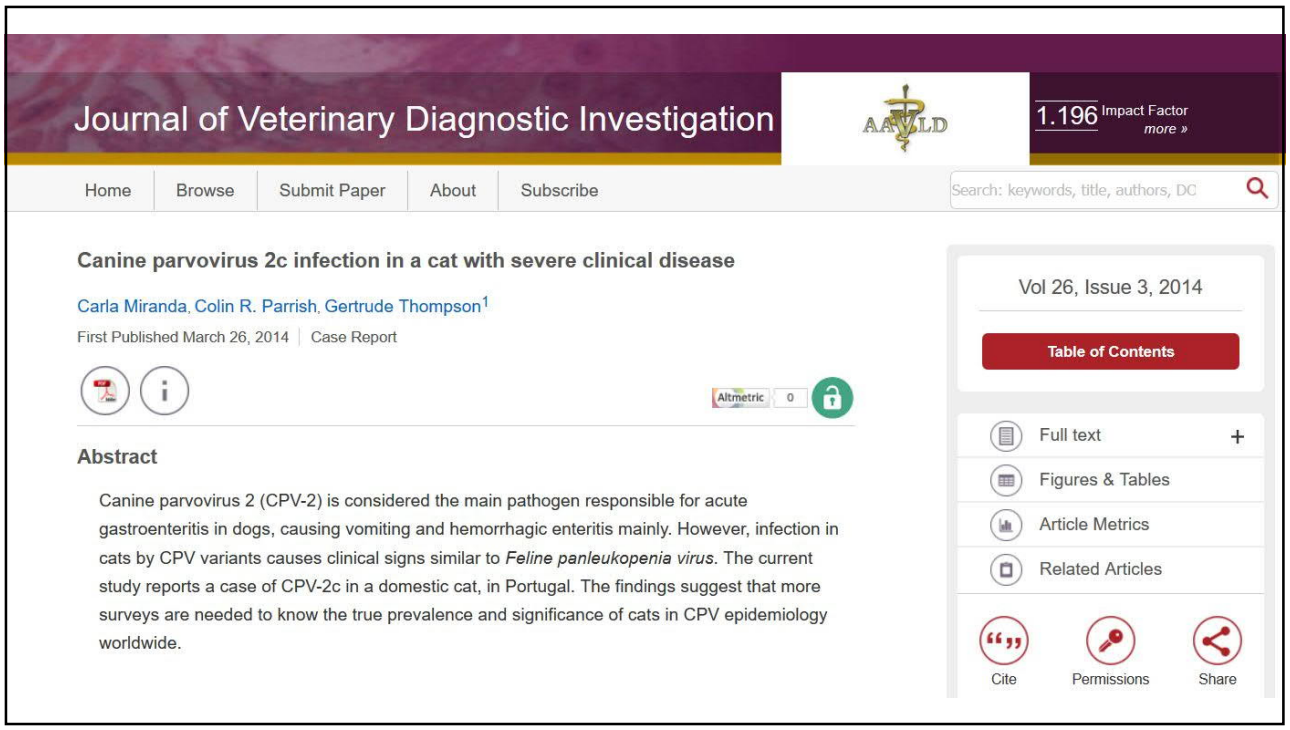
FPV & CPV-2 variants

In vivo

- FPV-type viruses replicate efficiently in cat tissues and are shed in faeces
- FPV-type viruses replicate in thymus and bone marrow of dogs, not in gut
- CPV-2 variants all replicate efficiently in canine and feline tissues, including intestinal tissues

CPV-2a, -2b & -2c in felids

- Domestic cats (all 3)
- Cheetah in Namibia (2b)
- Siberian tiger in Germany (2a)
- Leopard cats in Vietnam and Taiwan (-2a and -2b)



Journal of Veterinary Diagnostic Investigation

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Canine parvovirus 2c infection in a cat with severe clinical disease

Carla Miranda, Colin R. Parrish, Gertrude Thompson[1]

First Published March 26, 2014 | Case Report

Altmetric 0

Abstract

Canine parvovirus 2 (CPV-2) is considered the main pathogen responsible for acute gastroenteritis in dogs, causing vomiting and hemorrhagic enteritis mainly. However, infection in cats by CPV variants causes clinical signs similar to *Feline panleukopenia virus*. The current study reports a case of CPV-2c in a domestic cat, in Portugal. The findings suggest that more surveys are needed to know the true prevalence and significance of cats in CPV epidemiology worldwide.

Vol 26, Issue 3, 2014

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Predominance of *Canine* Parvovirus (CPV) in Unvaccinated Cat Populations and Emergence of New Antigenic Types of CPVs in Cats

Vietnam, Taiwan

"...of feline parvovirus isolates in Vietnam and Taiwan... more than 80% of the isolates were of the canine parvovirus type, rather than feline panleukopenia virus"

Veterinary Microbiology 157 (2012) 78–85

Contents lists available at SciVerse ScienceDirect

Veterinary Microbiology

journal homepage: www.elsevier.com/locate/vetmic

Canine parvovirus in asymptomatic feline carriers

S.R. Clegg [a], K.P. Coyne [a], S. Dawson [a], N. Spibey [b], R.M. Gaskell [a], A.D. Radford [a,*]

[a] Institute of Infection and Global Health, School of Veterinary Science, University of Liverpool, Leahurst Campus, Chester High Road, Neston, South Wirral CH64 7TE, UK
[b] MSD Animal Health, Walton Manor, Walton, Milton Keynes MK7 7AJ, UK

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Carrier
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Emergence

ABSTRACT

Canine parvovirus (CPV) and feline panleukopaenia virus (FPLV) are two closely related viruses, which are known to cause severe disease in younger unvaccinated animals. As well as causing disease in their respective hosts, CPV has recently acquired the feline host range, allowing it to infect both cats and dogs. As well as causing disease in dogs, there is evidence that under some circumstances CPV may also cause disease in cats.

This study has investigated the prevalence of parvoviruses in the faeces of clinically healthy cats and dogs in two rescue shelters. Canine parvovirus was demonstrated in 32.5% (13/50) of faecal samples in a cross sectional study of 50 cats from a feline only shelter, and 33.9% (61/180) of faecal samples in a longitudinal study of 74 cats at a mixed canine and feline shelter. Virus was isolated in cell cultures of both canine and feline origin from all PCR-positive samples suggesting they contained viable, infectious virus. In contrast to the high CPV prevalence in cats, no FPLV was found, and none of 122 faecal samples from dogs, or 160 samples collected from the kennel environment, tested positive for parvovirus by PCR. Sequence analysis of major capsid VP2 gene from all positive samples, as well as the non-structural gene from 18 randomly selected positive samples, showed that all positive cats were shedding CPV2a or 2b, rather than FPLV. Longitudinally sampling in one shelter showed that all cats appeared to shed the same virus sequence type at each date they were positive (up to six weeks), despite a lack of clinical signs. Fifty percent of the sequences obtained here were shown to be similar to those recently obtained in a study of sick dogs in the UK (Clegg et al., 2011).

These results suggest that in some circumstances, clinically normal cats may be able to shed CPV for prolonged periods of time, and raises the possibility that such cats may be important reservoirs for the maintenance of infection in both the cat and the dog population.

CPV was detected in faeces of 32.5% (13/50) of cats in feline-only shelter and 33.9% of cats in a mixed shelter. All healthy. No FPV!

Australian VETERINARY JOURNAL

THE JOURNAL OF THE AUSTRALIAN VETERINARY ASSOCIATION LTD

SMALL ANIMALS

Identification of parvovirus in the bone marrow of eight cats

SM Haynes[a]* and SA Holloway[b]

Objective To determine if canine parvovirus (CPV) or feline panleucopenia virus (FPV) genomic sequences are present in adult feline bone marrow samples.

Design Bone marrow samples were obtained from 32 semi-feral cats that were euthanased at an animal shelter. DNA was extracted and subjected to conventional polymerase chain reaction (PCR) designed to determine if CPV or FPV DNA was present. Positive PCR products were purified, cloned and sequenced to differentiate between CPV and FPV.

Results Eight of the bone marrow samples contained parvoviral DNA (7 CPV, 1 FPV).

Conclusion CPV and FPV DNA can be found in the bone marrow of healthy adult cats.

Keywords bone marrow; canine parvovirus; cats; feline panleucopenia virus; polymerase chain reaction

Abbreviations bp, base pairs; CPV, canine parvovirus; FPV, feline panleucopenia virus; PCR, polymerase chain reaction; SNP, single nucleotide polymorphism

Aust Vet J 2012;90:136–139 doi: 10.1111/j.1751-0813.2012.00899.x

Parvoviruses are small, encapsulated viruses of the family *Parvoviridae* and contain a single negative-stranded DNA genome of approximately 5200 bases.[1,2] The genome encodes

than 6 weeks and myocarditis in puppies.[2] CPV-2 was found to be very similar, both antigenically and genetically, to FPV and these viruses were classified as host-range variants of feline parvoviruses in the genus *Parvovirus*.[2,6,7] In 1979 and around 1985, two new variants of CPV were discovered, CPV-2a and CPV-2b, respectively.[8–10] These variants became the prevalent parvoviruses infecting dogs throughout the world, although the proportions of the two types varied in their geographic distribution.[9,11,12] The host range for these viruses also differed from the original CPV-2, which could replicate in feline cell lines, but not in vivo.[7] CPV-2a and CPV-2b were found to replicate in vitro in feline cell lines and in vivo in cats after experimental inoculation.[11–15] Numerous natural CPV-2a and CPV-2b infections in cats have also been reported.[5,6,16–20] More recently, a third variant, CPV-2c, has been isolated from domestic and wild cats, as well as dogs, in many locations throughout the world.[5,6,18,19,21–25]

The pathogenicity of the CPV variants in cats is somewhat variable, with some cats displaying severe clinical signs similar to those caused by the more common FPV[5,17,18,20,21,25] and others displaying minimal to no clinical signs.[11–13,15,16,19,25] Although faecal shedding generally ceases after the development of high titres of virus-neutralising antibody within 1–2 weeks post-infection, both CPV and FPV have been isolated from faecal samples of healthy cats.[5,16,26] Small amounts of CPV DNA have been found in peripheral blood mononuclear cells of experimentally infected domestic cats up to 4 weeks post-infection,[25] in a kitten with neurological deficits[21] and in apparently healthy large cats, despite the presence of virus-neutralising antibodies.[27] Isolation

32 semi-feral cats

11/32 PCR +ve

8/32 good DNA

7 CPV
1 FPV

(25% def. +ve)

Persistent CPV infections in cats

- Inside PBMC despite neutralizing antibodies

RAPID COMMUNICATION

Predominance of Canine Parvovirus (CPV) in Unvaccinated Cat Populations and Emergence of New Antigenic Types of CPVs in Cats

Yasuhiro Ikeda,*[,1] Masami Mochizuki,† Risako Naito,* Kazuya Nakamura,* Takayuki Miyazawa,* Takeshi Mikami,*[,2] and Eiji Takahashi*[,3]

Virology **278**, 13-19 (2000)

Molecular screening by PCR detects panleukopenia virus DNA in formalin-fixed hearts from cats with idiopathic cardiomyopathy and myocarditis.

Meurs KM. Fox PR. Magnon AL. Liu S. Towbin JA.

Department of Veterinary Clinical Sciences, The Ohio State University College of Veterinary Medicine, Columbus, OH 43210, USA. meurs.1@osu.edu

Cardiovascular Pathology **9**(2):119-26, 2000

Molecular screening by PCR detects panleukopenia virus DNA in formalin-fixed hearts from cats with idiopathic cardiomyopathy and myocarditis

"...Panleucopenia virus was identified by PCR in 10 of 31 cats with cardiomyopathy but in <u>none</u> of the controls…"

HUMAN DATA

High Prevalence of Viral Genomes and Multiple Viral Infections in the Myocardium of Adults With "Idiopathic" Left Ventricular Dysfunction

Uwe Kühl, PhD, MD; Matthias Pauschinger, MD; Michel Noutsias, MD; Bettina Seeberg, MD; Thomas Bock, PhD; Dirk Lassner, PhD; Wolfgang Poller, MD; Reinhard Kandolf, PhD, MD; Heinz-Peter Schultheiss, MD

(*Circulation*. 2005;111:887-893.)

EV=23 (9.4%),
ADV=4 (1.6%),
PV B19=126 (51.4%) Parvovirus B19
HHV-6=53 (21.6%),
EBV=5 (2.0%),
HCMV=2 (0.8%),
(27.3% with multiple infections.

Garigliany *et al. BMC Veterinary Research* (2016) 12:28
DOI 10.1186/s12917-016-0657-0

BMC Veterinary Research

RESEARCH ARTICLE Open Access

Feline panleukopenia virus in cerebral neurons of young and adult cats

Mutien Garigliany[1*†], Gautier Gilliaux[1†], Sandra Jolly[1], Tomas Casanova[1], Calixte Bayrou[1], Kris Gommeren[2], Thomas Fett[3], Axel Mauroy[3], Etienne Lévy[1], Dominique Cassart[1], Dominique Peeters[2], Luc Poncelet[4] and Daniel Desmecht[1]

Abstract

Background: Perinatal infections with feline panleukopenia virus (FPV) have long been known to be associated with cerebellar hypoplasia in kittens due to productive infection of dividing neuroblasts. FPV, like other parvoviruses, requires dividing cells to replicate which explains the usual tropism of the virus for the digestive tract, lymphoid tissues and bone marrow in older animals.

Results: In this study, the necropsy and histopathological analyses of a series of 28 cats which died from parvovirus infection in 2013 were performed. Infections were confirmed by real time PCR and immunohistochemistry in several organs. Strikingly, while none of these cats showed cerebellar atrophy or cerebellar positive immunostaining, some of them, including one adult, showed a bright positive immunostaining for viral antigens in cerebral neurons (diencephalon). Furthermore, infected neurons were negative by immunostaining for $p27^{Kip1}$, a cell cycle regulatory protein, while neighboring, uninfected, neurons were positive, suggesting a possible re-entry of infected neurons into the mitotic cycle. Next-Generation Sequencing and PCR analyses showed that the virus infecting cat brains was FPV and presented a unique substitution in NS1 protein sequence. Given the role played by this protein in the control of cell cycle and apoptosis in other parvoviral species, it is tempting to hypothesize that a cause-to-effect between this NS1 mutation and the capacity of this FPV strain to infect neurons in adult cats might exist.

Conclusions: This study provides the first evidence of infection of cerebral neurons by feline panleukopenia virus in cats, including an adult. A possible re-entry into the cell cycle by infected neurons has been observed. A mutation in the NS1 protein sequence of the FPV strain involved could be related to its unusual cellular tropism. Further research is needed to clarify this point.

Risks of cross-species transmission?

Outline of this talk

- Canine leptospirosis – including some unusual clinical presentations
- Parvovirus update, including infections of cats
- An update on feline immunodeficiency and the FIV vaccine
- Update on global companion animal vaccination recommendations

Contents lists available at ScienceDirect

Vaccine

journal homepage: www.elsevier.com/locate/vaccine

The protective rate of the feline immunodeficiency virus vaccine: An Australian field study

M.E. Westman [a], R. Malik [b], E. Hall [a], M. Harris [c], J.M. Norris [a,*]

[a] Faculty of Veterinary Science, The University of Sydney, NSW 2006, Australia
[b] Centre for Continuing Veterinary Education, The University of Sydney, NSW 2006, Australia
[c] Centre for Virus Research, The University of Glasgow, Scotland G61 1QH, United Kingdom

A world-first
A retrospective case-control field study
FIV status was carefully determined
Cats were "observed" for 3+ years

440 cats with outdoor access
139 vaccinated (cases)
301 unvaccinated (controls)

M.E. Westman et al./Vaccine 34 (2016) 4752–4758

JAMES COOK UNIVERSITY AUSTRALIA

FIV vaccine field study

- Strict inclusion criteria meant numbers were whittled down to:
  - 89 vaccinates
  - 212 unvaccinated controls
- 5/89 (6%) of vaccinates became infected over the study period
- 25/212 (12%) of unvaccinated controls
- Vaccine protective rate = 56%, but…

M.E. Westman et al./Vaccine 34 (2016) 4752–4758

JAMES COOK UNIVERSITY AUSTRALIA

FIV vaccine field study

- 95% confidence interval for degree of protection extended all the way from -22 to +84%
- p = 0.14, so…
- “Casts doubt on degree of protection afforded in the field…”
- “Retesting before annual revaccination may be prudent…”

FIV vaccine field study

- Power analysis a priori assumptions:
  - 3% prevalence in vaccinated cats
  - 16% in unvaccinated controls
- > 5-fold less FIV prevalence anticipated due to vaccination

- Would a lesser degree of protection be of interest to practitioners? To how high a standard should this particular vaccine be held?

Now, a story about myth busting!

- "Point-of-care diagnostic test kits for diagnosis of FIV infection cannot distinguish truly infected from vaccinated, uninfected cats"
- A workaround was developed (apart from PCR) but it was largely ignored

Misconceptions about PCR

- PCR got a "holistic" undeserved reputation for unreliability, emanating from North America, an accident of history...

The variability of serological and molecular diagnosis of feline immunodeficiency virus infection

D. Bienzle, F. Reggeti, X. Wen, S. Little, J. Hobson, S. Kruth

Abstract — Diagnosis of feline immunodeficiency virus (FIV) infection by polymerase chain reaction (PCR) has recently become available, but little is known about the performance of this assay. The purpose of this study was to determine the sensitivity and specificity of PCR diagnosis of FIV infection. Replicate aliquots of blood samples from cats identified as FIV positive or negative by 2 previous enzyme-linked immunosorbent assay (ELISA) results, and from clinically healthy dogs, were submitted to different laboratories for FIV serologic diagnosis and PCR. The PCR products obtained in 1 laboratory were sequenced to determine the FIV subtype. The PCR assays correctly identified 100%, 80%, and 50% of the FIV-positive samples, and 100%, 90%, and 70% of FIV-negative samples. Each dog sample was reported as FIV PCR positive at least once, and FIV subtypes A, B, and C were identified. It was concluded that PCR tests currently available for FIV infection are unreliable, with highly variable sensitivity and specificity.

Contents lists available at ScienceDirect

Comparative Immunology, Microbiology and Infectious Diseases

journal homepage: www.elsevier.com/locate/cimid

Determining the feline immunodeficiency virus (FIV) status of FIV-vaccinated cats using point-of-care antibody kits

Mark E. Westman[a,*], Richard Malik[b], Evelyn Hall[a], Paul A. Sheehy[a], Jacqueline M. Norris[a,*]

[a] Faculty of Veterinary Science, The University of Sydney, NSW 2006, Australia
[b] Centre for Veterinary Education, The University of Sydney, NSW 2006, Australia

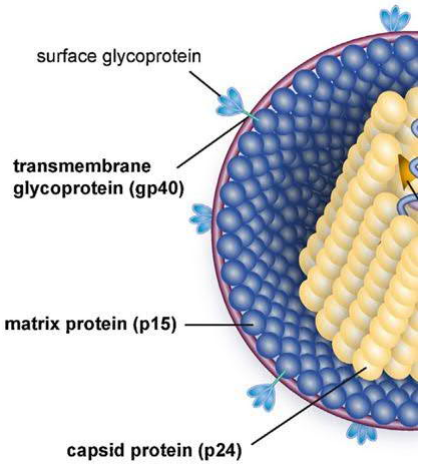
- Those different point-of-care antibody detection tests work quite differently...

JAMES COOK UNIVERSITY AUSTRALIA

Table 1
Summary of the antibodies detected using four different point-of-care FIV antibody test kits.

FIV antibody detection kit	FIV target antigen		
	p15	p24	gp40
SNAP FIV/FeLV Combo (Australia, NZ, North America)	●	●	
SNAP FIV/FeLV Combo Plus (Europe)[a]	●	●	●
Witness FeLV/FIV			●
Anigen Rapid FIV/FeLV		●	●

[a] Not used in this study, but used by Hartmann et al. [19].



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JAMES COOK UNIVERSITY AUSTRALIA

Determining the feline immunodeficiency virus (FIV) status of FIV-vaccinated cats using point-of-care antibody kits

Mark E. Westman[a,*], Richard Malik[b], Evelyn Hall[a], Paul A. Sheehy[a], Jacqueline M. Norris[a,*]

[a] Faculty of Veterinary Science, The University of Sydney, NSW 2006, Australia
[b] Centre for Veterinary Education, The University of Sydney, NSW 2006, Australia

- Whether detection of different proteins has much to do with the clinically important differences in performance between the tests is presently unclear.

M.E. Westman et al. / Comparative Immunology, Microbiology and Infectious Diseases 42 (2015) 43–52

JAMES COOK UNIVERSITY AUSTRALIA

Table 4
Results of three point-of-care FIV antibody test kits in FIV-vaccinated cats ($n = 119$). Confidence intervals (95%) are given in brackets.

Test kit	SNAP Combo	Witness	Anigen Rapid
True +ve	5	5	5
False +ve	114	6	0
True –ve	0	108	114
False –ve	0	0	0
Sensitivity (%)	5/5 = **100**	5/5 = **100**	5/5 = **100**
Specificity (%)	0/114 = **0**	108/114 = **95** (91–99)	114/114 = **100**
PPV (%)	5/119 = **4** (0–8)	5/11 = **45** (16–75)	5/5 = **100**
NPV (%)	0/0 = **0**	108/108 = **100**	114/114 = **100**

False +ve = vaccinated, uninfected cat.
PPV tells the story best, I think.

Further study...

JAMES COOK UNIVERSITY AUSTRALIA

Original Article

jfms Journal of Feline Medicine and Surgery

Journal of Feline Medicine and Surgery
1–10

DOI: 10.1177/1098612X16673292
jfms.com
This paper was handled and processed by the European Editorial Office (ISFM) for publication in *JFMS*
SAGE

Duration of antibody response following vaccination against feline immunodeficiency virus

Mark E Westman[1], Richard Malik[2], Evelyn Hall[1], Matthew Harris[3], Margaret J Hosie[3] and Jacqueline M Norris[1]

Abstract

Objectives Recently, two point-of-care (PoC) feline immunodeficiency virus (FIV) antibody test kits (Witness and Anigen Rapid) were reported as being able to differentiate FIV-vaccinated from FIV-infected cats at a single time point, irrespective of the gap between testing and last vaccination (0–7 years). The aim of the current study was to investigate systematically anti-FIV antibody production over time in response to the recommended primary FIV vaccination series.

Further study…

- Looking at cats during or shortly after primary vaccination, it doesn't work so well.
- 2 weeks post 2nd vaccination:
  - Anigen: 7/12 positive
  - Witness: 8/12 positive
- 1 month after 3rd (final) vaccination:
  - Only 2/12 positive (each test)
- All negative by 6 months post vaccination

Adult cats in for revaccination…

- There is a need to study what is the situation after annual boosting. (Work published to date considered only the primary vaccination program; 3 injections, 4 weeks apart)

"A similar longitudinal study to the current design is required in adult cats prior to and following annual FIV vaccination to determine whether this period of detectable antibody response with PoC test kits such as Witness extends beyond primary FIV vaccination."

Outline of this talk

JAMES COOK UNIVERSITY AUSTRALIA

- Canine leptospirosis – including some unusual clinical presentations
- Parvovirus update, including infections of cats
- An update on feline immunodeficiency and the FIV vaccine
- Update on global companion animal vaccination recommendations

Journal of Small Animal Practice • Vol 57 • January 2016 • © 2016 WSAVA

NIMAL PRACTICE

WSAVA Global Veterinary Community | Vaccination Guidelines Group

GUIDELINES FOR THE VACCINATION OF DOGS AND CATS

COMPILED BY THE VACCINATION GUIDELINES GROUP (VGG) OF THE WORLD SMALL ANIMAL VETERINARY ASSOCIATION (WSAVA)

M. J. Day[1], M. C. Horzinek[2], R. D. Schultz[3] and R. A. Squires[4]

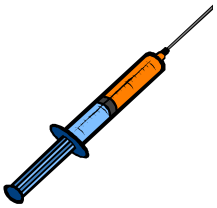
[1]University of Bristol, United Kingdom
[2](Formerly) University of Utrecht, the Netherlands
[3]University of Wisconsin-Madison, Wisconsin, USA
[4]James Cook University, Queensland, Australia

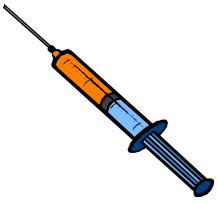
Journal of Small Animal Practice • Vol 57 • January 2016 • © 2016 WSAVA

http://www.wsava.org/guidelines/vaccination-guidelines

What are the updated recommendations?

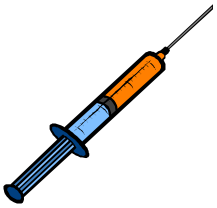
1. Last primary puppy and kitten vaccination goes up from 14 – 16 weeks to 16 weeks plus
2. "First annual booster" (so named) goes from 12 – 16 months to 6 – 12 months
3. FIV vaccine goes from being "not recommended" to "non core"

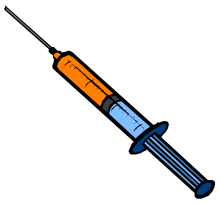




What are the updated recommendations?

4. "Low risk" and "high risk" situations and feline lifestyles are better defined
5. Updated consideration of anatomical sites for injection of vaccines in cats
6. Much more thoroughly referenced. Quality of evidence considered.



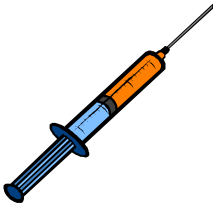


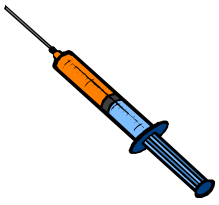
Explaining the updates

Last primary puppy and kitten vaccine goes from 14 – 16 weeks up to 16 weeks plus

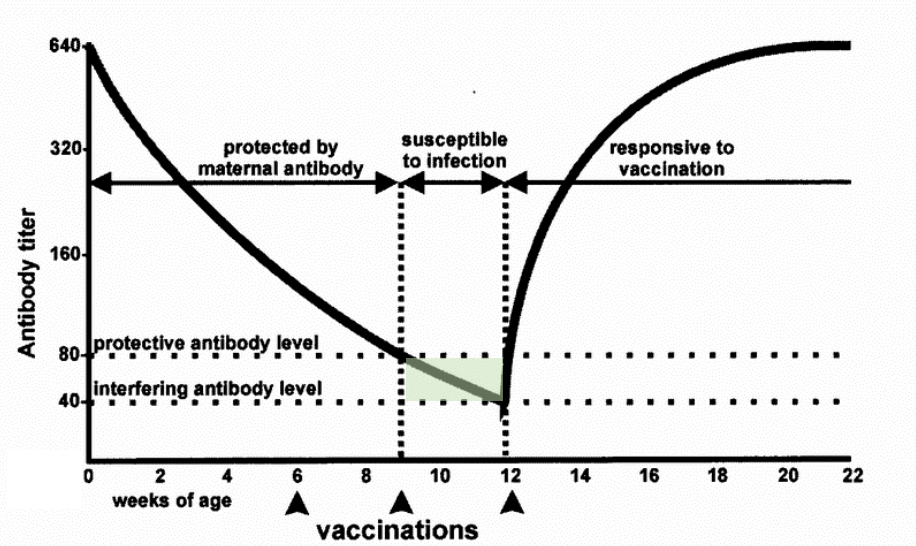
"First annual booster" goes from 12 – 16 months to 6 – 12 months

FIV vaccine goes from "not recommended" to "non core"





Effect of interfering maternal antibody



Original Article

Journal of Feline Medicine and Surgery
14(2) 118–123

DOI: 10.1177/1098612X11432239
jfms.sagepub.com
SAGE

Effects of maternally-derived antibodies on serologic responses to vaccination in kittens

Brian A DiGangi[1], Julie K Levy[2], Brenda Griffin[1], Michael J Reese[1], Patricia A Dingman[2], Sylvia J Tucker[2] and Edward J Dubovi[3]

Abstract

The optimal vaccination protocol to induce immunity in kittens with maternal antibodies is unknown. The objective of this study was to determine the effects of maternally-derived antibody (MDA) on serologic responses to vaccination in kittens. Vaccination with a modified live virus (MLV) product was more effective than an inactivated (IA) product at inducing protective antibody titers (PAT) against feline panleukopenia virus (FPV). IA vaccination against feline herpesvirus-1 (FHV) and feline calicivirus (FCV) was more effective in the presence of low MDA than high MDA. Among kittens with low MDA, MLV vaccination against FCV was more effective than IA vaccination. A total of 15%, 44% and 4% of kittens had insufficient titers against FPV, FHV and FCV, respectively, at 17 weeks of age. Serologic response to vaccination of kittens varies based on vaccination type and MDA level. In most situations, MLV vaccination should be utilized and protocols continued beyond 14 weeks of age to optimize response by all kittens.

Accepted: 12 November 2011

Jakel *et al. BMC Veterinary Research* 2012, **8**:62
http://www.biomedcentral.com/1746-6148/8/62

BMC Veterinary Research

RESEARCH ARTICLE Open Access

Vaccination against Feline Panleukopenia: implications from a field study in kittens

Verena Jakel[1*†], Klaus Cussler[1], Kay M Hanschmann[1], Uwe Truyen[2], Matthias König[3†], Elisabeth Kamphuis[1] and Karin Duchow[1]

Abstract

Background: Feline Panleukopenia (FPL) is a serious disease of cats that can be prevented by vaccination. Kittens are routinely vaccinated repeatedly during their first months of life. By this time maternally derived antibodies (MDA) can interfere with vaccination and inhibit the development of active immunity. The efficacy of primary vaccination under field conditions was questioned by frequent reports to the Paul-Ehrlich-Institut on outbreaks of FPL in vaccinated breeding catteries. We therefore initiated a field study to investigate the development of immunity in kittens during primary vaccination against FPL.
64 kittens from 16 litters were vaccinated against FPL at the age of 8, 12 and 16 weeks using three commercial polyvalent vaccines. Blood samples were taken before each vaccination and at the age of 20 weeks. Sera were tested for antibodies against Feline Panleukopenia Virus (FPV) by hemagglutination inhibition test and serum neutralisation assay in two independent diagnostic laboratories.

Results: There was a good correlation between the results obtained in different laboratories and with different methods. Despite triple vaccination 36.7% of the kittens did not seroconvert. Even very low titres of MDA apparently inhibited the development of active immunity. The majority of kittens displayed significant titres of MDA at 8 and 12 weeks of age; in some animals MDA were still detected at 20 weeks of age. Interestingly, the vaccines tested differed significantly in their ability to overcome low levels of maternal immunity.

Conclusions: In the given situation it is recommended to quantify antibodies against FPV in the serum of the queen or kittens before primary vaccination of kittens. The beginning of primary vaccination should be delayed until MDA titres have declined. Unprotected kittens that have been identified serologically should be revaccinated.

Veterinary Record (2006)
159, 733-736

PAPERS & ARTICLES

39 dogs

2 vaccines

1:320 highest puppy titre

Comparative trial of the canine parvovirus, canine distemper virus and canine adenovirus type 2 fractions of two commercially available modified live vaccines

J. G. H. E. BERGMAN, M. MUNIZ, D. SUTTON, R. FENSOME, F. LING, G. PAUL

The results of vaccinating two groups of puppies with commercial vaccines, both of which claimed to provide adequate protection with a final vaccination at 10 weeks of age, were compared. Groups of 19 and 20 puppies with similar titres of maternally derived antibodies against canine parvovirus (CPV), canine distemper virus (CDV) and canine adenovirus type 2 (CAV-2) at four weeks of age were vaccinated at six and 10 weeks of age and their responses to each vaccination were measured by comparing the titres against CPV, CDV and CAV-2 in the serum samples taken immediately before the vaccination and four weeks later. After the vaccination at six weeks of age, all 19 of the puppies in group 1 had responded to CPV and CDV, and 14 had responded to CAV-2; in group 2, 17 of the 20 had responded to CPV, 19 to CDV and 15 to CAV-2. In both groups the puppies that did not respond to the first vaccination had responded serologically to CPV, CDV and CAV-2 at 10 weeks of age.

63 pups

6 vaccines

Am J Vet Res. 1997 Apr;58(4):360-3.

Comparison of selected canine vaccines for their ability to induce protective immunity against canine parvovirus infection.

Larson LJ[1], Schultz RD.

Author information

Abstract

OBJECTIVE: To compare the ability of 6 commercially available multicomponent canine vaccines to stimulate antibody production in pups with variable amounts of maternally derived canine parvovirus (CPV) antibody and to induce protective immunity against challenge exposure.

ANIMALS: Sixty-three 5- to 6-week-old Beagle pups with passively acquired CPV antibody titer between 1: 20 and 1:320.

PROCEDURE: 9 pups were assigned to each of 6 vaccine groups and 1 control group. Eight pups in each group were inoculated with vaccine or saline solution twice, with 3 weeks between administrations. The ninth pup served as an uninoculated contact control. Serum samples were obtained weekly and tested for CPV antibody by hemagglutination-inhibition assay. All pups were challenge exposed with virulent CPV-2a and CPV-2b at 14 to 15 weeks of age.

RESULTS: 3 of the vaccines failed to provide protective immunity against challenge exposure because all pups in these groups became infected and most died. A fourth vaccine protected against death, but not infection and disease. Two of the 6 vaccines induced an immune response that was protective against infection and disease.

CONCLUSION AND CLINICAL RELEVANCE: Substantial differences existed among commercial vaccines available in 1994 in their ability to immunize pups with maternally derived CPV antibody. These differences caused many vaccinated pups to be susceptible to CPV disease for variable periods because some vaccines failed to immunize. Importantly, all 4 of the vaccines that performed poorly have recently been replaced by more effective products so that the 6 vaccines now perform similarly.

388 dogs

58 litters

4 manufacturers

Aus dem Institut für Medizinische Mikrobiologie, Infektions- und Seuchenmedizin der Tierärztlichen Fakultät der Ludwig-Maximilians-Universität München

Untersuchung der Wirksamkeit von Parvovirusimpfstoffen und der Effektivität zweier Impfschemata

Katrin Friedrich und U. Truyen

Praktischer Tierarzt 81: 12, 988–994 (2000)

ISSN 0032-681 X

ZUSAMMENFASSUNG: In dieser Feldstudie wurde an 388 Welpen aus insgesamt 58 Würfen verschiedener Hunderassen die Wirksamkeit verschiedener in Deutschland zugelassener Impfstoffe auf ihre Fähigkeit zur Induktion von Antikörpern gegen das canine Parvovirus untersucht. Dabei wurden zwei Impfschemata verglichen, die entweder nur Kombinationsvakzinen oder zusätzlich eine Impfung mit einer Parvovirus-Monovakzine beinhalteten. Nach korrekter Grundimmunisierung mit insgesamt zwei beziehungsweise drei Immunisierungen gegen CPV wiesen 92 Prozent der Hunde protektive Antikörperspiegel auf, 8 Prozent blieben ungeschützt. Nach einmaliger Immunisierung mit einer Lebendvakzine in der 6. Woche waren bereits 63 Prozent der Welpen geschützt. Die Ergebnisse dieser Studie implizieren, dass die Grundimmunisierung gegen die Parvovirose in der 6. Lebenswoche der Welpen beginnen sollte, damit der Großteil der Welpen in der kritischen Phase geschützt ist. Sie zeigen aber auch, dass zu einer abschließenden Impfung in der 15. bis 16. Lebenswoche zu raten ist. Die Nutzung des Muttertiertiters als Basis zur Berechnung des günstigsten Zeitpunk-

Einleitung

Die Parvovirose des Hundes wird durch das canine Parvovirus (CPV) hervorgerufen und ist heute die wichtigste Infektionskrankheit des Hundes. Neben seiner großen veterinärmedizinischen Bedeutung besitzt das CPV auch Modellcharakter für das Studium der viralen Evolution, da es sich um ein Virus handelt, das erst vor relativ kurzer Zeit (1978) erstmals in den Hundepopulationen nachgewiesen wurde. Nach seinem plötzlichen Auftreten breitete es sich innerhalb weniger Monate in einer von einer hohen Mortalität gezeichneten Pandemie weltweit aus. Während seiner Adaption an den Wirt Hund kam es 1979 und 1984 zum Auftreten umschriebener Mutationen im Strukturproteinen des Virus, die antigenetische Unterschiede bewirkten (Truyen 1994). Diese Unterschiede ließen sich durch monoklonale Antikörper nachweisen, und die betreffenden Isolate wurden daher als neue „antigene Typen" CPV-2 a und CPV-2 b bezeichnet. Zwischen den antigenen Typen des CPV besteht eine vollständige Kreuzprotektion, obwohl Unterschiede im Neutralisationsverhalten beobachtet werden. Als wichtige biologische Eigenschaft der neuen antigenen Typen ist die Erweiterung des Wirtsspektrums um den Wirt Katze festzustellen (Truyen 1996).

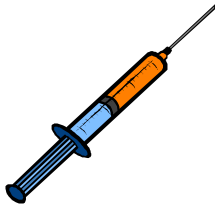
…92.2% seroconverted to CPV after the 12-week vaccination. Possible reasons for the non-responsiveness of nearly 10% of the puppies are discussed."

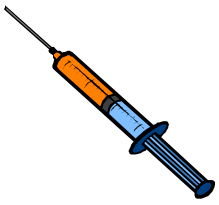
Explaining the updates

Last primary kitten vaccine goes from 14 – 16 weeks up to 16 weeks

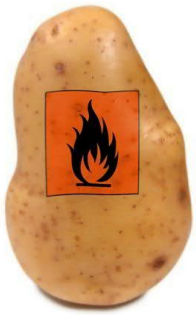
"First annual booster" recommendation goes from 12 – 16 months to 26 – 52 weeks

FIV vaccine goes from "not recommended" to "non core"





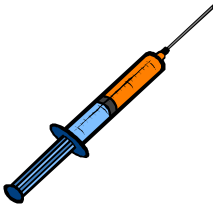
"First annual booster"

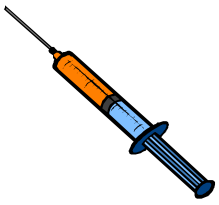


- Change to ~ 26 weeks of age (but guidelines are pragmatic and state up to 52 weeks in this iteration)
- The only immunological rationale for the 12-16 month "booster" (when using modern MLV core vaccines) has been to catch the small percentage of puppies and kittens that fail to respond immunologically at 16 weeks.
- So why leave them open to infection until they are 12 – 16 months of age?
- Absolutely does not preclude a first annual health check

Explaining the updates

1. Last primary kitten vaccine goes from 14 – 16 weeks up to 16 weeks
2. "First annual booster" goes from 12 – 16 months to 6 – 12 months
3. FIV vaccine goes from "not recommended" to "non core"



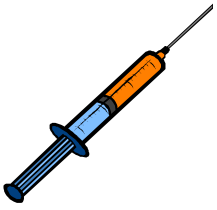


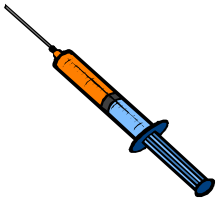
Explaining the updates?

"Low risk" and "high risk" situations and feline lifestyles are better defined

Updated consideration of anatomical sites for injection of vaccines in cats

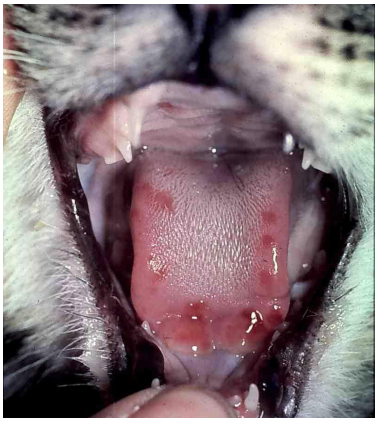
Much more thoroughly referenced. Quality of evidence considered.





What is “high risk”?

- Cats that go into boarding catteries should be vaccinated against FCV / FHV-1 annually, with the injection preferably in the months leading up to boarding



Duration of Immunity (DoI) data

FPV, FHV-1, FCV

- Scott FW, Geissinger CM. (1997) Duration of immunity in cats vaccinated with an inactivated feline panleukopenia, herpesvirus and calicivirus vaccine. Feline Practice 25: 12-19.
- Scott FW, Geissinger CM. (1999) Long-term immunity in cats vaccinated with an inactivated trivalent vaccine. American Journal of Veterinary Research 60: 652-658.

Veterinary Microbiology 177 (2015) 123–131

Contents lists available at ScienceDirect

Veterinary Microbiology

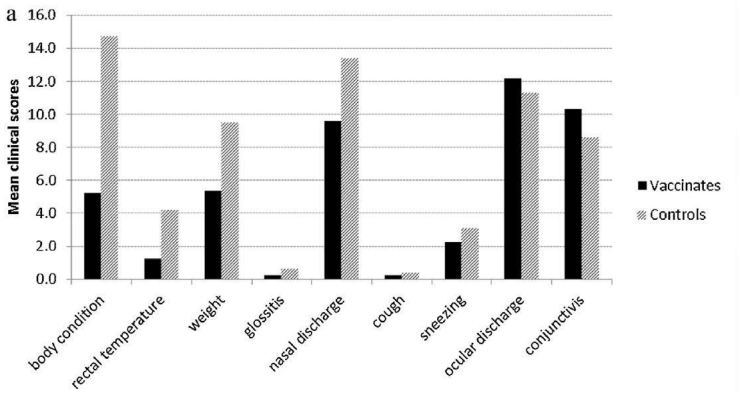
journal homepage: www.elsevier.com/locate/vetmic

Three-year duration of immunity for feline herpesvirus and calicivirus evaluated in a controlled vaccination-challenge laboratory trial

Dominique Jas, Valérie Frances-Duvert, Delphine Vernes, Pierre-Michel Guigal, Hervé Poulet *

Merial S.A.S., R&D, 254 avenue Marcel Mérieux, 69007 Lyon, France

Despite the title of this paper, vaccinated cats developed more significant illness than did the cats in the much earlier Scott & Geissinger studies. Control cats were worse affected, but protection was limited, esp. against FHV-1



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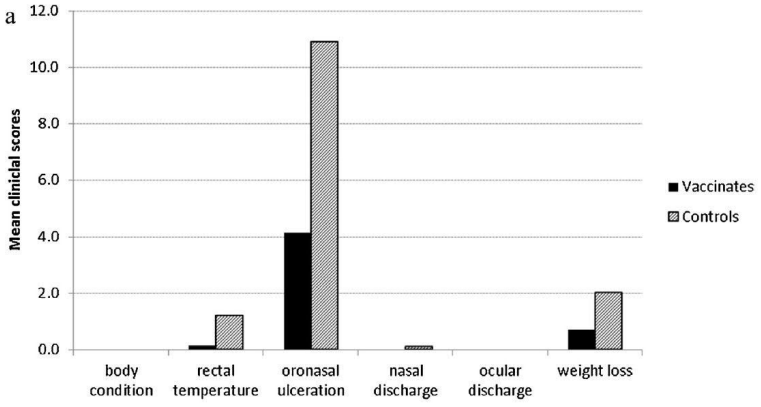


Despite the title of the paper, vaccinated cats developed more significant illness than did the cats in the much earlier Scott & Geissinger studies. Control cats were worse affected than vaccinates, but protection was limited, esp. against FHV-1

Vet. Res. 38 (2007) 337–354
© INRA, EDP Sciences, 2007
DOI: 10.1051/vetres:2006063

Review article

Feline herpesvirus

Rosalind GASKELL[a]*, Susan DAWSON[b], Alan RADFORD[b], Etienne THIRY[c]

[a] Department of Veterinary Pathology, Faculty of Veterinary Science, University of Liverpool, Leahurst, Chester High Road, Neston, S. Wirral, CH64 7TE, United Kingdom
[b] Department of Veterinary Clinical Sciences, Faculty of Veterinary Science, University of Liverpool, Leahurst, Chester High Road, Neston, S. Wirral, CH64 7TE, United Kingdom
[c] Virology, Department of Infectious and Parasitic Diseases, Faculty of Veterinary Medicine, University of Liège, Boulevard de Colonster 20, B43b, 4000 Liège, Belgium

(Received 6 October 2006; accepted 14 December 2006)

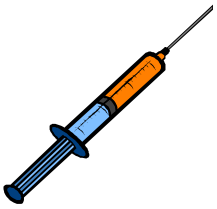
"After primary FeHV-1 infection, cats are largely resistant to disease following further challenge but after six months or more, protection may only be partial."
"Thus in cats with a previously low risk of exposure going into a high risk situation such as a boarding or rescue shelter for example, annual vaccination might still be considered appropriate."
"...the relative efficacy was shown to decrease from 95% shortly after primary vaccination, to 52% after 7.5 years".

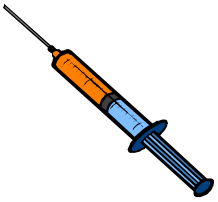
Explaining the updates?

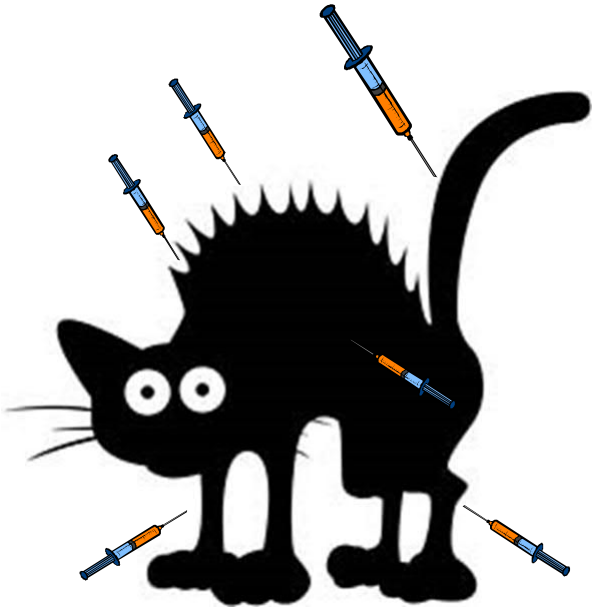
"Low risk" and "high risk" situations and feline lifestyles are better defined

Updated consideration of anatomical sites for injection of vaccines in cats

Much more thoroughly referenced. Quality of evidence considered.







Original Article

Journal of Feline Medicine and Surgery
2014, Vol. 16(4) 275–280

DOI: 10.1177/1098612X13505579
jfms.com
SAGE

Tail vaccination in cats: a pilot study

Cleon G Hendricks[1], Julie K Levy[1], Sylvia J Tucker[1], Shaye M Olmstead[2], P Cynda Crawford[1], Edward J Dubovi[3] and Cathleen A Hanlon[4]

Abstract

Feline injection site sarcomas affect 1–10 cats per every 10,000 vaccinated and are associated with high mortality. Radical resection may be curative, but is often associated with prolonged recovery, disfigurement and loss of function when tumors occur at currently recommended injection sites. The objective of this study was to assess alternatives to currently recommended vaccination sites in terms of preference by oncology practitioners, ease of injection and serological responses. Surgical, radiation and medical oncology practitioners were surveyed regarding their preference for vaccination sites based on the ease of tumor resection. A six-point Likert scale was used to measure each cat's behavioral reaction to vaccination when injected subcutaneously in the distal hind limb or the distal tail. Serum collected before and 1–2 months after vaccination was tested for antibody titers against feline panleukopenia virus (FPV) and rabies virus (RV). The preferred sites for vaccination by 94 oncology practitioners were below the stifle (41%) and the tail (30%). There were no significant differences in the cats' behavioral reaction to vaccination below the stifle (n = 31) and in the distal tail (n = 29). Of the cats seronegative for FPV at the time of vaccination, 100% developed protective antibody titers (≥40) against FPV 1–2 months following vaccination. For cats seronegative for RV, all but one cat (tail vaccine) developed acceptable antibody titers (≥0.5 IU/ml) against RV. Tail vaccination was well tolerated and elicited similar serological responses to vaccination in the distal limbs.

Accepted: 22 August 2013

Surprisingly good tolerance reported in this study

Evidence-based guidelines

- Guidelines are much more thoroughly referenced than previously
- Quality of evidence is considered using a specifically developed scale for publications in veterinary vaccinology

Category 1 evidence: a recommendation supported by peer-reviewed scientific publication of either experimental or field data. Evidence within this category might still be of variable scientific quality despite peer review, as the peer review process does not conform to a universal standard.
Category 2 evidence: a recommendation supported by unpublished commercially sensitive studies submitted as part of a regulatory package for licensed veterinary vaccines. The assumption for this level of evidence is that information appearing on the datasheets of licensed products has been through competent peer review by regulatory authorities.
Category 3 evidence: a recommendation supported by commercial or independent experimental or field data that have not been published in the peer reviewed scientific literature or were not included in a formal regulatory package and subjected to scrutiny by regulators.
Category 4 evidence: a recommendation unsupported by experimental or field data, but assumed from knowledge of the 'first principles' of microbiology and immunology or supported by widely-held expert opinion.

Lastly…

Virology 421 (2011) 67–77

Contents lists available at SciVerse ScienceDirect

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Comparison of differing cytopathic effects in human airway epithelium of parainfluenza virus 5 (W3A), parainfluenza virus type 3, and respiratory syncytial virus

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ABSTRACT

Parainfluenza virus 5 (PIV5) infects a wide range of animals including dogs, pigs, cats, and humans; however, its association with disease in humans remains controversial. In contrast to parainfluenza virus 3 (PIV3) or respiratory syncytial virus (RSV), PIV5 is remarkably non-cytopathic in monolayer cultures of immortalized epithelial cells. To compare the cytopathology produced by these viruses in a relevant human tissue, we infected an in vitro model of human ciliated airway epithelium and measured outcomes of cytopathology. PIV5, PIV3 and, RSV all infected ciliated cells, and PIV5 and PIV3 infection was dependent on sialic acid residues. Only PIV5-infected cells formed syncytia. PIV5 infection resulted in a more rapid loss of infected cells by shedding of infected cells into the lumen. These studies revealed striking differences in cytopathology of PIV5 versus PIV3 or RSV and indicate the extent of cytopathology determined in cell-lines does not predict events in differentiated airway cells.