**Small Animal Disease Surveillance Report: Small animal disease surveillance**

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**Introduction to SAVSNET**

Disease surveillance can be described as the ongoing systematic collection and analysis of data to characterise the current health and disease situation within a given population. In veterinary medicine, most surveillance activity has been focussed on food animals, often driven by a need to report notifiable diseases to the World Organization for Animal Health (OIE).

In the UK, farmed livestock including bird populations have a long tradition of disease surveillance, published as tabulated summaries of diagnoses recorded at Animal and Plant Health Agency (APHA) Veterinary Investigation Centres in England and Wales, Disease Surveillance Centres in Scotland, and the Agri-Food & Biosciences Institute (AFBI) in Northern Ireland. For the equine population, the Animal Health Trust publishes quarterly reports of diagnostic and post mortem results from diagnostic laboratories (AHT 2015).

In contrast, despite their estimated UK population of 11.6 million dogs and 10.1 million cats in 2011 (Murray and others 2015), small companion animal populations largely lack coordinated disease surveillance. This represents a missed opportunity to increase our understanding of their current disease burden, both in terms of the full range of endemic diseases they suffer, but also in terms of new global emerging diseases such as influenza H3N8 in dogs and H1N1 in felids. Lack of disease surveillance also has implications for human health, with approximately 75% of new and emerging diseases being zoonotic (Taylor and others 2001).

Recognising this opportunity, the Small Animal Veterinary Surveillance Network (SAVSNET) was developed to improve companion animal-disease surveillance at local, regional and national levels. SAVSNET Ltd is a not-for-profit charitable company, a partnership between the University of Liverpool and the British Small Animal Veterinary Association. SAVSNET harnesses the growing volumes of patient electronic health records (EHRs) available from small animal practices and complementary data from diagnostic laboratories, to improve animal and human health through enhanced surveillance and research. The scale and research potential of these datasets is a reflection of the high proportion of veterinary practices with computerised records (Asher and others 2011), and the high population coverage often achievable with laboratory data (Dorea and others 2014).

*SAVSNET data from veterinary practices*

SAVNSET collects EHRs in real-time from veterinary practices that volunteer to take part and that are using a compatible version of practice management software (currently Premvet, Robovet and Teleos). Owners attending these SAVSNET practices are given the option to opt out at the time of their consultation, thereby excluding their data. For those that participate, data are collected on a consultation-by-consultation basis, and include animal signalment (species, breed, sex, neutering status, age, vaccination and treatment history, weight, insurance and microchipping status), clinical notes written by the attending veterinary practitioner or nurse and owner’s post-code.

A compulsory, single-question questionnaire is appended at the end of each consultation allowing the attending veterinary surgeon or nurse to categorise the main reason for the animal’s presentation into syndromes (currently gastrointestinal, respiratory, pruritus, tumour and renal) or other routine veterinary interventions (i.e. trauma, “other sick”, vaccination, “other healthy” or post-operative check-up). This allows SAVSNET to collect a usable syndrome badge in real-time for every consultation it receives. A short, syndrome-specific questionnaire is randomly assigned to approximately 5% of consultations to collect additional information on each syndrome (e.g. duration, other clinical signs, diagnostic plans).

*SAVSNET data from* diagnostic labs

Complementary to these practice-derived data, SAVSNET also collates diagnostic data from those laboratories that consent to participate. Data are collected between near real-time and monthly and include for each test performed the species tested, sampling and diagnosis dates, sample type, diagnostic method and result, postcode area (first 1 or 2 letters of the postcode) of the submitting veterinarian, and in some cases, the breed, gender and neuter status of the animal tested.

*Use of SAVSNET data*

Veterinary surgeons in practice and diagnostic laboratories are encouraged to participate in SAVSNET by offering them personalised benchmarking of their key performance indicators and disease data through a private web-based secure portal; such portals can become a valuable aid in clinical audit.

In order to get the best possible value from these data, researchers can apply to access data for their own research through a Data Access and Publication Panel. Results of analyses are published in peer reviewed journals and also through www.savsnet.co.uk.

*Format of the reports*

The SAVSNET UK Small Animal Disease Surveillance (SADS) report will be published quarterly and will comprise four sections. The first two sections will summarise some practice- and laboratory-based surveillance data. This inaugural report is focused on gastrointestinal (GI) disease in practices and on laboratory-confirmed diagnosis of canine parvovirus type 2 (CPV-2) infection. A third section will describe other recent and relevant infectious disease events occurring in a global context, which have the potential to impact on small animal populations in the UK. The final section will provide an update on one of the diseases analysed in the surveillance report section – in this first report, an update on CPV-2 infection.

# **Syndromic surveillance of gastrointestinal disease**

GI disease may occur in all pet animals, ranging from acute life-threatening, to mild acute self-limiting, to chronic. Causes are varied and include both infectious and non-infectious aetiologies. GI disease affects animal welfare, can be expensive to manage and may be transmissible to other pets or owners. Here we describe animals presented primarily with GI disease to 133 veterinary practices (306 premises) between January 2014 and September 2015.

In total, EHRs for 654,431 consultations were collected (including repeat consultations for the same animal), of which 69.9% were from dogs, 25.6% cats, 1.7% rabbits, 1.5% other species and 1.3% where the species was not noted. Presentation for GI disease comprised 4.5%, 3.4% and 3.6% of canine, feline and rabbit consultations, respectively.

The clinical signs at presentation for 5,492 dogs and cats based on a single questionnaire per patient are shown in Table 1. A high proportion of dogs (53.7%) and cats (39.0%) were presented after only a short history of illness over the previous two days. Diagnostic tests were planned in 18.2% of dogs and 23.6% of cats with GI disease, with haematological/biochemical analyses being most common.

The spatial distribution of the relative risk for GI disease was evaluated in dogs and cats in England and Wales for each season of the year (Figure 1). Estimates for Scotland and Northern Ireland are not included in this report because SAVSNET geographical coverage in these areas is currently limited. Animals were considered as “cases” if, during the season assessed, they were presented for GI disease at one or more consultations. A kernel smoothing method was used to smooth the spatial variation of the relative risk for GI disease throughout England and Wales. The relative risk of dogs being presented with GI disease was estimated as the ratio of kernel-smoothed intensities (i.e. mean number of events estimated per unit area) of dogs presented with GI disease (cases) compared with all dogs presented to SAVSNET veterinary practices for a cause other than GI disease (controls); the same approach was conducted for cats. Estimations were made using a grid cell of 5 km and a bandwidth of 50 km.

In dogs, many zones of increased relative risk were identified. Most areas appeared transient, although areas around the north and west of Greater London seemed to have a higher relative risk throughout the year. In contrast, the picture in cats appeared more stable, with lower numbers of zones at high relative risk for GI disease compared with dogs, with autumn having no zones of high relative risk. Together these data reaffirm the different pattern of presentation for GI disease between cats and dogs, and suggest that the relative risk for GI disease varies spatially and temporally. It should be noted these zones may not equate to outbreaks; SAVSNET is currently developing models to allow outbreaks to be identified.

**Laboratory-based surveillance of canine parvovirus type 2 in the UK**

Canine parvovirus type 2 (CPV-2) is a major cause of severe haemorrhagic diarrhoea and the infection is endemic in dogs. SAVSNET data gathered from participating laboratories were used to identify temporal and geographical trends in the proportion of samples submitted for CPV-2 testing that were positive (Figure 2). Proportions are currently used to protect the case loads of participating laboratories.

Samples were submitted from 117 of the 121 postcode areas in use in the UK. The mean percentage of samples testing positive for CPV-2 was 13.2% between October 2013 and September 2014 and 10.7% between October 2014 and September 2015. Interestingly, January had the highest percentage of positive samples in both 2014 (19.5%) and 2015 (22.3%); we will look to see if this trend persists over future years.

From postcode areas that submitted 25 or more samples in each year, the highest percentage of samples testing positive were from Worcester (45% in 2013-14) and Newport (32% in 2014-15). In total, 33 postcode areas in 2013-14 and 41 in 2014-15 submitted only negative samples; some areas only had low sample numbers. However, Redhill in 2013-14 and Watford in 2014-15 submitted the highest numbers of these negative samples (37 and 74, respectively). These apparent temporal and spatial variations suggest a different epidemiology for CPV-2 infection across the country, and will need further study. Results presented here should currently be interpreted with caution as some postcode areas of the UK had small numbers of submissions during this sample period.

# **Global perspective**

This section of the small animal surveillance report briefly reviews some topical global trends in companion animal infection. Many of these reports were first notified globally on ProMED (www.promedmail.org).

*Canine distemper*

In January 2015, four adult pandas in a zoo in China were reported as contracting canine distemper virus (CDV), with one animal dying (ProMed 2015a). CDV may infect the gastrointestinal and respiratory tracts as well as the spinal cord and brain of a growing range of host species. Affected animals present with variable signs including pyrexia, conjunctivitis, coughing, vomiting, diarrhoea and lethargy. CDV is highly contagious, with a high fatality rate, and is commonly spread through direct or indirect contact with infected body. In the UK, disease in dogs is well controlled by vaccination, but the virus is still regularly diagnosed, confirming the need for ongoing vaccination (www.savsnet.co.uk/realtimedata).

*Neosporosis*

Rising levels of *Neospora caninum* infection in the UK and Ireland have led to it being added to Cattle Health Certification Standards (CHeCS) (ProMed 2015b). *Neospora* is carried by dogs, but it is an important cause of abortion in cattle if they ingest oocysts shed by infected dogs. The infection is usually asymptomatic in dogs, but may cause neurological signs in some animals. Dog owners have an important role to play in control of the infection; by picking up their dog's faeces to prevent cattle exposure to any oocysts it may contain. Similarly, dogs should be kept away from calving animals to prevent them becoming infected, especially by aborting animals.

*Canine influenza*

First described in dogs in the USA in April this year, influenza virus H3N2 is believed to have spread to dogs from birds (AHDC 2015, ProMed 2015c). It is thought to have been introduced to the USA from Asia, where it is circulating more widely, possibly through the importation of rescued dogs from Asian meat markets. This is the second influenza virus to infect dogs, joining H3N8, which itself was first introduced into the dog population from horses around 2004 in the USA (Crawford and others 2005). Neither virus is believed to be currently circulating in the UK. This confirms the importance of dog migration as a potential route to spread viruses globally - and as dogs become more mobile, these risks will only increase.

# **Update on canine parvovirus**

CPV-2 is a major cause of severe haemorrhagic diarrhoea in dogs. Since its emergence in 1978, there have been further mutations in the viral capsid leading to the development of types CPV-2a, 2b and more recently 2c. The original CPV-2 no longer appears to be circulating in the dog population, although it is present in some modified live vaccines. CPV-2 is thought to have emerged from feline panleukopenia virus, possibly via some wildlife intermediate host like the fox, and possibly on more than one occasion (Allison and others 2013). Within the UK, as in much of the world, types 2a and 2b seem to predominate (Clegg and others 2011); in some countries (e.g. Italy) subtype 2c is the most common (Decaro and others 2007). The biggest significance of the recent evolution of CPV is its ability to infect the cat; CPV-2 could not, whereas CPV-2a, -2b and -2c can, raising important questions for disease control, and also the diagnosis of parvovirus in cats (Clegg and others 2012).

There is considerable variation in the response of dogs to CPV-2 infection, depending on factors such as their age, breed, stress levels and immune status. The most severe disease tends to occur in puppies, during the ‘immunity gap’ where they lose their maternally-derived antibodies and before vaccination takes effect. The virus targets actively dividing cells – in the dog, this is largely the intestinal crypt epithelial cells, and cells in the bone marrow and lymphoid tissues. Clinical signs therefore generally include some or all of the following: severe haemorrhagic diarrhoea, vomiting, lethargy, and rapid onset of dehydration; sepsis and disseminated intravascular coagulation may also occur. Leukopenia may be present, especially in severe cases. The mortality rate in untreated animals is high. In puppies born to bitches without immunity, myocarditis can occur, but nowadays this is very rare.

Rapid diagnosis can be achieved with faecal ELISA antigen detection kits, which have reasonable sensitivity and specificity (Decaro and others 2013). Most laboratories use either conventional or real-time PCR, both of which are more sensitive than other methods (Desario and others 2005). The period of virus shedding in clinical cases is quite short (5-7 days of clinical signs), and false negatives can therefore occur if testing is delayed. Most vaccinations are modified live, and animals recently vaccinated with these vaccines may test positive on faecal analysis, even though shedding levels are generally lower than in clinical disease.

Serum antibody tests (e.g. haemagglutination inhibition tests) can be used to confirm response to vaccination and are advocated by some as an alternative to routine core revaccination of adult dogs (Day and others 2015).

Control of CPV-2 infection relies on a combination of preventative vaccination and case management procedures. Modified live parenteral CPV-2 vaccines are available in the UK, based on CPV type 2 or 2b. All licensed vaccines appear highly effective, and are generally considered to induce cross protection against all three types (2a, 2b and 2c). Data sheets vary somewhat in their recommendations for timing of the initial primary course. The last puppy dose is generally given at 10-12 weeks of age, but international vaccine guidelines recommend later finishes (16 weeks of age or older) (Day and others 2015). The first annual booster is important, but thereafter recommendations for revaccination are generally 3- 4 years.

Treatment of affected animals is focussed on active management of shock. One commercially produced anti-viral interferon is licensed for the reduction of mortality and clinical signs of parvovirosis (enteric form) in dogs from one month of age. Known or suspect cases should be isolated and barrier nursed. Because the virus is shed in high titre in faeces, and is very resistant, care should be taken to reduce environmental contamination (e.g. on personnel, the dog’s coat, and fomites such as feed bowls and grooming kits). The virus is resistant to many commonly used disinfectants, such as quaternary ammonium compounds, but can be inactivated by 1 in 32 dilution of hypochlorite (bleach), and also by some commercially available disinfectants.

**Conclusion**

This is a first UK SADS report. In the future, we will expand to other syndromes and diseases. As data are collected for longer, the estimates of changes in disease burden will become more refined, allowing more targeted local and perhaps national interventions. Anonymised data can be accessed for research by contacting the authors. SAVSNET welcomes your feedback.

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**Tables**

Table 1. Percentage of clinical signs in 4,286 dogs and 1,206 cats presenting with gastrointestinal disease to veterinary practices in the UK. The same animal could present with more than one sign per consultation.

|  |  |  |
| --- | --- | --- |
| Clinical signs | Number (%) of dogs | Number (%) of cats |
| Diarrhoea without blood  Diarrhoea with blood  Vomiting without blood  Vomiting with blood  Melaena  Weight loss/failure to gain weight  Poor appetite  Other signs | 1,774 (41.4)  1,026 (23.9)  1,537 (35.9)  131 (3.1)  53 (1.2)  110 (2.6)  577 (13.5)  510 (11.9) | 416 (34.5)  109 (9.0)  447 (37.1)  40 (3.3)  7 (0.6)  146 (12.1)  197 (16.3)  187 (15.5) |

**Figures**



Figure 1. Kernel intensity ratio surface of England and Wales showing the relative risk of dogs and cats being presented with gastrointestinal disease by season. The colours for relative risk have been categorised using the four cut-offs that divide the results obtained from dogs during spring into five equal-size groups (quintiles) each containing 20% of all results.



Figure 2. Temporal and geographical trends in canine parvovirus type 2 (CPV-2) diagnosis. A) and C): geographical distribution of the percentage of samples testing positive for CPV-2 by postcode area. An asterisk (\*) identifies the area with the highest percentage of positive CPV-2 samples in each year from those postcode areas that submitted at least 25 tests. A triangle (▲) identifies the CPV-2 negative postcode area which submitted the highest number of samples in each time period. B) and D): percentage of samples which test positive for CPV-2 by month (the dotted line indicates the mean percentage of samples testing positive in each year).