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Non-neoplastic anal sac disorders in UK dogs: Epidemiology and management aspects of a research-neglected syndrome

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Abstract

Background: Non-neoplastic anal sac disorders (ASD) are frequent presentations for dogs in primary-care practice but evidence-based information on disease occurrence and risk is sparse. This study estimates prevalence, breed associations and other risk factors as well as reporting on clinical management.

Methods: A cohort study of dogs attending VetCompass practices between 1 January 2013 and 31 December 2013. Risk factor analysis used multivariable logistic regression methods.

Results: Of 104,212 dogs attending 110 UK practices, the 1-year period prevalence of ASD was 4.40% (95% CI: 4.22–4.57). Compared to crossbreds, six breeds showed increased odds of ASD (Cavalier King Charles spaniel, King Charles spaniel, Cockapoo, Shih-tzu, Bichon Frise and Cocker spaniel), and six breeds showed reduced odds (Labrador Retriever, Border collie, Staffordshire Bull Terrier, Lurcher, German Shepherd Dog and Boxer). Brachycephalic types had 2.6 times the odds for ASD compared to dolichocephalic types. Medication prescribed for ASD included antimicrobials (n = 480, 20.24%) and analgesics (n = 284, 11.97%). Anal sacculectomy was performed in under 1% of cases.

Conclusions: High prevalence, strong breed predispositions and evidence of severity suggested from the antimicrobial and analgesic therapies combined with current substantial knowledge gaps identify ASD as a key research-neglected syndrome in dogs.

KEYWORDS
anal gland, anal sac impaction, anal sac infection, anal sacculitis, brachycephalic, first opinion, general practice, spaniel, VetCompass

INTRODUCTION

Consultations involving examination or expression/emptying of anal sacs in dogs are routine events for most small animal veterinary practitioners: anal sac impaction was the third most common disorder recorded in an analysis of first-opinion electronic patient records (EPRs) in England.[1] While studies on factors affecting owner-dog relationship generally exclude aspects related to disease or other physical attributes,[2] the plethora of online resources for dog owners relating to dog malodour and anal sac problems[3] would indicate that behaviours and odour associated with ASD are of significant concern to owners. It is therefore surprising that very little evidence-based information is published on the epidemiology or clinical management of non-neoplastic anal sac disorders (ASD). Despite this lack of research focus, ASD can be associated with significant disease severity, as in the case of abscessation and subsequent cellulitis, and also impairs quality of life through unpleasant perineal sensations manifesting as perianal self-trauma (scooting, rubbing, licking, biting), discomfort when sitting, tenesmus or dyschezia.[4,5] Variations of ASD that are distinguished in the literature include impaction, inflammation (sacculitis), infection and abscessation. These are typically considered as a continuum of a shared underlying process starting with a disturbance in the normal emptying process of the anal sacs.[6,7]

Abbreviations: ASD, anal sac disorder; CI, confidence interval; EPR, electronic patient record; IQR, interquartile range; KC, The Kennel Club; NSAID, nonsteroidal anti-inflammatory drug; OR, odds ratio

Criteria for diagnosis of, and differentiation between, individual types of ASD are poorly defined. Resolution of scooting after emptying of anal sacs seems an obvious marker for ASD and can help to discriminate ASD from perianal self-trauma due to other causes.[8] Perianal lesions are not pathognomonic for ASD.[9] Haemorrhagic anal sac content may be a distinguishing marker for ASD, while colour, consistency and smell were not definitely associated with ASD.[10,11] Laboratory tests appear to be of limited diagnostic value. Results from cytological examination and bacterial culture have shown that bacteria, yeasts and inflammatory cells can be found in content from clinically normal anal sacs and could not reliably be used to distinguish normal content from pathology.[8,10–12]

Despite these clinical and diagnostic uncertainties, ASDs are consistently reported as one of the most common disorders affecting dogs under veterinary care. Studies published over several decades, and using differing study designs, report period prevalence estimates for unspecified anal sac disease, or more specifically of anal sac impaction, ranging from 2% to 13%.[1,13–16] There is also some limited evidence on breed as a risk factor for ASD. Cavalier King Charles Spaniels were reported with prevalence estimates up to 19% for unspecified anal sac disease.[5,17]

Plausible risk factors along with therapeutic and preventative recommendations are frequently described but rarely substantiated in the veterinary literature. Episodes of diarrhoea, diet type and changes and obesity were proposed, based on limited evidence, as potential triggers for ASD over 40 years ago but little has been added to our understanding of ASD since.[6,18] Investigations on the effectiveness of fibre-rich diets are challenging to carry out and are frequently hampered by confounding factors[19,20] such that the true value of diet for prevention or therapy of ASD remains uncertain.

Manual expression alone as a relatively non-invasive procedure may be effective to relieve immediate anal sac impaction[21] but recurrence remains a problem. Repeated expression was necessary every 2 months in one study of 20 dogs with ASD, with signs reappearing within a median of 3 weeks.[8] While sacculectomy was previously advocated early on during the treatment of anal sacculitis,[22] this is now generally recommended only for recurrence of impaction, chronic sacculitis and abscessation, sinus formation and failure of medical therapy.[7] Recommendations on medical management of ASD, mostly from textbooks, include systemic or topical anti-inflammatory and/or antimicrobial drugs typically combined with manual expression, but recommendations on dosing, duration of treatment and efficacy remain largely anecdotal. The results from a case series[21] and a small trial comparing two systemic antimicrobials in combination with manual sac expression[23] suggest expected response rates between 60% and 85%. However, given the current urgent need for responsible antimicrobial stewardship, the use of systemic antimicrobial therapy in the absence of defined diagnostic criteria for a bacterial aetiology in a dog with ASD may not be sensible. Recent guidelines recommend topical treatment alone for anal sac inflammation or engorgement, and suggest restricting systemic antimicrobials for use only with evidence of abscessation and adherence to published tier systems.[24,25]

The scarcity of good quality information and evidence relating to non-neoplastic ASD is particularly surprising in view of the high prevalence, the increasing requirement for responsible antimicrobial prescribing and the multifaceted impact of ASD on dog and owner well-being.[25] In consequence, this study explores EPRs from first opinion practices in the UK to evaluate the prevalence, risk factors and interventions for non-neoplastic ASD in a large population of dogs. The study gave priority to identification of breed associations for ASD. These results can define knowledge gaps and guide future research into this research-neglected syndrome.[26]

MATERIALS AND METHODS

The VetCompass Programme collates anonymised EPR data from primary-care veterinary practices in the UK for epidemiological research.[1,27] This cohort study used a cross-sectional analysis and included dogs under 'veterinary care' within VetCompass during 2013 which were defined as any dogs with ≥1 EPR recorded from 1st January 2013 to 31st December 2013, or ≥1 EPR both before and after 2013. Sample size calculation estimated that 1010 dogs of a specific type (e.g. spaniel, brachycephalic or poodle) and 10,093 dogs not of that type were required to detect an odds ratio of 1.5 times or greater for ASD, assuming a 4.0% prevalence of ASD in the non-type dogs (10:1 ratio of non-type: type, two-sided 95% confidence interval [CI], 80% power).[28] Ethical approval was granted by the RVC Ethics and Welfare Committee (reference number 2015/T131).

An ASD case required a recorded diagnosis, clinical description or evidence of a clinical intervention for at least one ASD present during 2013. Exclusion criteria were recordings of anal sac neoplasia or unclassified anal sac masses, and where anal sac examination had an accompanying statement that there was no anal sac abnormality. Case-finding involved initial EPR screening of all dogs to identify candidate ASD cases by searching the clinical free-text field using search terms that were developed and refined iteratively in combination with manual review of the clinical records in line with the methods previously applied in several studies.[29–32] These terms included *anal g*, *anal s*, *saccul*, *eag*, *ag*, * ags*, * ag's* and *scoot*. The clinical notes of a requisite subset of candidate cases, determined by the samples size estimations, were reviewed for case inclusion and clinical data extraction. ASD terms were categorised as 'impaction', 'sacculitis' (inflammation or infection) and 'abscess' or otherwise as 'unspecified' with a hierarchy of clinical severity assumed from

impaction (least severe) to sacculitis to abscess (most severe). Classification of each case along the hierarchy of severity relied on the depth of information recorded in the clinical notes. Each ASD case was assigned the category with the highest severity as evidenced in the clinical records for 2013. Further data extraction included the status for manual anal sac evacuation, antibiosis and analgesic therapy usage, surgical intervention, referral for advanced clinical management and veterinary recommendation for weight loss and dietary change. Decision-making on whether the case definition had been met and on the further data extracted on each case was taken by two authors (Dan G. O'Neill and Jennifer A. Phillips). Dan G. O'Neill has over two decades of clinical experience. Jennifer A. Phillips was a final year veterinary undergraduate at the time of the study. Risk factor analysis grouped all dogs with confirmed ASD as *ASD cases* and all dogs originally screened as non-candidate cases as *non-cases*.

Breed was defined to include both currently recognised breeds[33] and also hybrid types with contrived names indicating purposive hybrid status (e.g. Labradoodle, Cockapoo).[34–36] A general crossbred term included dogs that were otherwise described with mixed breed parentage (e.g. xbreed, GSD-X, labrador-cocker cross). A *breed* variable included individual breeds with 10 or more ASD cases, along with general groupings for all crossbreds and for all remaining dogs. Recognised breeds were grouped as 'purebred' while designer types and the crossbred dogs were grouped as 'crossbred'. Purebreds were further categorized by UK Kennel Club (KC) breed-recognition (recognised/not recognised) and UK KC breed group (gundog, hound, pastoral, terrier, toy, utility, working).[35] Purebreds were also separately categorised based on skull conformation (dolichocephalic, mesocephalic and brachycephalic) (Supplementary A), spaniel status, poodle status and dachshund status (Supplementary B). Neuter status (final available EPR), age (years at 31 December 2013) and adult bodyweight (mean of all bodyweight [kg] values after 18 months old) were recorded. Mean adult bodyweight was derived for all combinations of breeds and sex with information available for at least 100 dogs in the overall VetCompass population. Each study dog was categorised (lower or higher) relative to their relevant breed/sex mean bodyweight to allow the effect of adult bodyweight to be assessed *within* each breed/sex combination. Cells with missing data were included in the analysis as 'unrecorded'.

Statistical analyses were conducted using Stata Version 13 (Stata Corporation). The 1-year period prevalence with 95% CI described the probability of ASD during 2013. Because the sampling design involved verification of a subset of candidate cases, the predicted total case count was calculated using the Stata *survey* function as previously described.[37] The CI estimates were derived from standard errors, based on approximation to the binomial distribution.[38]

Univariable associations between risk factors and diagnosis of ASD were evaluated using binary logistic regression with variables liberally associated ($p < 0.2$) taken forward for multivariable association. Because breed was a factor of primary interest for the study, purebred status, skull conformation, spaniel-type, poodle-type, dachshund-type, Kennel Club Breed Group (variables that are highly collinear with breed) and *adult bodyweight* (a defining characteristic of individual breeds) were excluded from the initial breed-based multivariable modelling. Instead, these variables individually replaced the *breed* variable in the final breed-based model to evaluate their effects after taking account of the remaining variables. Multivariable model development used manual backwards stepwise elimination. Pair-wise interaction effects were evaluated for the final model variables, and confounding effects from dropped variables were assessed by individual re-introduction to the final model. Clinic attended was evaluated as a random effect.[39] The area under the ROC curve and the Hosmer-Lemeshow test were used to evaluate the quality of the model fit and discrimination (non-random effect model).[39,40] Statistical significance was set at $p < 0.05$.

RESULTS

The study population comprised 104,212 dogs attending 110 UK primary-care practices during 2013. Of 6430 candidate cases identified, 3330 (51.79%) were individually checked to confirm 2372 ASD cases. After accounting for the subsampling protocol, the estimated 1-year period prevalence for ASDs in dogs overall was 4.40% (95% CI: 4.22–4.57). Breeds with the highest ASD prevalence were Cavalier King Charles Spaniel (14.83%, 95% CI 12.88–16.77), King Charles Spaniel (13.78%, 95% CI 9.72–17.83), Cockapoo (8.47%, 95% CI 5.21–11.73), Bichon Frise (7.29%, 95% CI 5.40–9.19) and Shih-tzu (6.93%, 95% CI 5.42–8.44) (Figure 1). The forms of ASD recorded during 2013 were unspecified disease (n = 7, 0.30%), impaction (1,864, 78.58%), sacculitis (289, 12.18%) and abscess (212, 8.94%).

The descriptive results for breed-related and general demographic risk factors associated with ASD are detailed in Tables 1 and 2. Data completeness varied between the variables assessed: breed 99.91%, age 99.79%, sex 99.98%, neuter 58.02%, bodyweight (any age) 91.65% and insurance 58.92%.

Manual evacuation of the anal sacs was recorded at least once during 2013 in 2256 (95.11%) cases. Antibiosis was used within anal sac clinical management for 480 (20.24%) cases: local antibiosis alone (19, 0.80%), systemic antibiosis alone (428, 18.04%), local and systemic antibiosis combined (33, 1.39%). Analgesic therapy contributed to anal sac clinical management for 284 (11.97%) cases: nonsteroidal anti-inflammatory drugs (NSAIDS) (274, 11.55%), NSAIDS and opioids (7, 0.30%) and opioids only (3, 0.13%). Anal sac irrigation was performed in 78 (3.29%) cases, and anal

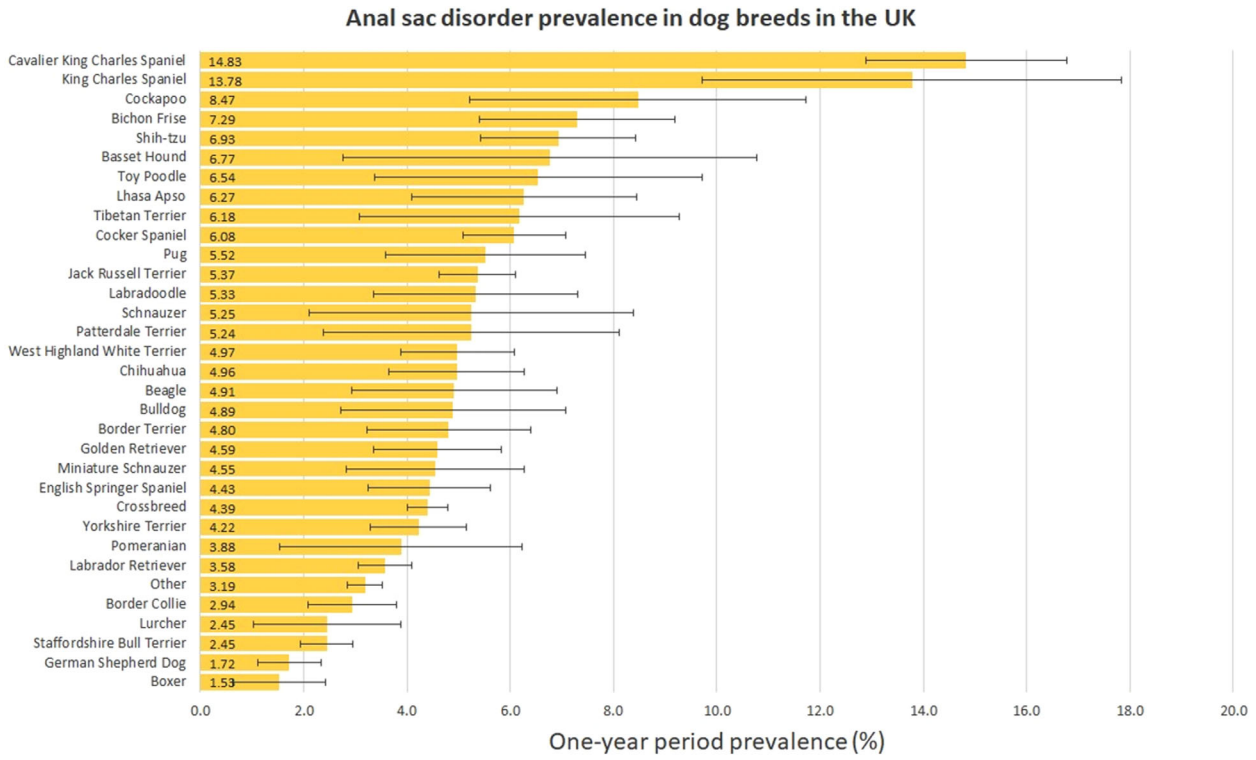

FIGURE 1 One-year (2013) period prevalence percentage (inset into bars) with 95% confidence intervals for ASD diagnosis in commonly affected dog breed-types attending primary-care veterinary practices in the VetCompass Programme in the UK. The error bars show the 95% confidence interval

sacculectomy was performed in 22 (0.93%) cases. There was no evidence that any ASD cases were referred for advanced clinical management during 2013. Dietary change was recommended in 194 (8.18%) cases, and weight loss was recommended in 27 (1.14%) cases.

Univariable logistic regression identified 11 variables liberally associated with ASD: breed, skull conformation, spaniel-type, poodle-type, dachshund-type, Kennel Club Breed Group, adult (>18 months) bodyweight (kg), bodyweight relative to breed/sex mean, age (years), neuter and insurance (Tables 1 and 2). The final breed-based multivariable model retained three risk factors: breed, age (years) and insurance. No biologically significant interactions were identified in the final model. The final unclustered model showed acceptable model-fit (Hosmer-Lemeshow test statistic: $p = 0.371$) and discrimination (area under the ROC curve: 0.647). The random effects model with clinic entered as a random effect was a better model of the data that the non-random effects model ($p < 0.001$) and these results are reported. The intraclass correlation coefficient indicated that 4.18% of the unaccounted-for variation in the data was due to unmeasured factors operating at the veterinary clinic level. After accounting for the effects of the other variables evaluated, six breeds (Cavalier King Charles spaniel, King Charles spaniel, Cockapoo, Shih-tzu, Bichon Frise and Cocker spaniel) showed increased odds of ASD compared with cross-bred dogs, and six breeds (Labrador Retriever, Border collie, Staffordshire Bull Terrier, Lurcher, German Shepherd Dog and Boxer) showed reduced odds. All age groups over 3 years had higher odds of ASD compared with dogs aged under 3 years (Table 3).

When variables that correlated highly with breed replaced the breed variable in the final multivariable logistic regression model, brachycephalic types and spaniel-types were significantly associated with a diagnosis of ASD with over twice the odds compared to dolichocephalic types and non-spaniel types, respectively (Table 4).

DISCUSSION

This retrospective, large-scale EPR-based study estimates the overall frequency (as the 1-year prevalence) for non-neoplastic ASDs as 4.40% in the overall population of dogs accessing primary veterinary care in the UK. Previously reported ASD frequencies for the UK dog population were derived from three differing study designs: 15% diagnosed with unspecified ASD in Halnan's study of 2000 dogs using a wide case definition,[13] 2.1% of consultations that recorded anal sac impaction[41] and 2.1% of dogs diagnosed with anal sac impaction from a survey of 2322 dogs presented for first opinion veterinary care.[15] Two previously published reports that drew on the same primary-care EPR database as the current study but that used differing methods provided prevalence estimates for a range of disorders, including unspecified ASD of 9% and 4.5%[1,5] and anal sac impaction specifically at 7%.[1] The true frequency of ASDs is likely to be even higher that reported in the current study because owners and groomers may undertake direct interventions without

TABLE 1 Descriptive and univariable logistic regression results for breed-related risk factors associated with ASD in dogs under primary veterinary care during 2013 in the VetCompass Programme in the UK. Column percentages shown in brackets

Variable	Category	Case number (%)	Control number (%)	Odds ratio	95% CI*	Category *p*-value	Variable *p*-value
Purebred status	Crossbred	546 (23.04)	21,916 (22.43)	Reference level			0.486
	Purebred	1,824 (76.96)	75,782 (77.57)	0.97	0.88–1.06	0.485	
Breed-type	Crossbreed	450 (18.97)	18562 (18.98)	Reference level			<0.001
	Cavalier King Charles Spaniel	177 (7.46)	1927 (1.97)	3.79	3.16–4.54	<0.001	
	King Charles Spaniel	36 (1.52)	427 (0.44)	3.48	2.44–4.95	<0.001	
	Cockapoo	23 (0.97)	471 (0.48)	2.01	1.31–3.09	0.001	
	Bichon Frise	51 (2.15)	1229 (1.26)	1.71	1.27–2.30	<0.001	
	Shih-tzu	73 (3.08)	1859 (1.9)	1.62	1.26–2.08	0.000	
	Basset Hound	10 (0.42)	261 (0.27)	1.58	0.83–2.99	0.160	
	Toy Poodle	15 (0.63)	406 (0.42)	1.52	0.90–2.57	0.115	
	Lhasa Apso	29 (1.22)	822 (0.84)	1.46	0.99–2.13	0.054	
	Tibetan Terrier	14 (0.59)	403 (0.41)	1.43	0.83–2.46	0.192	
	Cocker Spaniel	131 (5.52)	3838 (3.93)	1.41	1.16–1.72	0.001	
	Pug	29 (1.22)	940 (0.96)	1.27	0.87–1.86	0.215	
	Jack Russell Terrier	187 (7.88)	6247 (6.39)	1.23	1.04–1.47	0.017	
	Labradoodle	26 (1.1)	875 (0.89)	1.23	0.82–1.83	0.320	
	Schnauzer	10 (0.42)	342 (0.35)	1.21	0.64–2.28	0.563	
	Patterdale Terrier	12 (0.51)	411 (0.42)	1.20	0.67–2.15	0.531	
	Chihuahua	51 (2.15)	1852 (1.89)	1.14	0.85–1.52	0.395	
	West Highland White Terrier	73 (3.08)	2643 (2.7)	1.14	0.89–1.46	0.308	
	Beagle	22 (0.93)	807 (0.83)	1.12	0.73–1.74	0.596	
	Bulldog	18 (0.76)	663 (0.68)	1.12	0.69–1.81	0.642	
	Border Terrier	33 (1.39)	1239 (1.27)	1.10	0.77–1.57	0.607	
	Golden Retriever	49 (2.07)	1932 (1.98)	1.05	0.78–1.41	0.767	
	Miniature Schnauzer	25 (1.05)	995 (1.02)	1.04	0.69–1.56	0.864	
	English Springer Spaniel	51 (2.15)	2084 (2.13)	1.01	0.75–1.35	0.950	
	Yorkshire Terrier	74 (3.12)	3182 (3.25)	0.96	0.75–1.23	0.743	
	Pomeranian	10 (0.42)	469 (0.48)	0.88	0.47–1.66	0.691	
	Labrador Retriever	177 (7.46)	9043 (9.25)	0.81	0.68–0.96	0.017	
	Other purebred	335 (14.12)	19284 (19.72)	0.72	0.62–0.83	<0.001	
	Border Collie	43 (1.81)	2694 (2.76)	0.66	0.48–0.90	0.009	
	Lurcher	11 (0.46)	829 (0.85)	0.55	0.30–1.00	0.050	
	Staffordshire Bull Terrier	87 (3.67)	6562 (6.71)	0.55	0.43–0.69	<0.001	
	German Shepherd Dog	29 (1.22)	3138 (3.21)	0.38	0.26–0.56	<0.001	
	Boxer	11 (0.46)	1346 (1.38)	0.34	0.18–0.61	<0.001	
Skull conformation	Dolichocephalic	176 (9.25)	10,909 (13.88)	Reference level			<0.001
	Mesocephalic	1,255 (65.95)	55,571 (70.72)	1.40	1.19–1.64	<0.001	
	Brachycephalic	472 (24.80)	12,097 (15.40)	2.42	2.03–2.88	<0.001	
Dachshund-type	Not Dachshund-type	1,871 (97.45)	77,623 (98.09)	Reference level			0.054
	Dachshund-type	49 (2.55)	1,513 (1.91)	1.34	1.01–1.79	0.045	
Spaniel-type	Not Spaniel-type	1,455 (75.78)	68,959 (87.14)	Reference level			<0.001
	Spaniel-type	465 (24.22)	10,177 (12.86)	2.17	1.95–2.41	<0.001	
Poodle-type	Not Poodle-type	1,827 (95.16)	76,268 (96.38)	Reference level			0.007
	Poodle-type	93 (4.84)	2,868 (3.62)	1.35	1.10–1.67	0.005	
Kennel Club Breed Group	Not KC-Recognised	581 (24.51)	23332 (23.88)	Reference level			
	Gundog	454 (19.16)	19378 (19.83)	0.94	0.83–1.07	0.336	<0.001
	Hound	89 (3.76)	4295 (4.4)	0.83	0.66–1.04	0.110	

(Continues)

TABLE 1 (Continued)

Variable	Category	Case number (%)	Control number (%)	Odds ratio	95% CI*	Category *p*-value	Variable *p*-value
	Pastoral	92 (3.88)	6990 (7.15)	0.53	0.42–0.66	<0.001	
	Terrier	422 (17.81)	19128 (19.58)	0.89	0.78–1.01	0.061	
	Toy	464 (19.58)	11489 (11.76)	1.62	1.43–1.84	<0.001	
	Utility	228 (9.62)	8182 (8.37)	1.12	0.96–1.31	0.156	
	Working	40 (1.69)	4904 (5.02)	0.33	0.24–0.45	<0.001	

*CI: confidence interval.

TABLE 2 Descriptive and univariable logistic regression results for general demographic risk factors associated with ASD in dogs under primary veterinary care during 2013 in the VetCompass Programme in the UK. Column percentages shown in brackets

Variable	Category	Case number (%)	Control number (%)	Odds ratio	95% CI*	Category *p*-value	Variable *p*-value
Adult (>18 months) bodyweight (kg)	<10.0	755 (31.83)	19712 (20.16)	2.35	1.90–2.91	<0.001	<0.001
	10.0 - < <20.0	723 (30.48)	19337 (19.78)	2.29	1.85–2.84	<0.001	
	20.0 - << 30.0	323 (13.62)	15888 (16.25)	1.25	0.99–1.57	0.059	
	30.0 – < 40.0	242 (10.20)	11804 (12.07)	1.26	0.99–1.60	0.059	
	≥40.0	96 (4.05)	5891 (6.02)	Reference level	~	~	
	Unrecorded	233 (9.82)	25150 (25.72)	0.57	0.45–0.72	<0.001	
Bodyweight relative to breed/sex mean	Lower	1103 (46.5)	41081 (42.01)	Reference level	~	~	<0.001
	Higher	1124 (47.39)	38013 (38.88)	1.1	1.01–1.20	0.025	
	Unrecorded	145 (6.11)	18688 (19.11)	0.29	0.24–0.34	<0.001	
Age (years)	<3.0	467 (19.7)	28202 (28.9)	Reference level	~	~	<0.001
	3.0 - < 6.0	708 (29.86)	25401 (26.03)	1.68	1.50–1.89	<0.001	
	6.0 - < 9.0	597 (25.18)	19760 (20.25)	1.82	1.61–2.06	<0.001	
	9.0 - < 12.0	371 (15.65)	13499 (13.83)	1.66	1.45–1.91	<0.001	
	≥12.0	228 (9.62)	10710 (10.98)	1.29	1.10–1.51	0.002	
Sex	Female	1151 (48.57)	46,473 (47.66)	Reference level	~	~	0.386
	Male	1219 (51.43)	51,027 (52.34)	0.96	0.89–1.05	0.386	
Neuter	Entire	346 (14.59)	13,030 (13.33)	Reference level	~	~	<0.001
	Neutered	1386 (58.43)	42,743 (43.71)	1.22	1.08–1.38	0.001	
	Unrecorded	640 (26.98)	42,009 (42.96)	0.57	0.50–0.65	<0.001	
Insurance	Non-insured	619 (26.10)	27,515 (28.14)	Reference level	~	~	<0.001
	Insured	1,030 (43.42)	29,433 (30.10)	1.56	1.41–1.72	<0.001	
	Unrecorded	723 (30.48)	40,834 (41.76)	0.79	0.71–0.88	<0.001	

*CI, confidence interval.

seeking veterinary care, and some veterinarians may intervene but not record the ASD as a problem in the clinical records.[42]

While many breeds represented in the current sample had ASD prevalence values similar to the 4.4% overall prevalence, several breeds showed distinctly lower or higher prevalence that is suggestive of either predisposition or protection, respectively. The prevalence was well over twice the 4.4% level for the Cavalier (14.83%) and King Charles Spaniels (13.78%), while the prevalence was less than half of 4.4% for Boxer (1.53%) and German Shepherd Dog (1.72%). After accounting for other confounding factors, predispositions were shown for six smaller sized breeds, while protection was shown for six medium to larger sized breeds, compared with crossbred dogs. These findings support some previous evidence that smaller breeds may be predisposed to ASDs,[4,5,17,43] although the current study did not support predispositions in the small terrier breeds and some toy breeds as has been suggested previously.

Review of the clinical records in this study suggests that the majority of practitioners generally accepted ASD as a standalone diagnosis, which may reflect the current paucity of published information on the aetiology, pathogenesis and associations with other diseases for ASD. This renders proposals of biologically plausible explanations for the breed associations found in this study highly speculative. For Spaniel breeds, such explanations might include differences in the proliferative reaction to inflammatory stimuli of gland-rich tissue as similarly proposed for ear

TABLE 3 Final breed-based multivariable logistic regression model for risk factors associated with diagnosis of ASD in dogs attending primary-care veterinary practices in England

Variable	Category	Odds ratio	95% CI*	Category *p*-value	Variable *p*-value
Breed-type	Crossbreed	Reference level	~	~	<0.001
	Cavalier King Charles Spaniel	3.31	2.76–3.97	<0.001	
	King Charles Spaniel	3.30	2.31–4.71	<0.001	
	Cockapoo	2.59	1.68–4.01	<0.001	
	Shih-tzu	1.66	1.28–2.13	<0.001	
	Bichon Frise	1.63	1.21–2.20	0.001	
	Basset Hound	1.50	0.79–2.86	0.213	
	Toy Poodle	1.49	0.88–2.52	0.138	
	Pug	1.36	0.93–2.00	0.118	
	Lhasa Apso	1.35	0.92–1.98	0.130	
	Chihuahua	1.33	0.99–1.78	0.062	
	Cocker Spaniel	1.24	1.01–1.51	0.037	
	Bulldog	1.21	0.75–1.96	0.431	
	Jack Russell Terrier	1.18	0.99–1.41	0.062	
	Tibetan Terrier	1.10	0.64–1.90	0.729	
	Labradoodle	1.10	0.73–1.65	0.645	
	Patterdale Terrier	1.09	0.61–1.95	0.773	
	Beagle	1.08	0.70–1.67	0.720	
	Schnauzer	1.04	0.55–1.97	0.906	
	West Highland White Terrier	1.02	0.79–1.31	0.899	
	Border Terrier	0.98	0.68–1.40	0.900	
	Miniature Schnauzer	0.98	0.65–1.47	0.910	
	Yorkshire Terrier	0.97	0.76–1.25	0.838	
	Pomeranian	0.94	0.50–1.78	0.861	
	Golden Retriever	0.88	0.65–1.19	0.395	
	English Springer Spaniel	0.88	0.65–1.18	0.384	
	Labrador Retriever	0.70	0.59–0.84	<0.001	
	Other	0.69	0.60–0.80	<0.001	
	Border Collie	0.60	0.44–0.82	0.002	
	Staffordshire Bull Terrier	0.56	0.44–0.70	<0.001	
	Lurcher	0.51	0.28–0.93	0.027	
	German Shepherd Dog	0.37	0.25–0.53	<0.001	
	Boxer	0.29	0.16–0.53	<0.001	
Insurance	Non-insured	Reference level	~	~	<0.001
	Insured	1.53	1.37–1.70	<0.001	
	Unrecorded	0.83	0.73–0.94	0.004	
Age (years)	<3.0	Reference level	~	~	<0.001
	3.0 - < 6.0	1.42	1.26–1.62	<0.001	
	6.0 - < 9.0	1.57	1.38–1.79	<0.001	
	9.0 - < 12.0	1.47	1.27–1.71	<0.001	
	≥12.0	1.19	1.01–1.41	0.038	

*CI, confidence interval.

canal skin,[44] or a general primary, epidermal and glandular hyperproliferative state[45] that might contribute to stenosis of the sac ducts even if subclinical elsewhere on the body. It would seem likely that swelling and lichenification of (peri)anal skin through chronic inflammation with consequent self-trauma to this area, as for example in allergic skin disease, might also contribute to duct stenosis and thus impaction and its sequelae. However, Cavalier King Charles spaniels and King Charles spaniels are not generally recognised as particularly predisposed to atopic dermatitis[46] which is the most frequent allergic skin disease of dogs, whereas the Boxer as a predisposed 'atopic' breed appeared relatively protected from ASD in the current

TABLE 4 Results for variables that replaced the breed variable in the final multivariable logistic regression model that retained age category and insurance status and that were significantly associated with a diagnosis of ASD in dogs attending primary-care veterinary practices in England

Variable	Category	Odds ratio	95% CI*	Category *p*-value	Variable *p*-value
Skull conformation	Dolichocephalic	Reference level	~	~	<0.001
	Mesocephalic	1.38	1.18–1.62	<0.001	
	Brachycephalic	2.62	2.19–3.13	<0.001	
Spaniel-type	Not Spaniel-type	Reference level	~	~	<0.001
	Spaniel-type	2.09	1.87–2.32		
Dachshund-type	Not Dachshund-type	Reference level	~	~	0.032
	Dachshund-type	1.38	1.03–1.84	0.031	
Poodle-type	Not Poodle-type	Reference level	~	~	<0.001
	Poodle-type	1.46	1.17–1.81	0.001	
Kennel Club Breed Group	Not KC-Recognised	Reference level	~	~	<0.001
	Toy	1.63	1.44–1.85	<0.001	
	Utility	1.10	0.94–1.28	0.245	
	Terrier	0.85	0.74–0.96	0.010	
	Gundog	0.81	0.71–0.92	0.001	
	Hound	0.78	0.62–0.98	0.030	
	Pastoral	0.49	0.39–0.61	<0.001	
	Working	0.31	0.23–0.43	<0.001	

*CI, confidence interval.

study. Furthermore, a relatively small and also highly predisposed 'atopic' breed, the West Highland White terrier[46] did not emerge as predisposed to ASD in the current study. Much of the association between ASD and allergic skin disease could stem from the overlapping signs of self-traumatising behaviour and inflammation affecting this body region; this may result in misattribution of (peri)anal self-trauma to ASD in dogs with allergic perianal pruritus,[9] and perhaps less frequently the other way round.

Given that the current study also identified a strong predisposition for ASD in brachycephalic (2.62 times the odds of dolichocephalic types) and spaniel (2.08 times the odds of non-spaniel types) types of dogs, unravelling the relative roles of breed compared with conformation as a risk factor may help to expose more about the causes of ASD. Data can be explored at many levels of abstraction to elicit useful results. These levels of abstraction vary from high (e.g. skull conformation) to moderate (e.g. breed) to precise (e.g. colours within breeds).[47] Inference gained from each level of abstraction holds differing opportunities and limitations; there is rarely a single perfect level of abstraction that will holistically answer every specific research question. The current study aimed to explore breed effects at both moderate (i.e. breed) and high (i.e. skull conformation or spaniel type) levels of abstraction in order to give more than one perspective on breed effects for ASD. Although ASD associations were identified for brachycephalic and spaniel types, these should be taken as hypothesis generators for future studies that may explore these findings in greater detail and should not be interpreted as suggesting that all brachycephalic or spaniel breeds or subtypes of dog carry similar risk for ASD.

Obesity has previously been proposed as one of many possible factors promoting the development of ASD.[7,43] Adult bodyweight is likely to be affected by many factors other than obesity but the lack of an association between ASD and adult bodyweight relative to the mean for breed and sex in the current study fails to provide any evidence supporting an association between obesity and ASD. Indeed, in the context of management of ASD, the clinical records in this study mentioned clinical recommendations for weight loss in only 1% of dogs, and for dietary change in only 8% of dogs. This may reflect that these measures are perceived as speculative with regard to their value in the management plan or that veterinarians perceive that there may be barriers for owners to implement these changes. However, these low levels may also reflect under-reporting in the records.

The current study identified that insured dogs has 1.53 times the odds of ASD compared with uninsured dogs. There is no logical reason for insurance to directly cause ASD so this association presents the idea of a confounding diagnostic bias whereby insured dogs are presented more commonly for veterinary care, may undergo more detailed veterinary examination and clinical management and may have owners that are more emotionally invested in the welfare of their dogs.[48] This suggests some overall welfare benefits to dogs from pet insurance.

The current study was not designed to directly link the severity of each ASD event with the specific types of treatment interventions. However, keeping in mind

the diagnostic uncertainties within the spectrum of ASD, the current results imply that an infectious nature was diagnosed or suspected in around 20% of ASD cases. This is based on two main findings that give similar results: a combined diagnosis frequency of sacculitis (12%) and abscessation (9%), and on the use of either local or systemic antimicrobials in 20% of ASD cases. Extrapolation from the disease prevalence (4.4%) along with the level of systemic antibiosis (18.04%) suggests that ASD led to systemic antimicrobial use in around 1% of the study dogs overall. This is similar to the number of dogs receiving systemic antimicrobials in primary-care practice for treatment of pyoderma,[49] despite the ASD caseload having a comparatively limited evidence-base to justify antimicrobial use. Studies are urgently needed to define diagnostic criteria and the role of bacterial infection in ASD, followed by clinical trials on indications for, and best use of, antimicrobials. Such information will help to guide clinicians on medical management and reduce the risk of inappropriate prescribing of antimicrobial therapy in this common primary care problem.

This study had some limitations in addition to those discussed above. There were some risks of misclassification of cases and non-cases in the study. Identification of ASD cases from the study population relied on the application of search terms to the clinical records. It is possible that these search terms did not identify all ASD cases and therefore the prevalence values reported here may be an underestimate. Similarly, the inclusion and exclusion criteria that were applied in this study may have included some dogs that were not true cases and may have excluded some dogs that were true cases. The study relied on the record-making diligence and accuracy of the veterinary teams involved. It is possible that some cases and clinical recommendations were not recorded in the clinical records and therefore that the current results under-report the true values. Classification of cases to higher levels along the hierarchy of severity relied on recorded information to justify these decisions. It is possible that some cases were misclassified with lower severity because the clinical records failed to record the full extent of the anal sac disease. This may have inflated the proportions of cases at lower severity (anal sac impaction) and reduced the proportions at higher severity (abscess). The clinical parameters reported here should be taken as summary results that can be greatly expanded upon in more detailed studies that specifically focus on therapeutic and management strategies and on clinical outcomes. In this study, no dogs were recorded as referred for management of ASD; this may explain the paucity of published evidence so far and highlights the fact primary care data are needed to explore the full spectrum of disorders seen in dogs. Associations between ASD and bodyweight relative to the mean for that breed and sex were explored at only a binary weight level (lower or higher) to optimize statistical power. Future and larger studies that offer greater refinement of these weight categories (e.g. tertiles or quartiles) could offer greater inference on bodyweight effects.

CONCLUSION

This study based on routine primary-care veterinary clinical data provides generalisable evidence that non-neoplastic ASD is a common presentation in the overall population of dogs accessing primary care practice in the UK. The study highlights the importance of ASD to canine welfare, with 12% of dogs receiving analgesia, and suggests that further work may be needed on good antimicrobial stewardship in small animal practice with 20% of ASD cases receiving antibiosis.

This is the first study with a clear focus on breed effect as a risk factor for ASD in dogs and can serve as a basis for breed-health monitoring of the most affected breeds.[50] The study also highlights many knowledge gaps, including aetiopathogenesis, diagnostic criteria, therapy, prevention and risk factors. The paucity of published information on these aspects of such a common and high welfare disorder emphasises the status of ASD as a research-neglected syndrome in dogs.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was granted by the RVC Ethics and Welfare Committee (reference number 2015/T94).

CONSENT FOR PUBLICATION

Consent to publish was provided by the Royal Veterinary College (PPS_02161).

AVAILABILITY OF DATA AND MATERIAL

The datasets generated and analysed during the current study are publicly available on the RVC Repostiory https://researchonline.rvc.ac.uk/id/eprint/12669/.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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AUTHOR CONTRIBUTIONS

Dan G. O'Neill, Anke Hendricks and Jennifer A. Phillips were responsible for the conception and design. Dan G. O'Neill, David B. Church and Dave C. Brodbelt were responsible for the acquisition of data. Dan G. O'Neill and Jennifer A. Phillips were responsible for the extraction and preparation of data. Dan G. O'Neill car-

ried out the analysis. Dan G. O'Neill, Anette Loeffler, Jennifer A. Phillips, David B. Church, David B. Church and Anke Hendricks were responsible for interpreting the results, drafting the manuscript, revising the manuscript and gave final approval of the version to be published. Dan G. O'Neill, Anette Loeffler, Jennifer A. Phillips, David B. Church, Dave C. Brodbelt and Anke Hendricks agree to be accountable for all aspects of the accuracy and integrity of the work.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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