Epilepsy in the Petit Basset Griffon Vendeen: Prevalence, Semiology, and Clinical Phenotype

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Background: Epilepsy with a genetic background is increasingly being identified. In certain dog breeds, epilepsy occurs with a higher prevalence than the estimate of 1–2% reported in the general dog population.

Hypothesis: The Petit Basset Griffon Vendeen (PBGV) experiences an increased occurrence of epilepsy compared to the general dog population.

Animals: The target population consisted of all 876 PBGV dogs registered in the Danish Kennel Club from January 1, 1999 to December 31, 2008. The study population included 820 dogs that met the inclusion criteria.

Methods: A population study was conducted to estimate the prevalence of epilepsy in the Danish PBGV population. A mailed questionnaire was used to detect possible signs of epilepsy. The information was subsequently validated by telephone interviews of positive and possible positive responders and a negative responder control group, using an extensive questionnaire developed to detect epilepsy. Dogs evaluated as epilepsy positive after the telephone interview were offered a clinical investigation.

Results: The prevalence of epilepsy was estimated to be 8.9% (42/471) in the PBGV population. Average age of onset was 26.3 months. Sex and mode of response did not affect the prevalence, but a strong litter effect was seen. Among euthanized dogs, epilepsy was the predominant cause (6/45 = 13.3%).

Conclusion and Clinical Importance: Petit Basset Griffon Vendeen dogs experience an increased risk of epilepsy characterized by a relatively early onset and dominated by focal seizures with and without secondary generalization. With an estimated prevalence of 8.9% and substantial clustering within litters, a genetic factor associated with epilepsy is suspected.

Key words: Convulsions; Focal; Generalized; Seizure.

Epilepsy is one of the most common neurologic conditions in the dog population, as in humans. In a hospital population of dogs, the prevalence has been estimated to be 1–2%. Epilepsy is not a single disease entity, but a variety of clinical signs reflecting underlying brain dysfunction that may result from different causes. Seizures can arise from several intracranial and extracranial pathologies, whereas epilepsy as a term only comprises seizures originating from the brain. Epileptic seizures are defined by recurrent transient occurrence of clinical signs caused by abnormal excessive or synchronous neuronal activity in the brain.

Epilepsy expressed in specific dog breeds has been investigated using a variety of approaches, including population surveys based on owner-reported phenomenology in the individual dog. Several studies have combined different types of surveys and questionnaires with a clinical investigation, and examples include the Bernese Mountain Dog, Labrador Retriever, Border Collie, Irish Wolfhound, English Springer Spaniel, Vizsla, Lagotto Romagnolo, Belgian Shepherd, and the Standard Poodle. It is largely agreed that the accumulation of individuals with epilepsy within certain breeds, and thus a high prevalence, is precipitated by genetic influence.

The Petit Basset Griffon Vendeen dog (PBGV) originates from France and is closely related to the Grand Basset Griffon Vendeen from which it was established with a separate breeding standard in the 1950s. The original function of the breed was rabbit hunting. According to The Danish Dog Register, there are approximately 1300 PBGV in Denmark in a total dog population of 580,000 individuals. In recent years, breeders of PBGV around the world have observed an increase in the number of dogs diagnosed with epilepsy. However, no studies on epilepsy have been conducted in this breed. We therefore found it of interest to investigate epilepsy in this breed.

The aim of this study was to estimate the prevalence of epilepsy in the Danish PBGV and to characterize the clinical phenotype. We also investigated the possible effects of sex, year of registration, and
clustering of epilepsy within litters on the prevalence of epilepsy.

Materials and Methods

The study was designed as a retrospective (including both living and deceased dogs) population study and carried out from September 1, 2009 to June 30, 2010. The target population was all PBGVs in Denmark and the study population consisted of all PBGVs registered in the Danish Kennel Club (DKC) from January 1, 1999 to December 31, 2008. The DKC is responsible for registration of all purebred dogs under Federation Cynologie International (FCI) in Denmark. In the DKC files each dog has a unique code identifying it by name, microchip, date of birth, and parentage.

The study population included living and deceased dogs to insure that the estimated prevalence would reflect epilepsy in the breed in the 10-year period. Dogs not domiciled in Denmark were excluded from the study to avoid the risk of misinterpretation caused by translation. In the relevant 10-year period, 876 PBGV were registered in the DKC. Fifty-six dogs had been exported, leaving 820 dogs domiciled in Denmark to be included in the study.

The study was conducted in 3 steps.

Step I: Initial information campaign and creation of a homepage. Prior to the investigation, the Basset Club in Denmark was contacted and informed about the upcoming survey, and a short informational article about the study was published in the Basset Club magazine. In addition, a homepage containing information about the project was created and hosted under the name: http://www.petit-epilepsi.dk. During the informational campaign, it was stressed that all information obtained would be kept confidential and anonymous.

Step II: Mailed questionnaire survey. A written questionnaire with a separate instruction letter and an enclosed stamped addressed return envelope was sent to the owners of the 820 dogs included in the study. In the instruction letter, the owners were given a short introduction to the investigation including the confidentiality of all information given. In addition, they were informed about the possibility of returning their questionnaire electronically by using the homepage http://www.petit-epilepsi.dk (mode of response). A mailed reminder was sent out 10 weeks later to increase the number of responses.

The written questionnaire contained questions addressing possible ictal phenomenology typically associated with focal and generalized epileptic seizures. A short presentation of epilepsy and signs of epilepsy prefaced the questionnaire. In addition, it contained questions about dog identification, owner identification, if the dog was deceased and if deceased the cause of death. Year of registration, sex, and information about parentage were retrieved from DKC. All the returned questionnaires were evaluated by the investigators and divided into 4 groups: positive responders, possible positive responders, negative responders, and invalid responders. Positive responders were defined by having answered yes to the occurrence of at least 1 clinical sign that could be associated with epilepsy and the group of responders with possible positive status consisted of responders having reported a confusing mix of possible ictal signs along with answering in the same questionnaire that the dog had never experienced ictal signs. Negative responders had answered no to all of the clinical signs listed as possible signs of epileptic seizures. The group of invalid responders consisted of questionnaires from owners of dogs not belonging to the study population or that lacked information about dog and owner. The group of invalid responders was excluded from the study.

Step III – Validation of positive, possible positive and negative status responders. Telephone interview validation. In order to validate the information given by the owners in the written questionnaire, all responders who answered yes to positive or possible positive signs of epilepsy in their dog were contacted by phone. As a validating procedure, the owners’ initial answers in the mailed questionnaires were initially confirmed by conducting an oral repetition of the mailed questionnaire. If the investigator was able to sustain the suspicion of epilepsy, the owner was subsequently given an extensive interview regarding age of onset, ictal, post-ictal, and inter-ictal signs to establish whether these were in agreement with those of a typical epileptic seizure phenomenology and seizure history. It was also registered if the dog suffered from diseases other than epilepsy and if a diagnosis of epilepsy had been previously established by the family veterinarian. A typical telephone interview lasted between 45 and 60 minutes. The ability of the questionnaire to detect clinical signs of epilepsy had previously been tested in a process where it was given orally to (1) a group of veterinary students, (2) a group of volunteers with dogs with and without epilepsy, and (3) a group of owners of dogs with and without epilepsy that were patients at the Small Animal Hospital. During this process, the questionnaire was adjusted to correct ambiguities detected. The questionnaire has been used in other population studies of epilepsy in the past.

Thirty negative responders were selected at random. As for positive and possible positive responders, the owners’ initial answers were validated by conducting an oral repetition of the original questionnaire.

To ensure a uniform standard, all of the interviews were done by the primary investigator and a veterinary Master’s degree student trained by the primary investigator.

Clinical investigation. All dogs that were defined as epilepsy positive after the interview validation were invited to participate in a clinical evaluation at the Department of Small Animal Clinical Science, University of Copenhagen, Denmark. The dogs were subjected to physical and neurologic examinations. Paraclinical test tests included blood samples analyzed by CBC, serum biochemistry including thyroid hormone concentration and urinalysis, and ECG and ultrasound of the heart by a cardiologist. A few owners were offered an MRI of their dog’s brain.

Case definition and epilepsy phenotype. The diagnosis of epilepsy in the individual dog was based on the detailed information collected on seizure history, seizure phenomenology and development, duration, and characteristics of the disorder as also used in humans. Additional information obtained from the clinical investigation also was used to establish the diagnosis.

A dog could only be validated as positive if the information obtained in the validation procedure was in agreement with that of a typical epileptic seizure phenomenology and seizure history, and if the owner had observed at least 2 seizures with a minimum interval of 24 hours as defined by the Commission on Epidemiology and Prognosis, the International League Against Epilepsy (1993). For each dog, seizure phenomenology recorded in the extended questionnaire was analyzed and seizures were classified by applying the standards given by the International League Against Epilepsy and the modified canine model by Beredt and Gram.17,19 Seizures were classified into focal onset seizures with and without secondary generalization and primary generalized seizures.

In this study no attempt was made to classify epilepsy types.
**Statistical Analysis**

Initially, univariable analyses of the association between epilepsy and sex, year of registration, and mode of response (internet/mail) were examined using Fisher's exact test.

Subsequently, the association between epilepsy and sex, year of registration, and mode of response was examined in a logistic analysis, where the possible effect of clustering within litters was accounted for by including litter as a random effect. Initially, a model with all 3 factors (expressed as qualitative variables) was specified. The model was reduced using backward elimination, removing variables with P-values above .05, starting with the least significant. To assess confounding, all variables were reinserted into the final model to compare estimates. To assess the effect of clustering within litters, the model with random effect was compared to a model without random effect using the Pseudo-AIC. To assess the variation explained by litter in the analyses, the interclass correlation was calculated as described by Dohoo et al.\(^a\):

\[
\rho = \frac{\sigma_{\text{Litter}}}{\sigma_{\text{Litter}} + \pi^2 / 3},
\]

where \(\rho\) represents the interclass correlation, and \(\sigma_{\text{Litter}}\) is the between litter variation.

All multivariable analyses were carried out using PROC GLIMMIX in SAS 9.1.\(^a\)

**Results**

**Prevalence**

In total, 528 questionnaires were returned during the period of investigation. Twenty-two questionnaires lacked information on dog identification and owner data and were excluded from the analysis. Thirty-five questionnaires had been filled out for dogs that were not included in the study population and were excluded from the study. This left 471 usable questionnaires representing 238 males and 233 females that could be further analyzed, hence resulting in a response rate of 57.4%. Four hundred and six owners (86.2%) replied via mail and 65 (13.8%) replied via the internet. The distribution of responders on the initial contact and the reminder was 392 and 79, respectively, resulting in an increase in response rate of 9.6%.

The age of the 471 dogs ranged from 9 to 137 months, with a median of 59 months.

Of the 471 dogs included in the analysis, 426 were still alive at the time of the study and 45 dogs were deceased. When analyzing the cause of death for all 45 dogs, the most common cause was epilepsy (13.3%). Of the 6 dogs that had epilepsy as cause of euthanasia, 5 were euthanized because of lack of seizure control and 1 because of lack of breeding value. On average, the 5 dogs lived 36 months (range, 18-70) from 1st seizure to death. The last dog was euthanized because of lacking breeding value and lived only shortly after seizure onset. All 6 dogs appeared normal inter-ictally according to the owners.

Sixty-nine responders answered yes to positive signs of epilepsy in their dogs. Forty of these dogs were confirmed to be positive based on the telephone-conducted validation procedure, whereas 29 dogs were validated as negative. The group of dogs defined as negative during the validation procedure consisted of dogs with clinical signs typically originating from other diseases such as arthritis or general anxiety and 6 dogs that had experienced only 1 seizure. One of the 6 dogs experienced a cluster seizure episode and was put on daily phenobarbital treatment immediately with no further seizures observed.

Of 19 responders with possible positive epilepsy status, 18 were evaluated to be negative and 1 was positive.

During the validation interview for one of the negative responders, an owner reported that their dog had shown typical signs of epilepsy and was therefore given the extended questionnaire interview. Based on this, the dog was evaluated as epilepsy positive.

The 42 dogs that were validated as epilepsy positive at the telephone interview were invited to participate in the clinical investigation. Eleven dogs were deceased and for 12 dogs, the owners did not wish to participate mainly because of distant location or lack of time. This left 19 dogs to be examined by the investigator during spring 2010. In all dogs, the physical and neurologic examinations were normal, and the cardiology examination and urinalysis did not reveal any cause of the seizures. The results of the CBC and thyroid testing generally were within reference ranges. A few results were marginally outside of the reference range and were considered to be of no clinical relevance.

Alkaline phosphatase (ALP) activity was mildly increased in 12 dogs. Bile acid stimulation tests were conducted in 9 of the dogs resulting in normal values for 7 dogs and marginally increased results in the last 2 dogs. Seven of these dogs had an abdominal ultrasound examination with no abnormalities detected.

Three dogs had MRI of the brain that did not identify any abnormalities causing seizures.

Extracranial causes of seizures in the 19 dogs participating in the clinical investigation thus were excluded.

Forty-two dogs were finally evaluated to be epilepsy positive resulting in a prevalence of epilepsy of 42/471 = 8.9% (95% CI, 6.3–11.5%). None of the dogs were known to suffer from other diseases that may mimic epilepsy. They all appeared normal inter-ictally according to the owners.

Twenty-five of the 42 dogs had been diagnosed with epilepsy by their family veterinarian before completion of the survey according to the owners. In 6 dogs, the family veterinarian was either uncertain of a diagnosis of epilepsy or did not think it was epilepsy. For 11 dogs, the owner never informed a veterinarian about the seizures.

**Semiology/Clinical Epilepsy Phenotype**

The age of onset could not be established in 6 of the 42 cases, but for 4 of them the answer was “adult”. For the remaining 36 dogs, an average age of onset was estimated to be 26.3 months (range, 2–78 months) with a median of 24 months based on numbers given in whole or half months. The age of onset is presented in Figure 1.
The 19 dogs that attended the clinical investigations had lived with their epilepsy on average 47 months (range, 10–113) with a median of 42 months when they were examined. Only 2 dogs had lived <2 years with epilepsy with durations of 10 and 22 months. Thirty-nine dogs experienced focal seizures. Seventeen dogs experienced focal seizures alone, whereas 22 experienced focal seizures with secondary generalization. Two dogs experienced primary generalized seizures and in 1 dog, information was too sparse regarding the onset of the seizure as to determine whether the onset was focal or generalized. The most commonly reported focal seizure phenomenology included motor signs such as ataxia and contractions of single muscle groups, autonomic signs such as vomiting and salivation and paroxysms of behavioral signs such as excessive attention seeking or standing with a blank stare not responding to external stimuli. A typical seizure lasted from 1 to 3 minutes. Typical ictal phenomenology of the 42 dogs is presented in Table 1.

### Risk Factors

The distribution of dogs with regard to sex, mode of response, and year of registration is given in Table 2 including the P-value of Fisher’s exact test.

In the univariable analyses, only the variable “year of registration” was significant (P = .0034). In the logistic analysis, the model including litter as a random effect was strongly favored by the Pseudo-AIC. The intraclass correlation (ρ) was $1.25/(1.25 + 3.142/3) = 0.28$, that is, 28% of the variation was caused by the effect of litter, thus supporting the hypothesis of a hereditary component of epilepsy in the PGBV. The nonsignificant P-value for year of registration, when adjusting for random effect of litter, was .1607.

Figure 2 shows the estimated prevalence of epilepsy by year of registration when adjusting for the effect of litter. The 471 dogs represented 175 different litters, but 19 of the 42 dogs with epilepsy originated from 7 specific litters. Three of these litters shared a common sire. One litter consisting of 3 dogs had a morbidity of 100%.

### Table 1. Seizure characteristics.

<table>
<thead>
<tr>
<th>Seizure Characteristics</th>
<th>Number of Dogs</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal (with or without</td>
<td>39</td>
<td>92.9 (39/42)</td>
</tr>
<tr>
<td>generalization)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor</td>
<td>33</td>
<td>84.6 (33/39)</td>
</tr>
<tr>
<td>Autonomic</td>
<td>17</td>
<td>43.6 (17/39)</td>
</tr>
<tr>
<td>Paroxysms of behavior</td>
<td>36</td>
<td>92.3 (36/39)</td>
</tr>
<tr>
<td>Secondary generalization</td>
<td>22</td>
<td>56.4 (22/39)</td>
</tr>
<tr>
<td>Generalized (primary)</td>
<td>2</td>
<td>4.8 (2/42)</td>
</tr>
<tr>
<td>Unclassifiable</td>
<td>1</td>
<td>2.4 (1/42)</td>
</tr>
<tr>
<td>Total number</td>
<td>42</td>
<td>100</td>
</tr>
</tbody>
</table>

### Table 2. Association between sex, mode of response, year of registration, and occurrence of epilepsy including P-value of Fisher’s exact test.

<table>
<thead>
<tr>
<th>RF</th>
<th>EP</th>
<th>PREV</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>26</td>
<td>212</td>
<td>238</td>
</tr>
<tr>
<td>Female</td>
<td>16</td>
<td>217</td>
<td>233</td>
</tr>
<tr>
<td>Total</td>
<td>42</td>
<td>429</td>
<td>471</td>
</tr>
<tr>
<td>Mail</td>
<td>35</td>
<td>371</td>
<td>406</td>
</tr>
<tr>
<td>Mode of response</td>
<td>Internet</td>
<td>7</td>
<td>58</td>
</tr>
<tr>
<td>Total</td>
<td>42</td>
<td>429</td>
<td>471</td>
</tr>
<tr>
<td>Year of registration</td>
<td>1999</td>
<td>7</td>
<td>30</td>
</tr>
<tr>
<td>2000</td>
<td>4</td>
<td>24</td>
<td>28</td>
</tr>
<tr>
<td>2001</td>
<td>8</td>
<td>32</td>
<td>40</td>
</tr>
<tr>
<td>2002</td>
<td>2</td>
<td>48</td>
<td>50</td>
</tr>
<tr>
<td>2003</td>
<td>6</td>
<td>29</td>
<td>35</td>
</tr>
<tr>
<td>2004</td>
<td>1</td>
<td>49</td>
<td>50</td>
</tr>
<tr>
<td>2005</td>
<td>6</td>
<td>53</td>
<td>59</td>
</tr>
<tr>
<td>2006</td>
<td>3</td>
<td>46</td>
<td>49</td>
</tr>
<tr>
<td>2007</td>
<td>4</td>
<td>64</td>
<td>68</td>
</tr>
<tr>
<td>2008</td>
<td>1</td>
<td>54</td>
<td>55</td>
</tr>
<tr>
<td>Total</td>
<td>42</td>
<td>429</td>
<td>471</td>
</tr>
</tbody>
</table>

RF, risk factor; EP, epilepsy; PREV, prevalence.

### Discussion

#### Prevalence

The present study estimated a prevalence of epilepsy in the PGBV to be 8.9% (CI, 6.3–11.5). This is high compared to the 1–2% estimate originating from hospital-based population studies of dogs in general. It
is, however, very close to what has been reported from other epidemiological studies of specific breed-associated epilepsy, and raises a suspicion of a genetic background for epilepsy in the breed.\textsuperscript{5,7}

Investigations of the prevalence of epilepsy have been carried out in various ways in several breeds. In the Labrador Retriever and the Belgian Shepherd, the studies were carried out as population studies using designs similar to the one used in the present study and estimated prevalences of 3.1 and 9.4\%, respectively.\textsuperscript{5,8} Among others, family studies have been carried out for the Irish Wolfhound, the English Springer Spaniel, the Border Terrier, the Standard Poodle, and the Belgian Shepherd with prevalences as high as 18.3\% for the Irish Wolfhound and 33.2\% for the Belgian Shepherd.\textsuperscript{7,9,10,14,16} It should be noted that familial studies explore families with accumulated epilepsy, and thus cannot be used as a measure of epilepsy prevalence in the general population of the breed. Breed-associated epilepsies eventually may be described as specific epileptic syndromes as we begin to understand the pathogenesis of canine epilepsy.\textsuperscript{21}

Being an epidemiologic study, screening for epilepsy in a large population of dogs both living and dead animals must be included to assure that the prevalence is not underestimated. It was not possible to examine dogs that were deceased and therefore the investigators had to rely on the owners' observations regarding these dogs. This was also the case for the living dogs whose owners did not accept the invitation to participate in the clinical investigation. The study was not designed to classify epilepsy types. Therefore, although nothing in the detailed history of these dogs indicated symptomatic epilepsy, we cannot exclude that this was the case for single individuals.

In the present study, epilepsy was the most common cause of death, and euthanasia was in the majority of dogs motivated by insufficient seizure control, which stresses the severity of the disease in the PBGV.

An important aim of the study design was preventing an overestimation of the prevalence. Exclusion of all responders where the suspicion of epilepsy could not be confirmed in the validation procedure served to insure that no dog erroneously was initially included as epilepsy positive based on the mailed questionnaire alone. Had the prevalence estimate been calculated based upon self-reported epilepsy from owners in the mailed questionnaire (Step I) alone, the estimated prevalence would have been considerably higher than 8.9\%. The validation of 30 negative responders indicated that 1 dog had been falsely put in the negative responder group. However, there is a risk that other positive dogs may be hidden in the negative group. An example could be if breeders were unwilling to report epilepsy in their breeding stock or offspring. If this was the case, the true prevalence of epilepsy in the PBGV would be higher than what is reported in this study.

If the 42 dogs were actually the total true number of dogs with epilepsy in the study population consisting of 820 animals, the prevalence estimate would be lowered considerably from 8.9 to 5.1\%, which still is higher than the 1–2\% reported in the general population. However, the 5.1\% would be a very conservative estimate.

There could be a tendency of owners with epileptic dogs to have a greater interest in returning the questionnaire compared to owners of dogs without epilepsy. This could also apply to owners of deceased epileptic dogs and would falsely increase the estimated prevalence in the population. On the contrary, it could be argued that owners of epileptic dogs would be less interested in returning the questionnaire, as a consequence of a parallel stigma of epilepsy in dogs, as has also been observed in humans.\textsuperscript{22}

During the planning of the study, the age of onset was obviously unknown, but with the knowledge now gained from our study, the chosen study population may contain individuals that had not yet experienced their first seizures as the youngest dog was only 9 months old. This may have resulted in an underestimation of the true prevalence estimate of epilepsy in the PBGV.

The major increase in response rate gained by sending reminders stresses the importance of this step in mailed questionnaire investigations because it increases not only the response rate but also the reliability of the results.

It is important to specify that the initial inclusion of a dog as epilepsy positive in this study was based on the owner's subjective observations. Because careful and detailed history taking remains the cornerstone in the accurate diagnosis of epilepsy an oral interview served to support the diagnosis.\textsuperscript{23,24} During this investigation, standard procedures were followed to insure uniform retrieval of information in each case, and the telephone interview allowed the investigators to obtain additional detailed information that could be used to establish a tentative diagnosis of epilepsy. It has been shown in a selected group of human patients with temporal lobe epilepsy that seizure identification by clinical history is highly accurate.\textsuperscript{25} Epileptologists rarely missed seizures but however tended to over-diagnose nonepileptic events as seizures, thus giving this type of diagnostic tool a high sensitivity but lower specificity.\textsuperscript{25} Moreover, screening instruments for ascertainment of epilepsy have been shown to have high sensitivity but low positive predictive value.\textsuperscript{26} Consequently, there is a potential risk that the estimated prevalence of this study is overestimated as a result of the fact that the initial diagnosis was based on the clinical history reported by the owner. However, 19 dogs participated in the clinical phase of the study and had no evidence of extracranial causes of their seizures. Furthermore, 60\% of the dogs were already diagnosed by the family veterinarian as suffering from epilepsy before entering the study. This further supports the validity of the diagnosis.

Eleven owners never reported the seizures of their dog to a veterinarian. This phenomenon also is known from human studies in which 14–48\% of patients suf-
ferring from epilepsy or seizures had never consulted a doctor.27-30 Such cases may result in falsely low prevalence estimates if the estimate is based on medically confirmed diagnosis of epilepsy. This stresses the importance of the questionnaire survey as a sensitive tool in the prevalence investigation.

**Clinical Investigation**

The clinical investigations did not identify any extracranial causes of the seizures in the 19 dogs, but an interesting finding was the mildly increased ALP activity observed in 12 dogs. This could indicate a breed-associated increase in ALP activity in the PBGV as also seen in the Bernese Mountain Dog, and it is currently under further investigation.31

**Semiology/Epilepsy Phenotype**

With an average age of onset estimated to be 26.3 months (range, 2–78), the breed experiences a rather early onset of epilepsy compared to other breeds such as the Belgian Shepherd, the Vizsla, and the Border Terrier.5,7,15 However, an onset as early as 0.41 year has been observed in Border Collie individuals and in the Lagotto Romagnolo the average age of onset is 6.3 weeks.11,12

A focal onset of seizures was reported in 92.9% of the dogs diagnosed with epilepsy in this study. This also has been reported in several other breeds such as the Belgian Shepherd, Labrador, Border Collie, Vizsla, and Standard Poodle.5,8,9,11,14,15

Seizure phenomenology experienced by the dogs included in the study was in accordance with what has been reported from other studies on canine epilepsy.5,9,11,14,15

**Risk Factors**

The year of registration seemed to have an impact on the prevalence before the numbers were adjusted for the impact of litter. Accumulated occurrence in specific litters with several littermates being affected leads to an increased prevalence of epilepsy in the year they were born, which explains the lack of significance of “year of registration” when adjusting for litter. This strongly supports the suspicion of genetic epilepsy in the PBGV and is currently being further investigated.

The prevalence was not significantly affected by sex, although males tended to be overrepresented. This tendency also has been reported in Beagle, Golden Retriever, Irish Wolfhound, and English Springer Spaniel.10,16,32,33

Mode of response did not affect the prevalence, which indicates that the answer was not biased by selection of either mail or internet as mode of response. Internet-based surveys are a cheap, fast, and easy way of conducting an investigation and could be recommended for future epidemiological studies.

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**Conclusion**

In conclusion, PBGV dogs have an increased risk of epilepsy characterized by a relatively early onset and dominated by focal seizures with or without secondary generalization. The present study was not designed to classify types of epilepsy, but the information obtained from the mailed questionnaire, oral interview, and the clinical investigation combined with the findings of a high prevalence estimate and a significant effect of litter on prevalence indicate a strong genetic influence and thus suspected idiopathic (genetic) epilepsy.

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**Footnote**

a SAS, 2002, SAS/STATS software (version 9.1), SAS Institute, Cary, NC

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**Acknowledgments**

The authors are grateful for the financial and practical support provided by the Danish Kennel Club and the patience and support given by the dog owners. The study was supported by a grant from the Danish Kennel Club.

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