Norges veteriærhøgskole
Institutt for sports- og familiedyrmedisin

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Fordypningsoppgave for

Kristin Paaske Anfinsen, Therese Rymoen Haagensen og
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Veiledere: Mette Berendt, Astrid Indrebø

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Innholdsfortegnelse

Sammendrag

Formålet med denne oppgaven var å fastslå insidensen av aseptisk meningitt (AM) hos nova scotia duck tolling retrievere (NSDTR) i Norge i årene 1994-2003. Motivasjonen for prosjektet var en tilsynelatende overrepresentasjon av NSDTR med AM observert av veterinærer ved Norges veterinærhøgskole.

Eiere av 362 tilfeldig utvalgte NSDTR ble intervjuet via telefon i løpet av 2005 for å identifisere hunder som hadde vært rammet av denne sykdommen. Basispopulasjonen på 1525 hunder var alle NSDTR født mellom 01.01.1994 og 31.12.2003 registrert i Norsk Kennel Klub.

En hund ble definert som positiv for AM hvis eieren hadde observert nakkesmerter, hunden hadde respondert på kortisonbehandling, og diagnosen ble bekreftet av veterinær. Ni hunder oppfylte disse kriteriene. Dette ga en insidens på 2,5% (95% KI 0,9% - 4,1%).

Det ble ikke funnet noen kjønnspredisponering for AM og heller ingen sammenheng mellom denne sykdommen og mulige risikofaktorer som tidligere vaksinasjoner, infeksjonssykdommer, løpetid og jakt.

Eiere og oppdrettere av AM positive hunder ble intervjuet om kjennskap og eventuelt slektskap til andre AM positive hunder. Slektskap mellom samtlige positive hundene gir en indikasjon på at sykdommen er arvelig.
Videre undersøkelse av sykdommens eventuelle arvegang kan resultere i klarere avlsanbefalinger.

Forord

Denne fordypningsoppgaven er skrevet på engelsk i form av en vitenskapelig artikkel. Dette er gjort fordi vi mener at arbeidet er av en slik karakter at det kan være interessant lesning for flere enn de vi ville ha nådd med en norsk oppgave. Etter at oppgaven er godkjent av sensor, vil veilederne Mette Berendt og Astrid Indrebø i samarbeid med epidemiolog Lis Alban bearbeide den engelskspråkelige artikkelen i henhold til spesifikke krav for en fagartikkel i et vitenskapelig tidsskrift.

Vi har gjennomført en spørreundersøkelse vedrørende et utvalg av nova scotia duck tolling retrievere registrert i Norsk Kennel Klub (NKK), med det formålet å finne insidensen av aseptisk meningitt i denne populasjonen.

Vedlagt fordypningsoppgaven er spørreskjemaet vi brukte, samt utkast til en artikkel som skal sendes til Retrievernytt (medlemsblad for retriever-eiere) etter at den engelskspråkelige artikkelen er publisert.

Vi ønsker å takke alle som har gjort det mulig å gjennomføre undersøkelsen og å skrive oppgaven. Mette Berendt, nevrolog ved den Kongelige Veterinær- og Landbohøjskole, Danmark, har vært vår hovedveileder. Hun har med sin store entusiasme gitt oss følelsen av at arbeidet vårt var verdifullt og således gitt oss drivkraften vi trengte. Den geografiske avstanden mellom oss har vært en ekstra utfordring som har vært overkommelig blant annet fordi Mette var villig til å reise til Norges veterinærhøgskole utelukkende for å møte oss, og ved at vi har fått ringe henne også privat på kveldstid. Astrid Indrebø har fungert som medveileder og hadde en spesielt viktig rolle med å få hele prosjektet i gang, men har fulgt prosjektet hele veien og kommet med innspill der det trengtes. Lis Alban, epidemiolog ved Danske Slagterier, har vært vår rådgiver innen statistikk og epidemiologi. Ellers ønsker vi å takke Vidar Grundetjern fra NKK for oversendelse av NSDTR databasen og tilgang på Dogweb, samt Anna Eggertsdottir og Øyvind Stigen for villigheten til å stille opp da vi slet litt med å komme i gang.

Oslo 01.12.05

K. P. Anfinsen T. R. Haagensen F. J. H. Liste

Risk of Aseptic Meningitis in a population of 1525 Nova Scotia Duck Tolling Retrievers in Norway: An epidemiological study

Introduction

Aseptic meningitis (AM) is an inflammation of the meninges that surround the brain and spine. Histopathologically, AM is characterized by inflammatory cell infiltration in the meninges and by inflammatory stenotic lesions in the meningeal arteries.[1-11] Affected dogs have been shown to have an overrepresentation of B lymphocytes in meningeal lesions and in peripheral blood. In inflamed arteries, only T lymphocytes have been detected. This finding can be used to distinguish between viral, bacterial and aseptic meningitis as the distribution of lymhocytes varies with the aetiology.[12]

This non-infectious form of meningitis is probably the most common type of meningitis in dogs, and it is usually seen in medium to large breed dogs, 7-16 months old.[13-15] There is no apparent sex-predilection.[2,14] The syndrome has been described in Bernese Mountain Dogs, Boxers and Beagles.[2-8,10,16-20]

The aetiology of aseptic meningitis is unknown. Immune mediated mechanisms are most probably causing the disease.[1-5,7,8,10,15-18,21] Excessive immunoglobulin (Ig)A production seems to play a central role in the pathogenesis of this condition.[8,9,17,22-24] It has been suggested that repeated injections of live multivalent vaccines may trigger the disease.[13] Oestrus may influence the onset of the disease in bitches.[16]

Two clinical forms are recognized. The typical acute condition is characterized by one or more of the following clinical signs: Fever, depression, anorexia, severe neck pain, pain when opening the mouth, hunched back, reluctance to move and stiff, stilted gait. The cerebrospinal fluid (CSF) shows a significant increase in the number of polymorph nucleated cells and an elevated protein concentration.[7-9,15-18,21,23,24] In the more severe protracted form of meningoencephalitis, there may be additional neurological signs such as generalised ataxia, tetra- or para- paresis, and mixed or mononuclear pleocytosis in the CSF. A peripheral neutrophilia may be apparent on a complete blood count.[1,2,4,5,7,8,14,16-21,24] Affected dogs may also have immune mediated polyarthritis.[16,25,26]

In most cases, clinical signs disappear rapidly when steroid therapy is instituted. However, approximately 50% of affected dogs have recurrence of clinical signs following discontinuation of corticosteroid therapy, and in some dogs lifelong medication is needed to avoid relapse.[13]

During the last years an increasing number of Nova Scotia Duck Tolling Retrievers (NSDTRs) in Norway have expressed clinical signs resembling what is found with AM. The Norwegian population of NSDTRs originated from dogs imported from Sweden and Denmark in the mid to late eighties. Later on dogs from other European countries and Canada were brought in to the Norwegian population. The first Norwegian litter was born in 1989 and the population has increased quickly as 1525 dogs have been born between 1994 and 2003.

The aim of the present study is to estimate the incidence, and identify any risk factors associated with AM in the Norwegian population of NSDTRs. Additionally, we were interested in studying

possible risk factors and possible familiar predisposition.

Materials and Methods

Study Design and Description of the Population

The study was designed as a retrospective cohort study. The central register of the Norwegian Kennel Club (NKC) contains information about all purebred dogs registered in Norway. Dogs are registered chronologically by date of birth.

A random sample, stratified by year of birth, was drawn from the population of the 1525 registered dogs born in the 10 year period from 01.01.1994 to 31.12.2003. The number of dogs registered each year can be obtained from table 1. At the time of the investigation, none of the dogs were younger than one year old. The authors obtained the following data from the NKC register: *registration number, date of birth, name and sex of the dog, and the name, address and telephone number of the owner.*

Table 1. Number of Nova Scotia Duck Tolling Retrievers registered in the Norwegian Kennel Club

Year of birth	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003
Number of dogs registered	65	106	142	129	175	171	158	167	221	191

Sampling Size

A precision (L) of 2% was considered appropriate for this study. The prevalence (p) was expected to be around 5%, based on the authors' best guess. According to this, a sample (n) of 362 dogs was needed to achieve the desired precision. The calculations were carried out as follows:

A preliminary sample is calculated:

$n^*=4 \times p \times q / L2$

p=estimated prevalence=0.05

$q=1-p=0.95$

L2= precision to the power of $2=0.02^2$

$n^*= (4 \times 0.05 \times 0.95) / (0.02 \times 0.02) = 475$

This formula is based on the assumption that the population in infinite. In a finite population, like the present, a smaller sample is needed. Therefore, the following formula corrects for this taking into account that the entire population (N) consisted of 1525 dogs:

$1/n=1/n^* + 1/N = 1/475 + 1/1525 \Rightarrow n=362$

The sample of 362 dogs were selected randomly, stratified by year of birth. Both living and dead dogs born in 1994 or later were included in the study.

A selected dog was replaced if the owner had moved from the address listed in the NKC register and could not be traced by the Norwegian telephone register or a transfer of ownership had occurred and the new owner could not be traced. The next dog on the list from the NKC replaced the excluded dog.

This sample was presumed to be representative of the total Norwegian population of NSDTRs with regard to gender, reproductive status, health status and other factors of possible interest.

Questionnaire

The owners of the dogs were interviewed by telephone using a standardized questionnaire. They were questioned regarding clinical signs related to AM, diagnosis and treatment.

Telephone Interview

An experienced neurologist (MB) conducted interviewer training, including instructions regarding the study design and principles of interviewing. The questionnaire evolved to its final form through several stages of discussion and testing.

Throughout the questioning, the interviewers' veterinary outlook was set aside, aiming at perceiving the disease from the owners' point of view. The language was kept as simple as possible, and the questioning was adapted to the owner's level of understanding.

The interviews were conducted by three interviewers (KPA, TRH, FJHL). After introducing themselves, they stated the objective of the interview and described the disease. Initially it was established whether the dog was alive or not. If the dog had died, the owner was questioned about year and cause of death. The following group of questions aimed at categorizing the dogs as preliminary positive or negative for AM. Four questions were posed to detect neck pain, as neck pain is a cardinal sign of AM. The owners were asked if the dog actively expressed signs of pain related to the neck area, were unwilling to reach for the food bowl, unwilling to turning the head,

or/and if the dog had been walking with a lowered head at that particular time. They were also asked whether their dog had been diagnosed with AM by their veterinarian.

The owners were then presented with a list of additional clinical signs which may also be seen in dogs suffering from AM. It was recorded whether the owners had observed fever, a stiff gait, depression, unwillingness to go for walks or play, or excessive panting (as a sign of pain). Owners who had observed any of the above described signs of AM in their dogs were asked if these additional signs were seen in conjunction with clinical signs of neck pain. This part of the questionnaire was designed to detect more subtle signs of AM.

Owners of dogs who responded positively to one or more of the four clinical signs of neck pain were also asked questions concerning risk factors. Hunting was considered a possible risk factor, since hunting dogs are more prone to tick bites and tick borne infections and thus to have a triggered immunesystem. These dogs may also be exposed to an increased amount of stress. Information regarding use of the dog for hunting was therefore obtained.

Infection and vaccination trigger the immune system. As AM is believed to be caused by immune mediated mechanisms, information regarding previous infections and vaccinations was recorded on AM positive dogs.[1]

Owners of female AM positive dogs were also asked whether the AM episode occurred related to heat. Dogs in heat have different hormone levels, and a connection between altered hormone levels and AM was investigated.

Owners of the preliminary positive dogs were also questioned if a medical treatment had been instituted while the dogs expressed clinical signs of AM. Finally, they were asked whether they had knowledge of any AM positive dogs familiarly related to their own dog.

To detect any familial predisposition, the breeders of positive AM dogs were contacted to obtain information about the genealogical tree of dogs with a positive diagnosis of AM and the outcome of the treatment. They were asked if they had knowledge of any other AM positive dogs related to each of the positive AM dogs in this study.

To further investigate any familial predisposition, the breeders of positive AM dogs were contacted and asked whether they had any knowledge of other AM positive dogs.

The treating veterinarians of all preliminary positive dogs were contacted by the interviewers in order to confirm a clinical diagnosis of AM.

To be finally included in the study with a positive diagnosis of AM, the dog should fulfil the following criteria: 1) Express clinical signs of neck pain. 2) The local veterinarian having verified clinical signs corresponding to AM. 3) Respond to corticosteroid treatment.

Statistical Analysis

A level of significance of 0.05 was used for all statistical calculations. The 95% confidence interval (CI) for the true incidence of AM in the Norwegian population of NSDTRs was calculated as $I \pm 2$ $SE = I \pm 2\sqrt{pq/n}$, (where SE = standard error, p = prevalence estimate, $q = 1 - p$, and n = sampling

size). To determine if a gender predisposition could be found in this study, a chi square test was performed.

Results

Study Design

Of the initially 362 sampled dogs, 42 (11.6%) had to be replaced because they could not be traced through the central Norwegian telephone register. The final material consisted of 172 female and 190 male dogs, ranging from one to eleven years of age at the time of the survey.

Incidence Risk

Nine dogs fulfilled the criteria for a positive AM diagnosis. This resulted in an incidence risk of 2.5% (95% CI 0.9% - 4.1%). Four females (4/172=2.3%) and five males (5/190=2.6%) were affected.

Clinical Signs

The clinical signs of the nine positive AM dogs appear from table 2. Six dogs showed all four expressions of neck pain. Additionally, all of the nine dogs showed four or more of the less specific clinical signs which can be observed in dogs suffering from AM.

In one dog the owner gave a positive answer regarding neck pain and difficulty in opening the mouth. This dog also had fever, a stiff gait, depression and did not want to go for walks or play at this time. The treating veterinarian of this dog was not familiar with the disease (AM), but however treated the dog with corticosteroids for months and the dog recovered from its disease. When the authors contacted this veterinarian and described AM, the veterinarian was positive that the dog had suffered from this disease.

Table 2. Clinical signs in nine Nova Scotia Duck Tolling Retrievers suffering from aseptic meningitis identified in a study on 362 dogs.

	Dog number	1	2	3	4	5	6	7	8	9
Clinical signsof neck pain	Neck pain	+	+	+	+	+	+	-	+	+
	Lowered head while walking	+	0	+	+	+	+	+	+	-
	Reluctance to turn the head	+	0	+	+	+	+	+	+	+
	Difficulty in lowering head to food bowl	+	0	+	+	+	+	+	+	-
Subtle clinical signs	Fever	+	+	+	+	+	+	+	+	+
	Stiff gait	+	+	+	+	-	+	+	+	+
	Unwillingness to go for walks or play	+	+	+	+	+	+	-	+	+
	Depression	+	+	+	+	+	+	+	+	+
	Difficulty in opening the mouth, e.g. carrying a ball	+	+	+	+	+	+	0	+	+
	Excessive panting	+	0	+	-	+	+	+	0	-

Dog number 4 and 9 also showed signs of hyperesthesia during the AM episode.

The age of the nine dogs at the time of the onset of the disease ranged from 4 to 19 months. All dogs showed improvement after corticosteroid treatment. Two dogs recovered during the first week of corticosteroid treatment, and three dogs recovered within three weeks. One dog was still on medication, occasionally showing signs after more than six months of medication, and one dog relapsed several times and was finally euthanized. One other dog had two recurrences, the first one a few days after finishing a corticosteroid treatment of 15 days. Additional two weeks of corticosteroid treatment was prescribed, and the second recurrence occurred ten months later. This time the dog was treated with corticosteroids for three months. Dog number 9, whose veterinarian was not familiar with the disease, received a relatively low dose of corticosteroids and suffered from mild signs of AM for several months. In the nine AM positive dogs, the recurrence frequency after withdrawal of treatment was 33.3% (3/9).

Familial Predisposition

The pedigrees of the in total 16 AM positive dogs were obtained from the central register of the Norwegian Kennel Club. With this information a genealogical tree revealing relations between all the positive dogs was drawn up (figure 1).

The owners of two dogs reported that dogs familiarly related to their own dog had been diagnosed with AM. One dog had one sister and several half-siblings with AM, and one dog had one half sibling with the disease. The veterinarian of a third dog had treated other dogs from the same breeder for AM, but did not know whether these dogs were related. This information in addition to the interviews with the breeders yielded the identities of an additional 7 AM-positive dogs.

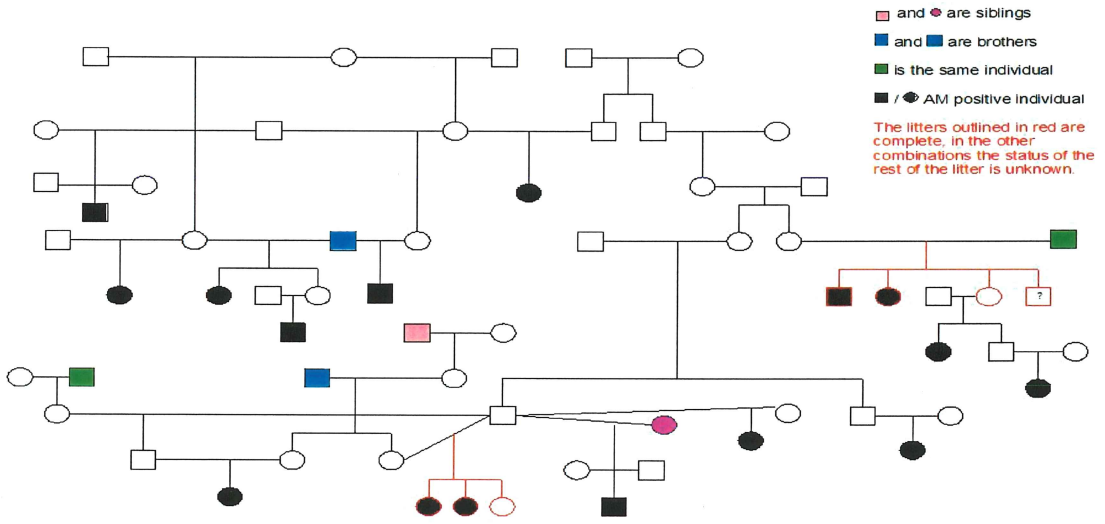

Figure 1. Genealogical tree of 16 Norwegian Nova Scotia Duck Tolling Retrievers suffering from aseptic meningitis.

The pedigree of each dog was drawn up as a genealogical tree, with five generations. Four ancestors appearing frequently were highlighted in all the genealogical trees. The most interesting of these ancestors, A, was found on both sides of two dogs and one side of seven dogs. The next step of the work was based upon the hypothesis that this dog, A, might hold a mutation that might be responsible for AM and that this dog was found as an ancestor on both the maternal and the paternal side of all 16 AM positive dogs. The pedigrees were extended where needed, by consulting the NKC register. It was found that all 16 dogs had A on both sides within the 7th generation (counting the AM positive dog as the first generation. However the investigation of a controlgroup of 14 negative dogs revealed that these also had A on both sides, within the 8th generation.

Risk Factors

Gender predisposition ($\chi^2 = 0.034$, $p = 0.8$) was not found, as five affected dogs were male (2.6% of all male dogs) and four were female (2.3% of all female dogs).

No indication of a relationship between vaccination, heat, infectious diseases or hunting and AM was found.

Discussion

Study Design

Both living and dead dogs born in 1994-2003 were included in this study. Excluding dead dogs would pose a risk of excluding a group of dogs with a higher incidence of AM than the rest of the population, thus creating selection bias. Regarding recall bias, it was decided that it would be no more difficult for an owner to remember ten years back whether the dog in question was old or dead. In order to avoid recall bias, it was however decided that no dog included in this study should be born prior to 1994.

Incidence Risk

In this study an incidence risk of 2.5% (95% CI 0.9% - 4.1%) was found. This can be interpreted as the risk an individual NSDTR in Norway has of developing AM some time during its life. In this study, none of the affected dogs were older than 19 months when the first signs of the disease appeared. This indicates that when a dog reaches a certain age, its risk of developing AM decreases.

Case Definition

To make a certain diagnosis of AM, a sample of CSF is needed. There will be an increased amount of polymorph nucleated cells and proteins in this sample in AM positive dogs. This is however not a standard procedure in Norway, as many veterinarians do not have the skills or access to the equipment needed. For this reason, CSF had not been obtained from the dogs diagnosed with AM in this study. However, we presume that the very typical clinical signs found in the nine positive dogs causing the veterinarians to diagnose them with AM and the quick response to corticosteroid therapy provided solid evidence to support that these dogs actually suffered from AM.

The dogs were initially classified as preliminary positive or negative regarding AM based upon whether the owners had observed any clinical expressions of the four initial questions regarding neck pain or not. Owners of dogs classified as negative were questioned if their dog had shown other, more unspecific signs of AM; fever, stiff gait, unwillingness to go for walks or play, depression or excessive panting. Regardless of the answers to these questions, these dogs could not

be classified as positive to AM. Information regarding these clinical signs was still recorded since this might reveal a more subtle form of AM. However, no such form can be described based on the results of this study.

Familial Predisposition

The work with the genealogical trees and comparison of common ancestors has so far not revealed any conclusive information. However, the theory of ancestor A being responsible can not be entirely discarded. Having ancestor A as a great-grandparent on both sides, assuming that AM is an inherited disease with an autosomal recessive mode of inheritance, and assuming A is a carrier, would give a 0.03% risk of having AM. Thus a control group of 14 negative dogs could easily have A on both sides without disproving the theory. While investigating the dogs beyond the 5th generation it appeared that there were a number of other ancestors that would be worthwhile to study further. This was not pursued on account of time limitation.

However, figure 1, revealing the relationship between the positive dogs, gives rise to the suspicion of familial predisposition.

Other Risk Factors

Information about the risk factors in the AM negative dogs was not obtained, and the number of AM positive dogs was low, disabling the possibility of concluding anything on the putative risk factors; Vaccination, heat, infectious diseases or hunting.

It has been suggested that vaccination and onset of heat could represent a risk factor for developing AM.[13,16] This could neither be confirmed nor discarded in the present study.

Future Perspectives

It would be interesting to further pursue the hypothesis of AM having an autosomal recessive mode of inheritance. This could be done by continuing to systematically compare the pedigrees of the AM positive dogs. It would also be interesting to get further knowledge on early cases of AM, in the NSDTRs native country Canada. Interviewing the littermates, halfsiblings and uncles and aunts of the positive dogs would also be useful in unravelling a possible pattern.

Another point worth pursuing is finding a model to calculate the prevalence of AM within a lineage, thus being able to determine the prevalence within the family described in figure 1. If this turns out to be greater than the prevalence in the breed it would be an indication that AM is indeed an inheritable disease.

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UNDERSØKELSE AV FOREKOMSTEN AV HJERNEHINNEBETENNELSE HOS NOVA SCOTIA DUCK TOLLING RETRIEVER I NORGE

I de senere årene har det vært et økt antall henvendelser til Norges veterinærhøgskole (NVH) fra dyrleger og eiere angående nova scotia duck tolling retrievere med symptomer som svarer til de man finner ved sykdommen ikke-smittsom hjernehinnebetennelse (aseptisk meningitt). Sykdommen forekommer spontant hos alle hunderaser, men det er registrert en økt forekomst hos visse raser. I Sverige har det vært mistanke om økt forekomst av denne sykdommen hos tollere.

Symptomer, behandling og prognose
Sykdommen opptrer typisk hos unge individer (7-16 mnd) med symptomer fra nervesystemet som feber, smerter i nakken og nedsatt appetitt. Symptomene reduseres ved behandling med kortison, men sykdommen kommer ofte tilbake. Prognosen er derfor avventende. Det vil si at sykdommen kan være invalidiserende og i noen tilfeller er det nødvendig med livslang behandling. Ikke alle tilfeller lar seg behandle og avlivning kan bli eneste utvei. Tidlig diagnose med riktig behandling bedrer prognosen. Det er derfor viktig å kontakte dyrlege med en gang dersom slike symptomer observeres.

Gjennomføring av undersøkelsen
Høsten 2004 til våren 2005 gjennomførte tre studenter ved Norges veterinærhøgskole en spørreundersøkelse angående denne sykdommen hos tollere i Norge. Et tilfeldig utvalg tollereiere, totalt 362, ble oppringt og spurt om de hadde observert symptomer som kunne relateres til denne sykdommen. Ettersom sykdommen spesielt karakteriseres av sterke nakkesmerter og høy feber, ble det stilt flere spørsmål vedrørende disse symptomene. Eiere av hunder som hadde fått diagnostisert sykdommen ikke smittsom hjernehinne betennelse, ble spurt om mulige utløsende faktorer som løpetid, vaksinering, tidligere infeksjonssykdommer og jakt. Telefonintervjuene ble utført etter veiledning fra veterinær og nevrolog Mette Berendt.

Resultater
Dataene fra undersøkelsen er bearbeidet i løpet av høsten 2005. Totalt ni hunder hadde fått stilt diagnosen ikke smittsom hjernehinnebetennelse hos veterinær. Dette gir en forekomst på 2,5% i utvalget på 362 hunder. Alle de ni positive hundene var mellom fire og nitten måneder gamle da de ble syke.

Forekomsten hos den totale hundepopulasjonen er ikke undersøkt, men det kan se ut til at tollerne er overrepresentert med hensyn til denne sykdommen. Man kan derfor ikke utelukke at arv har betydning for utvikling av sykdommen.

Det ble ikke identifisert noen utløsende faktorer i denne undersøkelsen.

Avlsanbefalinger
Nova scotia duck tolling retriever er en relativt liten rase i sterk vekst i Norge. Det er viktig å bevare rasens generelt gode helse. Selv om man på bakgrunn av undersøkelsen ikke med sikkerhet kan si at det foreligger en arvelig disposisjon for utvikling av sykdommen hos denne rasen, vil vi anbefale at hunder som har hatt denne sykdommen ikke brukes i avl. Det er grunn til å mistenke en arvelig disposisjon for en sykdom som trolig har høyere forekomst innen visse raser. Dersom man bruker nære slektninger av de syke hundene i avl, bør man pare med hunder som kommer fra linjer med lav eller ingen forekomst av sykdommen. For å kunne få dette til, er det nødvendig at raseklubben

får oversikt over hvilke hunder som har hatt sykdommen. Vi vil derfor oppfordre alle som har en hund som har fått diagnosen hjernehinnebetennelse (aseptisk meningitt) til å gi beskjed til klubben slik at klubben til enhver tid har best mulig oversikt til bruk i avlsarbeidet.

Vi gjør oppmerksom på at alle opplysningene i vår undersøkelse som kan identifisere hund og eier, behandles konfidensielt.

Vi vil gjerne takke alle hundeeiere som tålmodig har besvart våre spørsmål. Responsen har bare vært positiv når vi har tatt kontakt, og alle som har blitt oppringt har vært villige til å delta i undersøkelsen. Dette har gjort at vi har fått et sikkert resultat, og vi er takknemlige for all velvilje!

Veterinærstudentene
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SPØRREUNDERSØKELSE – IKKE SMITTSOM HJERNEHINNEBETENNELSE HOS NOVA SCOTIA DUCK TOLLING RETRIEVER

O. Generelle opplysninger om hund og eier, fra Norsk Kennel Klubs dataregister

P. Er hunden din (navn:.....) i live i dag? ja ☐ nei ☐

Hvis **JA,** gå til spm. **S.**

Q. Hva var dødsårsaken

.....

R. Hvor gammel var den da den døde?

0 år	1 år	2 år	3 år	4 år	5 år	6 år	7 år	8 år	9år	10 år +
0	1	2	3	4	5	6	7	8	9	10

FOR OSS: Hvis hunden har vært død i relativt kort tid må vi få eksakt dødsdato for å finne ut om den har vært død i mer enn 6 mnd, må da finne erstatningshund.

Selv om hunden din er død, vil vi gjerne stille deg noen spørsmål fordi det vil hjelpe oss i undersøkelsen vår.

S. Har hunden din fått stilt diagnosen ikke smittsom hjernehinnebetennelse (eller aseptisk meningitt) hos veterinær? ja ☐ nei ☐

Hvis **JA:** Navn på veterinær og evt klinikk.....

T. Har hunden din noen gang i en periode hatt symptomer på vondt i nakken uten at du kjenner noen direkte årsak til at den hadde vondt? ja ☐ nei ☐

Har du noen gang lagt merke til at hunden din i en periode..:

U. Har gått med hodet senket? ja ☐ nei ☐

V. Ikke har villet snu hodet til siden eller har virket stiv i nakken? ja ☐ nei ☐

W. Har hatt problemer med å senke hodet ned mot matskålen? ja ☐ nei ☐

Hvis **JA** på ett eller flere av spm:

X. Hvor gammel var hunden da?

0 år	1 år	2 år	3 år	4 år	5 år	6 år	7 år	8 år	9år	10 år +
0	1	2	3	4	5	6	7	8	9	10

Y. Hvor lenge varte det?

<1 uke	1-3 uker	1uke-2mnd	2-6mnd	6mnd-1år	Over 1år
1	2	3	4	5	6

De som har svart **JA** på **S, T, U, V** eller **W** → **JA** -skjema
De som har svart **NEI** på **S, T, U,V** og **W** → **NEI**-skjema

NEI-SKJEMA

Z. **Jeg vil nå lese opp en liste med ulike symptomer for deg. Jeg vil gjerne vite om du har observert noen av disse hos hunden din.**

1. Feber
2. Stiv gange
3. Liten lyst til å gå på tur eller leke
4. Vanskelig for å åpne munnen, f.eks holde en ball i munnen
5. Mye pesing (uten at det er åpenbart at det var fordi den var for varm)

Hvilke har du sett? (skriv tallene)_____

AA.Kjenner du årsaken til disse symptomene? (diagnose hos vet) ja □ nei □

Hva var årsaken?_____

Hvis ja: -takk for hjelpen

AB. **Hvor lenge varte disse symptomene?**

<1 uke	1-3 uker	1uke-2mnd	2-6mnd	6mnd-1år	Over 1år
1	2	3	4	5	6

AC. **Når var det hunden din viste disse symptomene?**

0 år	1 år	2 år	3 år	4 år	5 år	6 år	7 år	8 år	9år	10 år +
0	1	2	3	4	5	6	7	8	9	10

Takk for at du tok deg tid til å svare på våre spørsmål!

JA-SKJEMA

Jeg vil nå lese opp en liste med ulike symptomer for deg. Så vil jeg gjerne vite om du har sett noen av disse symptomene hos hunden din samtidig med de nakke-problemene du har nevnt tidligere.

AD. 1.Feber? ja ☐ nei ☐

AE. 2.Stiv gange? ja ☐ nei ☐

AF. 3.Liten lyst til å gå på tur eller leke? ja ☐ nei ☐

AG. 4Vært nedstemt? ja ☐ nei ☐

AH. 5.Hatt vanskelig for å åpne munnen, f.eks holde en ball i munnen? ja ☐ nei ☐

AI. 6.Mye pesing? (uten at det åpenbart var fordi den var varm) ja ☐ nei ☐

(**7.** Har gått med hodet senket **8.** Ikke har villet snu hodet til siden eller har virket stiv i nakken? **9.**Har hatt problemer med å senke hodet ned mot matskålen, **10.** Andre symptomer på smerter i nakken)

AJ. **Tisper: har noen av de symptomene oppstått i forbindelse med løpetid?** ja ☐ nei ☐

AK. Hvis **JA**, hvilke symptomer gjelder dette?(skriv tall).....

AL . **Har noen av symptomene oppstått umiddelbart etter vaksinasjon?** ja ☐ nei ☐

AM. Hvis **JA:** Hvilke symptomer gjelder dette?(skriv tall).....

AN. **Har noen av symptomene oppstått umiddelbart etter en annen infeksjonssykdom, for eksempel diaré eller halsbetennelse?** ja ☐ nei ☐

AO. Hvis **JA:** Hvilke symptomer gjelder dette?(skriv tall).....

AP. **Har du oppsøkt veterinær i forbindelse med noen av symptomene?** ja ☐ nei ☐

Hvis **JA:** Hvilken veterinær var du hos? .....

Hvis nei- hopp til AX

AQ. **Fikk hunden din behandling?** ja ☐ nei ☐

AR. Hvis **JA:** Husker du hva slags behandling?

1.antibiotika ☐ **2.**kortison ☐ **3.**smertestillende ☐ **4.**annet ☐ .....

Husker du navnet på preparatet? .....

AS. **Hvor lenge fikk hunden din medisiner?**

<1 uke	1-3 uker	1uke-2mnd	2-6mnd	6mnd-1år	Over 1år
1	2	3	4	5	6

AT. **Ble hunden din bedre av behandlingen?** ja ☐ nei ☐

AU. Hvis **JA:** Ble hunden din verre igjen etter avsluttet behandling? ja ☐ nei ☐

AV. **Fikk hunden din en diagnose hos veterinæren?** ja ☐ nei ☐

Hvis **JA:** Hva var diagnosen? .....

AW. **Kjenner du til at hunder i slekt med din hund har hatt ikke smittsom hjernehinne-betennelse?** ja ☐ nei ☐

AX. Hvis **JA:** spesifiser:.....(bror, søster, mor, far, besteforeldre....)

AY. **Bruker du hunden din som jakthund?** ja ☐ nei ☐