

Studies of serum total immunoglobulin E concentrations in atopic and non-atopic dogs

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The serum total immunoglobulin E (IgE) concentrations of two groups of atopic dogs and three groups of non-atopic dogs were compared. There was a wide range of concentrations with a high degree of overlap between the groups. The serum total IgE concentrations of a group of 15 non-atopic racing greyhounds were significantly higher than those of all the other groups. Atopic and non-atopic dogs receiving stringent parasite control treatments could not be differentiated on the basis of their serum total IgE concentrations. In the non-atopic dogs there was no correlation between their serum total IgE concentrations and the number of allergen-specific positive results obtained in an ELISA, or between their serum total IgE concentrations and their age.

IN human medicine, serum total immunoglobulin E (IgE) concentrations are used as a diagnostic indicator of atopic dermatitis (Juhlin and others 1969, Gurevitch and others 1973, Jones and others 1975). In general, atopic people have significantly higher concentrations of serum total IgE than non-atopic people. However, no such differences have been found in dogs, which have been shown to have much higher serum total IgE levels than people (Schwartzman and Rockey 1967). It has been suggested by Halliwell and Kunkle (1978) that this difference is due to the high levels of parasites to which dogs are exposed, because the major antibody response to parasites is IgE. The first aim of this study was to find out whether atopic and non-atopic dogs which were considered parasite free, could be differentiated in terms of their serum total IgE concentrations alone.

It has been suggested by Griffin and others (1990) that, in non-atopic dogs with high serum total IgE concentrations, non-specific binding of IgE can occur in an ELISA and lead to false positive results. The second aim of this study was to find out whether non-atopic dogs with high serum total IgE concentrations were more likely to have positive reactions on an allergen-specific ELISA than non-atopic dogs with low concentrations.

In people serum total IgE concentrations have been shown to increase with age up to 23 years (Johansson and others 1970). The final aim of this study was to measure serum total IgE concentrations in non-atopic dogs of different ages.

MATERIALS AND METHODS

Dogs

The serum total IgE concentrations of five groups of dogs were examined: they consisted of 55 non-atopic and 12 atopic dogs from the Guide Dogs for the Blind Association (GDBA) (guide dogs), 15 non-atopic racing greyhounds, 25 non-atopic laboratory beagles and 28 atopic dogs referred to the dermatology department at the Glasgow University Veterinary School (GUVS).

Samples were obtained from the non-atopic dogs at various ages but they were not allocated to this group until they had reached three years of age without any history of multiple episodes of atopic type skin disease on the basis of the criteria established by Willemse (1986).

The skin of any dog showing evidence of pruritus was thoroughly examined by the collection of coat brushings, skin scrapings, tape strips and hair pluckings which were examined microscopically and by culture for fungi.

All of the dogs were treated for ectoparasites with prescription-only medicines (POMs) and all of the guide dogs with recurrent pruritus were treated with phosmet (VetKem

Sponge On; Sanofi Animal Health), which is part of the GDBA protocol. Dogs with pyoderma were treated with 30 mg/kg cephalixin (Ceporex; Schering-Plough) twice daily for a minimum of two weeks. Yeast infections were treated with chlorhexidine/enilconazole shampoo (Malaseb; Leo Laboratories), according to the manufacturer's instructions, twice weekly for a minimum of two weeks. Dogs with recurrent episodes of pruritus were placed on a restriction diet of either home-prepared chicken and rice or commercially available catfish and rice (Pedigree Select Protein; Pedigree), and were allowed only this diet and water for a minimum of six weeks up to a maximum of 12 weeks.

The two groups of atopic dogs were diagnosed as such if they continued to show signs of pruritus after the above treatment regimens, did not show any improvement on a restriction diet, and gave at least one positive reaction on intradermal skin testing.

Guide dogs The guide dogs were predominantly labrador retrievers, golden retrievers (and their crosses) and German shepherd dogs, with a few curly coated retrievers, flat-coated retrievers and collie crosses. They were either kept in kennels in the north east of Scotland or in their owner's homes throughout Scotland, and were therefore exposed to a wide variety of allergens. They were fed proprietary diets and were kept under a strict regimen of veterinary care and parasite control. They were vaccinated against distemper, parvovirus (Nobi-vac Puppy DP; Intervet) and leptospirosis (Nobi-vac L; Intervet) at six weeks of age, against distemper, parvovirus, hepatitis, parainfluenza (Nobi-vac DHPPi; Intervet) and leptospirosis at 12 weeks of age and finally against leptospirosis at 16 weeks. Full booster vaccinations were then given annually. They were not routinely vaccinated against kennel cough.

Parasite control began with treatment of the nursing bitches and their pups with fenbendazole (Panacur; Intervet), at a dose of 1 g/2.2 kg at two, five and eight weeks after whelping. Subsequently, the pups received a combination of febantel, praziquantel and pyrantel (Drontal Plus; Bayer) at a dose of 5 mg praziquantel/kg, that is, one tablet/10 kg, at 12 weeks, six months and 12 months of age, and then at six-month intervals. Faecal samples taken from all the dogs at eight to 10 months of age were uniformly negative for parasites. All the dogs were treated monthly with fipronil (Frontline; Merial) or permethrin (Exspot; Schering-Plough) spot-on once a month to control ectoparasites, and no parasites were found on clinical examination.

Greyhounds The greyhounds were registered with the National Racing Greyhound Club (NRGC) and were being blood sampled as part of an investigation of their poor rac-

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ing performance. They were examined by the referring veterinary surgeon and were free of clinical skin disease when they were blood sampled and had no history of recurrent skin disease. They were housed in outdoor kennels built of either concrete or wood and were kept on newspaper or sawdust bedding. In general, they were not allowed into a household environment, but they were exposed to fleas both in the kennels and at the race track. They were all treated regularly, at least three to four times a year, with proprietary POM anthelmintics (M. Boyce, personal communication). They were all up to date with vaccinations against distemper, parvovirus, leptospirosis and hepatitis.

Beagles The beagles were from a closed colony of dogs in England which were being examined serologically for the assessment of antiviral antibodies; excess serum was made available for this study. It was not possible to examine them directly but the referring veterinary surgeon confirmed that they were free of skin disease when they were sampled and had no history of recurrent skin disease. They were kept indoors at all times and had no access to outdoor allergens. No ectoparasites were detected and the dogs did not receive any ectoparasite therapy. They were housed in concrete and steel buildings with sawdust and paper bedding and were allowed plastic toys. They had all been vaccinated against distemper, hepatitis, parvovirus, parainfluenza (Nobivac DHPPi; Intervet) and leptospirosis (Nobivac L; Intervet) at six, nine and 12 weeks of age, and these vaccinations were repeated annually. The breeding bitches were also vaccinated by the same regimen at every oestrus. Endoparasites were controlled with fenbendazole (Panacur; Intervet) at two, five, eight and 12 weeks of age at a dosage of 50 mg fenbendazole/kg bodyweight for three consecutive days. At 28 weeks of age and every four months thereafter, they received a single dose of fenbendazole at 100 mg/kg bodyweight. Pregnant bitches were treated with fenbendazole at a dosage of 25 mg/kg bodyweight daily from day 42 of pregnancy until two days after whelping. These dogs were believed to be free of parasites.

Atopic GUVS dogs This group of dogs contained a wide variety of breeds, including Staffordshire bull terriers, English setters, labrador retrievers and German shepherd dogs. They had been referred with clinical signs consistent with atopic dermatitis and at least one positive result on intradermal skin testing. They were kept as household pets, were exposed to both indoor and outdoor allergens, and had access to areas of grass and other animals. The measures used to control parasites in these dogs varied widely, often being irregular or unknown, but they had all received up-to-date vaccinations against distemper, leptospirosis, parvovirus and hepatitis.

Serology

Blood samples were taken from the atopic dogs for diagnostic serological testing. Blood samples were taken from the non-atopic dogs either for health profiles before elective surgery or for routine health checks. Serum from the laboratory beagles was obtained from samples taken to measure viral antibody titres.

Blood samples of at least 5 ml were taken by jugular venepuncture, placed in a glass tube without any additives and allowed to clot at room temperature. The samples were then centrifuged at 2000 g for 10 minutes and the serum was removed and divided into 0.5 ml aliquots which were stored at -20°C until required for testing.

Immunodot Serum total IgE concentrations were measured by using an Immunodot serological test kit (HESKA) which consisted of multiple nitrocellulose strips with spots con-

taining various substances which, when incubated with serum, bind to IgE and immunoglobulin G (IgG). Following the addition of an anti-IgE antibody, which binds specifically to IgE, and an enzyme, colour is developed in proportion to the amount of IgE present.

The test kit also contained Tris buffered saline (TBS), 4 per cent Microcide III, horseradish peroxidase monoclonal anti-IgE antibody, chromogen (4-chloro-1-naphthol) and an enzyme substrate containing 3 per cent hydrogen peroxide.

Before assessing the clinical samples, serial dilutions of a known serum total IgE concentration were applied to Immunodot test strips to validate the test. The serum samples were defrosted at room temperature, and 0.5 ml was added to 0.5 ml TBS which had previously been diluted one in 10 with distilled water and 4 per cent Microcide III. The resulting 1 ml of solution was then added to a well containing the Immunodot strip and incubated at 22°C on a horizontal shaker for 12 hours.

After this time, the serum was removed from the well and the strips were washed. To each strip, 1 ml of the horseradish peroxidase monoclonal anti-IgE antibody mixture was added, and it was incubated at 22°C on the horizontal shaker for two hours. The antibody was then removed and the strips were washed. Developer was prepared by adding 15 graduated drops of chromogen and two graduated drops of enzyme substrate containing 3 per cent hydrogen peroxide to 10 ml distilled water. To each strip, 1 ml of this developer was then added and incubated for 15 minutes. The developer was then removed and the strips were washed under running tap water for one minute. The strips were then blotted dry and allowed to dry completely at room temperature for 24 hours.

Colour formation on this test strip indicated a positive result, with the degree of colour formation being proportional to the concentration of serum total IgE. The colour was assessed with an optical densitometer (Scanner model 420oe; Agfa), to give a reflective density value.

ELISA Allergen-specific ELISA analyses of the serum samples were carried out on 43 of the non-atopic dogs, using a commercially available ELISA (ALerCHECK). There was too little serum to assess the other 12 non-atopic dogs. The allergens incorporated into this test are designed specifically for the European market and the test itself has been validated by the company (R. Robinson, personal communication).

The serum samples were defrosted at room temperature and 0.6 ml of each was mixed with 2.4 ml of the diluent provided. This mixture was incubated at 4°C for 24 hours, to bind serum proteins which can bind non-specifically to the plates (Kemeny and Challacombe 1988). Following this incubation, 100 µl of each diluted sample was added to each allergen-coated well and incubated at 22°C for two hours. Each serum was tested against outdoor and indoor antigen panels, a negative control and a positive control (Table 1). The wells were then decanted and washed. To each well, 100 µl

TABLE 1: Allergens included in the ELISA

Outdoor allergens	Indoor allergens
Orchard grass	Mixed housedust mites –
Timothy grass	<i>Dermatophagoides farinae</i> and
Kentucky blue	<i>Dermatophagoides pteronyssinus</i>
Fescue	Mixed feathers
Poplar	<i>Alternaria</i> species
Birch	<i>Aspergillus</i> species
Sheep's sorrel	<i>Rhizopus</i> species
English plantain	Kapok
Mugwort	Housedust
Dandelion	Cat epithelium
Nettle	Human epithelium
Flea	<i>Mucor</i> species

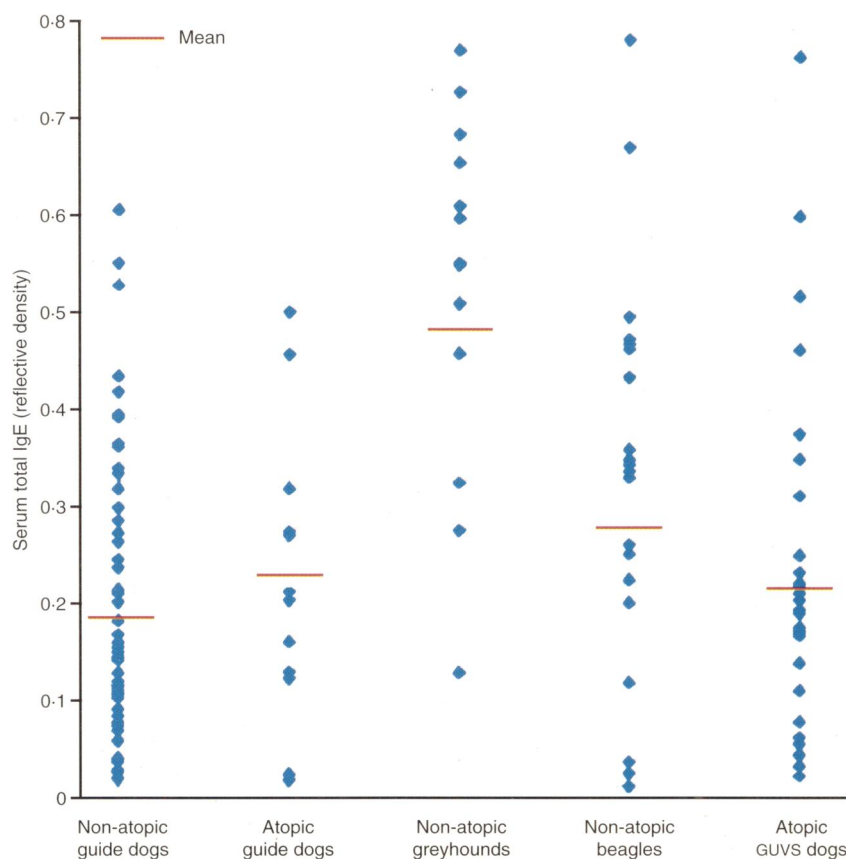


FIG 1: Individual serum total immunoglobulin E (IgE) reflective densities in five groups of atopic and non-atopic dogs. GUVS Glasgow University Veterinary School

of polyclonal rabbit anti-canine IgE was added, followed immediately by 100 µl of conjugated peroxidase goat anti-rabbit IgG. This mixture was incubated at 22°C for three hours. The well contents were then decanted and washed. To each well was added 100 µl of 3,3',5,5'-tetramethylbenzidine hydrochloride (TMB)/ peroxide and the plate was incubated at 22°C for 30 minutes; 100 µl of 0.18M sulphuric acid was then added to each well, resulting in the formation of a yellow colour if allergen-specific IgE was detected. The optical density was measured on a microplate reader (Bio-tek Instruments) at 450 nm, and was correlated with the degree of positivity.

Statistical analysis

The results were analysed by a one-way analysis of variance and the Newman-Keuls multiple range test. Trends of association between variables were examined by using Pearson's correlation coefficient, with *r* values close to 0 indicating no evidence of association. The analyses were made by using the Minitab version 11.21 statistical software package.

RESULTS

Serum total IgE concentrations of the different groups of dogs

There was a wide range of serum total IgE concentrations in the atopic and non-atopic dogs with extensive overlaps between the groups (Fig 1). There was no significant difference between the IgE concentrations of the atopic and non-atopic guide dogs (Table 2) and both groups contained dogs with high and low concentrations of IgE.

TABLE 2: Mean (sd) and ranges of serum total immunoglobulin E reflective densities in the five groups of dogs

Group	Number of dogs	Mean (sd)	Range
Non-atopic guide dogs	55	0.182 (0.143)	0.008-0.567
Atopic guide dogs	12	0.203 (0.144)	0.006-0.467
Non-atopic greyhounds	15	0.498 (0.167)	0.011-0.684
Non-atopic beagles	25	0.275 (0.202)	0-0.735
Atopic GUVS	28	0.206 (0.172)	0.010-0.718

GUVS Glasgow University Veterinary School

There were no significant differences between any combination of non-atopic beagles, atopic GUVS dogs, or non-atopic or atopic guide dogs. The non-atopic guide dogs with excellent parasite control could not be differentiated from the atopic GUVS dogs receiving variable parasite control measures. However, the serum total IgE concentrations of the non-atopic greyhounds were significantly ($P<0.05$) higher than those of all the other groups (Table 2).

Serum total IgE concentrations and false positive ELISA results

Many positive results were observed in the allergen-specific ELISAs of the non-atopic guide dogs, against a wide variety of allergens, with no particular allergen predominating. There was no significant correlation between the number of these positive ELISA results for each dog and that dog's serum total IgE concentration (Pearson's correlation coefficient -0.102) (Fig 2). Some dogs with serum total IgE reflective density levels up to 0.567 did not produce any positive ELISA results.

Serum total IgE concentrations in relation to age

There was no correlation between the age of the non-atopic guide dogs and their serum total IgE concentrations, with both young and old dogs having a wide range of serum total IgE levels (Fig 3).

DISCUSSION

The production of IgE antibodies is directed mainly against parasites and is involved in hypersensitivity reactions (Bloch and others 1972, Tizard 1987). Serum IgE levels in non-atopic human beings are relatively low at 300 ng/ml (Johansson and others 1970), whereas in dogs they can be as high as 350 µg/ml (Rockey and Schwartzman 1967).

Serum IgE levels have been shown to be significantly higher in atopic people than in non-atopic people (Juhlin and others 1969, Gurevitch and others 1973, Jones and others 1975). However, no such difference has been observed in dogs (Halliwell and Kunkle 1978). The high level of total serum IgE in dogs is assumed to be due to the high level of parasitism to which they are exposed and their infestation (Halliwell and Kunkle 1978), so that parasitised atopic and non-atopic dogs have similar serum total IgE concentrations.

It is known that guide dogs are generally free of endoparasites, and it would therefore be expected that atopic guide dogs would have significantly higher serum total IgE concentrations than non-atopic guide dogs. However, the non-atopic guide dogs had a wide range of serum total IgE concentrations similar to that observed in the atopic guide dogs. Similar findings were reported by DeBoer and Hill (1999) who found a wide range of serum total IgE concentrations in a group of six- to 12-week-old West Highland terriers, and the terriers which went on to develop atopic dermatitis could not be differentiated from those that did not on the basis of their serum IgE concentrations.

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The groups of non-atopic guide dogs and non-atopic beagles both contained dogs with high levels of serum total IgE. Both groups underwent stringent parasite control measures and it is unlikely that parasite burdens were responsible for inducing the production of serum IgE. A more likely reason for these serum total IgE concentrations is the 'high' and 'low' responder theory put forward by de Weck and others (1998), following original work by Katz (1978). Katz demonstrated that a group of inbred mice had a wide range of serum total IgE concentrations and he divided them into groups of high or low responders; the phenotype was restricted to the IgE antibody class and did not occur with IgG. Katz (1978) also observed that the low responder phenotype corresponded to the non-atopic population. De Weck and others (1998) observed that similar groups of dogs had high and low levels of serum total IgE. The results from the non-atopic guide dogs and beagles were similar. Although parasitism and hypersensitivity reactions may influence serum IgE levels, there may also be a genetically dominant factor affecting them. The division of animals into groups of high or low responders suggests that there may be a bimodal distribution of serum total IgE response, but this is not the case, because there was some overlap between the high and low responders (Fig 1). A genetic influence on serum IgE production has also been observed by Griot-Wenk and others (1999) who found significant differences in serum total IgE concentrations between different groups and breeds of dogs.

The non-atopic guide dogs did not show any evidence of being atopic, but some of them were high responders. As de Weck and others (1998) suggested that only the dogs with high serum total IgE concentrations went on to develop atopic dermatitis, it would be reasonable to expect that these dogs could develop atopic dermatitis. However, the results of this study suggest that although a dog may have the ability to become atopic, that is, it is a high responder, it may not necessarily become so and other controlling factors must be involved. Mayer and others (1998) suggested that genes other than those controlling serum IgE concentrations may be involved in the development of atopic dermatitis.

It is possible that IgE is not the only antibody involved in the pathogenesis of atopic dermatitis. Willemse and others (1985) detected allergen specific IgG₄ antibodies in atopic dogs, but not in normal dogs. However, Lian and Halliwell (1998) have suggested that this antibody has a protective role rather than a pathogenic role.

De Weck and others (1998) suggested that only high responders develop atopic dermatitis, but some of the atopic guide dogs had low serum total IgE concentrations. The ears of the dog with the lowest concentration had been treated with prednisolone two weeks before it had been skin tested and blood sampled and it had received prednisolone tablets intermittently for three months until eight weeks before the skin test. The dog with the second lowest serum total IgE concentration had received an injection of dexamethasone four months before being skin tested and blood sampled. Dexamethasone has been shown to have approximately 20 times the anti-inflammatory activity of prednisolone (Intervet data sheet) and it is possible that these steroid therapies were responsible for the low serum total IgE concentrations. Although it has been stated that there is no requirement to stop corticosteroid therapy before ELISA testing (Anderson and Sousa 1993), recent work has suggested that such treatment may influence serum IgE concentrations (McCall and others 1998).

There was no significant difference between the non-parasitised, atopic guide dogs and the possibly parasitised atopic GUVs dogs. If serum total IgE concentrations are influenced by factors other than parasitism then this result

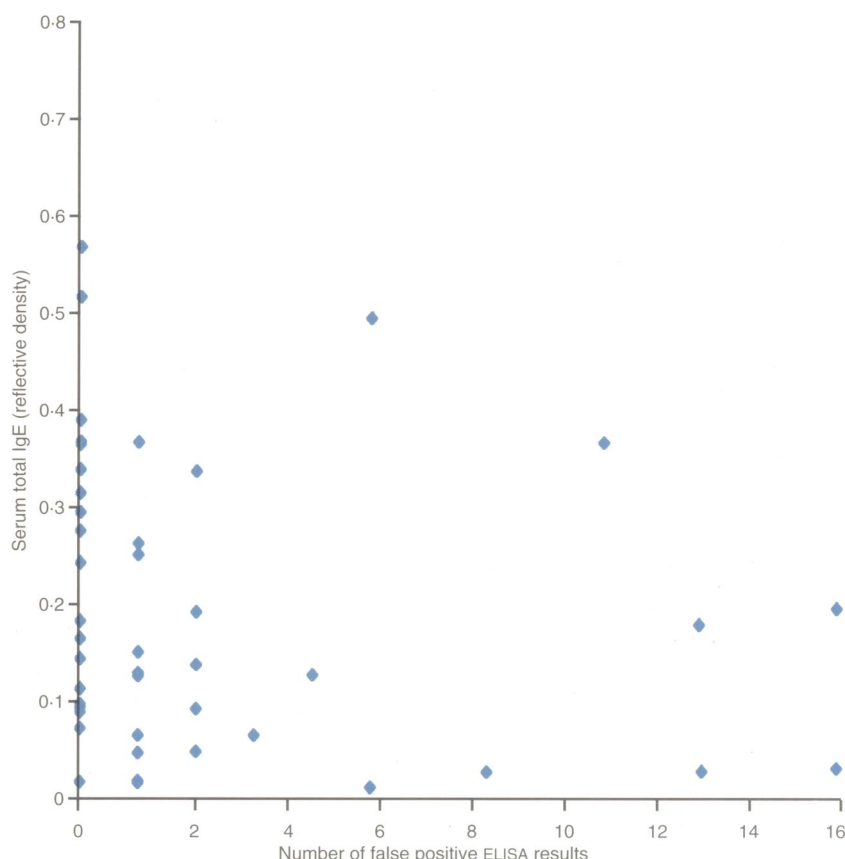


FIG 2: Serum total immunoglobulin E (IgE) reflective density as determined by Immunodot in relation to the number of false positive ELISA results in individual guide dogs

would be expected. The non-atopic greyhounds had significantly higher levels of serum total IgE than all the other groups. Greyhounds are known to be exposed to high levels of parasites (Jacobs 1978, Walker and Jacobs 1982) and it is possible that parasitism was involved in their production of IgE. It was not possible to count the parasite eggs

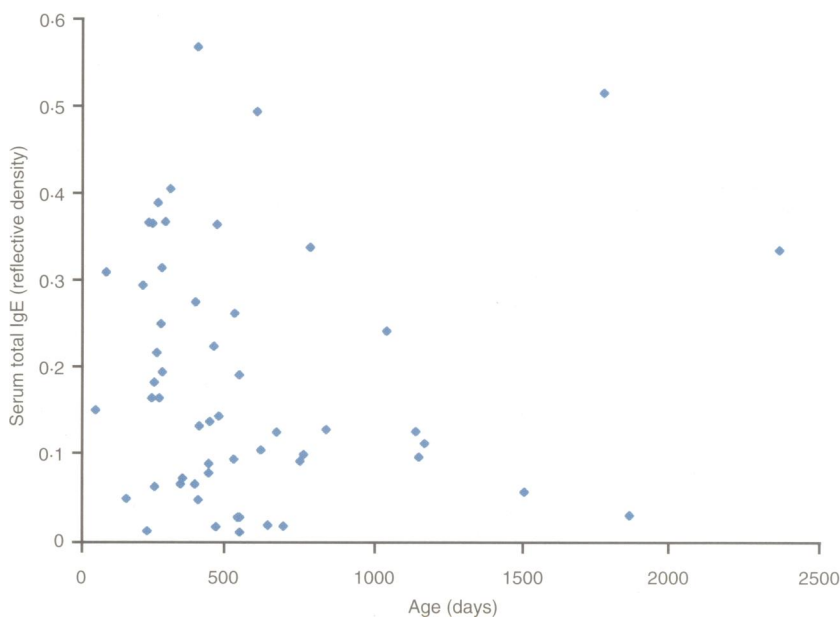


FIG 3: Serum total immunoglobulin E (IgE) reflective density of the non-atopic guide dogs in relation to their age

in the faeces of these dogs, but a low count does not prove that dog does not have a parasite burden. It is possible that dogs with latent larvae of *Toxocara canis* may have high serum IgE levels.

Fleas can be a problem in many greyhound kennels (M. Boyce, personal communication) and it is possible that this may have an influence on the dogs' serum total IgE concentrations. However, these greyhounds had no history of flea allergy. Moreover, Halliwell and others (1987) showed that dogs which were continually exposed to fleas had low levels of serum total IgE.

It is also possible that greyhounds may produce higher levels of serum total IgE than other breeds, and that because only 15 greyhounds were examined (in comparison with 55 non-atopic guide dogs), the high concentrations may have been a chance observation.

Griffin and others (1990) suggested that there may be a correlation between the serum total IgE concentration in non-atopic dogs and the number of so-called false positive ELISA results, that is, non-atopic dogs giving positive ELISA results. They suggested that high levels of serum total IgE would be more likely to induce non-specific binding of IgE to the test well, and a positive result. No such correlation was observed in this study, possibly because 43 non-atopic dogs were examined whereas Griffin and others examined only 15. However, Fig 2 indicates that so-called positive reactions can be shown by normal dogs with no clinical signs of atopic dermatitis. This result supports previous findings which suggested that serological tests cannot be used alone in the diagnosis of atopic dermatitis.

In non-atopic people, serum total IgE levels increase until 20 to 30 years of age (Johansson and others 1970). In the non-atopic dogs there was no such correlation with age, possibly because the high and low responders become differentiated when the dogs are very young, or because there were few samples from dogs less than six months of age. In contrast, Racine and others (1999) found that sex and age did significantly influence IgE levels up to four years of age, possibly because all their dogs belonged to similar genetic pools and could be classified as either low or high responders. In this study, the lack of correlation between serum total IgE concentrations and age may have been due to the mix of high and low IgE responders in the non-atopic group.

The results of this study show that dogs can have a wide range of serum total IgE concentrations, and that it was not possible to differentiate atopic and non-atopic dogs on the basis of their serum total IgE concentrations, even when they underwent rigorous parasite control measures. It therefore appears that serum total IgE concentrations cannot be used in the diagnosis of atopic dermatitis. There was no correlation between the serum total IgE concentrations and the incidence of positive serological results in the non-atopic dogs, and there was no correlation between the age and the serum total IgE concentrations of dogs up to three years of age.

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