

The efficacy of commercially available veterinary diets recommended for dogs with atopic dermatitis

Katharina Glos*, Monika Linekt,
Christine Loewenstein†, Ursula Mayer*
and Ralf S. Mueller*

*Medizinische Kleintierklinik, Ludwig Maximilian University, Munich, Germany

†Tierärztliche Spezialisten, Hamburg, Germany

‡Tierärztliche Klinik für Kleintiere, Bereich Dermatologie, Hofheim, Germany,

Correspondence: Ralf S. Mueller, Medizinische Kleintierklinik, Ludwig Maximilian University, Munich, Germany. E-mail: ralf.mueller@med.vetmed.uni-muenchen.de

Abstract

The classical treatments for dogs with atopic dermatitis have traditionally been oral antipruritic drugs, allergen-specific immunotherapy and topical therapy. Fifty dogs with atopic dermatitis were included in this multicentred, double-blinded, randomized study to compare clinical response to an 8-week period of feeding one of three commercial veterinary foods marketed for dogs with atopic dermatitis (diets A–C) or a widely distributed supermarket food (diet D). Atopic dermatitis was diagnosed using Willemse's criteria and through the exclusion of differential diagnoses. Fourteen dogs were assigned to diet A and 12 dogs each to diet B, C or D. Flea and tick control using a monthly fipronil spot-on product was administered for a minimum of 4 weeks prior to inclusion in the study and during the study period. Evaluations were made monthly. These included lesion scores, using an established scoring system (canine atopic dermatitis extent and severity index, CADESI-03) and owner evaluation of pruritus level using a visual analogue scale. After 8 weeks on the new diets, there was a significant improvement in CADESI and pruritus scores with diet B (Wilcoxon test, $P = 0.043$ and paired t -test, $P = 0.012$, respectively), in pruritus scores with diet A (paired t -test, $P = 0.019$) and in CADESI scores with diet D (Wilcoxon test, $P = 0.037$). No significant changes were detected with diet C. Based on the results of this study, in addition to the conventional therapies, changing the diet of dogs with atopic dermatitis may be a useful adjunctive therapeutic measure.

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Conflict of Interest

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Introduction

Canine atopic dermatitis is a common skin disease in small-animal practice.^{1,2} It is defined as a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic features and most commonly associated with IgE antibodies to environmental allergens.³ Atopic dermatitis can be treated symptomatically with anti-inflammatory drugs or with allergen-specific immunotherapy (ASIT). ASIT is to date the only specific treatment for canine atopic dermatitis.^{4,5} Symptomatic treatment comprises oral and topical glucocorticoids,⁶ cyclosporine A,^{6,7} antihistamines,^{8,9} fatty acid supplementation^{10,11} as well as topical treatment with antipruritic shampoos.^{6,12}

In humans with atopic dermatitis, polyunsaturated fatty acids (PUFA) have been reported to modulate the inflammatory response.^{13,14} Similarly, oral PUFAs decrease inflammation and pruritus in atopic dogs.^{15–17} Supplementation with n-6 fatty acids such as gamma-linoleic acid (GLA),¹⁸ n-3 fatty acids¹⁰ such as eicosapentaenoic acid (EPA), and combinations of GLA and n-3 fatty acids^{16,19} have been reported to be beneficial. In recent years, specific diets for allergic dogs with increased PUFA content have been developed. However, clinical response to these diets has been variable. One retrospective open study reported almost half of the patients showing a good or excellent response.²⁰ In contrast, in another prospective and double blinded study, n-3 fatty acid supplementation did not appear to have any added benefit on clinical signs when compared to thorough clinical management.²¹

The purpose of this multicentred, double-blinded, randomized study was to evaluate three veterinary diets marketed for allergic dogs and a diet widely distributed in supermarkets for their influence on the clinical signs of atopic dermatitis.

Materials and methods

Study participants

Fifty dogs with nonseasonal atopic dermatitis were included in this multicentred, double-blinded, randomized study. Atopic dermatitis was diagnosed using Willemse's criteria²² in addition to exclusion of differential diagnoses. At least three major and three minor criteria were needed for a diagnosis. Impression smears and skin scrapings ruled out secondary infections and ectoparasites as the dogs had to be free of dermatitis or otitis caused by microorganisms or ectoparasites at the time of study enrollment. Before inclusion in the study, 5 dogs were fed a home-prepared diet, 9 dogs a commercial diet for allergic dogs and 36 dogs an over-the-counter commercial food. Dogs were permitted to remain on medications prescribed prior to beginning the study as long as dose and frequency did not change in the 4 weeks immediately prior to and during the study period. Flea control started 4 weeks before study entry with monthly fipronil spot-on (Frontline®, Merial, Hallbergmoos, Germany) and was maintained throughout the study. ASIT initiated 1 year or more prior to the study was also permitted as long as the injection protocol was not changed during the study. Forty-two of the 50 dogs had been fed an elimination diet in the past without improvement. However, as the purpose of this study was not to evaluate the value of these diets as an elimination diet, but rather to evaluate any improvement on clinical signs of atopic dermatitis, a previous elimination diet was not regarded as a necessary inclusion criterion.

Exclusion criteria were veterinary health concerns and/or a concurrent disease. Dogs with known adverse reactions to fish, potatoes or venison were not included in the study. Dogs that were diagnosed with concurrent diseases necessitating treatment and pregnancy or likely pregnancy at time of inclusion in the study were also excluded.

Randomization

Dogs were assigned to one of four study diets in a double-blinded and randomized fashion. Randomization was carried out using a randomization table and dogs were assigned to their groups consecutively by the study supervisor, independent of the study site. Sites included three dermatology referral practices in Hamburg, Hofheim, and Munich, Germany, and two general practices near Munich, Germany.

Parameters evaluated

Dogs were evaluated both by owners and clinicians and each time by the same individual. Veterinary evaluation consisted of a physical examination and scoring of lesions on day 0 and at weeks 4 and 8 of the study using a previously established clinical scoring system for canine atopic dermatitis, the canine atopic dermatitis extent and severity index (CADESI-03).²³ In addition, veterinarians evaluated the coat regarding a number of criteria on a scale from 1 to 5 (dull/lustrous, patchy/full, rough/soft, dry/oily, thin/thick) and weighed the patients.

Owners were asked to evaluate the dogs' pruritus on day 0 and on weeks 4 and 8 of the study using a visual analogue scale (0–10), the left end representing a normal dog without any pruritus, the right end severe and constant pruritus. They also estimated the stool volume, the odour of the dogs' faeces and the dogs' flatulence on a scale from 1 to 5.

Diets

Diet A^a was based on fish, hydrolysed chicken and rice. Diet B^b contained fish and potato. Diet C^c was a diet with rice and hydrolysed chicken and soy. The control diet (diet D^d) was a widely distributed

over the counter food based on animal meat by products, poultry and cereals. All diets underwent analysis in an independent laboratory (Masterlab Analytical Services, Putten, Netherlands, Table 1). The food and the feeding guidelines were provided for the 8-week feeding period.

Withdrawal criteria

Dogs were removed from the study if they refused to consume the assigned diet for more than 24 h or if concurrent diseases with a need for treatment occurred. Moreover, dogs were withdrawn if they ate treats, table scraps or other foods during the study.

Laboratory testing

Blood samples were taken for a complete blood count (CBC) and serum biochemistry profile at the beginning and end of the study. Prothrombin time, activated partial thromboplastin time (aPTT) and thrombin time (TZ) were obtained for coagulation profiles at the same time.

Statistics

For descriptive statistics, median values and 95% confidence intervals were determined. CADESI and pruritus scores, weight and scores for additional evaluated parameters such as stool and coat qualities were tested with an analysis of variance (ANOVA) and Tukey post-test if the data were normally distributed; otherwise, a Kruskal–Wallis test and a Dunn's multiple comparison test were used. A paired *t*-test (with parametric data) or Wilcoxon test (with nonparametric data) was performed to compare the CADESI and pruritus scores between weeks 0 and 8 within each group. To identify any possible correlation between the CADESI and pruritus scores, a Spearman rank correlation coefficient was used. All data were evaluated using intention to treat analysis (ITT). In the ITT, dogs that completed the study and dogs withdrawn from the study were included in the analysis. The ITT analysis was performed to avoid bias towards the intervention. In this analysis, the scores of the last examination day were carried forward to subsequent examination time points for animals that terminated the study prematurely. Dogs that had undergone an elimination diet trial without improvement prior to the study were also evaluated separately. A Fisher exact test was used to compare the number of blood test results outside the normal range at the beginning (day 0) and end (week 8) of the study. Statistical analyses were performed with the program GRAPHPAD IN-STAT 3.06 from GraphPad Software, San Diego, USA. For all tests, *P* < 0.05 was considered significant.

Results

Study participants

Forty-three of the 50 enrolled dogs completed the study. Of the seven dogs withdrawn from the study, three dogs showed increased pruritus, erythema and papules and required additional treatment. These three dogs had negative cytology and skin scrapings at the withdrawal time. All three dogs improved when placed on their original diet, two of them did not receive any additional medication, one received concurrent trial antibiotic therapy. One of these dogs was eating a hydrolysed chicken and soy dog food before inclusion in the study, and was also on a hydrolysed chicken and soy diet (diet A) during the trial. The second dog received home-cooked food based on vegetables, horse, ostrich, pig and fish and deteriorated on fish and potato (diet B). The third dog received an over-the-counter commercial dog food based on beef, poultry, vegetables and cereals and deteriorated on the control diet (diet D). One dog was lost to follow-up; another dog refused to eat the assigned diet; one dog developed lethargy and one dog died of sepsis secondary to arthritis. The breed, age and sex of the 50 dogs are presented in Tables 2 and 3.

^aHill's prescription diet Canine d/d Salmon & Rice™, Hill's Pet Nutrition GmbH, Hamburg, Germany.

^bEukanuba Dermatitis F/P. Procter & Gamble Pet Care, Schwalbach, Germany.

^cRoyal Canin Hypoallergenic DR21, Royal Canin Nederland, BV, Netherlands.

^dPedigree Complete Maxi Chicken & Rice, Masterfoods, Verden, Germany.

Table 1. Analysis of diets used in the study

Analytical results	% of dry matter	Diet A	Diet B	Diet C	Diet D
Raw ash	%	4.3	8.1	7.9	6.6
Crude fibre	%	1.4	2.2	< 1.0	2.3
Moisture (4H)	%	7.7	6.9	6.6	8.6
Crude protein	%	15.2	23.2	22.6	21.0
Crude fat	%	13.9	13.0	17.2	12.7
Starch ewers	%	57.1	39.4	39.6	36.3
Calcium	%	0.71	1.25	1.11	1.31
Chloride	%	0.75	1.1	0.43	0.99
Phosphorus	%	0.57	0.95	0.87	0.87
Iron	mg kg ⁻¹	136	448	217	152
Potassium	%	0.69	1.61	0.98	0.56
Copper	mg kg ⁻¹	9	21	21	8
Magnesium	%	0.0912	0.11	0.0594	0.13
Manganese	mg kg ⁻¹	17	66	72	97
Sodium	%	0.3	0.45	0.42	0.65
Zinc	mg kg ⁻¹	160	258	272	214
Vitamin A	IU g ⁻¹	6.9	21	34	5.7
Vitamin E	mg kg ⁻¹	546	150	822	203
Saturated fatty acids	%	33.3	38.6	32.1	33.2
Unsaturated fatty acids	%	65.6	60.3	67.4	66.2
Mono unsaturated fatty acids	%	36.7	43.4	44.3	41.9
Poly unsaturated fatty acids	%	28.9	16.9	23.1	24.3
n-3 fatty acids (of fatty acids)	%	5.1	4.6	3.6	1.6
n-6 fatty acids (of fatty acids)	%	24.1	12.6	19.8	2.9
Arginine	%	0.99	1.29	1.62	1.13
Cystine	%	0.21	0.21	0.3	0.29
Phenylalanine	%	0.63	1.04	1.18	0.92
Glycine	%	1.03	1.34	0.96	1.53
Glutamic acid	%	2.1	3.12	4.02	3.77
Histidine	%	0.42	0.65	0.67	0.53
Iso-leucin	%	0.59	0.93	1.07	0.76
Leucin	%	1.06	1.64	1.81	1.77
Lysin	%	0.89	1.63	1.26	0.85
Methionine	%	0.55	0.65	0.81	0.36
Proline	%	0.72	0.92	1.08	1.72
Serine	%	0.71	0.97	1.16	0.92
Threonine	%	0.56	0.94	0.83	0.71
Tyrosine	%	0.56	0.78	1.12	0.71
Valine	%	0.78	1.2	1.18	0.98
Alanine	%	1.02	1.32	0.99	1.31
Aspartic acid	%	1.28	2.58	2.46	1.44
n-6 in total food	%	3.34	1.63	3.37	2.90
n-3 in total food	%	0.71	0.60	0.62	0.20
n-6/n-3		4.7 : 1	2.72 : 1	5.44 : 1	14.25 : 1
LA (18 : 2n-6)	%	23	11.3	18.7	21.6
GLA (18 : 3n-6)	%	< 0.1	< 0.1	< 0.1	< 0.1
DHGA or DGLA (20 : 3n-6)	%	0.1	0.2	0.1	0.1
AA (20 : 4n-6)	%	0.4	0.3	0.4	0.4
ALA (18 : 3n-3)	%	2.2	1.2	2.0	1.2
EPA (20 : 5n-3)	%	1.0	0.9	0.7	< 0.1
DPA (22 : 5n-3)	%	0.4	0.2	0.2	0.2
DHA (22 : 6n-3)	%	1.1	1.8	0.5	0.1
LC-PUFAs	%	2.9	3.2	1.8	0.8
Caloric value	Kcal kg ⁻¹	4842	4741	4892	4848

Twenty-two of the 50 dogs received concurrent therapy for their atopic dermatitis. This therapy was initiated more than 4 weeks and, in case of ASIT more than 1 year, prior to the dogs' inclusion in the study. Concurrent therapy included: ASIT (11), weekly shampoos (11), prednisolone at anti-inflammatory doses (8), antihistamines (5), cyclosporine (1).

Randomization

Fourteen dogs received diet A, and 12 dogs each, diet B, C and D. No significant differences (Kruskal–Wallis test)

among the groups regarding CADESI and pruritus scores as well as coat quality scores were observed at enrollment, indicating a successful randomization (data not shown).

CADESI and pruritus scores

The median CADESI and pruritus scores and confidence intervals for all diets are listed in Table 4. There was a significant improvement in CADESI and pruritus scores with diet B (Wilcoxon test, $P = 0.043$ and paired t -test, $P = 0.012$, respectively), in pruritus scores with diet A

Table 2. Breed distribution of dogs enrolled in the study

Breed	Number of dogs
Mix	14
German shepherd dog	6
Golden retriever	5
Labrador retriever	5
West Highland white terrier	3
Deutsche Wachtel	2
Magyar Vizsla	2
Boxer	2
Airdale terrier	1
Bearded collie	1
Dogo Argentino	1
Gordon setter	1
Hovawart	1
Husky	1
Jack Russel terrier	1
Kleiner Münsterländer	1
Rhodesian Ridgeback	1
Schnauzer	1
Total	50

(paired *t*-test, $P = 0.019$) and in CADESI scores with diet D (Wilcoxon test, $P = 0.037$). Exclusive evaluation of the 42 dogs without response to an elimination diet prior to the study revealed that CADESI scores did not change significantly with any of the diets, but pruritus scores improved significantly in the dogs receiving diets A (paired *t*-test, $P = 0.007$) and B (paired *t*-test, $P = 0.0137$). The Spearman rank correlation showed a significant correlation between the improvement of CADESI and pruritus scores for all dogs ($r = 0.423$; $P = 0.002$). Dogs receiving diets A and D showed a significant mean weight increase during the study (3.2 kg, 12%, paired *t*-test, $P = 0.039$ and 1.8 kg, 5%, Wilcoxon test, $P = 0.002$, respectively). Stool

volume increased (Wilcoxon test, $P = 0.016$) and the coat became more lustrous (paired *t*-test, $P = 0.0354$) in dogs receiving diet A. There was no significant ($P > 0.05$) change in flatulence, odour of faeces and any of the other evaluated coat parameters.

Laboratory testing

No consistent and marked abnormalities could be detected on complete blood count, serum biochemistry and coagulation profiles of any dog. There were no significant ($P > 0.05$; Fisher exact test) changes in regard to the number of dogs with abnormal values at the beginning (day 0) and at the end (week 8) of the study in any of the groups.

Discussion

In this double-blinded, randomized, multicentred study, four different commercial canine diets were evaluated. Three of these diets were specifically formulated for allergic dogs, one widely distributed supermarket food was chosen as a control. After 8 weeks, there was a significant improvement in pruritus and/or CADESI with some of the diets indicating that dietary modulation may be helpful in some dogs with atopic dermatitis.

Seven of 50 dogs did not complete the study. Three of these dogs were excluded for reasons not related to the diets or their clinical signs of atopic dermatitis. One dog did not eat diet A. Palatability of commercial diets is usually tested carefully.²⁴ However, individual preferences may lead to refusal of a particular diet as seen in this dog. Three additional dogs showed an increase in pruritus and erythema and developed an eruption characterized by small erythematous papules. Bacterial pyoderma is a common

Table 3. Ages and sexes of dogs enrolled in the study

	Diet A	Diet B	Diet C	Diet D
Age in years				
Median	7	5	6	5
Range	1–11	1–7	2–10	2–11
Sex*	3 FE, 5 FN, 1 ME, 5 MN	2 FE, 3 FN, 2 ME, 5 MN	3 FE, 5 FN, 4 ME, 0 MN	3 FE, 6 FN, 3 ME, 0 MN
Number enrolled on Day 0	14	12	12	12

*FE, entire female; FN, neutered female; ME, entire male; MN, neutered male.

Table 4. Descriptive data of CADESI and pruritus scores of dogs in the different diet groups before, four and 8 weeks after the study and *P*-values of comparisons between CADESI and pruritus scores before and after the study

	Week	Diet A (14 dogs)		Diet B (12 dogs)		Diet C (12 dogs)		Diet D (12 dogs)	
		Median	C.I.	Median	C.I.	Median	C.I.	Median	C.I.
CADESI (0–1220)	0	34.0	27–60	22.5	11–42	19.0	16–27	17.0	11–34
	4	33.0	21–48	8.0	3–32	24.0	15–31	17.5	9–23
	8	30.5	19–50	5.5	0–30	20.0	11–29	8.5	5–21
<i>P</i> -value		0.208		0.043		0.774		0.037	
Pruritus (0–10)	0	6.6	4.8–7.5	3.2	3.0–6.6	8.1	4.7–8.2	4.2	2.8–5.6
	4	5.2	3.7–7.1	2.8	1.7–5.3	6.3	4.1–7.6	6.0	3.6–7.8
	8	4.0	2.9–5.9	2.3	1.2–4.0	4.7	3.3–7.6	4.2	2.3–5.5
<i>P</i> -value		0.019		0.012		0.062		0.674	

Numbers in parenthesis indicate score range, C.I. = 95% confidence interval, < 0.05 was considered significant.

complication in atopic dermatitis,²⁵ but in these dogs, cytology evaluation was negative and two of them resolved the lesions without any antimicrobial therapy. It is possible that these dogs reacted to ingredients in their trial diet. Flares of atopic dermatitis resulting from changes in the environmental allergen load are also commonly seen²⁶ and are another explanation for the deterioration. The possibility of concurrent allergies is always a confounding factor in clinical trials.²⁶ All dogs in this study underwent uniform treatment for fleas, which began 4 weeks prior to the start of the diet. Although monthly application of fipronil would not be an adequate flea control in many places around the world, this type of flea control is typically adequate in the vast majority of cases in the temperate climate of southern Germany. In addition, no dog exhibited the classic tail base pruritus seen in flea-bite hypersensitivity. However, it is not possible to reliably rule out the influence of occasional flea bites on the pruritus of some dogs in this study.

Although the majority of dogs in the study underwent an elimination diet prior to inclusion, it is possible that some dogs with undiagnosed food adverse reaction were included. Thus, to avoid erroneous interpretation of results, those dogs in which an adverse reaction to the food was ruled out prior to inclusion in the study were analysed separately. Pruritus improved significantly in dogs receiving diets A and B. CADESI scores improved in dogs receiving diets B and D. However, this difference was not significant when only dogs were evaluated in which food adverse reaction was ruled out. Performing a statistical analysis on a smaller data set decreases the power of a study to detect a significant difference and is a possible explanation for the lack of significance in the difference of CADESI scores in the subgroup of dogs receiving diet B or D after a previous unsuccessful elimination diet. Of course, given the waxing and waning nature of atopic dermatitis, the improvement seen in some dogs may not have been the result of the diet, but rather environmental factors. Some of the dogs that did not undergo an elimination diet prior to enrolment could have improved as a result of diet changes addressing an adverse food reaction. However, in this case one would expect deterioration upon re-introduction of the previous diet, which did not occur in any of these dogs. Overall, diets A and B seem to decrease pruritus and diet B and D lesional scores in the atopic dogs included in this study.

It is possible, that seasonal changes could have caused an improvement in this 8-week trial. However, all dogs were essentially included in June, July and August of 2006 and the trial was concluded by late summer. Pollination during 2006 did not show prominent changes during this time, and the temperature and rainfall did not dramatically vary. In addition, one would expect that a climate-induced or seasonal improvement would have been seen in all of the groups.

In clinical studies, PUFA supplementation was shown to be an alternative to glucocorticoid therapy in the treatment of canine AD.¹⁰ Diets recommended for dogs with atopic dermatitis typically have a higher content of PUFA, which may contribute to the influence of such diets on allergic skin disease.²⁰ Proposed mechanisms of action include modulation of eicosanoid production, inhibition of cellular

activation and an increase in epidermal barrier function. EPA and Dihomo-gamma-linolenic acid (DHGLA) both compete with arachidonic acid as a substrate for 5-lipoxygenase and cyclooxygenase, which converts these fatty acids to leukotrienes and prostaglandins.^{11,27} Vaughn et al. reported a decrease of proinflammatory LTB-4 in favour of increased LTB-5 production in normal dogs with n-3 and n-6 enriched diets.²⁸ Subsequently, an omega-6 to omega-3 ratio of 5 : 1 was recommended. However, normal beagles and not dogs with atopic dermatitis were used in that study and lower ratios of 2 : 1 and 1 : 1 were not evaluated. It is therefore possible that a lower ratio may be more beneficial. In addition, although leukotriene inhibitors have been shown to be beneficial for human allergic rhinitis,²⁹ similar studies with leukotriene-inhibitors used in dogs with AD failed to demonstrate satisfactory efficacy.^{6,30,31} Thus, leukotrienes may not be crucial in the pathogenesis of atopic dermatitis in the dog, and effects of PUFA may not be the result of an influence on eicosanoid production.

Recently, a correlation between a filaggrin gene defect and atopic dermatitis was shown in humans.^{32,33} In addition, cytokine expression of lymphocytes in atopic skin may decrease filaggrin expression in keratinocytes.³⁴ Filaggrin (filament-aggregation protein) promotes the aggregation of keratin filaments and is thus instrumental for the barrier function of the stratum corneum. Abnormalities of the stratum corneum in dogs with atopic dermatitis have been demonstrated.³⁵ Percutaneous penetration of allergens leads to clinical signs in atopic dogs.³⁶ Oral fatty acids may alter the cutaneous fatty acid profile.³⁷ Increased concentrations of n-3 fatty acids may improve the epidermal barrier function of atopic dogs and thus improve clinical signs of canine atopic dermatitis.

PUFA can influence the immune system by acting as second messengers, regulators of signal transducing molecules or transcription factors.³⁸ They have been reported to suppress interleukin-2 production and mononuclear cell proliferation in humans. The decreased production of IL-1 and TNF- α was accompanied by a decreased ratio of arachidonic acid to EPA in the membrane phospholipids of mononuclear membrane.³⁹ In a recent study, it was shown that PUFA inhibit T-cell proliferation in dogs.⁴⁰ This may contribute to the clinical effects of PUFA supplementation. Thus, it is possible that improvement in dogs of this study was the result of the feeding of a good-quality fatty acid source.

Diet B showed a significant improvement of both CADESI and pruritus compared to baseline. This diet contained a relatively low n-6 : n-3 ratio. If benefits are indeed related to the fatty acid content of the diet, the improvement in group B may support lower ratios than 5 : 1, particularly for lesion scores such as CADESI. Calculation of n-6 : n-3 ratios may, however, be misleading and insufficient, as the long-chain fatty acids (LC-PUFAs) are more potent substrates for eicosanoid production than their shorter counterparts.⁴¹ For example, at a constant concentration of 18 : 2n-6, greater amounts of 18 : 3n-3 must be fed to achieve a similar effect regarding eicosanoid production than that obtained by using a corresponding amount of 20 : 3n-3. Additionally the amount of fatty acids in the diets varied. Diets A and B contained the highest percentage of EPA, DHA and LC-PUFAs and diet A the most

DPA. It is possible that this content of EPA, DHA and DPA was contributing to the clinical effects seen with these two diets.

The Spearman rank correlation showed a significant association between the CADESI and pruritus scores. This is most likely explained by the fact that most lesions of atopic dermatitis are the result of self-trauma and thus increased pruritus should lead to increased lesion scores. It confirms a previous study with allergic dogs showing a good correlation between pruritus and lesions.¹⁰

Nine dogs in the study had been through a commercial hydrolysed poultry-based elimination diet without any improvement. Hydrolysing proteins renders them less allergenic, but the degree of residual large proteins may be dependent on the particular allergen as well as on the degree of hydrolyzation.^{42,43} Although many animals seem to respond to hydrolysed diets,^{44,45} some dogs may still react to hydrolysed antigen.⁴⁶ It could have been possible that some dogs had a chicken allergy and the reason for their clinical improvement during the study was the switch to a chicken-free diet such as diet B. However, all four dogs that were originally fed an elimination diet with hydrolysed chicken as a protein source and which showed clinical improvement when fed the diet B were subsequently rechallenged. None of these four dogs deteriorated in the first 2 to 4 weeks on their old food after completion of the study. This may be due to the fact that the improvement was caused by different fatty acid composition of the test diet B. Clinical changes may be delayed for a few weeks as fatty acids are incorporated in the body and similarly may outlast a change to the previous food for a few weeks. Another possibility, supported by the analysis of dogs previously not responding to elimination diets, is a change in environmental allergen load in these atopic dogs.

Laboratory testing did not reveal any abnormalities on CBC, serum biochemistry and coagulation profile. In humans, diets rich in mixtures of saturated and unsaturated fats may raise the risk of occlusive thrombosis in the event of atheromatous plaque rupture.⁴⁷ However, two studies in dogs could not identify clinically relevant changes in haemostasis after fatty acid-supplemented diets for 8 weeks.^{48,49} Similarly, there was no negative influence on coagulation blood parameters measured in this study.

The clinical improvement observed in this study may also be the result of hitherto unidentified factors. Interactions between the many ingredients of the individual diets are difficult to evaluate and could have contributed to the results reported. In this study, the diets of four manufacturers were evaluated. Although protein and fat content of the diets was approximately the same based on analysis by an independent laboratory, there were subtle differences in certain minerals and vitamins as well as amino acids. In addition, the quality of ingredients is not evaluated by such an analysis and may also contribute to clinical effects.

Dogs receiving diets A and D showed a weight increase during the study, although all four diets had approximately the same caloric value. However, recommendations regarding the amount of food administered every day varied from diet to diet. The caloric intake of dogs on diet A and D according to the recommendations was 54–104% and 96% higher, respectively, than that of dogs receiving diet C. The caloric intake with diet B was 36–185% higher than that of diet C. Digestibility of diets also depends on

the concentration of unsaturated fatty acids in the diet. More unsaturated fatty acids lead to an increased digestibility. Diets A, C and D had approximately the same amount of unsaturated fatty acids.⁵⁰ The concentration of these fatty acids in diet B was 5% lower and may have contributed to the fact that despite a potentially higher caloric intake, the dogs did not gain weight. Stool volume increased with dogs receiving diet A. This correlates with the different feeding guidelines and the fact that these dogs were fed a higher amount of food than dogs with diet B. The food amounts of diets C and D were nearly the same.

The coat became more lustrous with dogs receiving diet A. This diet contains the highest amounts of PUFA, LA and ALA. In previous reports, the coat lustre increased with diets containing increased LA.⁵¹

In summary, feeding a special formulated canine skin diet may be helpful in decreasing pruritus and skin lesions in individual dogs with atopic dermatitis.

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Résumé Les traitements classiques de la dermatite atopique canine regroupent les molécules antiprurigineuses par voie orale, l'immunothérapie spécifique d'allergènes et les traitements topiques. Cinquante chiens souffrant de dermatite atopique ont été inclus dans cette étude randomisée, en double aveugle, multicentrique, pour comparer la réponse clinique obtenue en alimentant les chiens pendant huit semaines avec un parmi trois régimes commercialisés pour les chiens atopiques (régimes A–C) ou un aliment de supermarché (aliment D). La dermatite atopique était diagnostiquée en suivant les critères de Willemse et après exclusion des diagnostics différentiels. Quatorze chiens ont reçu l'aliment A et 12 chiens ont été inclus dans chaque autre groupe. Un contrôle insecticide et acaricide a été réalisé sur tous les chiens par l'application mensuelle d'une pipette de fipronil au moins un mois avant l'inclusion dans l'étude, et pendant toute l'étude. Les évaluations ont été faites tous les mois. Elles incluaient un score lésionnel, en utilisant le CADESI version 03 (Canine Atopic Dermatitis Extent and Severity Index) et l'évaluation par le propriétaire du prurit en utilisant une échelle analogique visuelle. Après huit semaines d'alimentation, une amélioration significative du CADESI et du score de prurit a été observée pour l'aliment B (Wilcoxon test, $P = 0.043$ et paired t test, $P = 0.012$, respectivement), pour le score de prurit avec l'aliment A (paired t test, $P = 0.019$) et pour le score CADESI avec l'aliment D (Wilcoxon test, $P = 0.037$). Aucune modification significative n'a été observée avec l'aliment C. En se basant sur les résultats de cette étude, l'utilisation d'un aliment spécialement formulé en association avec les traitements classiques peut être une mesure intéressante chez les chiens atopiques.

Resumen Los tratamientos clásicos frente a dermatitis atópica en perros han sido tradicionalmente fármacos antipruriginosos por vía oral, inmunoterapia específica de alérgeno y terapia tópica. Cincuenta perros con dermatitis atópica se incluyeron en un estudio al azar, doble-ciego y multicéntrico para poder comparar la respuesta clínica a tres dietas comerciales distribuidas para perros con dermatitis atópica y administrada durante ocho semanas (dietas A, B y C), comparada con otra dieta comercial ampliamente distribuida en supermercados (dieta D). La dermatitis atópica se diagnosticó utilizando los criterios de Willemse y tras la exclusión de otros diagnósticos diferenciales. Catorce perros se asignaron a la dieta A y doce a cada una de las dietas B, C o D. Se administró fipronil tópico mensualmente para el control de pulgas y ácaros al menos cuatro semanas previas a la inclusión en el estudio y durante el estudio. Se realizaron exámenes mensuales. Estos incluyeron la valoración de lesiones utilizando un sistema establecido (índice de extensión y severidad de dermatitis atópica canina, CADESI-03) y evaluación por el propietario del prurito utilizando una escala análoga visual. Tras ocho semanas en las nuevas dietas hubo una mejora significativa en los valores de CADESI y prurito con la dieta B (prueba de Wilcoxon, $P = 0.043$ y la prueba t -pareada, $P = 0.012$, respectivamente), en los valores de prurito con la dieta A (prueba t -pareada $P = 0.019$) y en los valores CADESI con la dieta D (prueba de Wilcoxon, $P = 0.037$). No se detectaron cambios significativos con la dieta C. Basado en los resultados de este estudio, además de las terapias convencionales, cambiar la dieta en los perros con dermatitis atópica puede ser una medida terapéutica adicional de utilidad.

Zusammenfassung Die klassischen Behandlungen für Hunde mit atopischer Dermatitis waren traditionsgemäß orale juckreizstillende Medikamente, Allergen-spezifische Immuntherapie und topische Therapie. Fünfzig Hunde mit atopischer Dermatitis wurden in diese Multicenter, doppelblinde, randomisierte Studie inkludiert, um die klinische Auswirkung einer acht-wöchigen Fütterungsperiode zu vergleichen. Es wurde eines von drei kommerziellen Veterinärfuttermitteln, die für Hunde mit atopischer Dermatitis am Markt sind (Diäten A–C) oder ein weitverbreitetes Supermarkt-Futter (Diät D) gefüttert. Die atopische Dermatitis wurde mittels Willemse's Kriterien und durch Ausschluss von Differentialdiagnosen diagnostiziert. Vierzehn Hunde wurden für Diät A und jeweils 12 Hunde für Diät B, C und D eingeteilt. Floh- und Zeckenkontrolle wurde mit einem monatlichen Fipronil Spot-on Produkt mindestens für 4 Wochen vor der Aufnahme in die Studie sowie während der Dauer der Studie durchgeführt. Es wurden monatliche Evaluierungen gemacht. Diese beinhalteten eine Bewertung der Läsionen unter Verwendung eines etablierten Bewertungssystems (Caniner Atopische Dermatitis Extent and Severity Index oder CADESI-03), sowie eine Evaluierung des Juckreizes mit einer visuellen analogen Skala durch den Besitzer. Nachdem acht Wochen lang die neuen Diäten gefüttert worden waren, bestand eine signifikante Verbesserung der CADESI und der Juckreiz Werte bei Diät B (Wilcoxon Test, $P = 0.043$ bzw. gepaarter t -Test, $P = 0.012$), der Juckreiz Werte bei Diät A (gepaarter t -Test, $P = 0.019$) und bei den CADESI Werten bei Diät D (Wilcoxon Test, $P = 0.037$). Mit Diät C wurden keine signifikanten Veränderungen beobachtet. Basierend auf den Ergebnissen dieser Studie kann man sagen, dass zusätzlich zu den konventionellen Therapien, eine Veränderung der Diät der Hunde mit atopischer Dermatitis eine hilfreiche zusätzliche therapeutische Maßnahme sein kann.