RESEARCH

Exploring the potential link between vegetable oil supplementation and adverse food reactions in dogs: a preliminary study

Elisa Maina[1*] and Eric Cox[2,3]

Abstract

Background Adverse food reactions (AFR) are a common cause of skin diseases in dogs, with a prevalence of up to 24%. Research in humans and mice has shown that a high intake of vegetable oils rich in omega-6 polyunsaturated fatty acids (n-6 PUFA) can increase the risk and severity of allergic diseases. This study aimed to investigate the association between AFR and the supplementation of vegetable oils in the canine diet.

Results A total of 459 dogs were analyzed, with atopic dermatitis (22.66%) and adverse food reactions (AFR) (8.06%) being the most common diagnoses. Oil supplementation was recorded in 14.16% of dogs, with extra-virgin olive oil being the most common (70.77%). Dogs with AFR and concurrent AFR and CAD showed significantly higher oil supplementation rates compared to other conditions ($P < 0.05$; $P < 0.001$). No significant association was found between oil type and diagnosis ($P = 0.563$). After excluding EFA-supplemented dogs and those on prescription dermo diets, oil supplementation was still more prevalent in dogs with AFR compared to other conditions (29.31% vs. 10.10%, $P < 0.001$).

Conclusions Vegetable oil supplementation was more frequent in dogs with AFR than in those with other dermatological conditions. This is the first study to evaluate the relationship between vegetable oil supplementation and AFR in dogs. While vegetable oil supplementation is associated with AFR, further studies are needed to establish causality and better understand the role of n-6 PUFA in the development of food allergies in dogs.

Keywords Adverse food reactions (AFR), N-6 Polyunsaturated Fatty Acids (PUFA), Canine Atopic Dermatitis (CAD), Vegetable Oils, Dietary Supplements, Essential Fatty Acids (EFA), Skin Diseases in Dogs, Omega-6/Omega-3 Ratio, Dietary Intake Survey

*Correspondence:
Elisa Maina
elisa.maina@unibe.ch
[1]Department of Clinical Veterinary Medicine, Division of Clinical Dermatology, Vetsuisse Faculty, University of Bern, Länggassstrasse 128, Bern CH-3012, Switzerland
[2]Department of Animal Health and Biomedical Sciences, SLU - Swedish University of Agricultural Sciences, Uppsala, Sweden
[3]Laboratory of Immunology, Faculty of Veterinary Medicine, Ghent University, Salisburylaan 133, Merelbeke 9820, Belgium

Background

Adverse food reactions (AFR) are defined as any clinically abnormal response caused by the ingestion of a food or food additive. These reactions can be categorized into immune-mediated food allergies and non-immune-based food intolerances. AFR is a common condition in dogs, with studies reporting that it is the underlying cause of skin diseases in up to 24% of affected dogs [1]. Similarly, AFRs are increasingly prevalent in humans, raising significant concerns for food safety [2]. However, measuring the true prevalence of AFR remains challenging, as many studies rely on self-reported data based on food allergy perceptions, often without the diagnostic gold standard, the oral food challenge (OFC). While food allergies are more common in children (3.9–8%) compared to adults (3.7%) [3–7], food intolerances and reactions to food additives affect a broader population, with prevalence estimates ranging from 5 to 20% [8–10].

The growing incidence of AFR in humans has prompted considerable research to understand its underlying causes [11–14]. Epidemiological studies suggest that environmental factors, such as changes in lifestyle, hygiene (hygiene hypothesis), and the timing and method of food exposure, may contribute to the increasing prevalence [15–17]. Other risk factors include co-existing conditions like atopic dermatitis, vitamin D insufficiency, oxidative stress, and changes in dietary fat intake, particularly the imbalance between omega-6 and omega-3 polyunsaturated fatty acids (PUFAs), which may exacerbate inflammatory responses [18–26]. Recent dietary shifts in Western countries, characterized by higher consumption of vegetable oils rich in omega-6 PUFAs and lower intake of omega-3-rich oily fish, may further contribute to this rise [27–29].

The impact of dietary n-6 and n-3 PUFAs on allergic diseases has been well studied in humans. Long-chain n-3 PUFAs, found in oily fish, have immunomodulatory effects that may reduce allergy risk, while omega-6 PUFAs, such as linoleic acid (LA) and arachidonic acid (AA), promote pro-inflammatory eicosanoids like PGE2, which increase IgE production and contribute to allergic responses [30–44]. A disrupted n-6/n-3 ratio is considered a critical factor in modulating immune responses, with excessive omega-6 intake linked to a higher risk of developing allergic diseases. Therefore, understanding the role of n-6 PUFA-rich oils in the diet of dogs with AFR could provide valuable insights into the potential causes and clinical implications of this condition.

While the prevalence of AFR in dogs varies widely across studies, environmental factors and differences in research methodology could contribute to these discrepancies. It remains unclear whether n-6 PUFA-rich dietary oils influence the risk of developing AFR in dogs. Notably, we observed a higher prevalence of AFR in Italy compared to other countries, possibly linked to cultural dietary practices where oil supplementation is common in dog diets [45–46]. The aim of this study was to investigate the association between oil supplementation and AFR in dogs, confirming our hypothesis that oil-enriched diets may contribute to the development of AFR in dogs.

Methods

Study population

The study included dogs presented by referral owners for dermatological examination at ten clinics in Northern Italy. Information was collected through dietary intake interviews and dermatological exams performed during initial consultations and follow-up visits. Additional details were gathered by phone when necessary. All dogs with a confirmed dermatological diagnosis were sequentially enrolled in the study. Dogs with unclear diagnoses were excluded. The diagnostic process for suspected allergic reactions involved ruling out ectoparasites (via skin scrapings, trichography, and coat brushing) and flea bite hypersensitivity (using isoxazoline treatment). Microbial infections were excluded through cytological examinations and cultures for bacterial and fungal infections, with antimicrobial treatment when required.

After excluding parasitic and microbial causes, dogs with allergic symptoms underwent a homemade elimination diet consisting of a single novel protein and carbohydrate source, based on the owner's dietary history. If a homemade diet with a novel protein and carbohydrate source was not feasible due to availability issues or owner preference, a commercial hypoallergenic diet (Anallergenic diet, Royal Canin) was used for at least eight weeks, with no additional foods or flavored supplements.

Dogs that showed improvement on the elimination diet underwent a provocation test. A relapse of clinical signs upon reintroduction of food indicated an adverse food reaction. Dogs that did not respond to the elimination diet but displayed clinical signs of atopic dermatitis were diagnosed as atopic dogs if they met the diagnostic criteria for canine atopic dermatitis (CAD).

This structured diagnostic approach ensured that only dogs with clear, definitive diagnoses were included, allowing for accurate differentiation between food-induced (AFR) and non-food-induced allergic conditions (CAD).

Survey

Dietary intake was assessed using a standardized questionnaire (Table 1), which gathered detailed information about the dog's daily diet, including the type of diet, ingredients, oil supplementation, and the timing of dietary changes in relation to the onset of clinical signs. The questionnaire included the following questions:

Table 1 Questionnaire about dietary intake posted to owners before the referral consultation

Category	Questions	Response options
Dietary type and changes	What type of diet is fed daily to the dog?	Commercial pet food, Home-made diet, Commercial and home-made diet
	Is the commercial diet a veterinary prescription diet or a maintenance diet?	Veterinary prescription diet, Maintenance diet
	Please, list all the ingredients in the dog's diet.	List
	Has the diet of your dog been changed before or after clinical signs onset?	Before, After
Oil supplementation	Do you add any oil to your dog's diet?	No, Yes
	Which type of oil do you add to your dog's diet?	Olive, Extra-virgin olive, Corn, Mix seeds, Sunflower
	How many teaspoons of oil do you add per ration?	1, 2, >2
	How often do you integrate oil into your dog's diet?	1 = A few times/year, 2 = A few times/month, 3 = A few times/week, 4 = Daily, 5 = Multiple times/day
	Did you start oil supplementation before or after the onset of clinical signs?	Before, After
EFA supplementation	Do you feed any dietary supplement based on EFA to your dog?	No, Yes
	Did you start the dietary supplement based on EFA before or after the onset of clinical signs?	Before, After

1. **Dietary type**: Was the diet commercial (veterinary prescription or maintenance diet), homemade (cooked or raw), or a combination of both?
2. **Diet composition**: A detailed list of the diet's ingredients, including the quantities and nutritional information.
3. **Dietary changes**: Was the diet modified before or after the onset of clinical signs?
4. **Oil supplementation**: What type of oil was used (e.g., extra-virgin olive, corn, sunflower)? How much was given (in teaspoons per meal)? How frequently was oil added (rated on a scale of 0–5, where 0 = never and 5 = multiple times per day)?
5. **Dietary supplements**: Were essential fatty acid (EFA) supplements included in the diet? If so, did supplementation begin before or after the onset of clinical signs?

Additional details regarding the dog's signalment, medical history, and previous treatments were also collected. Table 1 provides the full questionnaire and response options used during the study.

A specific numerical score (0–5) was assigned to different frequencies for oil integration: 0 = never, 1 = a few times/year, 2 = a few times/month, 3 = a few times/week, 4 = daily, 5 = multiple times/day.

To ensure accurate temporal associations, dogs whose oil supplementation began after the onset of clinical signs, as well as those whose diets were changed before the onset of clinical signs, were excluded from the analysis.

Given the hypothesis that excessive omega-6 fatty acid (n-6) intake may contribute to the onset of food allergies, particular attention was paid to the composition of oils used in the diets. For example, extra-virgin olive oil

has an n-6/n-3 ratio of approximately 13:1, while corn oil has a much higher ratio of 50:1. Other oils, such as sunflower oil (n-6/n-3 ratio around 40:1) and mixed seed oils (ranging from 20:1 to 30:1), also showed high levels of omega-6.

In a second analysis, dogs receiving EFA supplements and those fed prescription dermo diets rich in omega-3 (content > 0.6%) were excluded. The remaining dogs were then divided into two groups: those with AFR (including only AFR, AFR with CAD, and AFR with FAD (flea allergy dermatitis)) and those without AFR (all other non-allergic conditions). The prevalence of oil supplementation was then compared between these groups to assess any association with AFR.

Results

Study population

A total of 550 dogs were initially enrolled in the study, but 91 dogs were excluded due to the lack of a final diagnosis. The remaining 459 dogs, representing 69 different breeds, are summarized in Table 2. The sample included an equal number of males and females.

Diagnoses

A total of 50 different dermatological diagnoses were made across the 459 dogs. The most prevalent diagnoses were atopic dermatitis (CAD) (22.66%), adverse food reactions (AFR) (8.06%), primary superficial idiopathic pyoderma (7.84%), concurrent CAD and AFR (5.88%), demodicosis (5.88%), skin tumors (4.29%), and localized deep pyoderma (4.29%) (Table 2). The number and percentage of dogs receiving oil supplementation for each diagnosis are also shown in the table.

Table 2 Gender, age, weight, and breed of 459 dogs included in the study. Only dog breeds represented by three or more animals are listed

Category	Details	% of total dogs (459)	n
Gender	Females	47.49	218
	Females intact	17.74	86
	Males	52.51	241
	Males intact	44.23	203
Age	Mean ± SD = 6.07 ± 3.87 years (range: 5 months – 17 years)	-	-
Weight	Mean ± SD = 19.14 ± 12.97 kg (range: 1–38.5 kg)	-	-
Breed	Mongrel	26.58	122
	Labrador retriever	6.75	31
	German shepherd	5.45	25
	Dachshund	4.79	22
	Jack Russell terrier	4.14	19
	French bulldog	3.49	16
	Staffordshire bull terrier	3.27	15
	Miniature pinscher	2.83	13
	English bulldog	2.83	13
	Shih tzu	2.40	11
	Lagotto	1.96	9
	Boxer	1.96	9
	English cocker spaniel	1.74	8
	Bull terrier	1.74	8
	Golden retriever	1.53	7
	Chihuahua	1.53	7
	Breton	1.53	7
	Pug	1.53	7
	Poodle	1.53	7
	West Highland white terrier	1.31	6
	English setter	1.31	6
	Cavalier King Charles spaniel	1.31	6
	Yorkshire terrier	1.09	5
	Beagle	0.87	4
	Corso dog	0.87	4
	Argentine dogo	0.87	4
	Rottweiler	0.87	4
	Maltese	0.65	3
	Shar pei	0.65	3
	Pomeranian dog	0.65	3
	Pointer	0.65	3

SD, Standard deviation

Survey

Dietary intake data for the dogs is detailed in Table 3. Most dogs were fed commercial pet food (59.69%), while 31.37% received a combination of commercial and homemade diets, and 8.93% were exclusively fed homemade food. Of the commercial diets, 91.24% were maintenance diets, and 8.76% were veterinary prescription diets. Among the dogs fed homemade diets, rice (62.75%) was the most common carbohydrate source, followed by potatoes (5.66%) and wheat (4.79%). The primary protein

Table 2 Dermatological diagnoses of 459 dogs included in the study. Number and percentages of dogs supplemented with oil among animals with the same diagnosis

Diagnosis	Total n.	Oil n.	%
Canine atopic dermatitis (CAD)	104	15	14.4
Adverse food reactions (AFR)	37	11	29.7
Primary superficial idiopathic pyoderma	36	5	13.9
CAD + AFR	27	9	33.3
Demodicosis	27	4	14.8
Skin tumour	24	2	8.3
Localized deep pyoderma	24	3	12.5
Sarcoptes mange	13	1	7.7
Keratinization defects	13	1	7.7
Hormonal disease	12	1	8.3
Flea allergic dermatitis (FAD)	11	1	9.1
Hair follicle dysplasia	11	1	9.1
Otitis (no underlying cause)	9	1	11.1
Recurrent flank alopecia	9	1	11.1
Leishmaniasis	8	1	12.5
Pemphigus foliaceous	7	0	0
Dermatophytosis	6	1	16.7
Intertrigo	6	1	16.7
Metatarsal fistulae	6	0	0
Vasculitis	6	0	0
Pulicosis	6	1	16.7
Lupus complex	5	0	0
Acral lick dermatitis	4	1	25
Actinic keratosis	4	1	25
Cheyletiella mange	4	0	0
Non-neoplastic neoformation	4	0	0
Eosinophylic furuncolosis	3	0	0
Kerion	3	0	0
Reactive histiocytosis	2	0	0
Perianal fistulae	2	0	0
Hereditary cutaneous hyaluronosis	2	0	0
Adverse drug reaction	2	0	0
Alopecia X	2	0	0
Migratory necrolytic dermatitis	2	0	0
Behavioural disorders	2	0	0
Lupoid onychodystrophy	2	1	50
AFR + FAD	2	1	50
CAD + FAD	1	1	100
Phalangeal fracture	1	0	0
Trombiculosis	1	0	0
Vitiligo	1	0	0
Vogt-Koyanagi-Harada Disease	1	0	0
Zinc-responsive dermatosis	1	0	0
Panniculitis	1	0	0
Foreign body	1	0	0
Arthropods bite	1	0	0
Canine dermatomyositis	1	0	0
Viral papilloma	1	0	0
Dermanyssus gallinae	1	0	0
Total cases	**459**	**65**	**-**

Abbreviations: AFR, adverse food reactions; CAD, canine atopic dermatitis; FAD, flea allergic dermatitis; CAFR, canine adverse food reaction; n., number of dogs

Table 3 Dietary intake data of 459 dogs included in the study

Category	Subcategory	%	n
All dogs		**100**	**459**
Diet			
Dietary type	Commercial pet food	59.69	274
	Maintenance diet	91.24	250
	Veterinary prescription diet (all conditions)	8.76	24
	Veterinary prescription diet for skin problems	6.93	19
	Hydrolyzed veterinary prescription diet	0.73	2
	Home-made diet	8.93	41
	Commercial and home-made diet	31.37	144
	Maintenance diets	95.14	137
	Veterinary prescription diet (all conditions)	6.94	10
	Veterinary prescription diet for skin problems	6.94	10
	Hydrolyzed veterinary prescription diet	2.78	4
Diet changes before clinical signs onset	From commercial to home-made diet	5.01	23
	From home-made to commercial diet	2.44	11
Main protein/carbohydrate in the diet			
(Reported for ≥ 3 dogs)	Chicken	73.86	339
	Rice	62.75	288
	Beef	31.59	145
	Fish	31.37	144
	Lamb	5.66	26
	Potatoes	5.66	26
	Pork	4.79	22
	Wheat	4.79	22
	Turkey	3.49	16
	Duck	1.74	8
	Eggs	1.09	5
	Hydrolyzed food	1.09	5
	Dairy products	0.87	4
	Only vegetal	0.87	4
	Rabbit	0.65	3
Oil supplementation		14.16	65
Type of oil	Extra-virgin olive	70.77	46
	Corn	10.77	7
	Mix seeds	10.77	7
	Olive	4.62	3
	Sunflower	3.08	2
Amount of teaspoons of oil per ration	1	64.62	42
	2	30.77	20
	> 2	4.62	3
Frequency of oil supplementation	1 = A few times/year	0	0
	2 = A few times/month	13.84	9
	3 = A few times/week	13.31	8
	4 = Daily	50.77	33
	5 = Multiple times/day	23.08	15
Onset of oil supplementation/clinical signs	Supplementation before clinical signs	90.77	59
	Supplementation after clinical signs	9.23	6
Dietary supplements containing EFA		2.40	11
	Supplementation before clinical signs	18.18	2
	Supplementation after clinical signs	81.82	9

source was chicken (73.86%), followed by beef (31.59%), fish (31.37%), and lamb (5.66%).

Regarding oil supplementation, 14.16% of dogs received vegetable oils (which do not contain essential fatty acids, EFA) as a supplement, while only 2.40% received dedicated EFA-based supplements. Among those receiving EFA supplements, 81.82% started supplementation after the onset of clinical signs, minimizing its relevance in preclinical associations. In contrast, 90.77% of dogs that received vegetable oils began supplementation before the onset of clinical signs, suggesting a temporal association. The most commonly used oil was extra-virgin olive oil (70.77%), followed by corn oil (10.77%), mixed seed oil (10.77%), olive oil (4.62%), and sunflower oil (3.08%). Oil was supplemented at least once daily in 73.85% of cases.

Statistical analysis and grouping

After excluding dogs whose oil supplementation started after the onset of clinical signs (1.3%) and those whose diets changed before clinical signs (7.2%), the final analysis included 420 dogs. Among these, 58 dogs (13.81%) received oil supplementation: 9 of 33 (27.27%) dogs with AFR, 7 of 22 (31.81%) with concurrent AFR and CAD, 14 of 94 (14.89%) with CAD, and 45 of 385 (11.60%) with other conditions. The frequency of oil supplementation was significantly higher in dogs with AFR and concurrent AFR and CAD/FAD compared to those with other diagnoses ($P < 0.05$; $P < 0.001$, respectively) (Fig. 1). No significant association was found between oil supplementation and other dermatological conditions. Additionally, oil supplementation was not associated with a higher frequency of AFR across different dosage groups (group

1: 35%, group 2: 92%, group 3: 56%, group 4: 40%). There was no significant association between the type of oil used and the diagnoses ($P = 0.563$).

After excluding dogs receiving EFA supplementation (2.1%) and those fed prescription dermo diets (1.2%), the analysis was performed on 406 dogs. Among these, 58 dogs (14.3%) had AFR (including only AFR, AFR with CAD, and AFR with FAD) and 348 dogs (85.7%) had other diseases. The proportion of dogs with AFR who received oil supplementation was higher compared to those with other diseases (29.31% vs. 10.10%). This difference was statistically significant ($P < 0.001$) (Fig. 2). Additionally, 70.69% of dogs receiving oil supplementation had other diseases, compared to 88.22% of dogs not receiving oil supplementation, indicating a higher prevalence of non-allergic conditions in the latter group.

Discussion

The aim of this study was to explore the association between vegetable oil inclusion in the diet and the occurrence of adverse food reactions (AFR) in dogs. Our findings suggest a significant association between vegetable oil supplementation and an increased prevalence of AFR in this population. To the best of the authors' knowledge, this is the first veterinary study specifically addressing the relationship between oil supplementation and AFR in dogs. While extensive human literature has examined the effects of vegetable oil, particularly in relation to allergic diseases, such studies in veterinary medicine remain scarce.

In humans, high dietary intake of margarine and vegetable oils rich in n-6 polyunsaturated fatty acids (PUFA)
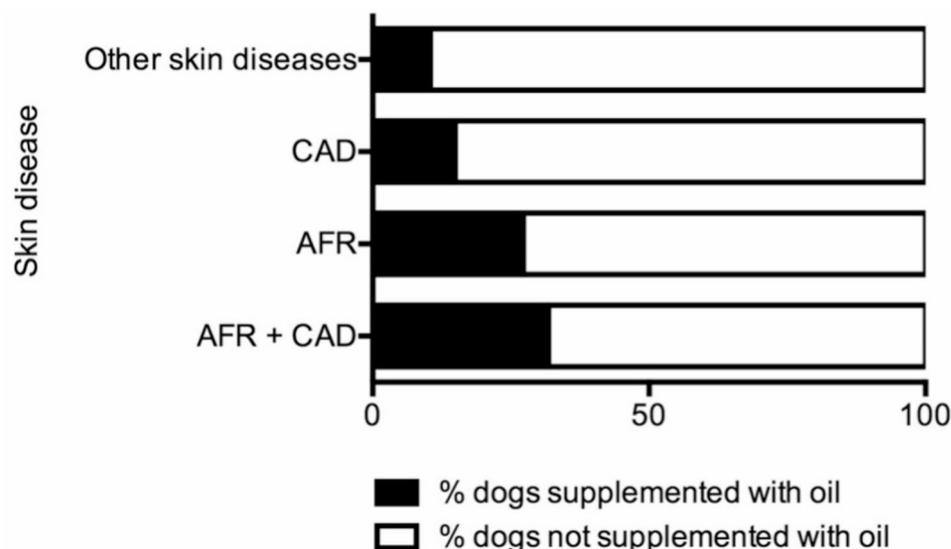
Fig. 1 Percentages of dogs supplemented with vegetable oil among animals with AFR, concurrent AFR and CAD, CAD, and other skin diseases. The percentages are based on the following numbers of dogs supplemented with vegetable oil: 11 out of 37 dogs with AFR (29.7%), 9 out of 27 dogs with concurrent AFR and CAD (33.3%), 15 out of 104 dogs with CAD (14.4%), and 30 out of 291 dogs with other skin diseases (10.3%). Only dogs supplemented with vegetable oil before the onset of clinical signs are included in the graphic. The total number of dogs under each condition is reported in Table 3
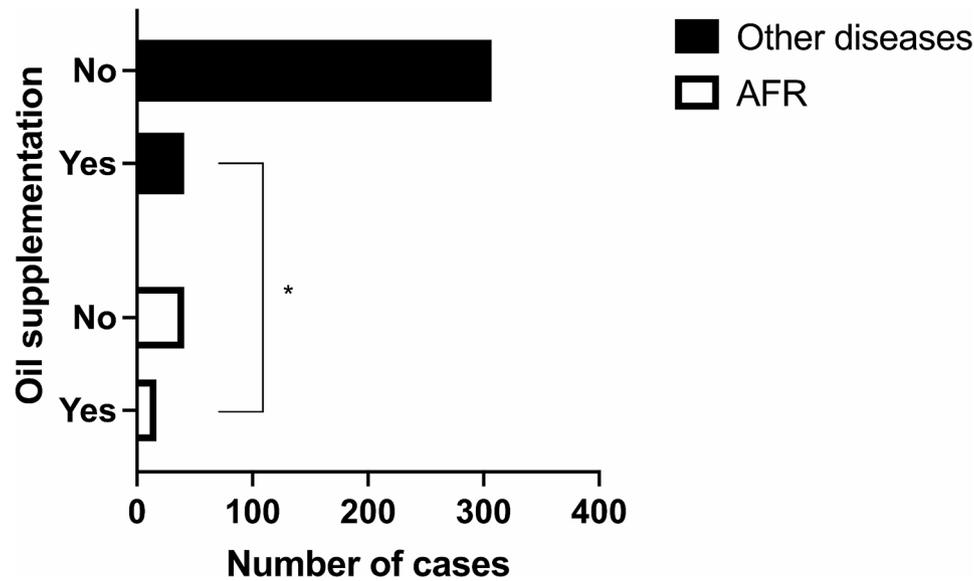
Fig. 2 Distribution of cases by oil supplementation and diagnosis (AFR (white bars) vs. other diseases (black bars). The bar chart shows the number of cases of AFR and other diseases based on oil supplementation. The asterisk (*) indicates a statistically significant difference ($p = 0.0010$): The comparison reveals that the prevalence of AFR is significantly higher in the group receiving oil supplementation

has been linked to an increased incidence of asthma, allergic rhinitis, eczema, and allergic sensitization [47–53]. N-6 PUFA intake is known to enhance the risk of atopic diseases in children compared to non-atopic children [54]. While the potential impact of n-6 PUFA-rich vegetable oils on the development of food allergies in humans remains unclear, recent studies have shown that increased consumption of n-6 PUFA-rich oils can suppress tolerance induction and exacerbate allergic responses in a murine model of cow's milk allergy [55]. N-6 PUFA may influence allergic disease by promoting the formation of inflammatory mediators [56]. Specifically, linoleic acid, a precursor of arachidonic acid, leads to the production of pro-inflammatory eicosanoids, including prostaglandin E2 (PGE2) and prostaglandin D2 (PGD2). Diets high in omega-6 oils disturb the omega-6/omega-3 ratio, tipping the balance towards pro-inflammatory mediators and resulting in increased systemic inflammation. Omega-3 fatty acids, which have anti-inflammatory properties, compete with arachidonic acid for the same enzymes (cyclooxygenase and 5-lipoxygenase), thus mitigating the inflammatory response when in balance. Disruption of this balance, particularly an increase in omega-6, promotes a stronger inflammatory response, activates dendritic cells, and shifts T-helper cell responses from type 1 to type 2. This imbalance may, in turn, increase the incidence of IgE-mediated allergic diseases [27, 43, 57–60]. It has also been demonstrated that n-6 PUFA decreases the IFNg/IL4 ratio in mice and humans [61]. N-6 PUFA acts mainly by increasing the humoral response against allergens.

Interestingly, while n-6 PUFA has been implicated in the exacerbation of allergic diseases, our study did not find an association between n-6 PUFA and canine atopic dermatitis (CAD). This may be due to the lack of enzymatic activity of D6- and D5-desaturase in the epidermis of atopic dogs, making them less susceptible to the effects of n-6 PUFA supplementation [62–64]. Additionally, the sensitization mechanisms may differ between AFR and CAD. In CAD, epicutaneous sensitization is more critical, while food allergens primarily sensitize via the gastrointestinal tract [65–66]. Furthermore, dietary long-chain triglycerides (LCT), such as n-6 PUFA, can slow gastric emptying and prolong allergen exposure to the immune system [67]. This prolonged exposure might increase the dose of allergens presented to the immune system, promoting the development of sensitization.

Moreover, LCTs may enhance the chylomicron-dependent absorption of food proteins and bacterial lipopolysaccharides (LPS) through mesenteric lymph nodes, which play a crucial role in oral tolerance induction [68]. LPS is a potent immune activator and could contribute to T- and B-cell activation, further enhancing allergic sensitization [69]. Additionally, chylomicrons can be phagocytosed by macrophages, facilitating antigen uptake [70–71]. These mechanisms suggest that n-6 PUFA-rich oils in the diet might enhance allergic responses by increasing the sensitivity of mast cells, possibly without increasing IgE levels.

Our study also revealed that the prevalence of AFR was higher in dogs fed veterinary prescription diets compared to those on maintenance diets, and this association was noted before the onset of clinical signs. Veterinary

prescription diets are often enriched with essential fatty acids (EFAs), including both n-6 and n-3 PUFAs, which are commonly used to manage inflammatory and dermatological conditions. However, since the exact EFA composition of these diets was not analyzed through advanced laboratory techniques, the precise concentrations of omega-6 and omega-3 fatty acids in the diets were not determined, making this hypothesis speculative. Commercial maintenance diets are typically formulated to meet basic nutritional needs and may contain lower levels of EFA compared to prescription diets, which are specifically designed for clinical conditions.

Homemade diets and diets supplemented with oils can vary widely in EFA content, depending on the ingredients and types of oils used. Although the decision to feed veterinary prescription diets to clinically healthy dogs could be influenced by factors such as breed predisposition to allergies, breeder recommendations, or early subtle clinical signs not yet diagnosed as AFR, this hypothesis cannot be confirmed due to the lack of detailed dietary composition and feeding behavior data.

This study's findings emphasize the need for future research to analyze the EFA content in both prescription and maintenance diets to better understand their potential relationship with AFR. While our results suggest a significant association between prescription diets and AFR, the causal relationship has yet to be established.

Another limitation of this study is the heterogeneous distribution of oils used in the diets. Olive oil was the most commonly used oil, though not exclusively. This variability might have influenced the results, but it also offers an intriguing perspective. Despite the predominance of olive oil, which has been linked to anti-allergic effects in murine models of ovalbumin (OVA)-induced sensitization, our study did not observe a protective effect of olive oil in dogs. In the study by Yu Ma et al., olive oil supplementation alleviated allergic symptoms in sensitized mice by increasing IL-10 levels and reducing Th2 cell-associated factors [72]. However, our findings in dogs did not support these results, suggesting that the anti-allergic effects observed in mice and humans may not directly translate to dogs.

Moreover, although studies suggest that olive oil may slow gastrointestinal transit, this could potentially influence allergen exposure time in the immune system, which might increase the risk of allergic reaction [73–75]. A slower gastrointestinal transit could enhance allergen contact with the immune system, contributing to the development of food allergies. However, this remains speculative and warrants further investigation.

Finally, the study population consisted of dermatological referral cases, which may not represent the general canine population, limiting the generalizability of these results. Future studies should include healthy dogs and dogs with non-dermatological conditions to assess the broader impact of oil supplementation across different populations.

Despite these limitations, our study provides valuable insights into the potential association between vegetable oil supplementation and AFR in dogs, highlighting the need for further research to establish causal relationships and explore the underlying mechanisms.

Conclusion

Despite its exploratory nature, this study offers valuable insights into the potential link between vegetable oil supplementation and adverse food reactions (AFR) in dogs. It is important to note that these findings only establish an association and do not confirm a causal relationship. As such, the results should be interpreted with caution.

Nonetheless, the observed associations underscore the need for more in-depth research to clarify the role of vegetable oils and essential fatty acids in the development or exacerbation of AFR. Future studies should focus on analyzing the specific composition and dosage of oils, as well as their interactions with other dietary components. Such research will be crucial to confirm or refute these preliminary findings and will help guide dietary recommendations for dogs with dermatological conditions.

Abbreviations

AFR	Adverse Food Reactions
CAD	Canine Atopic Dermatitis
FAD	Flea Allergic Dermatitis
CAFR	Canine Adverse Food Reaction
EFA	Essential Fatty Acids
PUFA	Polyunsaturated Fatty Acids
n-6 PUFA	Omega-6 Polyunsaturated Fatty Acids
n-3 PUFA	Omega-3 Polyunsaturated Fatty Acids
OFC	Oral Food Challenge
LCT	Long Chain Triglycerides
MLN	Mesenteric Lymph Nodes
LPS	Lipopolysaccharides
SPSS	Statistical Package for the Social Sciences
SD	Standard Deviation
$kg^{0.75}$	Metabolic Weight (used to normalize energy requirements)
PGE2	Prostaglandin E2
PGD2	Prostaglandin D2
IFNg	Interferon-gamma
IL4	Interleukin-4

Data availability
The data that support the findings of this study are not openly available due to reasons of sensitivity and are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The owners of dogs interviewed in this study were part of the study approved by the Ethical Committee of Ghent University, Belgium (EC 2013/189 for healthy dogs and EC 2013/198 for allergic dogs) and by the Deontological Committee of the Belgian government (232663/13_11_2/14). This study involved gathering information from clients regarding their dogs' health and perspectives and is not considered experimental. Therefore, no additional ethical approval was required. The animals were treated in accordance with high ethical standards and national legislation. Informed consent to participate in the study was obtained from all clients, who were fully informed about the nature and purpose of the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

References
1. Olivry T, Mueller RS. Evidence-based veterinary dermatology: a systematic review of the treatment of canine atopic dermatitis. Vet Dermatol. 2016;27:105–14.
2. Nwaru BI, Hickstein L, Panesar SS, Muraro A, Werfel T, Cardona V, et al. Prevalence of common food allergies in Europe: a systematic review and meta-analysis. Allergy. 2014;69:992–1007.
3. Branum AM, Lukacs SL. Food allergy among children in the united States. Pediatrics. 2009;124:1549–55.
4. Gupta RS, Springston EE, Warrier MR, Smith B, Kumar R, Pongracic J et al. The prevalence, severity, and distribution of childhood food allergy in the united States. Pediatrics. 2011;128.
5. McGowan EC, Keet CA. Prevalence of food allergies in children and adults in the united States. JAMA. 2013;309:2574–5.
6. Skypala IJ, Venter C, Meyer R, de Jong NW, Fox AT, Groetch M, et al. The prevalence of pollen food syndrome and food allergy in adults in the UK. Clin Transl Allergy. 2011;1:11.
7. Venter C, Laitinen K, Vlieg-Boerstra B. Nutritional aspects in diagnosis and management of food hypersensitivity—the dietitian's role. J Allergy Clin Immunol Pract. 2016;4:33–8.
8. Hodge L, Salome CM, Peat JK, Haby MM, Xuan W, Woolcock AJ. Consumption of oily fish and childhood asthma risk. Am J Respir Crit Care Med. 1998;157:1018–22.
9. Lessof MH, Wraith DG, Merrett TG, Merrett J, Buisseret PD. Food allergy and intolerance in 100 patients—local and systemic effects. QJM. 1983;52:361–74.
10. Zopf Y, Baenkler HW, Silbermann A, Hahn EG, Raithel M. The differential diagnosis of food intolerance. Dtsch Arztebl Int. 2009;106:359–69.
11. Kool B, Ameratunga S, Walker N, Jackson R, Lee A, Robinson E. The incidence of acute food allergy reactions in new Zealand. Clin Exp Allergy. 2016;46:548–56.
12. Mullins RJ, Dear KBG, Tang MLK. Characteristics of childhood peanut allergy in the Australian capital territory, 1995 to 2007. J Allergy Clin Immunol. 2009;123:689–93.
13. Rudders SA, Banerji A, Vassallo MF, Clark S, Camargo CA Jr. Trends in pediatric emergency department visits for food-induced anaphylaxis. J Allergy Clin Immunol. 2010;126:385–8.
14. Turner PJ, Baumert JL, Beyer K, Boyle RJ, Chan CH, Clark AT, et al. Can we identify patients at risk of life-threatening allergic reactions to food? Allergy. 2016;71:1241–55.
15. Kotz D, Simpson CR, Sheikh A. Incidence, prevalence, and trends of general practitioner-recorded diagnosis of food allergy in the united Kingdom. J Allergy Clin Immunol. 2011;127:623–30.
16. Lack G, Penagos M. Long-term relationship between peanut allergy and sensitization to Ara h 2 and Ara h 6 in an unselected cohort. J Allergy Clin Immunol. 2013;132:1444–6.
17. Untersmayr E, Vestergaard H, Malling HJ, Wöhrl S, Bindslev-Jensen C, Skov PS, et al. Incomplete digestion of codfish represents a risk factor for anaphylaxis in patients with allergy. J Allergy Clin Immunol. 2008;122:295–301.
18. Brough HA, et al. Epicutaneous sensitization in the development of food allergy: what is the evidence and how can this be prevented? Allergy. 2020;75:2185–205.
19. Oyoshi MK, et al. Epicutaneous sensitization to food allergens in atopic dermatitis: pathophysiology and clinical implications. J Allergy Clin Immunol. 2017;140:1602–8.
20. Nakamura T, et al. Role of skin and gut microbiota in the pathogenesis of atopic dermatitis and food allergy. Clin Exp Allergy. 2021;51:4–11.
21. Allen KJ, et al. Vitamin D insufficiency is associated with challenge-proven food allergy in infants. J Allergy Clin Immunol. 2013;131:1109–16.
22. Devereux G. The increase in the prevalence of asthma and allergy: food for thought. Nat Rev Immunol. 2006;6:869–74.
23. Untersmayr E, Jensen-Jarolim E. The role of protein digestibility and antacids in food allergy. Curr Opin Allergy Clin Immunol. 2006;6:214–9.
24. Lu KD, et al. Obesity, asthma, and the microbiome: the role of systemic inflammation. Pediatr Allergy Immunol Pulmonol. 2016;29:104–11.
25. Calder PC, et al. Fatty acids and inflammation: the cutting edge between food and medicine. Eur J Clin Nutr. 2017;71:1–8.
26. Visness CM, London SJ, Daniels JL, Kaufman JS, Yeatts KB, Siega-Riz AM, et al. Association of obesity with IgE levels and allergy symptoms in children and adolescents: results from the National health and nutrition examination survey 2005–2006. J Allergy Clin Immunol. 2009;123:1163–9.
27. Black PN, Sharpe S. Dietary fat and asthma: is there a connection? Eur Respir J. 1997;10:6–12.
28. Devereux G. Early life events in asthma: an update. Acta Paediatr Suppl. 2006;95:11–4.
29. Devereux G, Turner SW, Craig LCA, McNeill G, Martindale S, Harbour PJ, et al. Low maternal vitamin E intake during pregnancy is associated with asthma in 5-year-old children. Am J Respir Crit Care Med. 2005;171:121–8.
30. Farjadian S, Moghtaderi M, Nasiri M, Shakiba AM, Kashef S. The role of omega-3 and omega-6 fatty acids in asthma and allergic diseases. Allergol Int. 2016;65:215–22.
31. Furuhjelm C, Warstedt K, Larsson J, Fredriksson M, Böttcher MF, Fälth-Magnusson K, et al. Fish oil supplementation in pregnancy and lactation May decrease the risk of infant allergy. Acta Paediatr. 2009;98:1461–7.
32. Horrobin DF. Nutritional and medical importance of gamma-linolenic acid. Prog Lipid Res. 1992;31:163–94.
33. Jenmalm MC, Duchen K. Timing of allergy-preventive and Immunomodulatory dietary interventions—are prenatal, perinatal or postnatal strategies optimal? Clin Exp Allergy. 2013;43:273–8.
34. Makino S, Kato T, Ito K, Kashiwabara Y, Shirakawa T. Enhancement of natural killer cell activity in male students with dietary supplementation of fish oil fatty acids—a randomized, double-blind, placebo-controlled trial. J Nutr Sci Vitaminol (Tokyo). 2005;51:252–6.
35. Miles EA, Calder PC. Modulation of immune function by dietary fatty acids. Proc Nutr Soc. 2015;54:591–602.
36. Muehleisen B, Gallo RL. Vitamin D in allergic disease: shedding light on a complex problem. J Allergy Clin Immunol. 2013;131:324–9.
37. Nagakura T, Matsuda S, Shichijyo K, Sugimoto H, Hata K. Dietary supplementation with fish oil rich in omega-3 polyunsaturated fatty acids in children with bronchial asthma. Eur Respir J. 2000;16:861–5.
38. Okamoto M, Mitsunobu F, Ashida K, Mifune T, Hosaki Y, Tsugeno H, et al. Effects of dietary supplementation with n-3 fatty acids compared with n-6 fatty acids on bronchial asthma. Intern Med. 2000;39:107–11.
39. Palmer DJ, Sullivan T, Gold MS, Prescott SL, Heddle R, Gibson RA, et al. Randomized controlled trial of fish oil supplementation in pregnant women to reduce the risk of allergic disease in their children. Pediatr Allergy Immunol. 2013;24:513–21.
40. Van den Elsen LWJ, van Esch BCAM, Hofman GA, Kant J, Harmsen HJM, Willemsen LEM, et al. Dietary long chain n-3 polyunsaturated fatty acids prevent allergic sensitization to cow's milk protein in mice. Clin Exp Allergy. 2013;43:798–810.

41. Wendel T, Willich SN, Balzer F. Omega-3 fatty acids and cardiac arrhythmias: preventive potential and possible mechanisms. Proc Nutr Soc. 2014;70:193–201.

42. Calder PC, Miles EA. Fatty acids and atopic disease. Pediatr Allergy Immunol. 2000;11:29–36.

43. Kankaanpää PE, Seppänen-Laakso TJ, Smit J, Hammarén JP, Räsänen L, Schmidt E, et al. Increased Linoleic acid and decreased α-linolenic acid in atopic eczema patients compared with controls. Br J Dermatol. 1999;141:72–8.

44. Simopoulos AP. Importance of the omega-6/omega-3 balance in health and disease: evolutionary aspects of diet. World Rev Nutr Diet. 2011;102:10–21.

45. Maina E, Galzerano M, Noli C. Perianal pruritus in dogs with skin disease. Vet Dermatol. 2014;25:204–9.

46. Proverbio D, Perego R, Spada E, Ferro E. Prevalence of adverse food reactions in 130 dogs in Italy with dermatological signs: a retrospective study. J Small Anim Pract. 2010;51:370–4.

47. Bolte G, Frye C, Hoelscher B, Meyer I, Wjst M, Heinrich J. Margarine consumption and allergy in children. Am J Respir Crit Care Med. 2001;163:277–9.

48. Haby MM, Peat JK, Marks GB, Woolcock AJ, Leeder SR. Asthma in preschool children: prevalence and risk factors. Thorax. 2001;56:589–95.

49. Nagel G, von Mutius E, Haider D, Weinmayr G, Björkstén B, Buchele G, et al. Effects of dietary intake of n-3 and n-6 fatty acids on hay fever, asthma, and allergic sensitization in children. J Allergy Clin Immunol. 2005;115:1202–8.

50. Sausenthaler S, Kompauer I, Borte M, Herbarth O, Schaaf B, Berg A, et al. Margarine and butter consumption, eczema and allergic sensitization in children. The LISA birth cohort study. Allergy. 2006;61:1151–7.

51. Trak-Fellermeier MA, Brasche S, Winkler G, Heinrich J. Food and fatty acid intake and atopic disease in adults. Eur Respir J. 2004;23:575–82.

52. Von Mutius E, Weiland SK, Fritzsch C, Duhme H, Keil U. Increasing prevalence of hay fever and atopy among children in Leipzig, East Germany. Lancet. 1998;351:862–6.

53. Winkler G, Brasche S, Heinze S, Heinrich J. Dietary intake of fatty acids and selected foods in relation to hay fever, specific and total serum IgE. Allergy. 1992;47:265–9.

54. Dunder T, Kuikka L, Turtinen J, Räsänen L, Uhari M. Diet, serum fatty acids, and atopic diseases in childhood. Allergy. 2001;56:425–8.

55. Van den Elsen LWJ, Garssen J, Willemsen LEM. Long chain n-3 polyunsaturated fatty acids in the prevention of allergic and cardiovascular disease. Curr Pharm Des. 2012;18:2375–92.

56. Wendell S, Halling A, Nilsson TK, Olsson R, Nyberg F. Omega-3 fatty acids reduce the risk of coronary heart disease and allergic disease in adults. Eur J Clin Nutr. 2014;68:277–82.

57. Gosset P, Janin A, Bureau F, Ang D, Croci C, Flamand V, et al. Immune response and transcription factor activities in bronchial epithelial cells exposed to organic dust extracts from swine confinement buildings. J Immunol. 2005;175:1051–63.

58. Kompauer I, Demmelmair H, Koletzko B, Heinrich J. Association of fatty acids in serum phospholipids with hay fever, specific and total Immunoglobulin E. Br J Nutr. 2004;91:91–9.

59. Prescott SL, Jenner PJ, Dunstan JA, Simmer K, Palmer DJ, Hiller J, et al. Role of Proinflammatory fatty acids in the development of allergic disorders. Pediatr Allergy Immunol. 2004;15:90–3.

60. Theiner S, Siegl U, Heinzl G, Wilders-Truschnig M, Truschnig-Wilders M, Körmöczi G, et al. Immunophenotyping of lymphocytes in blood of patients with food intolerance: evaluation of a whole blood method. Clin Exp Allergy. 2006;36:1482–9.

61. Mizota T, Takahashi K, Tokuyama K, Noma H, Kaneda M, Shibata H, et al. Dietary intake of n-6 polyunsaturated fatty acids reduces interferon-γ production by T cells in asthmatic patients. Eur Respir J. 2009;34:983–90.

62. Schlotter YM, Vroom MW, van Schaik S, Rutten VP, Bruijnzeel-Koomen CA, Willemse T. A prospective study of the effect of a standard oligoantigenic diet on the frequency and nature of food intolerance in dogs. J Small Anim Pract. 2008;49:75–8.

63. Campbell WC, Faure M, Germain P, Novak N, Roth MP, Torres-Almanas A. A prospective study of fish oil and asthma in 18,533 male participants. Allergy. 1993;48:292–8.

64. Fuhrmann H, Heine S, Hupe A, Eule C. The effects of n-3 fatty acids on cutaneous inflammatory reactions in dogs. J Vet Med Physiol Pathol Clin Med. 2006;53:206–11.

65. Marsella R, Olivry T, Carlotti DN. Current evidence of skin barrier dysfunction in canine atopic dermatitis. Vet Dermatol. 2006;17:223–31.

66. Weala OI, Choudhary SK, Commins SP. Food allergy. Curr Gastroenterol Rep. 2018;20:17–25.

67. Mackie A, Moutsatsou P, McDougall J, McKinley M, Simpson J, Melton L. Omega-3 and omega-6 polyunsaturated fatty acids modulate gastric emptying and satiety in obese women. Obesity. 2012;20:1034–8.

68. Worbs T, Bode U, Yan S, Hoffmann MW, Hintzen G, Bernhardt G, et al. Oral tolerance originates in the intestinal immune system and relies on antigen carriage by dendritic cells. J Exp Med. 2006;203:519–27.

69. Ghoshal S, Witta J, Zhong J, de Villiers W, Eckhardt E. Chylomicrons promote intestinal absorption of lipopolysaccharides. J Lipid Res. 2009;50:90–7.

70. Mamo JCL, Elsegood CL, Gennat HC, Yu K. Degradation of chylomicron remnants by macrophages occurs via phagocytosis. Biochemistry. 1996;35:10210–4.

71. Elsegood CL, Mamo JCL. An investigation by electron microscopy of chylomicron remnant uptake by human monocyte-derived macrophages. Atherosclerosis. 2006;188:251–9.

72. Ma Y, Li J, Guo Y, et al. Dietary Olive oil enhances the oral tolerance of the food allergen ovalbumin in mice by regulating intestinal microecological homeostasis. J Food Biochem. 2022;46:e14297.

73. Thomas JE. Mechanics and regulation of gastric emptying. Physiol Rev. 1957;37:453–74.

74. Hunt JN, Knox JT. Regulation of gastric emptying. In: Code CF, editor. Handbook of physiology. Volume IV. Baltimore: Waverly; 1968. pp. 1917–35.

75. Cooke AR. Control of gastric emptying and motility. Gastroenterology. 1975;68:804–16.

Publisher's note