Prevalence of IgE-Mediated Food Allergy Among Children With Atopic Dermatitis

Philippe A. Eigenmann, MD; Scott H. Sicherer, MD; Teresa A. Borkowski, MD; Bernard A. Cohen, MD; and Hugh A. Sampson, MD

ABSTRACT. Objective. There is a growing body of clinical and laboratory evidence to support the notion that food allergy plays a role in the pathogenesis of atopic dermatitis (AD). However, the incidence of IgE-mediated food allergy in children with AD is not well established.

Design. A prospective study to determine the prevalence of IgE-mediated food hypersensitivity among patients referred to a university-based dermatologist for evaluation of AD.

Setting. University hospital pediatric dermatology clinic.

Patients. A total of 63 patients with AD were recruited (35 male; 32 white, 24 African-American, 7 Asian).

Methods. Patients were assigned an AD symptom score (SCORAD) and were screened for food-specific serum IgE antibodies to six foods (milk, egg, wheat, soy, peanut, fish) known to be the most allergenic in children. The levels of food-specific serum IgE were determined by the CAP System fluoroscein-enzyme immunoassay (CAP); patients with a value ≥0.7 kUa/L were invited for an additional allergy evaluation. Those with CAP values below the cutoff were considered not food allergic. Patients were considered to be allergic if they met one of the following criteria for at least one food: 1) reaction on food challenge; 2) CAP value more than the 95% confidence interval predictive for a reaction; 3) convincing history of an acute significant (hives, respiratory symptoms) reaction after the isolated ingestion of a food to which there was a positive CAP or prick skin test.

Results. A total of 63 patients (median age, 2.8 years; median SCORAD, 41.1) were recruited; 22 had negative CAP values (without a significant difference in age or SCORAD score, compared with the 41 with positive specific IgE values). Further allergy evaluation was offered to the 41 remaining patients; 10 were lost to follow-up and 31 were evaluated further. Of these, 19 underwent a total of 50 food challenges (36 double-blind, placebo-controlled, and 14 open), with 11 patients experiencing 18 positive challenges (94% with skin reactions). Additionally, 6 patients had a convincing history with a predictive level of IgE; 5 had a convincing history with positive, indeterminate levels of IgE; and 1 had predictive levels of IgE (to egg and peanut) without a history of an acute reaction. Overall, 23/63 (37%; 95% confidence interval, 25% to 50%) had clinically significant IgE-mediated food hypersensitivity without a significant difference in age or symptom score between those with or without food allergy.

Conclusions. Approximately one third of children with refractory, moderate–severe AD have IgE-mediated clinical reactivity to food proteins. The prevalence of food allergy in this population is significantly higher than that in the general population, and an evaluation for food allergy should be considered in these patients. Pediatrics 1998;101(3). URL: http://www.pediatrics.org/cgi/content/full/101/3/e8; atopic dermatitis (eczema), food allergy, IgE-mediated, prevalence, hypersensitivity.

ABBREVIATIONS. AD, atopic dermatitis; DBPCFC, double-blind, placebo-controlled food challenge; RAST, radioallergosorbent test; CAP, CAP System FEIA (a quantitative antibody fluoroscein-enzyme immunoassay); CLA, cutaneous lymphocyte-associated.

A topic dermatitis (AD) is a chronic inflammatory skin disorder that affects between 10% and 12% of the pediatric population. There is an increasing body of clinical and laboratory evidence suggesting that food hypersensitivity plays a pathogenic role in AD in a subset of patients. However, the prevalence of clinically relevant food hypersensitivity among children with AD remains an unanswered question. Sampson and McCaskill found that ~60% of a highly selected group of pediatric patients with AD evaluated by double-blind, placebo-controlled food challenge (DBPCFC) experienced a positive reaction to food. Burks and colleagues used DBPCFC to evaluate a group of 46 children with moderate to severe AD identified in an allergy-treatment clinic (31 patients) and a dermatology clinic (15 patients). Fifteen patients (33%) were determined to have clinically relevant food allergy, but the study has been criticized because a majority of the patients were recruited from the allergy clinic, introducing some selection bias.

Several lines of laboratory and clinical evidence suggest a role for IgE-mediated hypersensitivity in the pathogenesis of AD. Laboratory evaluations of children with AD reveal that ~80% to 90% have elevated total IgE levels, with ~80% having positive immediate skin tests and radioallergosorbent test (RAST) results to dietary and environmental allergens. More important, IgE has been implicated in the cutaneous late-phase response and in antigen-processing and T cell activation by cutaneous anti-
gen-presenting cells10-12 (eg, dendritic cells). Clinical studies have shown that associated atopic disorders, such as asthma and allergic rhinitis, develop in 50% to 80% of children with AD.13,14 In challenge studies, skin symptoms can be reproduced in some subjects challenged with foods to which they have specific IgE antibodies. When the incubated foods are eliminated from the diet, patients typically have significant improvement in their symptoms, compared with children without food allergy or with those who do not adhere to the diet.15

Because the true prevalence of food hypersensitivity in AD remains unknown, we undertook a study to determine the prevalence of clinically relevant food hypersensitivity in patients who were referred to a pediatric dermatologist (B.A.C.). These patients were referred to the dermatologist without selection for any adverse reaction to foods.

**METHODS**

All children between the ages of 6 months and 20 years with a history of a persistent eczematous rash in two or more predilection sites despite the use of topical corticosteroids and who presented to the dermatology clinic at the Johns Hopkins Hospital were eligible for the study. Patients were evaluated by the dermatologist (B.A.C.) at the initial visit, and an AD symptom score was assigned using the SCORAD index.16 Parents were informed by the dermatologist that children with eczema may have worsening of their symptoms triggered by foods to which they are allergic and were offered the opportunity to participate in the study. Participants had blood drawn for determination of food-specific serum IgE to a battery of six foods (milk, egg, wheat, soy, peanut, fish) that have been shown in DBPCFC to be the most common food allergens in children7,16 and to account for ~85% of documented food allergy in this group. The levels of food-specific serum IgE were determined using CAP System fluorescent-enzyme immunoassay (Pharmacia, Uppsala, Sweden) (limit of the assay <0.35 kUA/L) (Pharmacia and Upjohn Diagnostics, Evansville, IN). This method provides greater sensitivity than the standard RAST.18 In a previous study of 196 patients with AD who underwent 494 DBPCFCs, this method was shown to provide useful positive and negative predictive values for clinical reactions to a number of foods, as shown in Table 1.15 Subjects with a value greater than the 95% predictive value of a specific food were considered to be allergic and not evaluated by oral challenge to that food. Conversely, subjects with a low level of specific IgE antibody (<0.7 kUA/L) to all six foods, a level generally less than the 95% negative predictive range for a food-related reaction,19 were considered not to be food-allergic and were not evaluated by the allergist.

- Subjects with at least one food-specific IgE value >0.7 kUA/L were evaluated in the allergy clinic. A detailed history was obtained and physical examination performed, and skin tests and DBPCFCs were performed as indicated clinically.

**Skin Tests**

Prick skin tests were performed using glycerinated food extracts at a 1:10 or 1:20 (weight to volume) concentration (Greer Laboratories, Lenoir, NC) along with appropriate positive (histamine) and negative (saline) controls as described previously.15,20 In some cases, fresh extracts were prepared from fruits and vegetables.21 Mean wheal and flare diameters were determined, and a wheal >3 mm was considered a positive reaction.22

**DBPCFC**

DBPCFCs were performed on the basis of history and skin test and/or CAP results. Subjects with a convincing history of an immediate, severe reaction after the isolated ingestion of a food to which they had a positive prick skin test or CAP value were not challenged, but were considered allergic to that food. Patients also were not challenged to foods to which they had a level of food antigen-specific IgE that exceeded the 95% predictive value. In some cases, open challenges were performed because of young age or parental request. DBPCFCs were performed as described previously.32 Patients were instructed to avoid the challenge food strictly for 2 weeks before the challenge and to discontinue antihistamine medications for the equivalent of at least five half-lives for the given medication. All challenges were performed in the Pediatric Clinical Research Unit of Johns Hopkins Hospital under physician supervision. Intravenous access was obtained, and emergency medications were available immediately. Up to 10 grams of dehydrated food were camouflaged in juice, infant formula, or a moist food (eg, cream of rice) and administered in graduated portions over 90 minutes. Two challenges separated by 4 hours were administered each day; one challenge contained the suspected food allergen, whereas the other contained only the camouflage food (placebo). Randomization and preparation of the challenges were performed by the dietitians on the Research Unit; thus, patients, parents, nursing staff, and investigators were unaware of the content of the challenges. All reactions were scored for type, time of onset, severity, and duration. Symptoms generally were not followed beyond a 4-hour observation period. Negative challenges were confirmed with open feeding with the quantity of the test food normally consumed in a meal.

**Statistical Analysis**

Statistical comparisons were performed using the Mann-Whitney U test, and a P value <.05 was considered significant.

**RESULTS**

A total of 63 patients were enrolled in the study (35 male; 32 white, 24 African-American, 7 Asian). The median age of these patients was 2.8 years (range, 0.4 to 19.4 years). The median SCORAD was 41.1 (range, 6.5 to 94.5), and the mean SCORAD was 41.8. Five patients had a SCORAD <20, and 7 had a SCORAD >60. Twenty-two of the 63 patients enrolled had CAP values to all six foods that were <0.7 kUA/L and were not evaluated further. Comparison of the 41 patients with at least one CAP value >0.7 kUA/L with the 22 patients without showed no significant difference in either the mean age (2.8 years vs 2.9 years; P = .33) or the mean SCORAD (43.6 vs 37.6; P = .47), respectively. Of the 41 patients, 10 did not undergo the allergy evaluation because of loss to follow-up or patient preference. None of these 10 patients had a CAP value more than the 95% cutoff value predictive of a positive reaction and, therefore, were considered not food allergic. The remaining 31 patients underwent additional allergy evaluation.

Table 1. Levels of Specific IgE-Yielding Predictive Values (kUA/L) for CAP–RAST Tests

<table>
<thead>
<tr>
<th>Food</th>
<th>95% Positive Predictive Value</th>
<th>95% Negative Predictive Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk</td>
<td>32</td>
<td>0.8</td>
</tr>
<tr>
<td>Egg</td>
<td>6</td>
<td>90% at 0.6</td>
</tr>
<tr>
<td>Peanut</td>
<td>15</td>
<td>85% at &lt;0.35</td>
</tr>
<tr>
<td>Soy</td>
<td>50% at 65</td>
<td>2</td>
</tr>
<tr>
<td>Wheat</td>
<td>75% at &gt;100</td>
<td>5</td>
</tr>
<tr>
<td>Fish</td>
<td>20</td>
<td>0.9</td>
</tr>
</tbody>
</table>

**TABLE 1.** Levels of Specific IgE-Yielding Predictive Values (kUA/L) for CAP–RAST Tests

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positive responses to placebo. There were no episodes of anaphylaxis and no false
patient experiencing an isolated respiratory reaction. seen in 15 (83%) of the positive challenges and 2
of the challenges. Isolated cutaneous symptoms were
(6%) and respiratory symptoms (wheezing, rhinitis,
sea, vomiting, diarrhea, and/or abdominal pain) in 1
cluded cutaneous (erythema, hives, and/or maculo-
Symptoms elicited during positive challenges in-
The results of these challenges are shown in Table 2.
There were 18 positive challenges in 11 patients.
Symptoms elicited during positive challenges included
cutaneous (erythema, hives, and/or maculo-
papular eruption) in 17 (94%), gastrointestinal (nau-
sea, vomiting, diarrhea, and/or abdominal pain) in 1
(6%) and respiratory symptoms (wheezing, rhinitis,
nasal congestion, and/or repetitive cough) in 2 (11%)
of the challenges. Isolated cutaneous symptoms were
seen in 15 (83%) of the positive challenges and 2
organ systems were involved in 2 (11%), and one
patient experiencing an isolated respiratory reaction.
There were no episodes of anaphylaxis and no false
positive responses to placebo.

Characteristics of Patients With Food Allergy

Table 3 summarizes the foods to which the 23
patients were allergic based upon challenge results, a
"convincing" history plus a positive food-specific
IgE antibody level as determined by CAP-System
FEIA, a "convincing" history plus a positive food-
specific IgE level and/or prick skin tests, or a CAP-
System FEIA value greater than the 95% predictive
cut-off. Eight patients were allergic to one food, 4
to two, 5 to three, and 5 patients to four or more foods.

**Table 2. Results of Food Challenges in 19 Patients**

<table>
<thead>
<tr>
<th>Food</th>
<th>DBPCFC</th>
<th>Open Challenge</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>Egg</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Milk</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Soy</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Wheat</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Barley</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Oat</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beef</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Chicken</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Pork</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Green beans</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Turkey</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Tangerine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Totals</td>
<td>13</td>
<td>23</td>
</tr>
</tbody>
</table>

Overall, 23 of the 63 patients were allergic to at
least one food (37%; 95% confidence interval, 25% to
50%). Comparison of these 23 food-allergic patients
with the 40 with a negative or incomplete evalu-
ation showed no significant difference in median
SCORAD among those with (46.9) or without (39.6)
food allergy (P = .92). The median ages of those with
(2.3 years) and without (3.0 years) food allergy also
were similar (P = .31).

**DISCUSSION**

AD is a form of eczema that usually begins in early
infancy; is characterized by a typical distribution,
extraordinary pruritus, and a chronic and relapsing
course; and frequently is associated with asthma,
alergic rhinitis, and a family history of allergic dis-
orders.6 The rash is typically an erythematous, pap-
ulovesicular eruption, frequently with exudation
early in infancy that progresses to a scaly, lichenified
rash later in childhood. The distribution of the rash
varies with age, involving the cheeks and extensor
surfaces of the extremities in infants with a predilec-
tion for the flexor surfaces of the elbows, knees,
hands, and feet of the older child and adult. The
pathogenic role of allergy in AD has been the focus of
much debate, but a number of clinical and laboratory
observations have suggested a significant role for
IgE-mediated mechanisms.5-9,13-15

The pathogenic role of food allergy in AD was first
suggested by clinical observations and uncontrolled
studies performed >80 years ago and has been sup-
ported further by the results from large controlled
studies using DBPCFC performed in the past 20
years. In a study in 1918, Talbot25 described a series
of patients with eczema and positive skin tests to
foods who experienced clearing of their skin with
elimination of the incriminated foods from their diet.
The issue of whether ingested food antigens could reach
the skin and cause immune-mediated symp-
toms was addressed in studies by Walzer and col-
leagues.26,27 They passively sensitized healthy sub-
jects by intracutaneous injection with serum from
food-allergic subjects. When passively sensitized
subjects ingested the relevant protein, they devel-
opped a wheal and flare reaction at the sensitized site
within 1 to 2 hours, but not at the control site. Ad-
ditional support for the relationship of AD to food
allergy was demonstrated in 1936 by Engman and
colleagues,28 who reported a patient with eczema in
whom ingestion of wheat caused intense pruritus.
The patient was admitted to the hospital on a wheat-
elimination diet with clear skin, had his left arm and
leg bandaged to prevent access for scratching, and
was fed wheat, which led to itching and scratching.
By the next morning, the patient had eczematous
lesions only in exposed areas where rubbing and
scratching occurred.

These studies gave way to larger, controlled stud-
ies of food allergy and AD. Sampson and colleagues
have investigated the role of IgE-mediated food
allergy in pediatric patients with AD using
DBPCFC.2,6,15,18,19,29-31 In general, positive DBPCFCs
elicit a variety of symptoms within minutes to 2
hours. Although a majority of reactions involve cu-

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taneous symptoms, the upper and lower respiratory tract and gastrointestinal system are frequently involved. At first evaluation, skin symptoms provoked by DBPCFC generally consisted of a pruritic, erythematous, morbilliform rash that developed in predilection sites for AD; at follow-up evaluations years after the incriminated food was eliminated from the diet, skin reactions in children who remained sensitive generally consisted of urticular lesions. Both immediate and late-phase effects of ingested food allergens have been described during DBPCFC, with some patients developing diffuse pruritus and, less frequently, an erythematous macular rash 4 to 8 hours after a positive challenge.2,15,31 When an appropriate elimination diet is undertaken in food-allergic children with AD, significant improvement has been documented in their skin symptoms, compared with food-allergic children with eczema who are not on an appropriate elimination diet or who have AD without evidence of food allergy.15 However, double-blind dietary intervention studies are needed to document further the efficacy of elimination diets in the treatment of AD.

Laboratory evaluations of patients with AD have provided additional evidence for the role of IgE-mediated hypersensitivity in the pathogenesis of this illness. The immunohistochemical analysis of chronic lesions of AD reveals infiltration with predominantly CD4-positive T cells, Langerhans’ cells bearing high-affinity receptors for IgE, mast cells, eosinophils, and eosinophil granule products.32–34 One explanation for the development of this infiltrate is that it represents the chronic stages of a late-phase response after the IgE-mediated immediate-phase allergic response. However, other IgE-mediated mechanisms are likely to play a role, because cutaneous Langerhans’ cells also bear high-affinity IgE receptors and these cells can elaborate cytokines that activate T cells. Evidence supporting the immunopathogenic role of IgE-dependent, food-allergic reactions in the development of cutaneous symptoms that may exacerbate AD include the sharp rise in plasma histamine30 and the rise in serum levels of eosinophil granule products and eosinophil activation markers35,36 accompanying positive food challenges. Biopsies obtained at the site of the morbilliform eruption 10 to 14 hours after a positive DBPCFC revealed eosinophil infiltration and deposition of eosinophil major basic protein, cells, and cellular products capable of promoting the inflammatory response. Using skin blister chambers, it was shown that the T cells infiltrating the skin are enriched for the cutaneous lymphocyte-associated (CLA) antigen, whereas lung T cells were CLA-negative.37 Interestingly, when the peripheral blood lymphocytes of AD patients with cow’s milk sensitivity were stimulated in vitro with cow’s milk antigen, there was a marked increase in proliferation of CLA-positive T cells not seen in patients with milk allergy without AD.38 Another mechanism believed to play a role in AD involves the generation of a cytokine called histamine releasing factor from mononuclear cells that results, in vitro, in increased spontaneous basophil histamine release.29,39 The spontaneous release of this cytokine was documented in food-allergic children with AD who were not on elimination diets and was shown to decrease to baseline values when the children were maintained on appropriate elimination diets for 6 to 9 months.

Thus, both clinical and laboratory evidence support a role for IgE-mediated food hypersensitivity in some patients with AD. The population studied by Sampson and McCaskill2 had severe AD and was selected on the basis of possible food allergy. Burks and colleagues4 sought to overcome this selection bias by recruiting patients from both a dermatology clinic and an allergy clinic, but the 33% prevalence rate of food allergy found in that study has been questioned because two thirds of the patients were referred to a pediatric dermatologist for AD. Despite the elimination of the selection bias inherent in the study by Burks and colleagues, we found a similar prevalence rate (37%) of food allergy among children with AD.

A study by Guillet and Guillet40 suggested an increased prevalence of food allergy among patients with increasing severity of AD. However, we did not detect a difference in the prevalence of food allergy in relation to severity of AD in our population; patients with and without food allergy had similar SCORAD scores. We may not have been able to detect a correlation of food allergy because of the smaller population studied and the selection of patients predominantly with moderate to severe symptoms.

There were several factors in the current study that actually may have caused an underestimate of the prevalence of IgE-mediated food allergy in children with AD. First, screening patients with the CAP-System FEIA to six foods may have missed 10% to 15% of patients in whom some other food would

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**TABLE 3. Sensitivities of the 23 Food-allergic Children to Major Food Allergens**

<table>
<thead>
<tr>
<th>Method of Determination</th>
<th>Food Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Challenge positive</td>
<td>—</td>
</tr>
<tr>
<td>History positive, IgE-positive</td>
<td>1</td>
</tr>
<tr>
<td>History positive, CAP–RAST more than 95% predictive</td>
<td>3</td>
</tr>
<tr>
<td>CAP–RAST more than 95% predictive</td>
<td>3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Peanut</th>
<th>Egg</th>
<th>Milk</th>
<th>Wheat</th>
<th>Soy</th>
<th>Fish</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>14</td>
<td>7</td>
<td>3</td>
<td>0</td>
<td>0</td>
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have been implicated. Second, 10 patients with indeterminate but positive CAP values were not evaluated fully and therefore considered not to be food-allergic. Third, 4 patients considered to have negative evaluations did not complete challenges to all of the foods to which they had food-specific IgE antibodies. Finally, we included children >5 years of age, a group that is likely to have a lower prevalence of food allergy than do infants and toddlers. Thus, a prevalence rate of 37% for food allergy in AD is conservative and probably represents an underestimate.

Considering that there are many triggers for AD, including both specific (aeroallergens, food allergens, and staphylococcal enterotoxins) and nonspecific (heat, humidity, irritants, and stress) triggers, the question of which child should be evaluated for food hypersensitivity arises. Given a prevalence rate of 25% to 50%, an evaluation for food allergy may be warranted in any AD patient requiring chronic treatment or responding poorly to a regimen of hydrating baths, topical corticosteroids, lubricating ointments, antihistamines, and antibiotics. Certainly, if the patient has a history of an acute, life-threatening reaction to a food, appropriate measures should be taken to confirm the allergy and provide information on food-allergy avoidance and emergency management in case of accidental ingestion. Additionally, patients with associated atopic illnesses may require additional allergy evaluation.

In addition to studies suggested by information gleaned from history, a reasonable approach for screening children with poorly responsive AD for IgE-mediated food allergy would include prick skin testing to the major food allergens and any other suspected foods. Foods eliciting positive responses could be evaluated with open or single-blind challenges to exclude clinical reactivity, but must be performed in a setting where severe reactions, including anaphylaxis, can be treated. Alternatively, quantitation of food-specific IgE levels (CAP System FEIA) could be performed. Positive and negative predictive values for four foods19 (Table 1) have been established that can help determine who may ingest the food safely and who is likely to react. For those with indeterminate values, challenges should be performed. Open oral food challenges are less labor-intensive and resulted in a similar proportion of positive and negative results as the DBPCFC in this study. However, if a patient appears to be allergic to more than one major food (egg, milk, wheat, soy) or a large number of other foods, DBPCFC should be performed to confirm reactivity. Strict avoidance of the food is the only treatment currently available.

The guidance of a dietitian often is required to ensure complete elimination of all hidden sources of food proteins and the nutritional adequacy of the diet. Studies suggest that strict avoidance results in improvement of skin symptoms15 and also may speed the acquisition of tolerance. Children typically outgrow their clinical reactivity to egg, milk, wheat, and soy (despite persistently positive skin tests), whereas clinical sensitivity to peanuts, tree nuts, fish, and shellfish is often lifelong.15,42,43 Because approximately one third of patients will outgrow their reactivity after 1 to 2 years of allergen avoidance, serial evaluations are an important part of follow-up.

In conclusion, a significant percentage (37%) of patients referred to a university-based pediatric dermatologist for AD have symptomatic food allergy. An evaluation for food allergy should be considered in patients with refractory, moderate to severe AD and in patients with a history of acute allergic reactions. Studies are underway to address further the efficacy of food allergen elimination in the treatment of AD.

ACKNOWLEDGMENTS

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