TABLE I. Characteristics of children from an inner-city allergy clinic who underwent food allergy evaluation

	Group A (history of immediate symptoms on food ingestion)	Group B* (no history of immediate symptoms on food ingestion)
No.	87	141
Male sex (%)	45 (52%)	81 (57%)
Median age (y), range	3 (0.25-18)	3 (0.25-17.0)
African American	34 (39%)	62 (44%)
Hispanic	36 (41%)	68 (48%)
Asthma	40 (46%)	66 (47%)
AD	33 (38%)	66 (47%)
Allergic rhinitis	41 (47%)	51 (36%)
Sensitized to any food†	55 (63%)	74 (52%)
Serum sIgE >95PDP for:		
Hen's egg white	12 (14%)	27 (19%)
Peanut	10 (11%)	22 (16%)
Cow's milk	8 (9%)	16 (11%)

*Group B had 1 or more of the following: (1) moderate-to-severe persistent AD or asthma not controlled with optimal therapy, (2) failure to thrive, and (3) family history of food allergy and avoidance of foods as a precaution.
†Statistically different rates of food sensitization between groups A and B (logistic regression model adjusted for AD and allergic rhinitis, $P = .02$).

Both food IgE sensitization and food allergy are common in the inner-city pediatric allergy clinic population, which is consistent with the National Cooperative Inner-City Asthma Study and National Health and Nutrition Examination Survey reports.[4,5] High rates of food sensitization and allergy are seen in children for whom acute allergic reactions might not be the chief complaint. It is possible that a history of acute reactions was not elicited because of fragmented childcare, leading to the guardian being unaware of exposures and reactions, language barriers, or fragmented health care causing poor control of AD, asthma, or both.

Children with AD are at higher risk for food allergy.[7] In up to 40% of children with moderate-to-severe persistent AD, ingestion of the offending foods might result in exacerbation of the existing skin lesions, acute allergic reactions, or both. We also found that children with AD were at significantly higher risk for food allergy and sensitization when compared with those without AD.

The majority of children underwent food allergy evaluations for reasons other than clinical reactivity (group B), suggesting that different racial groups might manifest food allergies with different symptoms. It has been reported that African American children have higher rates of AD.[8] Thus AD might be a more common presenting symptom of food allergy in this population, especially if the children demonstrate skin symptoms early in life. Children with early-onset severe AD are at higher risk of having increased food-specific IgE levels.[9]

Limitations to our study include the retrospective design and a study cohort that might not be representative of all inner-city children. Double-blind, placebo-controlled food challenges were not conducted in the majority of children. Although the double-blind, placebo-controlled food challenge is considered the gold standard for food allergy diagnosis, it is not practical in the epidemiologic studies because of high cost and limited availability. Therefore, the acceptable substitute is the determination of sIgE levels to foods, particularly the use of established 95PDP values. In the United States these diagnostic decision points are widely used, and oral food challenges are usually not performed when specific sIgE levels exceed 95PDP values. We limited our analysis to egg, milk, and peanut, for which 95PDP values are well established, although the accuracy of 95PDP values in children without a clear history of clinical reactivity is uncertain.[7] Finally, food allergy evaluations were performed if there was a suspicion of food allergy, and therefore the exact prevalence of food sensitization in our clinic cannot be determined.

In spite of these limitations, our data clearly show a very high rate of IgE sensitization to foods and IgE-mediated allergy to egg, milk, and peanut in the East Harlem inner-city pediatric allergy clinic population. Clinicians should maintain a high index of suspicion for food allergy when evaluating inner-city children with persistent AD and asthma resistant to standard medical therapies, even in the absence of definitive clinical reactions.

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Anaphylaxis to diphtheria, tetanus, and pertussis vaccines among children with cow's milk allergy

To the Editor:

Immediate-type hypersensitivity to cow's milk affects approximately 1% to 2% of young children, can persist, and can be

TABLE I. Clinical characteristics of the children who reacted to the tetanus vaccines

Age at time of reaction (y)	Sex	Milk allergy history	Asthma history	Milk-specific IgE level (kIU_A/L)	Symptoms of reaction after vaccination	Treatments given	Vaccine administered	Time to reaction after vaccine administration
4	Male	Proctocolitis from cow's milk–based formula at 3 mo of age	Yes	82.9	Diffuse hives, respiratory distress	Diphenhydramine, oral steroid	DTaP	10 min
5	Male	Hives, lip angioedema, wheezing at 3 y of age after eating turkey with milk contamination	Yes	>100	Repetitive cough, sneezing, rhinorrhea	Albuterol, diphenhydramine	DTaP	10 min
5	Male	Urticaria, edema of the face, and wheezing after contact with milk-based formula at 2 y of age	Yes	>100	Generalized urticaria, facial swelling, wheezing, retractions, difficulty breathing	Diphenhydramine, epinephrine	DTaP	Immediately
6	Male	Milk was eliminated based on allergy testing as an infant after presenting with atopic dermatitis	Yes	>100	Cough, hives, facial swelling	Levalbuterol, diphenhydramine	DTaP	Within 15 min
11	Male	Urticaria and vomiting at 11 mo of age after eating yogurt; facial swelling and diffuse rash at 8 y of age after eating turkey with milk contamination	Yes	58.9	Generalized urticaria, sneezing, congestion, wheezing	Levalbuterol, diphenhydramine, oral steroid	Tdap	Within 10 min
11	Female	Vomiting after milk-based formula as an infant; angioedema of hands after contact with spilled milk at 1 y of age	Yes	96.1	Throat tightness, wheezing, respiratory distress	Epinephrine	Tdap	Driving home from pediatrician
16	Female	Cough, wheezing, and vomiting after milk at 1 y of age; respiratory distress, vomiting, hypotension, and cardiac arrest after eating pasta sprinkled with cheese at 8 y of age	No	>100	Nasal congestion, watery eyes, hives, wheezing, chest tightness	Diphenhydramine, oral steroid	Tdap	Within 1 h
17	Male	Hives, lip, and tongue swelling; diarrhea; and abdominal pain at 4 y of age	Yes	>100	Wheezing, respiratory distress, flushing of face and neck	Epinephrine	Tdap	Within 20 min

fatal.[1,2] Vaccines against tetanus, diphtheria, and pertussis are routinely given to children in multiple doses throughout childhood. Anaphylactic reactions to these vaccines are rare and are generally attributed to the vaccine toxoids.[3,4] The US national Vaccine Adverse Events Reporting System lists 39 anaphylactic reactions to DTaP, DTP, or Tdap vaccines for patients aged 0 to 17 years from 2007-2010.[5] In this period we observed 8 children in our single center with a history of anaphylactic reactions to booster doses of these vaccines. We noted that these vaccines are labeled as being processed in medium containing casamino acids (derived from cow's milk), raising the concern that residual casein in the vaccines might have triggered these reactions. We are not aware of prior reports linking milk contamination to anaphylaxis from these vaccines. To investigate this possibility, we reviewed the medical history of the affected children and tested 8 lots of the vaccines for residual casein.

The clinical characteristics of the 8 children were obtained by means of chart review (approved by the institutional review board)

and are shown in Table I. These patients were selected based on reports of anaphylactic reactions to the vaccines and not because of a history of milk allergy. Each patient had symptoms consistent with anaphylaxis[6] within 1 hour of administration of the vaccine. Six of the patients had prior acute allergic reactions to cow's milk, including severe reactions in 5 patients and reactions to trace exposures in 4 patients. One patient was given a diagnosis of milk allergy based on serologic testing performed to evaluate atopic dermatitis, and another was given a diagnosis based on serologic testing to evaluate proctocolitis. Each of the patients had an increased milk-specific IgE level documented within 2 years of the reaction to the vaccine. Although milk-specific IgE levels do not necessarily correlate with severity, the recorded levels are far above those that typically correlate with reactivity.[7]

The vaccine package inserts for the DTaP and Tdap vaccines, including those for Adacel, Boostrix, Daptacel, Infanrix, Kinrix, Pediarix, and Pentacel, all state that either the tetanus toxin is produced by growing *Clostridium tetani* in a modified Latham medium derived from bovine casein or that the *C tetani* is grown in modified Mueller-Miller casamino acid medium.

Additionally, for the acellular pertussis vaccine components, all of the vaccines use a modified Stainer-Scholte medium, but only the Adacel, Pentacel, and Daptacel vaccines specifically mention this being modified with casamino acids. To determine whether there was casein in the vaccines, we performed assays on 5 different lots of the Tdap vaccine and 3 different lots of the DTaP vaccine. We also performed assays on 3 different lots of the influenza vaccine and 1 lot of the hepatitis B vaccine, which do not report processing in medium containing casamino acids, to act as negative controls.

For the assays, 96-well Immulon 4HBX plates (Fisher Scientific, Pittsburgh, Pa) were coated overnight with 50 μL/well of 1 μg/mL casein (Sigma-Aldrich, St Louis, Mo) diluted in 0.05 mol/L carbonate-bicarbonate buffer (pH 9.6). After washing (3 times) with phosphate-buffered saline containing 0.05% Tween-20 (PBS-T), the plates were blocked with 0.1 mL of PBS-T–ovalbumin 0.07% (OVA grade V, Sigma-Aldrich) per well for 1 hour. Samples (1:1 and 1:2 dilutions) and standards (0.19-190 ng/mL) were prepared and mixed (1:1) with rabbit anti-casein polyclonal antibody (a gift from Ross Laboratories, Columbus, Ohio) diluted at 1:50,000 in blocking buffer. This inhibition mix was incubated for 2 hours at 37°C. A mixture (1:1) of the same antibody with blocking buffer without competing casein was also prepared for cutoff determination. Fifty microliters per well of the test mixture was then pipetted in quadruplicates into the coated microtiter plates and incubated for 1 hour at 31°C. After washing (3 times) with PBS-T, 50 μL/well of horseradish peroxidase–labeled goat anti-rabbit IgG (Sigma-Aldrich) diluted to 0.4 μg/mL in blocking buffer was added and incubated for 1 hour at 31°C. Plates were washed (6 times) with PBS-T, and 50 μL/well of TMB (KPL, Inc, Gaithersburg, Md) was added and allowed to develop at room temperature for 60 minutes. Absorbance values were read at 650 nm with SoftMax Pro software, and the concentration of casein in analytes was obtained from the linear part of the standard curve by using a 5-parameter model. The presence of casein was confirmed in all samples of the Tdap and DTaP vaccines, whereas no casein was found in the negative controls (Table II).

In summary, we identified 8 patients with severe milk allergy who reacted with anaphylaxis to Tdap or DTaP vaccines, which are processed in a broth derived from casein. We identified casein in 8 lots of the vaccines, raising the concern that residual casein in the vaccines might result in reactions for highly sensitive patients with milk allergy.

TABLE II. Mean casein concentrations in vaccine samples examined

Vaccine	Brand name	Lot numbers	Casein (ng/mL)
Tdap	Adacel	C3727BA	12.7
Tdap	Adacel	C3518AA	13.6
Tdap	Adacel	C3819AA	8.1
Tdap	Adacel	C3246BA	17.3
Tdap	Adacel	C3448AA	11.8
DTaP	Daptacel	C3192BA	10
DTaP	Infanrix	AC14B099BA	18.3
DTaP	Infanrix	AC14B121BB	12.2
Influenza	Fluarix	AFLUA531BA	0
Influenza	Fluzone	UT3667AA	0
Influenza	Fluvirin	111814P1	0
Hepatitis B (recombinant)	Recombivax	1023Y	0

Allergic reactions have been noted to trace amounts of ingested milk protein.[8,9] We suspect that smaller injected doses, similar to insect sting–induced anaphylaxis, trigger reactions in sensitive patients because digestion is bypassed. Most children with cow's milk allergy receive these vaccines without incident, but the patients we identified have very severe milk allergy, very high milk-specific IgE levels, and, for 4 of them, past reactions to trace milk contamination. These children tolerated their initial vaccinations but reacted to booster doses. This observation is most likely explained by increasing milk sensitivity of the children as they aged, as reflected in the personal histories of several of the children. The number of patients at our single center and their clinical features of severe milk allergy and immediate severe reactions to the vaccine argue against alternative explanations for the reactions. Unfortunately, we were not able to test the lots of the vaccines associated with the reactions, and there might be variability in milk protein content influencing the risks.

In conclusion, our novel observation raises a concern regarding booster vaccination of children with high levels of milk allergy with Tdap and DTaP. Although the children we identified appear by history and testing to be exquisitely allergic to milk, we cannot accurately define a high-risk group based on this case series. Clearly, many highly sensitive children with milk allergy tolerate the vaccines because these reactions are apparently rare. Further studies will be necessary to make more definitive recommendations regarding which children might be at risk. Manufacturer investigation and possible labeling or elimination of casein from the vaccines might avoid this risk. In the interim, we recommend continuing the standard practice for DPT vaccination in all children, but advise caution when administering booster doses in highly sensitive milk-allergic children.

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Single-dose influenza vaccination of patients with egg allergy in a multicenter study

To the Editor:

Studies have suggested that the ovalbumin content in recent years' influenza vaccines is extremely low[1,2] and that most individuals with egg allergy can be safely vaccinated.[3,4] Although there are more conservative approaches, the majority of published studies use a 2-step protocol in which 10% of the dose is administered, followed 30 minutes later by the remaining 90%, a strategy used in the pivotal study by James et al[5] in 1998 and confirmed in several studies since then.[3,6] Some, however, have documented the safety of a single full dose when the vaccine contains a very low amount of ovalbumin.[7] Even patients with a history of severe egg allergy have been safely vaccinated by both strategies.

On average, 36,000 people die each year in the United States because of complications of influenza infection. Children under the age of 2 years are 9 times more likely to be hospitalized because of complications from influenza than children older than 5 years.[8] Vaccination remains the single most important tool available for prevention. Unnecessary avoidance of the influenza vaccine places undue risk on a significant proportion of one of our most vulnerable populations.

We performed a retrospective review of the safety of seasonal and H1N1 influenza vaccinations in patients with egg allergy at 4 university-based allergy and immunology clinics during the 2009 to 2010 influenza season to determine the tolerability of these vaccines in this population. The diagnosis of egg allergy was confirmed by an allergist and based on a clinical history consistent with an allergic reaction within 2 hours of the ingestion of egg and evidence of egg-specific IgE by skin or serum testing, or by an egg-specific IgE level or skin prick test (SPT) with >95% predictive value for type I hypersensitivity.[9]

A full-strength vaccine SPT was performed along with positive and negative controls. Patients who tolerated baked egg were only skin tested at the clinician's discretion; otherwise they were given the full dose in a single injection because individuals tolerant of baked egg regularly tolerate small amounts of egg protein. If SPT was negative, the patient was given a single injection containing 100% of the required dose on the basis of age. If the SPT was positive or equivocal, the patient was given the vaccine in a 2-step protocol (10% of the total dose followed 30 minutes later by the remaining 90%). If a patient required a booster dose and the first dose was tolerated without adverse reaction, the vaccine from the same manufacturer but not necessarily the same lot was administered as a single dose with no antecedent SPT. No intradermal testing was performed at any time. All patients were observed for 30 minutes after the final dose, and all patients or parents were contacted the next clinic day to ensure that no delayed reactions occurred. Only injectable influenza vaccines were used in this study.

A total of 292 vaccinations with seasonal and/or H1N1 influenza were performed on 152 patients. Thirty-four (22%) of the 152 patients had a convincing history of anaphylaxis to egg involving a drop in blood pressure or a combination of respiratory compromise, skin involvement, or prolonged gastrointestinal symptoms as defined by the Second Symposium on the Definition and Management of Anaphylaxis.[10] Eighty-seven (57%) patients had a history of immediate-type allergic reaction to egg affecting the skin or gastrointestinal system alone. Thirty-one (20%) patients had not knowingly consumed egg or no reaction was documented in the medical record, but had a level of egg IgE by SPT, serum *in vitro* test, or both that was >95% predictive of egg allergy.[9] The age of patients ranged from 7 months to 30 years with a median age of 3 years. The most recent SPT to egg showed a median wheal size of 8 mm (range, 0-28 mm). The median serum IgE to egg was 6.01 kU/mL (range, <0.35->100 kU/L) using the Phadia ImmunoCAP system (Phadia, Uppsala, Sweden).

Skin prick testing was performed before 85% of challenges. One patient had a positive SPT, and 6 were equivocal. One child received the vaccine in split dosing because of parent preference, despite a negative SPT. The only positive SPT occurred to the seasonal FluZone (Sanofi Pasteur Inc, Swiftwater, Pa) vaccine in a 22-month-old who had never knowingly ingested egg but who had a 15-mm wheal to egg SPT and serum egg IgE 2.57 kU/L. The child tolerated the full dose of the seasonal vaccine administered by split dosing. Of note, this same child with a positive SPT to the seasonal vaccine had a negative SPT to the H1N1 vaccine and tolerated the latter in a single dose on 2 occasions, including a booster H1N1 dose.

Two hundred eighty-five vaccinations were performed by using single-dosing (97%), including 65 vaccinations (23%) in patients with a history of severe egg allergy. One of the 34 patients with severe egg allergy received the vaccine in a divided dose because of an equivocal SPT to the seasonal FluZone vaccine. There were no systemic reactions in any of the patients undergoing vaccination in our study, including those with severe egg allergy. Two

AAAAI Ask the Expert
8/19/11

Q:

How are people handling giving a dTap immunization to a child with significant allergy to milk now that there has been at least one lot found to be contaminated with milk protein?

A:

Thank you for your recent inquiry.

A very pertinent question, and one for which I know of no precedent in the literature. I am therefore going to ask Dr. John Kelso, who is co-author of the most recent Parameters on vaccine reactions, to share his thoughts with us in this regard. As soon as I hear from Dr. Kelso, I will forward his comments to you.

Thank you again for your inquiry.

Sincerely,
Phil Lieberman, M.D.

We have received the response from Dr. John Kelso, which is copied below and attached. Thank you again for your inquiry and we hope this response is helpful to you.

Sincerely,
Phil Lieberman, M.D.

Response from Dr. John Kelso:
This stems from a report in the July 2011 JACI (http://www.jacionline.org/article/S0091-6749%2811%2900747-0/fulltext). Although these children's reactions may have been due to their extreme milk sensitivity, it is clearly unusual to react to nanogram quantities of protein and the children's actual sera were not available to test against the vaccines on a solid phase by RAST or to see if the vaccines could inhibit a milk RAST to actually demonstrate that the children's IgE could recognize the casein in the vaccines. Even if these reactions are due to milk specific IgE recognizing casein in the vaccines, this is clearly a very rare phenomenon, since the vast majority of even severely milk allergic children receive the vaccines uneventfully. Thus I would agree with the authors who state, "we recommend continuing the standard practice for DPT vaccination in all children, but advise caution when administering booster doses in highly sensitive milk-allergic children." In my opinion, this "caution" would only mean observing highly milk allergic children for 30 minutes after such immunizations administered in the usual manner.

John Kelso, M.D.