023 Investigating Food Allergy Reactions and Overall Impact on College Campuses



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RATIONALE: There is currently little food allergy (FA) awareness and accommodation on college campuses, despite the high prevalence of allergic reactions on campus.

METHODS: An online, cross-sectional survey was sent nationwide to college students with and without FA in July 2022. Descriptive statistics were used to investigate students' experiences with FA, evaluate FA awareness on campus, and determine common causes of allergic reactions in dining halls.

RESULTS: Of 193 respondents from 65 universities, 74 reported having FA. Exactly 50.0% of students with FA reported being more worried about their FA now in comparison to high school, which is supported by the fact that 32.5% of students with FA stated they have had an increase in allergic reactions since coming to college. The most common causes of allergic reactions in dining halls were food mislabeled with the allergen (45.0%), cross contact in buffet style (40.0%), and food not labeled (35.0%). The top feelings associated with having a food allergy are anxious (54.1%), frustrated (52.7%), and scared (28.4%). The top areas of college life impacted by FA were social life (55.4%) and on-campus dining experience (55.4%).

CONCLUSIONS: Universities need to better identify students with FA, especially considering the increase in allergic reactions among students since attending college. Further, food allergies significantly impact social life in addition to the on-campus dining experience. Universities must take steps to increase food allergy awareness and limit reactions in dining halls to improve safety for students with FA.

024 Sensitization Through Skin and Airways Mediates Distinct Mechanisms for Anaphylaxis in Mice.



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RATIONALE: The immunologic mechanisms of peanut allergy are not fully understood. The controversies remain regarding the route of sensitization. We compared the mechanisms of peanut allergy sensitized through skin and airways.

METHODS: Naïve BALB/c mice were exposed epicutaneously or intranasally to peanut flour (Golden Peanut Co.) without any adjuvants by painting or by inhalation twice a week for four weeks. Mice were challenged by intraperitoneal injection of peanut extract and monitored for acute anaphylaxis. Various gene knockout mice were used to dissect the mechanisms.

RESULTS: Regardless of the routes of sensitization, T follicular helper (Tfh) cells played a critical role as the mice deficient in Tfh cells (i.e., Bcl6fl/fl-CD4Cre mice) failed to produce peanut-specific IgE or IgG and were protected from anaphylaxis when challenged with peanut extract. IL-13 was dispensable for peanut allergy irrespectively of whether mice were sensitized through skin or airways. Distinct differences were also observed. In the airway model, high levels of peanut-specific IgE and IgG were generated, and the anaphylaxis was dependent on mast cells, IgG antibodies, and a high-affinity IgG receptor FcgRIII. In the skin model, peanut-specific IgE and IgG1 (but not other isotypes) were produced involving the IL-4Ra pathway, and the anaphylaxis was dependent on IgE and accompanied by marked elevation of serum mast cell protease-1.

CONCLUSIONS: Peanut allergy and acute anaphylaxis are dependent on Tfh cells but independent of the IL-13 pathway. Different isotypes of peanut-specific antibodies are likely responsible for anaphylaxis depending on the routes of sensitization.



025 Incidence of and risk factors for pediatric perioperative anaphylaxis in the United States, 2005-2014



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RATIONALE: Little is known about pediatric perioperative anaphylaxis (pPOA). The objective of this study is to determine the incidence of pPOA in the US and to identify risk factors for pPOA.

METHODS: Using the National Inpatient Sample from 2005 to 2014, we identified cases (patients aged <18 years) of pPOA utilizing the International Classification of Diseases, Ninth Revision, Clinical Modification codes.

RESULTS: Amongst 3,601,180 procedures (mean age 3.3 [SD 6], 78% male, 60% White, 85% Non-Hispanic), 297 (1 in 12,214) cases of pPOA were identified. When compared to pediatric patients who underwent procedures without developing anaphylaxis, those who developed pPOA were older (mean age 9.8 vs 3.3 years; p<.0001) and had an increased median length of stay (6 vs 2 days; p<.0001), median hospital cost (\$54,719 vs \$5,109; p<.0001), and mortality rate (1.7% vs 0.3%; p<.0001). Children undergoing transplant procedures (odds ratio [OR] 3.16; 95% confidence interval [CI] 2.27-8.64) had an increased risk of pPOA. Female sex (OR 1.16; CI 0.90-1.50) was not associated with pPOA.

CONCLUSIONS: The incidence of pPOA in the US found in our study was 1 in 12,214 procedures with a 1.7% mortality rate. We found that pPOA has worse outcomes compared to controls. Procedures performed in older children and transplant procedures pose an increased risk for pPOA. Contrary to what has been seen in the adult population, female sex was not determined to be a risk factor for pPOA.

026 Mast Cell Degranulation Links Anti-PEG IgE to Anaphylaxis Caused by PEGylated Drugs and PEG-contained LNP/mRNA COVID Vaccines



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RATIONALE: Anti-PEG IgE has been reported to be associated with PEG-associated anaphylaxis. However, there lacks mast cell degranulation evidence to validate anti-PEG IgE-mediated type 1 hypersensitivity in anaphylaxis caused by PEGylated drugs and PEG-contained LNP/mRNA COVID vaccines.

METHODS: Human mast cells were derived from CD34+ pluripotent progenitor cells and mast cell maturation was confirmed with expression of CD117/FceRI. Mature mast cells were primed with anti-PEG IgE or control antibodies. Mast cell degranulation was monitored in real-time by calcium influx assay upon PEG and LNP/mRNA COVID vaccine exposure. Anti-IgE antibody and Tyrode buffer were served as positive and negative cross-linker controls, respectively.

RESULTS: Anaphylactic mast cell degranulation was shown by calcium influx kinetics in anti-PEG IgE-primed mast cells upon PEG and LNP/ mRNA COVID vaccine exposure. Mast cell degranulation was not observed in controls including un-treated mast cells, anti-PEG IgG, anti-PEG IgM or isotype control IgE-primed mast cells upon PEG or vaccine exposure.

CONCLUSIONS: Evidence of Type I hypersensitivity caused by PEGylated Drugs and PEG-contained LNP/mRNA COVID Vaccines is demonstrated by specific anti-PEG IgE-mediated mast cell degranulation upon PEG and vaccine exposure. Mast cell degranulation assay and/or anti-PEG IgE assay is warranted for in vitro diagnosis of anaphylaxis caused by PEGylated Drugs and PEG-contained LNP/mRNA COVID Vaccines.