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Article

Far UV Exposure (UV_{222}) Decreases Immune-Based Recognition of Common Airborne Allergens

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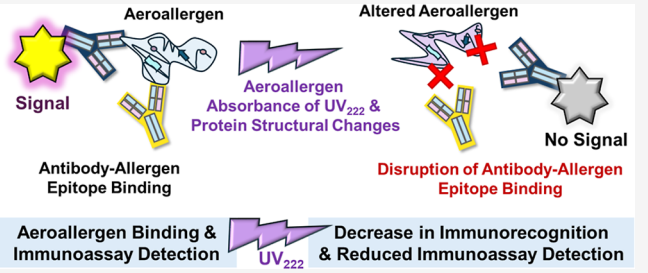
ABSTRACT: Airborne allergens (aeroallergens) significantly contribute to respiratory allergies and asthma. Traditional methods such as cleaning and allergen avoidance have shown mixed results in improving health outcomes in sensitized individuals, and effective control of airborne protein allergens remains a practical challenge within the built environment. To address this challenge, this study developed a controlled experimental system to generate respirable particles (≤ 10 μm) containing common aeroallergens from mites, pet dander, mold, and pollen, in both dust and purified forms. Allergens were aerosolized into a 10 m^3 controlled environment chamber where the contents were either exposed to a calibrated UV_{222} irradiation field or left untreated (control). Respirable aerosols containing allergens were subsequently collected by condensation capture at 10 min intervals over the course of an hour to evaluate allergen stability. Aeroallergens were quantified using an antibody-based immunoassay, which relies on intact protein conformation for antibody-allergen recognition, binding, and quantification. In a time frame relevant to indoor air exchange rates (30 min), statistically significant reductions in airborne allergen levels were observed in response to UV_{222} doses $\leq$ 16.8 mJ/cm^2 when compared to otherwise identical control conditions. These results suggest that UV_{222} may be engineered for use as an aeroallergen intervention strategy.



KEYWORDS: *aeroallergens, far-UVC, allergen intervention, indoor air quality, asthma, allergy*

INTRODUCTION

Allergen exposures contribute to severe negative health effects worldwide, and nearly one-third of US adults and children have been diagnosed with an allergic condition.[1,2] Many allergens can manifest within aerosols (called aeroallergens), which are often enriched in indoor environments and pose respiratory exposure hazards. Common aeroallergens are most frequently proteins derived from a variety of indoor and outdoor sources, including arthropods (dust mites, cockroaches), mammals (cats, dogs, mice, rats), fungi, and plants.[3−6] Most allergic reactions are the result of the human immune system recognizing and responding to one or more exogenous protein allergen(s) from these sources, although other nonprotein allergens may also trigger allergic responses. The immune system produces immunoglobulin E (IgE) antibodies that each recognize and bind to a specific region of an allergenic protein's three-dimensional structure, called the allergen epitope. In sensitized individuals, IgE antibody-allergen epitope "immunorecognition" initiates a pro-inflammatory immune cascade that leads to allergic reactions.[7,8]

Airborne allergen exposure reactions range from mild to severe, including sneezing, swollen sinuses, coughing, and difficulty breathing. Repeated exposure to aeroallergens is associated with development and/or exacerbation of asthma, which affects 262 million people worldwide, and contributes to approximately 1,000 deaths each day across the globe.[3−6] Aeroallergens often trigger asthma attacks, which were associated with 939,000 emergency department visits and 3602 deaths in 2022 in the US.[9] American economic losses associated with asthma were estimated at nearly $82 billion from 2008 to 2013, highlighting the need to reduce exposure to asthma triggers and develop more effective aeroallergen control strategies.[10]

Current strategies to manage respiratory allergies and asthma are complex, involving a combination of prevention, medication, engineering controls, and other interventions. Reducing exposure to airborne allergens is one approach to mitigate allergy and asthma symptoms; however, unlike other bioaerosols containing airborne bacteria and viruses, aeroallergens are nonviable macromolecules that pose unique monitoring and control challenges.[11−15] They are often structurally stable and can persist in indoor environments for years, yet methods to monitor aeroallergens, including

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sampling, processing, and quantification, are not standardized. Indeed, most of the work investigating environmental and indoor aeroallergens does not directly measure allergens within the air, instead relying on indirect measures, such as assessing allergen surrogates (mold spore counts, pollen granule counts) or evaluating allergen levels within settled dust. As many studies do not directly assess aeroallergens within the dynamic, respirable fraction of air, there is limited understanding of the airborne levels of allergens and the complexities of real-world exposure through inhalation.[11−15]

Although no exposure limits have been established for environmental or indoor airborne allergen levels, occupational settings offer some guidance. Direct aeroallergen air monitoring in workplaces where airborne allergen exposures may pose a respiratory risk to workers, such as in latex glove manufacturing, detergent enzyme production, laboratory animal handling, and grain and flour processing, has demonstrated that sensitization and allergy symptom-inducing concentrations likely occur when aeroallergens are present in the nanogram per cubic meter of air (ng/m^3) range. Therefore, to reduce sensitization risks and the development or exacerbation of occupational allergy and asthma, these specific work settings often aim to maintain airborne allergen concentrations below 10 ng/m^3.[16−19] These workplace airborne allergen levels associated with sensitization and allergic symptoms provide important context to evaluate exposure−response relationships and to understand the efficacy of allergen-intervention strategies within indoor environments.

Despite decades of research, effective and reproducible intervention strategies aimed at reducing environmental aeroallergen exposures, specifically in homes, have shown mixed results.[13−15,20−22] Overall, implementation of a single intervention strategy is generally not associated with health improvements or linked to reduced allergen levels in the environment; however, multifaceted interventions tend to reduce allergens and improve asthma and allergy symptoms.[13−15,20−22] For example, Bjornsdottir et al. demonstrated that eight months of implementing rigorous environmental control measures within the homes of cat-allergic individuals reduced settled dust levels of the cat allergen Fel d 1, and this reduction led to measurable clinical improvements. However, only 31 out of 219 volunteers completed the study due to the difficulty of sustaining the intensive intervention measures, including removing carpets, washing walls, vacuuming weekly, washing bedding at high temperatures, and biweekly cat bathing.[22] This highlights a critical challenge for developing aeroallergen mitigation strategies: interventions must be practical, effective, and multilayered. Moreover, both the settled dust fractions and the airborne allergen fractions must be targeted holistically, and more intervention strategies should be developed to target airborne allergens, which is the fraction most relevant to respiratory health.

Our current work addresses this gap by investigating ultraviolet (UV) light in this context, specifically "occupant safe" far-UVC centered at 222 nm (UV_{222}). We explore how UV_{222} may be used to reduce airborne allergens as measured by commercial immunoassays, which utilize antibody-allergen epitope binding, similar to the human allergic response, to quantify protein allergens directly from air. Conventional UV treatment of indoor air and surfaces is often distinguished by "germicidal" bandwidths centered around 254 nm (UV_{254}), which is absorbed by nucleic acids and causes lethal genomic damage. However, UV_{254} exposure to human skin and eyes can cause serious tissue damage; therefore, indoor UV_{254} applications are often used in unoccupied spaces or with protections that limit occupant exposure.[23,24] Narrow bandwidth UV_{222} also inactivates airborne microorganisms and is considered safer than UV_{254} for extended occupant exposures, as it causes less DNA and skin damage than UV_{254} and does not penetrate deep into human skin or eye tissues; however, further studies are suggested to understand its impact on skin regeneration.[23−27] Proteins strongly absorb UV_{222} light, which likely leads to photooxidation, cross-linking, and structural changes of proteins following UV_{222} exposure, altering enzymatic and structural functions, and causing lethal damage to microorganisms and minor damage to skin.[24,26−30] Although there has been some work investigating food processing methods that implement pulsed UV light (broad spectrum 100−1100 nm) to reduce foodborne allergens in soy, peanuts, and shrimp products,[31−33] there has been no investigation to date exploring the effects of UV_{222} light treatments on airborne allergens contained in respirable particles ($\leq$10 μm).

Therefore, we hypothesized that airborne protein allergens exposed to UV_{222} undergo structural changes that reduce antibody-allergen epitope binding, decreasing quantification of aeroallergens as measured by the commercially available antibody-based Multiplex Array for Indoor Allergens (MARIA, Indoor Biotechnologies (InBio); Charlottesville, VA),[34] which was used to measure up to seven allergenic proteins simultaneously for this work. We developed a controlled 10 m^3 chamber model to generate respirable aeroallergens at levels associated with sensitization, allergy, and asthma. We then evaluated the impact of UV_{222} on common airborne allergens, and used time-resolved condensation capture to collect respirable particles containing aeroallergens and measure them via the MARIA immunoassay. We observed significant UV_{222}-dependent reductions in airborne allergens averaging 20−25% following UV_{222} doses below the American Conference of Governmental Industrial Hygienists (ACGIH) established threshold limit value for skin exposure of 480 mJ/cm^2 and eye exposure of 160 mJ/cm^2,[35] where the majority of aeroallergen reductions occurred within 30 min of air treatment. Importantly, UV_{222}-dependent reductions were within time frames relevant to indoor air exchange rates and were comparable to other long-term allergen intervention studies that demonstrated allergy symptom relief in sensitized individuals. These findings suggest that UV_{222} exposure can reduce allergen immunorecognition within respirable particles, supporting its use as an integrated strategy for indoor aeroallergen control.

MATERIALS AND METHODS

Allergen Source Materials. All allergens described here are referenced in the World Health Organization/Union of Immunological Societies (WHO/IUIS) nomenclature database.[36] Allergens were sourced from InBio or generated in the laboratory. *Aspergillus fumigatus* ATCC 1022 (ATCC, Manassas, VA) cultures were grown in Yeast Peptone Dextrose (YPD) broth at room temperature (20−22 °C). The secreted *A. fumigatus* allergen Asp f 1 was measured by enzyme-linked immunosorbent assay (ELISA; InBio) within culture supernates that were harvested after 30 days of growth by decanting culture media from the mycelium. Supernates were filtered through a 0.22 μm PVDF membrane and stored at 4 °C for

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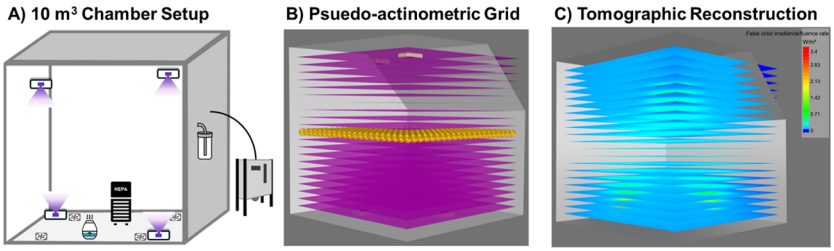

Figure 1. Environmentally controlled 10 m^3 bioaerosol chamber description and UV_{222} fluence modeling. All allergen-containing aerosols in this study were contained in a temperature- and humidity-controlled, well-mixed 10 m^3 chamber. The left section (A) is a schematic of the experimental chamber including four KrCl UV_{222} lights symmetrically juxtaposed in quadrants on the chamber ceiling and floor. HEPA filtration was performed before and after each experiment. Allergen-containing aerosol was introduced with a six-jet Collison precious fluids nebulizer and collected with BioSpot condensation capture. UV_{222} lamp emission measurements and aerosol exposure modeling were performed by LTI Optics, and the average UV_{222} doses (or fluences) that (bio)aerosol moving through the chamber experienced was estimated using Photopia software modeling of the well-mixed chamber condition. The middle section (B) outlines the average fluence rate of 10.2 $\mu W/cm^2$, which was calculated by splitting the chamber into quadrants outfitted with an array of 1458 actinometric spheres (exemplar in gold) that comprised a symmetrical fluence grid. The right section (C) is a tomographic reconstruction of the UV field, which was calibrated with boundary conditions obtained from semispherical (180°) measurements of UV_{222} light emissions from the KrCl lamps.

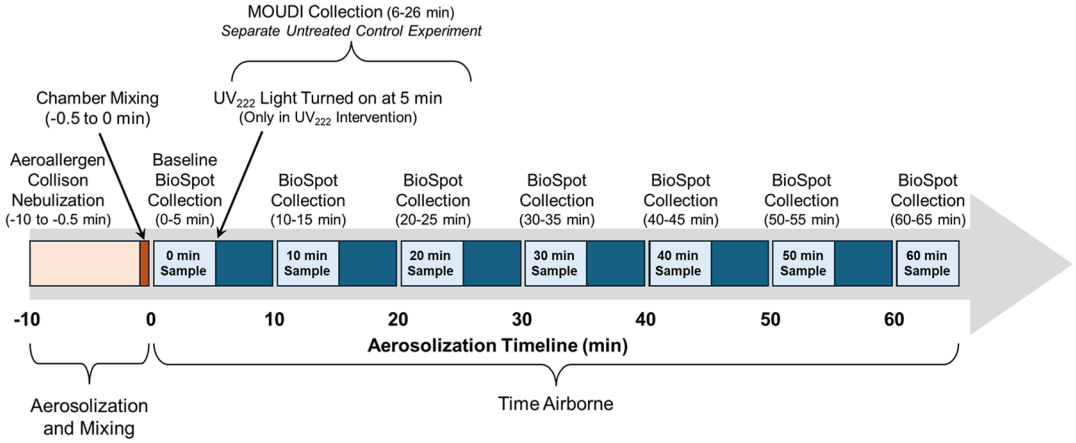

Figure 2. Aerosolization timeline and bioaerosol collection. Aeroallergens were generated from dustborne or purified sources using a six-jet precious fluids Collison nebulizer in a controlled 10 m^3 chamber. Composite aerosol samples were collected every 10 min for 5 min each across the 65 min time course using a BioSpot VIVAS 310. Following baseline 0−5 min sampling, UV_{222} lamps were turned on or no treatment was applied to the control chamber runs. In a separate set of experiments to characterize particle size distribution, aeroallergens from dustborne or purified sources were nebulized identically to the control BioSpot time course experiments described above, and a composite sample was collected by MOUDI from 6 to 26 min for each allergen source material.

short-term assessment (days) or −20 °C for long-term archives. Filtered supernates contained 20−80 μg Asp f 1/ml.

Dust-allergen conglomerates were purchased (InBio) containing Der p 1 (European house dust mite), Der f 1 (American house dust mite), Can f 1 (domestic dog), Fel d 1 (domestic cat), Phl p 5 (Timothy grass), and Bet v 1 (European white birch). Dustborne allergen concentrations ranged between 10 and 30 μg each allergen per gram of dust. Dust was sequentially sieved through American Society for Testing and Materials (ASTM) #20 (850 μm) and #40 (425 μm) screens to prevent clogging of the Collison nebulizer, and sieved dust was resuspended in phosphate-buffered saline (PBS; 137 mM NaCl, 2.7 mM KCl, 10 mM Na_2HPO_4, 1.8 mM KH_2PO_4) to a final dust concentration of 100 mg dust/ml and final allergen concentrations 1−3 μg/mL. Asp f 1 from *A. fumigatus* supernates described above were also added to the dustborne allergens for a final concentration of 1−3 μg Asp f 1/ml. Purified allergens (>95% pure), including natural Der p 1, natural Der f 1, natural Can f 1, natural Fel d 1, and natural Bet v 1, were purchased from InBio. Before aerosolization, purified allergens were thawed on ice then resuspended in stabilization buffer (PBS + 1% w/v Bovine Serum Albumin Fraction V (BSA; Sigma-Aldrich, St. Louis, MO) + 0.02% v/v Tween-20 (Sigma-Aldrich)) to final allergen concentrations 1−3 μg/mL.

Aeroallergen Generation and Collection. Aeroallergen experiments were performed in a well-mixed, temperature- and relative humidity (RH)-controlled (20−23 °C; 58−62% RH), 10 m^3 chamber (Figure 1A) as previously applied to observe airborne virus persistance.[37] In general, it is recommended to maintain 40−60% RH indoors for comfort and health; however, several allergens evaluated for these studies are more abundant in environments >50% RH, such as dust mite and mold allergens.[38−40] Therefore, the chamber was humidified to 60% RH for these experiments to represent a controlled environment where these aeroallergens are more likely to be present and pose a respiratory risk.

Before and after each allergen aerosol chamber run, airborne particles were removed from the chamber with HEPA filtration, and chamber aerosol content was confirmed to be <2 particles/L with a fluorescence-based optical particle counter (DetectionTek LLC, Boulder, CO). As outlined in

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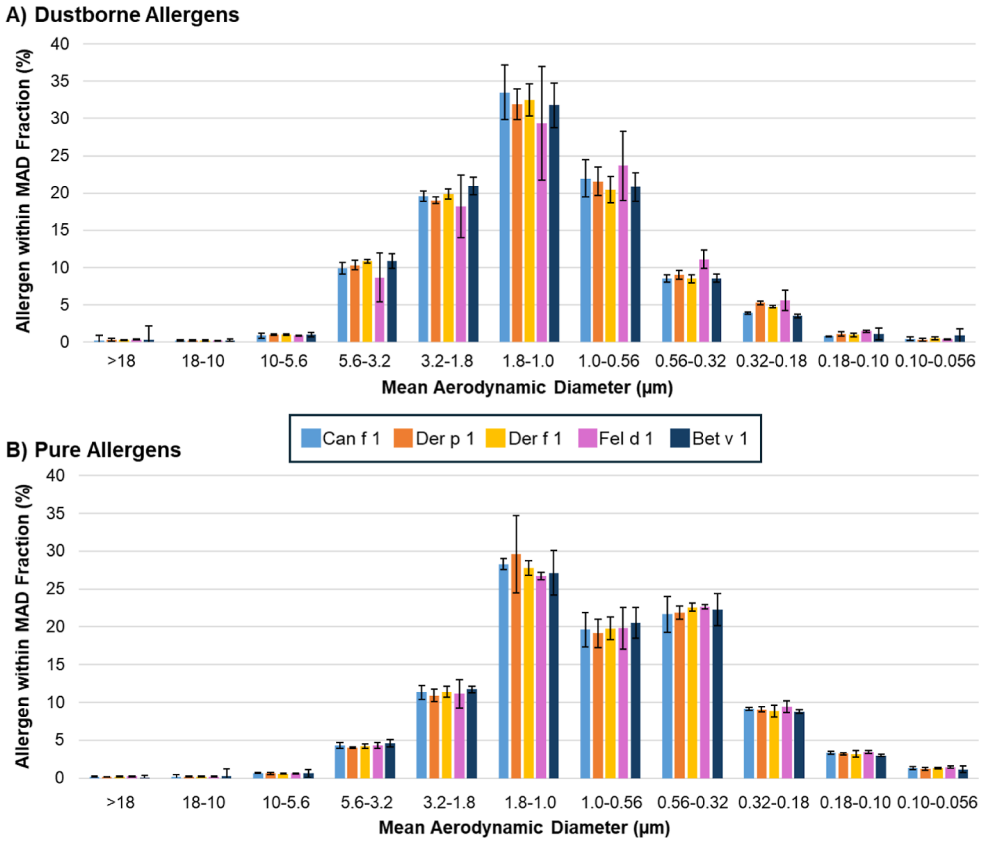

Figure 3. Particle size distribution of allergen-containing aerosols from both allergen source materials. Airborne allergens were collected using a MOUDI and resolved into 11 MAD bins. Total allergen levels across all MAD distribution sizes were added together, and the percent of each allergen found within each MAD distribution was plotted for (A) aeroallergens generated from dustborne allergens in PBS, or (B) aeroallergens generated from purified protein dissolved in PBS. Error bars represent percent standard deviation across analytical triplicate samples from undiluted, 1:5, or 1:20 dilutions. Over 99% of allergen-containing particles were ≤ 10 μm; average relative allergen concentration within any given bin was within $\pm$10% for each allergen, and no single MAD bin contained evidence of relative allergen enrichment.

the aerosolization timeline scheme in Figure 2, allergens were introduced to the chamber air from their resuspended source solutions (dustborne vs purified allergens), using a six-jet Collison precious fluids nebulizer, which generates polydisperse aerosol of a mean aerodynamic diameter (MAD) of 2.5 μm and a geometric standard deviation of 1.8, according to the manufacturer (CH Technologies, Westwood, NJ). The aerosolization time course began by nebulizing 5 mL of either allergen source solution at 20 psi with compressed air (AI B300, Airgas, Radnor, PA) for 9.5 min. Aerosol was mixed in the chamber with four small, floor-mounted 4 W fans during nebulization and for an additional 30 s after nebulization for a total of 10 min of generating and mixing aerosol before starting a "time airborne" clock and beginning sample collection (Figure 2).

A BioSpot VIVAS 310 (Aerosol Devices Inc., Fort Collins, CO) was used to serially collect composite samples of chamber air at a flow rate of 14.5 L per minute (LPM), which was confirmed using a calibrated DryCal Defender volumetric flow meter (Mesa Laboratories, Lakewood, CO). The temperature array of the BioSpot condensation growth tube was set to 5, 45, 18, or 25 °C for the conditioner, initiator, moderator, or nozzle temperatures, respectively. Particles were collected every 10 min for 5 min over the course of 65 min, resulting in seven composite aerosol samples for each experimental chamber run (Figure 2). Each composite aerosol sample was collected in the BioSpot reservoir containing 1 mL of PBS (dustborne allergens) or 1 mL stabilization buffer (purified allergens); aerosol samples were immediately placed on ice following collection, and allergen levels were quantified by MARIA within 4 h to prevent any analytical artifacts associated with potential protein degradation over time.

In separate control pilot experiments, a Micro-Orifice Uniform Deposit Impactor (MOUDI) model 110 (TSI Inc., Shoreview, MN) was used instead of a BioSpot to collect and separate the Collison nebulizer-generated, allergen-containing aerosols by their MAD. This was performed to characterize the particle size distribution for both allergen source materials used for this work and to determine if any size fractions were enriched with any specific allergen—important context that could be used to understand potential differences between allergen stability or susceptibility to UV_{222} light. Unlike the BioSpot time-course series, MOUDI collection cannot take multiple samples in rapid succession; therefore, a 20 min composite sample representing 6−26 min time airborne was collected for each source material (dustborne vs purified) to reflect the particles sizes and aeroallergen content within each MAD that would first be exposed to UV_{222} light (Figure 2).

In two control chamber runs, dustborne or purified allergens were nebulized as described above, and airborne particles were collected at a flow rate of 30 LPM to separate particles into size fractions based on their MAD, and allergen-containing particles were deposited onto Teflon collection substrates at each impactor stage. Following 20 min of collection

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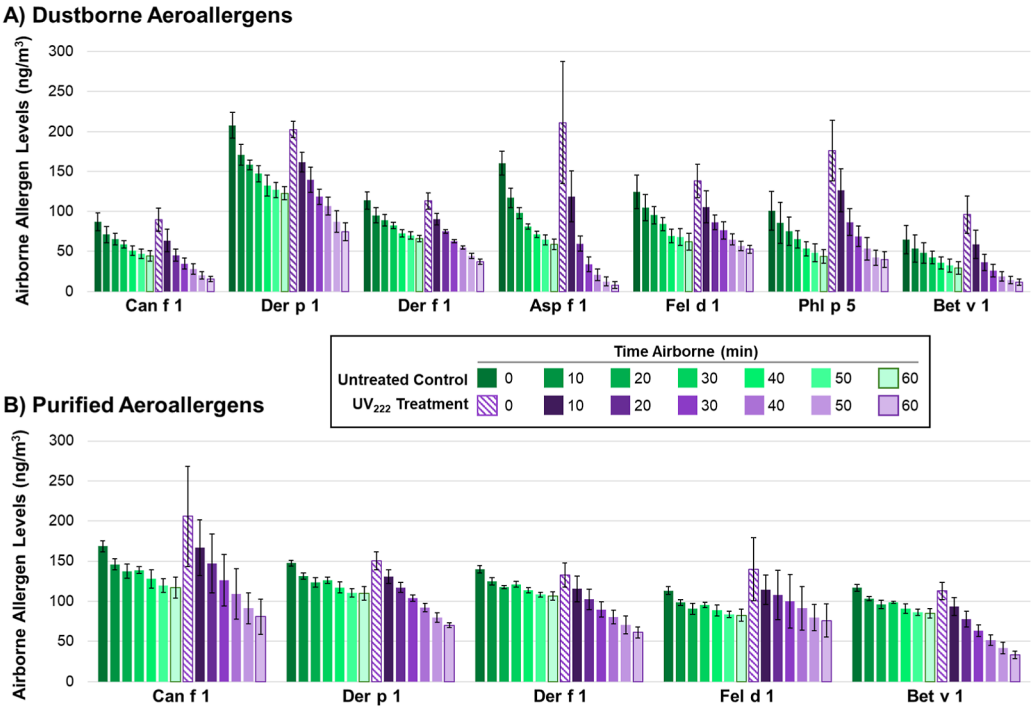

Figure 4. Airborne allergen levels across 10 m^3 chamber runs. The airborne allergen levels across experiments were calculated using the BioSpot collection parameters, collection volume, and MARIA allergen concentrations to convert each allergen into ng/m^3 air. Triplicate chamber runs were performed under control or UV_{222} treatment conditions using source materials from (A) dustborne allergens or (B) purified allergens. Each time point represents pooled analytical triplicates from three independent chamber runs (n = 9 per time point), represented as the mean airborne levels for each allergen ± standard deviation across runs.

corresponding to 6−26 min time airborne (Figure 2), the chamber was cleaned as described above, the MOUDI was disassembled, and aeroallergens from each MOUDI stage were extracted using 1 mL stabilization buffer, placed on ice, and immediately quantified using MARIA in analytical triplicate, as described below. The amount of each allergen within each stage was quantified and added together across stages to determine the total amount of each allergen collected by MOUDI. The percentage of each allergen within each MAD fraction size was then calculated by dividing the allergen amount within each stage by the total allergen observed across all the MOUDI stages and multiplied by 100 (Figure 3).

UV_{222} Intervention Treatment. Krypton chloride (KrCl) lamps (Far UV Technologies, Kansas City, MO; Ushio Care222-B1 222 nm filtered lamp module in Krypton 36) were used for UV_{222} chamber treatments. Four lamps were placed in a symmetric array: two were centered in diagonal quadrates on the ceiling pointing down, and the other two were placed in opposite quadrants on the floor of the chamber pointing upward (Figure 1A). Each 10.5 W lamp provided 99 mW total radiant watts, as averaged through a 180° irradiance scan using accepted methods. The lamps were activated at 5 min time airborne, immediately following the collection of the baseline aerosol sample (0−5 min), and they remained on for the subsequent hour, during which the UV_{222}-exposed aerosol samples were acquired (Figure 2). The spherical irradiance distribution in the chamber was reported based on the modeling calibrated measurements of USHIO lamp output (99 mW) using Photopia software (LTI Optics, Louisville, CO) and compared to default lamp outputs using Illuminate shareware (illuminate.osluv.org) with a 5% chamber surface reflectance imposed on the cubic chamber dimensions. The averaged fluence reported by the respective models were within 12% of each other. A tomographic rendition of the UV exposure field is presented in Figure 1B,C. As a result of the modeling, 10.2 $\mu W/cm^2$ average fluence rate was used for all exposure calculations reported here.

Commercial KrCl UV_{222} lamps can generate ozone;[41] thus, chamber ozone levels were monitored using a calibrated, single beam ozone absorbance instrument (model 106-L Ozone Monitor, 2B Tech, Broomfield, CO). Following 60 min of UV_{222} lamp operations, chamber ozone levels increased 60−80 ppb over background levels. Although ozone exposure is outside the scope of this work, these levels are near or above the environmental and occupational ozone exposure limits, which are 70 and 100 ppb, respectively;[42,43] thus, when implementing UV_{222} treatment in low ventilation conditions, operators should consider ozone monitoring.[41] To understand if these ozone levels were a process variable that could impact airborne allergen stability during UV_{222} exposures, a pilot chamber run was performed under low oxygen (O_2) conditions to reduce ozone formation during treatment. Before aerosolization, the chamber was purged with nitrogen until chamber air contained < 5% O_2, as verified by a calibrated oxygen monitor (Goyojo, Shenzhen, PRC), and dustborne allergens were aerosolized as described above, except that allergen nebulization used nitrogen instead of air. Under low oxygen conditions, ozone levels did not increase above background during UV_{222} treatment. Aeroallergen reductions in response to UV_{222} under low oxygen conditions showed similar reductions to those observed following UV_{222} treatment with ambient oxygen levels, effectively removing ozone as a process variable (Figure S1).

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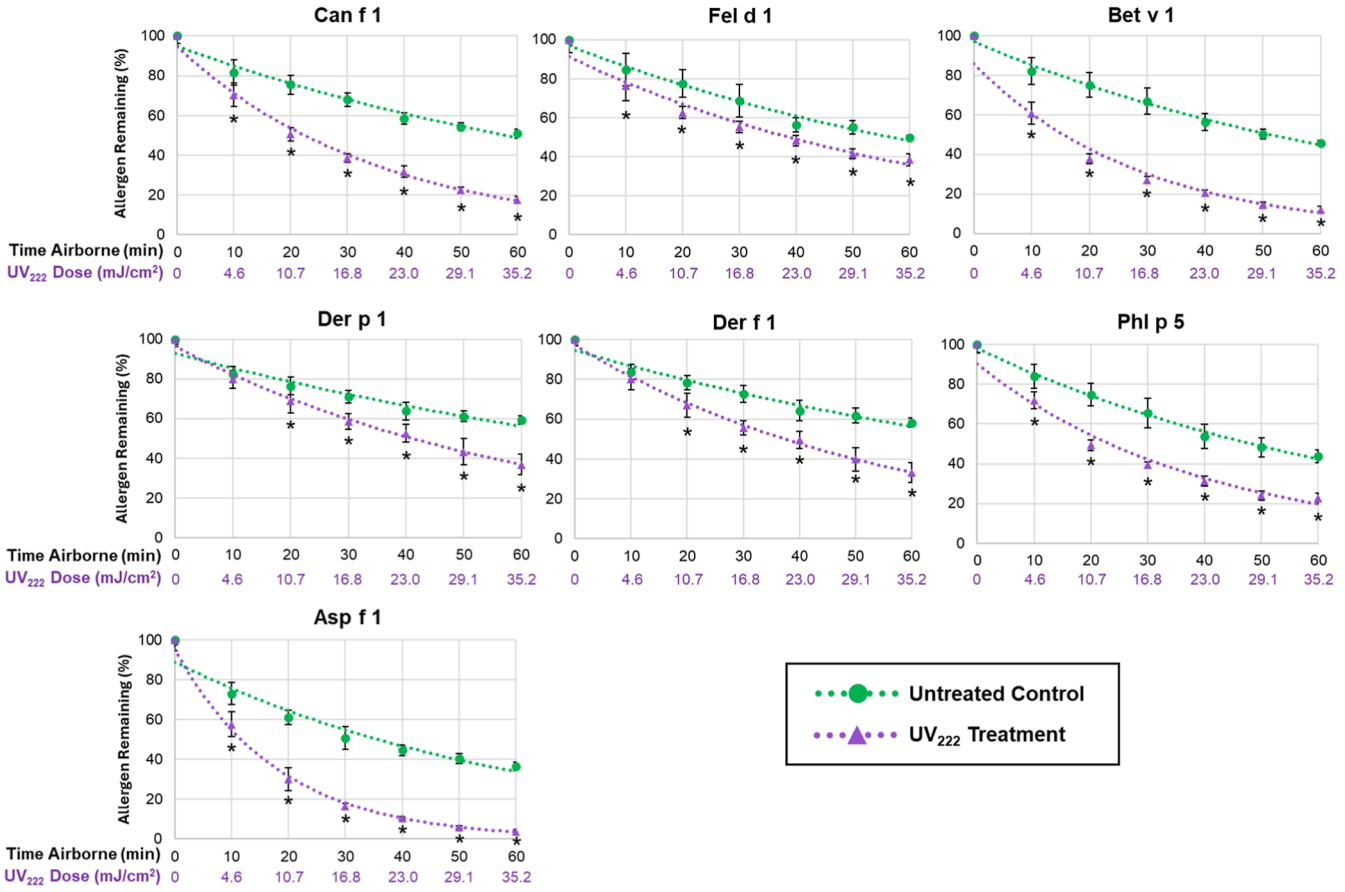

Figure 5. Dustborne aeroallergens are significantly reduced following UV_{222} air treatment as measured by immunoassay. Allergen-laden dust was resuspended in PBS and nebulized into respirable particles contained in a controlled 10 m^3 chamber. Airborne allergens were quantified by MARIA in a time-series of aerosol samples collected every 10 min over 65 min. Following baseline sample collection, airborne allergens were continuously exposed to UV_{222} or were maintained under otherwise identical environmental conditions as an untreated control. Data represent averages ± standard deviation of the percent allergen remaining at each time point of three independent experimental chamber trials, each incorporating MARIA analytical triplicates at every time point (n = 9). As evaluated by a two-tailed Student's t-test, an asterisk (*) indicates percent allergen reduction levels within the UV_{222} treatment group that are significantly different from the control group at that time point ($p < 0.05$).

Aeroallergen Quantification by MARIA Immunoassay. Five- or 7-plex MARIA assays were used to simultaneously quantify Der p 1, Der f 1, Can f 1, Fel d 1, Asp f 1, Phl p 5, and Bet v 1 within each sample using a MAGPIX multiplex immunoarray instrument (Luminex, Austin, TX). The MAGPIX was calibrated weekly following the manufacturer's instructions, and QC standards (InBio) were used to confirm allergen detection was within acceptable analytical thresholds. Following the manufacturer's instructions, standard allergen curves were generated using a five-parameter logistic (5PL) model, and each undiluted BioSpot sample was quantified by MARIA in triplicate. Lower limit of detection (LLOD) ranges for the MARIA were between 0.02 and 0.2 ng allergen/ml depending on the specific allergen, corresponding to an LLOD of 0.3−3 ng allergen/m^3 within BioSpot samples collected.

Data Processing and Statistical Analyzes. Unless otherwise noted as a pilot experiment, all chamber runs were performed in experimental triplicate for each condition and allergen source material. To understand the range of aeroallergen levels across experiments and compare between source materials, the BioSpot MARIA allergen levels (ng/mL) were converted to airborne concentrations (ng/m^3) using the BioSpot collection parameters (14.5 LPM x 5 min) and collection volume (1 mL). The averages and standard deviation of airborne allergen levels (ng/m^3) were calculated by pooling all analytical triplicates from three independent chamber runs (n = 9) for each time point under each condition (control vs UV_{222}) and for each source material (dustborne vs purified).

As seen in Figure 4, there was some variability in aeroallergen baseline levels (ng/m^3) across chamber runs for both aeroallergen source materials and for each condition. Therefore, baseline normalization was performed for each chamber run experimental replicate to normalize each time point as a percent reduction of the original baseline levels for each allergen. First, the MARIA analytical replicates were averaged for the 0−5 min baseline sample for each run to establish the average allergen baseline levels for each allergen. Then, each analytical replicate for each time point in that chamber run was converted to a percent reduction of the average allergen baseline. This was performed for each chamber run's analytical replicates at every time point for each allergen using the equation

$$\left(\frac{(\text{Avg. allergen baseline}) - (\text{allergen at time point } x)}{\text{Avg. allergen baseline}}\right) \times 100 = \text{percent allergen reduction at time point } x$$

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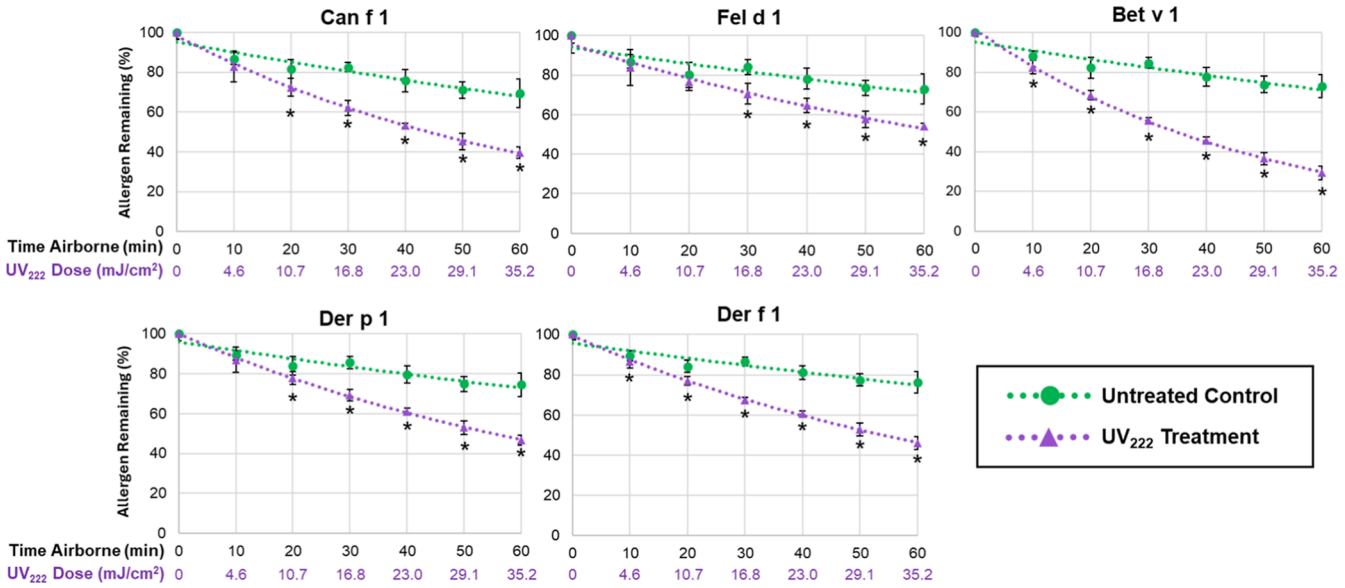

Figure 6. Purified aeroallergens are significantly reduced following UV_{222} air treatment as measured by immunoassay. Commercially purified allergens, resuspended in a stabilization buffer (PBS + 1% BSA + 0.02% Tween-20), were nebulized into respirable particles contained in a 10 m^3 chamber. Airborne allergens were quantified by MARIA in a time-series of aerosol samples collected every 10 min over 65 min. Following baseline sample collection, airborne allergens were continuously exposed to UV_{222} or were maintained under otherwise identical environmental conditions as an untreated control. Data represent averages ± standard deviation of the percent allergen remaining at each time point of three independent experimental chamber trials, each incorporating MARIA analytical triplicates at every time point (n = 9). As evaluated by a two-tailed Student's t-test, an asterisk (*) indicates percent allergen reduction levels within the UV_{222} treatment group that are significantly different from the control group at that time point ($p < 0.05$).

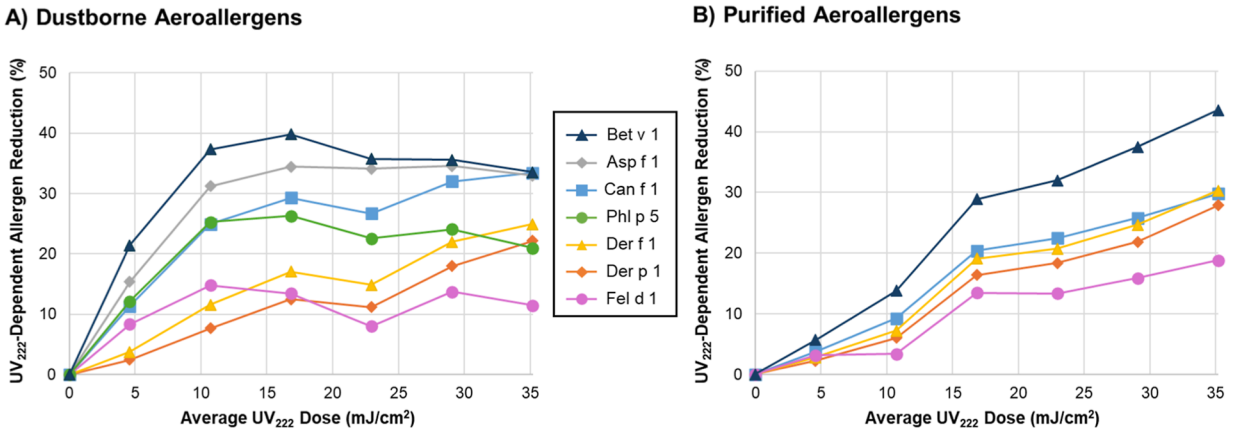

Figure 7. UV_{222}-dependent aeroallergen reductions as a function of fluence dose. UV_{222}-specific aeroallergen reductions were determined by subtracting the average percent reduction observed under control conditions from the average percent reduction observed at the corresponding time point under UV_{222} treatment conditions and plotted against the corresponding average UV_{222} dose (mJ/cm^2). The average UV_{222}-dependent aeroallergen percent reductions for each allergen at each time point are presented for (A) dustborne allergens aerosolized in PBS or (B) purified allergens aerosolized in stabilization buffer.

Following baseline normalization and conversion to percent reductions for each chamber run, the percent allergen reduction was averaged across the triplicate analytical samples and triplicate experimental chamber runs for each time point, condition (control vs UV_{222} treatment), source material (dustborne vs purified), and allergen. The standard deviation of the percent reduction was also calculated across experimental and analytical replicates using these normalized percent allergen reduction values. A two-tailed Student's t-test was used to compare the percent allergen reduction under UV_{222} exposure vs control conditions at each corresponding time point across experimental and analytical replicates for each allergen source material, and significant differences between the control vs UV_{222} at each time point were determined at a threshold p-value < 0.05. For visualization purposes, each aeroallergen's reduction data were converted to a percentage of aeroallergen remaining, compared to baseline at each time point (100% − percent allergen reduction at time point x = percent allergen remaining at time point x), plotted for the untreated control (green lines) or UV_{222} treated conditions (purple lines), and separated by dustborne allergens (Figure 5) or purified allergens (Figure 6).

As expected, airborne allergen levels gradually decreased over time in the untreated controls for both allergen source materials due to deposition and natural decay (Figures 5 and 6). To determine the enhanced allergen reductions attributable

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Table 1. UV_{222}-dependent Percent Aeroallergen Reductions for Dustborne or Purified Aeroallergens

		UV_{222}-Dependent Dustborne Aeroallergen Reduction (%)							
Avg. Dose (mJ/cm^2)	Time Point (min)	Can f 1	Der p 1	Der f 1	Asp f 1	Fel d 1	Phl p 5	Bet v 1	Avg. All Allergens
4.6	10	11.4	2.5	3.8	15.4	8.4	12.2	21.4	10.7
10.7	20	25.0	7.7	11.6	31.2	14.8	25.3	37.3	21.8
16.8	30	29.3	12.5	17.1	34.5	13.4	26.3	39.8	24.7
23.0	40	26.7	11.2	14.9	34.1	8.1	22.6	35.8	21.9
29.1	50	32.0	18.0	22.0	34.6	13.7	24.1	35.6	25.7
35.2	60	33.4	22.2	25.0	33.0	11.5	21.0	33.5	25.6

		UV_{222}-Dependent Purified Aeroallergen Reduction (%)					
Avg. Dose (mJ/cm^2)	Time Point (min)	Can f 1	Der p 1	Der f 1	Fel d 1	Bet v 1	Avg. All Allergens
4.6	10	3.8	2.2	2.8	3.2	5.7	3.6
10.7	20	9.2	6.0	7.2	3.4	13.8	7.9
16.8	30	20.4	16.4	19.1	13.5	28.9	19.7
23.0	40	22.5	18.4	20.7	13.3	32.0	21.4
29.1	50	25.8	21.9	24.7	15.9	37.5	25.1
35.2	60	29.8	27.9	30.3	18.8	43.6	30.1

to UV_{222} treatment, each allergen's control percent reduction was subtracted from the percent reduction in the UV_{222} samples at each corresponding time point (Figure 7 and Table 1). To understand the dose-dependent UV_{222} effects on airborne allergen reductions, the average fluence dose (mJ/cm^2) was calculated for each 5 min composite UV_{222} sample. Since the UV_{222} light was activated at 5 min (Figure 2), the 10 min UV_{222} sample, representing a composite aerosol collection from 10 to 15 min time airborne, was exposed to UV_{222} for 5−10 min. Based on UV_{222} fluence modeling above, the average fluence rate was 10.2 $\mu W/cm^2$; therefore, the UV_{222} dose after 5 min of exposure was 3.1 mJ/cm^2 and the UV_{222} dose at 10 min of exposure was 6.1 mJ/cm^2 (and so on); thus, the average UV_{222} dose for the 10 min UV_{222} sample was 4.6 mJ/cm^2. This approach was used to calculate the average dose for each UV_{222}-treated time point, and the UV_{222}-dependent aeroallergen reductions were visually plotted as a function of UV_{222} dose for each source material (Figure 7) or represented in a tabular format (Table 1).

RESULTS AND DISCUSSION

Respirable Airborne Allergens Generated for This Study Reflect Real-World Allergenic Levels. As expected, > 99% of the allergen-containing particles generated by the Collison nebulizer were ≤ 10 μm, regardless of allergen source material, confirming the manufacturer-specified MAD distribution (CH Technologies), and corroborating similar bioaerosol-generating methods using this nebulizer.[44,45] Aerosols generated from pure allergens demonstrated a slight shift toward smaller MAD bins with respect to their dustborne counterparts, potentially due to the higher densities of the dustborne conglomerates. Importantly, these MOUDI pilot experiments confirmed that the aeroallergen-containing aerosol generated for these experiments were within a respirable particle size range and that no single allergen was enriched within any MAD fraction (Figure 3).

For both allergen sources, airborne allergen baseline levels were approximately 50−200 ng/m^3 on average in control and UV_{222}-treated conditions (Figure 4). There was slightly more variation between different aeroallergens generated using the heterogeneous dust mixture (Figure 4A) as compared to the baseline aeroallergen levels generated from purified allergens (Figure 4B). This was likely due to the variation of allergen levels within the source material itself. Despite the small differences in baseline levels of airborne allergens between allergen source materials, all airborne allergen levels represent levels that have been associated with sensitization and allergic disease in indoor environments and occupational settings, thus the respirable aeroallergen levels generated for these controlled chamber experiments represent levels relevant to real-world exposures.[16−19,46−49]

Aeroallergen Stability Generated from Different Source Materials and Carrier Aerosols. It is important to compare the airborne stability of heterogeneous dustborne allergen conglomerates to that of their purified counterparts, as environmental dusts may contain components that promote the decay of allergens, or conversely, stabilize allergens. For example, components of pet dander may stabilize cat or dog protein allergens, or dust may harbor proteases that may degrade allergens. Therefore, we compared the airborne allergen decay rates under control conditions from two different allergen source materials (dustborne vs purified aeroallergens). As anticipated, regardless of UV_{222} exposure, airborne allergen levels decreased over the observation period, likely due to a combination of deposition, desiccation, oxidation, and/or other environmental factors that may contribute to airborne protein denaturation and/or degradation (Figures 5 and 6, green lines).

Some allergens appeared to be more stable in an airborne state than others. For example, in control dustborne aeroallergen experiments, dust mite allergens Der f 1 and Der p 1 remained at about 60% of their baseline levels in the 60 min sample, yet dustborne Asp f 1 mold allergen was only present at 37% of its baseline level during the same period (Figure 5, green lines).

Purified allergens nebulized in stabilization buffer (PBS + 1% BSA + 0.02% Tween-20) remained relatively stable in control conditions, with allergen levels averaging 69−76% remaining of their baseline levels in the 60 min sample (Figure 6, green lines). However, pilot experiments that removed the stabilizing

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agents and nebulized purified allergens in PBS alone demonstrated a loss of the ability to measure Der p 1 and Der f 1 within BioSpot samples, as their levels were below the LLOD in all aerosol samples collected under both control and UV_{222} treatment chamber runs (<3 ng/m^3; Figure S2). Interestingly, purified Fel d 1 cat allergen remained relatively stable in the air under control conditions when nebulized in either stabilization buffer or in PBS alone (Figures 6 and S2, green lines).

These results suggest that some allergens may be inherently more stable in the air than others, likely due to complex internal interactions of the proteins (hydrogen bonds, disulfide bonds, ionic interactions, hydrophobic interactions, etc.) as well as interactions with the external environment (mechanical forces, desiccation, oxidation, stabilizing factors, etc.). Although outside of the scope of these experiments, further investigation into the role of these internal and external factors and their effects on aeroallergen stability under normal conditions is warranted.

UV_{222} Exposure Significantly Reduces Immunodetection of Respirable Aeroallergens. With respect to otherwise identical untreated control conditions, UV_{222} doses $\geq$ 16.8 mJ/cm^2 significantly reduced immunoassay detection of all airborne allergens tested in dust conglomerates and in purified form. Importantly, several aeroallergens showed statistically significant reductions with UV_{222} doses as low as 4.6 mJ/cm^2 (Figures 5 and 6, purple lines). These effective doses of UV_{222} are well below the ACGIH established threshold limit for skin (480 mJ/cm^2) and eyes (160 mJ/cm^2).[35] UV_{222}-dependent decreases in aeroallergens were calculated by subtracting the percent reductions of the UV_{222}-treated samples from the control percent reductions (i.e., the difference between the green lines and the purple lines in Figures 5 and 6). As presented in Figure 7 and outlined in more detail in Table 1, aeroallergen levels showed a UV_{222}-dependent decrease of 20−25% on average by the 30 min sample (25−30 min of UV_{222} exposure averaging 16.8 mJ/cm^2). Airborne Fel d 1 appeared to be the least impacted by UV_{222} treatment, regardless of allergen source material, and the birch allergen Bet v 1 was the most sensitive to UV_{222} exposure when aerosolized in dust or purified form (Figure 7 and Table 1). Overall, dustborne aeroallergens decreased more rapidly in response to UV_{222} doses $\leq$ 16.8 mJ/cm^2, but treatments > 16.8 mJ/cm^2 showed (mixed) additional UV_{222}-dependent reductions in aeroallergen levels (Figure 7A and Table 1A). Conversely, purified allergens were less impacted by lower UV_{222} doses than dustborne allergens, but they demonstrated more consistent reductions in response to increasing UV_{222} doses (Figure 7B and Table 1B).

As mentioned above, Fel d 1 from both dustborne and purified sources was the least impacted by increasing UV_{222} doses (Figures 5−7), and purified Fel d 1 was relatively stable when nebulized in PBS alone under control conditions (Figure S2, green line); however, when purified Fel d 1 was nebulized in PBS alone, then exposed to UV_{222}, this allergen showed enhanced susceptibility to UV_{222} treatment, with a 61% UV_{222}-dependent decrease in Fel d 1 levels following 35−40 min of UV_{222} exposure averaging 23.0 mJ/cm^2 (Figure S2, purple line) compared to its untreated control. This suggests components of the stabilization buffer (BSA and/or Tween-20) provided some protection for purified airborne Fel d 1 against UV_{222}-mediated effects, and the removal of these stabilizing agents from the carrier aerosol solution increased Fel d 1 susceptibility to UV_{222} treatment.

Although not further explored here, these observations highlight the critical role that interactions between aeroallergens and their carrier aerosols may play in determining airborne allergen stability, under both normal environmental conditions and in response to intervention treatments. Moreover, there are other fundamental questions about aeroallergen particle size and composition as well as UV_{222} penetration within those aerosols and the underlying mechanism of reduced immunodetection that were not addressed in these experiments. The methods developed in this study establish a reproducible and controlled approach to generate and collect respirable aeroallergens at biologically relevant concentrations. This approach will enable future investigation into how intrinsic protein characteristics and extrinsic factors such as aerosol composition and size, stabilizing agents, humidity, and temperature may influence allergen persistence and susceptibility to denaturation and/or degradation. Importantly, this is the first study to the authors' knowledge to generate respirable aeroallergens within a controlled environment at levels that reflect real-world airborne concentrations associated with allergic sensitization and disease (50−200 ng/m^3).[16−19,46−49] As such, the foundational methods developed here provide a powerful new approach to study the complex dynamics of airborne allergens and support the development of effective, evidence-based aeroallergen intervention strategies.

IMPLICATIONS

Overall, this study demonstrates that UV_{222} exposure well below the ACGIH threshold limit can significantly reduce airborne allergens within short time frames relevant to indoor air exchange rates. UV_{222}-dependent aeroallergen reductions averaged 25% for dustborne allergens and 20% for purified allergens after just 30 min of exposure to an average UV_{222} fluence dose of 16.8 mJ/cm^2 (Table 1). Although more research is needed to understand the mechanism of UV_{222}-mediated aeroallergen immunodetection reduction, these results suggest that UV_{222} may induce permanent structural changes in allergenic proteins, altering antibody−epitope interactions, and reducing immunodetection. The observed patterns of allergen susceptibility to UV_{222} exposure may reflect differences in protein structure, source material (dustborne vs purified), or the composition of carrier aerosols (heterogeneous components of dust, stabilizing agents like BSA). Future work utilizing the methods developed here should explore how these internal and external variables, as well as environmental factors, such as temperature and RH, may affect allergen stability and UV_{222}-dependent aeroallergen reductions.

It is important to note that the human immune system also relies on antibody-allergen immunodetection to elicit an allergic response; therefore, the reduced immunodetection observed in the commercial MARIA assay following UV_{222} intervention may translate to symptom relief for allergic individuals; however, further research is needed to characterize the biological relevance of allergen reductions in the air. Future work examining Type I hypersensitivity responses *in vitro* and *in vivo*, as well as research evaluating allergic and asthma responses within sensitized individuals, would be valuable to explore the clinical relevance of UV_{222}-dependent aeroallergen reductions.

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Studies investigating aeroallergen intervention strategies often evaluate allergic outcomes in sensitized individuals over much longer time frames than in the work performed here, often over weeks or months.[13−15,20−22,50−53] For example, the study described above by Bjornsdottir et al. observed a 93% reduction in settled dust levels of Fel d 1 after eight months of rigorous, multicomponent environmental control implementation. While this intervention timeline and approach was effective, the study acknowledged that it "may have chosen a biased population, a very "hardy" group of individuals with stamina and endurance, and this may explain the reason for the positive response to compliance with otherwise complicated and time-consuming EC [environmental control] measures",[22] highlighting the challenges in developing and implementing effective aeroallergen control methods.

Other long-term allergen intervention studies have shown similar allergen reductions as the UV_{222}-dependent reductions observed here. For example, research investigating the Purina Pro Plan LiveClear cat food, which uses anti-Fel d 1 egg antibodies to reduce Fel d 1 allergen in cat saliva and fur, showed 20−47% reductions in Fel d 1 after 4−10 weeks of feeding this product to cats.[50,51] These Fel d 1 reductions were also associated with improvements in allergy symptoms,[52,53] suggesting the UV_{222}-dependent reductions observed in our work within just 30 min of exposure may also lead to clinically meaningful symptom relief for allergic individuals.

Long-term interventions over weeks and months of applying rigorous environmental controls or implementing specific pet diets are difficult to execute and maintain. In contrast, UV_{222} offers a fast-acting, passive intervention strategy that acts directly on the exposure-relevant, respirable particles in the air. While no single intervention is likely to eliminate aeroallergen exposure risks entirely, UV_{222} may complement other strategies such as air filtration, surface cleaning, or allergen source removal to holistically improve indoor air quality and advance respiratory health. Taken together, these findings suggest that UV_{222} treatment has strong potential as an intervention strategy to reduce aeroallergens by targeting them within respirable airborne particles.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/acsestair.5c00080.

Figure S1 demonstrates that UV_{222}-dependent reductions in dustborne aeroallergens occur similarly under both low (<5%) and ambient (21%) oxygen conditions, removing ozone generation (≤80 ppb over background) as a process variable in this pilot experiment. Figure S2 shows that some purified aeroallergens, when nebulized in PBS, are less stable and more susceptible to UV_{222} than those nebulized in stabilization buffer. Some purified allergens, like Der p 1 and Der f 1 were not detectable in the air when nebulized in PBS in these pilot experiments (PDF)

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Notes

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■ ABBREVIATIONS

ACGIH	American Conference of Governmental Industrial Hygienists
ASTM	American Society for Testing and Materials
BSA	bovine serum albumin
ELISA	enzyme-linked immunosorbent assay
IgE	immunoglobulin E
KrCl	krypton chloride
LLOD	lower limit of detection
LPM	liters per minute
MAD	mean aerodynamic diameter
MARIA	multiplex array for indoor allergens
PBS	phosphate-buffered saline
ppb	parts per billion
PVDF	polyvinylidene fluoride
RH	relative humidity
UVC	ultraviolet-C light (100−280 nm)
UV_{222}	ultraviolet light at 222 nm
UV_{254}	ultraviolet light at 254 nm
WHO/IUIS	World Health Organization and the Union of Immunological Societies
YPD	yeast peptone dextrose

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