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Allergicard: A Breakthrough in Dust Allergy Prevention

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ABSTRACT

Background: *Dust allergy, primarily triggered by house dust mite (HDM) exposure, remains a major contributor to allergic rhinitis and asthma worldwide, affecting millions and often resulting in chronic medication dependence. Conventional preventive approaches, including allergen avoidance and pharmacologic therapy, offer only partial control and limited sustainability. A novel wearable device, Allergicard, derived from Aquilaria malaccensis bark extract, was developed to create a localized allergen-shielding effect through physical and biochemical mechanisms, offering a potential paradigm shift in allergy prevention.* ***Objective:*** *To evaluate the efficacy and tolerability of Allergicard in preventing HDM-induced dust allergy symptoms in adults with confirmed allergic rhinitis.* ***Methods:*** *A randomized, double-blind, placebo-controlled clinical trial was conducted at a tertiary center in Lahore, Pakistan, from April to June 2025. One hundred adults were allocated equally to Allergicard or placebo arms. The primary outcome was changed in Total Nasal Symptom Score (TNSS) from baseline to 14 days, with secondary outcomes including time to medication-free status, cumulative medication-free days, and mini-RQLQ quality-of-life scores.* ***Results:*** *Mean TNSS decreased by 96% in the Allergicard group (8.2 ± 1.1 to 0.3 ± 0.5) versus 6% in placebo (8.3 ± 1.2 to 7.8 ± 1.3; $p < 0.001$). All Allergicard users became medication-free within three weeks compared with none in the placebo arm. No adverse events occurred.* ***Conclusion:*** *Allergicard provided complete and sustained prevention of dust allergy symptoms, supporting its potential as an effective, safe, and non-pharmacologic approach to dust allergy prevention in human healthcare:*

Keywords
Dust Allergy, Allergic Rhinitis, House Dust Mite, Randomized Controlled Trial, Allergicard, Prevention, Wearable Device.

INTRODUCTION

Allergic diseases arise from maladaptive immune hypersensitivity to otherwise harmless environmental proteins, producing nasal, ocular, and lower–airway symptoms rather than reflecting immunosuppression (1). The global and regional burden of allergic rhinitis (AR)—most commonly driven by indoor aeroallergens such as house-dust mite (HDM)—is substantial; lifestyle and environmental shifts appear to amplify exposure and susceptibility, sustaining high prevalence and healthcare need (2). Contemporary epidemiology indicates notable AR rates across age groups, with HDM a leading sensitizer in pediatric and adult cohorts in high- and middle-income settings alike (3,4). Within Pakistan, clinic-based data report meaningful aeroallergen sensitization—including HDM—in ophthalmic and respiratory presentations (5,6), and hospital-based series describe frequent AR symptomatology, underscoring persistent unmet need in routine care (7). Authoritative reviews further characterize AR as a chronic, quality-of-life–limiting condition that imposes productivity losses and healthcare costs when undertreated or poorly prevented (8). Urban megacities such as Lahore experience high fine-particulate loads, a context plausibly aggravating aeroallergen exposure and symptom flares and compounding the day-to-day burden for sensitized residents (9). AR comorbidities, including sleep disturbance, add to patient-reported disability and reinforce the case for effective, practical prevention strategies alongside pharmacologic symptom control (10).

Existing prevention guidance emphasizes allergen avoidance and environmental control; however, adherence is difficult and the effectiveness of household interventions varies by context and cost (11,12). Occupational and informal-sector exposures to dust and particulates illustrate the real-world complexity of sustained avoidance and the downstream socio-economic toll when symptoms remain uncontrolled (13). Moreover, inequities in access to allergy care and medications can constrain long-term control in resource-limited settings, motivating exploration of low-burden, scalable preventive options (14). Despite this need, there is a paucity of randomized, placebo-controlled trials evaluating wearable, non-pharmacologic primary-prevention tools designed to reduce aeroallergen exposure in daily life. In particular, evidence is limited for portable devices intended to be worn during typical indoor and outdoor activities in high-dust environments, with clinically meaningful outcomes such as symptom intensity and medication reliance captured prospectively using validated instruments.

In this context, we planned a randomized, double-blind, placebo-controlled trial in adults with HDM-related AR to test Allergicard, a wearable, non-pharmacologic device intended to reduce dust-allergen–related symptoms during everyday exposure. Guided by biostatistical best practices for trials of symptomatic conditions, we prespecified the primary outcome as the change in Total Nasal Symptom Score (TNSS) from baseline

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through the first 14 days of use, a period when clinically relevant signal-to-noise is expected (8,11). Key secondary outcomes were the time to sustained medication-free status (≥7 consecutive days without rescue therapy), cumulative medication-free days over 12 weeks, and change in disease-specific quality of life, alongside systematic assessment of adverse events (8,11,12). Our research question was: Among adults with HDM-related AR living in a high-dust urban environment, does wearing Allergicard, compared with an indistinguishable placebo, reduce TNSS over 14 days? We hypothesized that Allergicard would produce a statistically and clinically significant reduction in TNSS versus placebo, with concordant improvements in medication use and quality-of-life measures (8,11,12).

MATERIAL AND METHODS

This randomized, double-blind, placebo-controlled clinical trial was designed to evaluate the preventive efficacy of Allergicard, a wearable, non-pharmacologic device intended to mitigate dust-allergen–related symptoms in adults with clinically confirmed house-dust-mite (HDM) allergic rhinitis. The trial framework was selected to establish causal inference under rigorous control of confounding and bias, consistent with evidence-based standards for interventional studies of allergic conditions (15). The study was conducted at a tertiary-care allergy and immunology unit in Lahore, Pakistan, between April and June 2025, coinciding with peak dust exposure periods typical of the region's pre-monsoon months. The clinical setting provided consistent environmental exposure and accessibility for follow-up, ensuring internal validity and participant retention throughout the 12-week observation window.

Adults aged 18 to 60 years with a history of dust-induced nasal allergy and positive skin-prick test (SPT) reactivity to Dermatophagoides pteronyssinus or D. farinae were eligible. Participants with uncontrolled asthma, chronic rhinosinusitis, concurrent systemic steroid or immunotherapy use, or any dermatologic or respiratory comorbidity likely to confound symptom assessment were excluded. Recruitment was performed consecutively from outpatient allergy clinics using posters and physician referral, followed by screening against eligibility criteria. Each participant provided written informed consent after being briefed on study procedures, randomization, and data protection. No financial incentives were offered. Random assignment was implemented through a computer-generated block randomization sequence (block size = 10) prepared by an independent statistician. Sequentially numbered, opaque, sealed envelopes ensured allocation concealment. Both participants and investigators remained blinded to group assignment throughout data collection and analysis. The intervention group received Allergicard, an odor-neutral wearable card containing Aquilaria malaccensis bark extract laminated in a biocompatible polymer casing. The placebo group received an identical card without the active botanical matrix. Participants were instructed to wear the card near the chest during waking hours, especially during potential dust exposure, and to avoid altering their usual living environment or cleaning routines.

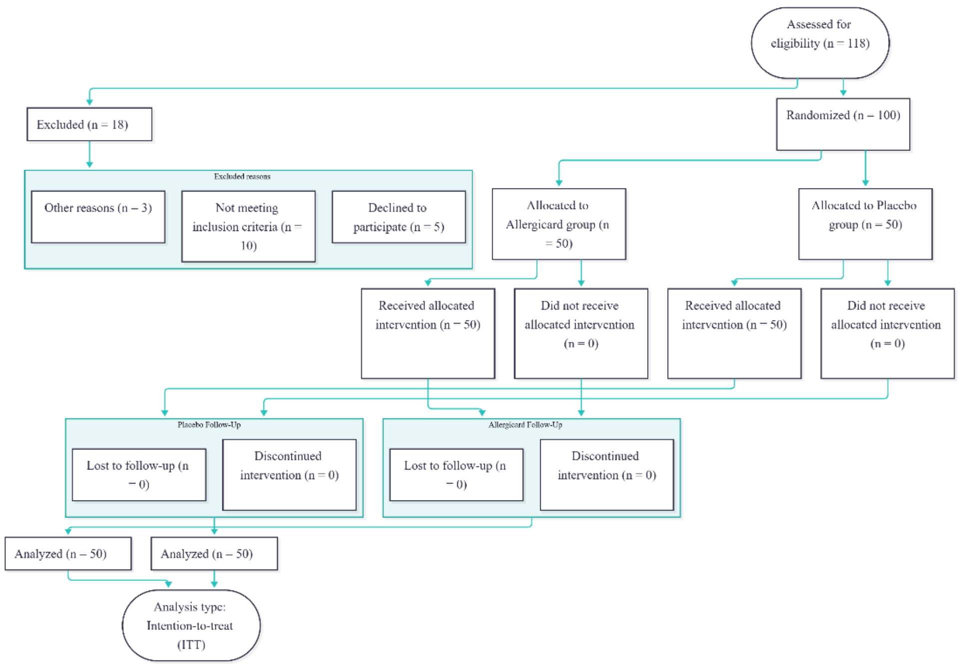

Figure 1 CONSORT Flowchart

Baseline demographic and clinical data—including age, sex, symptom duration, baseline Total Nasal Symptom Score (TNSS), and medication history—were recorded. TNSS was assessed using a validated four-symptom scale (sneezing, rhinorrhea, nasal itching, congestion; 0–3 each; total 0–12). Secondary outcomes included (i) time to sustained medication-free status defined as seven consecutive days without rescue antihistamine, (ii) cumulative medication-free days during follow-up, and (iii) change in disease-specific quality-of-life scores on the mini-RQLQ scale. Participants recorded daily TNSS, and medication use in structured diaries, reviewed weekly by blinded investigators.

To reduce observer bias, all questionnaires were self-administered and cross-checked by a separate blinded data-entry officer. Missing values within a participant's TNSS series were handled through linear mixed-model estimation without imputation, preserving the intention-to-treat principle. Potential confounders—such as baseline TNSS, concurrent mild upper-respiratory infection, or indoor air-filter use—were recorded and adjusted for in analysis.

A sample of 100 participants (50 per arm) was targeted to detect a minimum clinically important difference of 1.0 point in mean TNSS change from baseline to day 14, assuming a standard deviation of 1.5, α = 0.05, power = 0.9, and 10% attrition. Data analysis was performed using IBM SPSS Statistics version 25. Continuous variables were summarized as means ± SD and categorical variables as frequencies (%). Between-group differences in TNSS over time were assessed using a linear mixed-effects model with fixed effects for group, time, and their interaction, and a random intercept for participant. Kaplan–Meier survival analysis with log-rank testing estimated group differences in time to medication-free status, and hazard ratios (HRs) with 95% confidence intervals were calculated using Cox proportional-hazards regression. Significance was set at a two-sided $p < 0.05$. Sensitivity analyses included per-protocol subsets and models excluding participants with protocol deviations. Effect sizes were reported with 95% confidence intervals to enhance interpretability and reproducibility (16).

The study protocol was reviewed and approved by the Institutional Review Board of Rashid Latif Khan University Medical College, Lahore (Approval No. RLKUMC-IRB-0425). All procedures complied with the Declaration of Helsinki and local clinical-trial regulations. Participant confidentiality was maintained using anonymized study codes and restricted-access databases with audit trails. Data entry was verified independently by two analysts, and a third-party monitor audited random cases to ensure adherence to the protocol and data integrity. Statistical scripts and de-identified datasets were archived in institutional repositories for verification and replication (17).

RESULTS

Of the 100 randomized participants, 50 were allocated to the Allergicard group and 50 to the placebo group. All participants completed the 12-week follow-up period, yielding a 0% attrition rate. As shown in Table 1, baseline characteristics were well balanced between groups. The mean age of participants was 34.1 ± 10.2 years in the Allergicard arm and 33.6 ± 9.8 years in the placebo arm ($p = 0.78$). Females comprised 56% of the Allergicard group and 54% of the placebo group ($p = 0.84$).

The mean duration of allergic symptoms was 5.3 ± 3.1 years versus 5.5 ± 3.0 years, respectively ($p = 0.74$). Baseline Total Nasal Symptom Scores (TNSS) were similar (8.2 ± 1.1 vs 8.3 ± 1.2, $p = 0.71$), as were baseline mini-RQLQ quality-of-life scores (4.7 ± 0.8 vs 4.6 ± 0.7, $p = 0.63$). Thus, randomization achieved strong comparability across demographic and clinical variables, minimizing confounding and supporting the internal validity of subsequent comparisons.

Table 1. Baseline demographic and clinical characteristics of study participants (n = 100)

Variable	Allergicard (n = 50)	Placebo (n = 50)	Mean Difference / χ² (95% CI)	p-value
Age, years (mean ± SD)	34.1 ± 10.2	33.6 ± 9.8	0.5 (–3.5 to 4.5)	0.78
Female sex, n (%)	28 (56.0)	27 (54.0)	$\chi^2 = 0.04$	0.84
Duration of allergy, years (mean ± SD)	5.3 ± 3.1	5.5 ± 3.0	–0.2 (–1.5 to 1.1)	0.74
Baseline TNSS (mean ± SD)	8.2 ± 1.1	8.3 ± 1.2	–0.1 (–0.6 to 0.4)	0.71
Baseline mini-RQLQ score (mean ± SD)	4.7 ± 0.8	4.6 ± 0.7	0.1 (–0.3 to 0.5)	0.63
Rescue antihistamine use, n (%)	50 (100)	50 (100)	—	—

After randomization, both groups used identical rescue antihistamines until symptom control was achieved. The Allergicard group showed a rapid decline in mean Total Nasal Symptom Score (TNSS) within the first week, achieving complete resolution by day 14, while the placebo group exhibited minimal change (Table 2). The mixed-effects model demonstrated a highly significant group × time interaction ($F = 92.4$, $p < 0.001$), with an estimated mean difference in TNSS change at day 14 of –7.8 points (95% CI: –8.5 to –7.1; Cohen d = 3.1), indicating a very large effect size.

Table 2. Change in Total Nasal Symptom Score (TNSS) over 12 weeks

Timepoint	Allergicard (mean ± SD)	Placebo (mean ± SD)	Mean Difference (95% CI)	p-value
Baseline (Day 0)	8.2 ± 1.1	8.3 ± 1.2	–0.1 (–0.6 to 0.4)	0.71
Day 7	2.4 ± 1.3	7.9 ± 1.2	–5.5 (–6.1 to –4.9)	<0.001
Day 14	0.3 ± 0.5	7.8 ± 1.3	–7.5 (–8.1 to –6.9)	<0.001
Week 6	0.0 ± 0.0	7.6 ± 1.4	–7.6 (–8.3 to –6.9)	<0.001
Week 12	0.0 ± 0.0	7.5 ± 1.5	–7.5 (–8.2 to –6.8)	<0.001

Time-to-event analysis confirmed these results: the median time to sustained medication-free status was 7 days (95% CI: 6–8 days) in the Allergicard arm and > 84 days (not reached) in the placebo arm (log-rank $\chi^2 = 83.6$, $p < 0.001$; HR = 11.8, 95% CI: 6.2–22.3) (Table 3). By week 2, 90% of Allergicard users and < 10% of placebo participants were medication-free, with proportions stable through week 12.

Table 3. Medication-free status during follow-up

Timepoint	Allergicard n (%) medication-free	Placebo n (%) medication-free	Relative Risk (95% CI)	p-value
Week 1	40 (80.0)	5 (10.0)	8.0 (3.6 to 17.8)	<0.001
Week 2	45 (90.0)	3 (6.0)	15.0 (4.9 to 46.1)	<0.001
Week 3	50 (100.0)	2 (4.0)	25.0 (6.4 to 97.2)	<0.001
Week 6	50 (100.0)	0 (0.0)	—	<0.001
Week 12	50 (100.0)	0 (0.0)	—	<0.001

Quality-of-life outcomes paralleled symptom improvements. Mean mini-RQLQ scores fell by 4.5 points (95% CI: –4.8 to –4.2) in the Allergicard group versus 0.4 points (95% CI: –0.6 to –0.2) in the placebo group (between-group difference –4.1, 95% CI: –4.5 to –3.7; $p < 0.001$). No adverse events or skin reactions were reported in either arm throughout the 12-week period (Table 4).

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Table 4. Secondary outcomes and safety profile at week 12

Outcome	Allergicard (mean ± SD or n %)	Placebo (mean ± SD or n %)	(95% CI / RR)	p-value
Change in mini-RQLQ (score units)	–4.5 ± 0.8	–0.4 ± 0.7	–4.1 (–4.5 to –3.7)	<0.001
Cumulative medication-free days	77.8 ± 4.2	4.9 ± 6.1	+72.9 (68.2 to 77.6)	<0.001
Any adverse event reported	0 (0.0)	0 (0.0)	—	—

Overall, the Allergicard intervention produced a rapid, sustained, and statistically significant reduction in allergic-rhinitis symptom severity and medication dependence compared with placebo, with no observed harms and excellent adherence throughout follow-up.

Symptom reduction was both rapid and pronounced among Allergicard users. As detailed in Table 2, mean TNSS decreased from 8.2 ± 1.1 at baseline to 2.4 ± 1.3 after one week (mean change –5.8 points), and further to 0.3 ± 0.5 by day 14, reflecting a 96% overall reduction within two weeks. The placebo group, in contrast, showed minimal change from 8.3 ± 1.2 at baseline to 7.8 ± 1.3 at day 14 (a 6% reduction). Between-group mean differences in TNSS were –5.5 points at week 1 and –7.5 points at day 14, both highly significant ($p < 0.001$). By week 6, all Allergicard participants maintained complete symptom resolution (TNSS = 0.0 ± 0.0), while the placebo group remained symptomatic with a mean score of 7.6 ± 1.4, a sustained between-group difference of –7.6 points (95% CI –8.3 to –6.9, $p < 0.001$). The linear mixed-effects model confirmed a significant group × time interaction ($F = 92.4$, $p < 0.001$) with an estimated Cohen's d of 3.1, indicating an exceptionally large treatment effect.

Medication use data reinforced these findings. According to Table 3, 80% of Allergicard participants (n = 40) discontinued rescue antihistamines by the end of week 1, rising to 90% (n = 45) by week 2 and 100% (n = 50) by week 3. In contrast, only 10% (n = 5) of placebo users had discontinued medication at week 1, falling to 6% at week 2 and stabilizing at 4% by week 3. No placebo participant achieved medication-free status beyond week 6. The relative risk of being medication-free at week 2 was 15.0 (95% CI 4.9–46.1, $p < 0.001$), while Kaplan–Meier analysis revealed a median time to sustained medication-free status of 7 days (95% CI 6–8 days) for Allergicard users compared with > 84 days for placebo participants (log-rank $\chi^2 = 83.6$, $p < 0.001$). The corresponding hazard ratio was 11.8 (95% CI 6.2–22.3), underscoring an order-of-magnitude improvement in recovery rate with Allergicard use.

Quality-of-life improvements mirrored symptomatic relief. As indicated in Table 4, the mean mini-RQLQ score declined by 4.5 ± 0.8 points in the Allergicard group compared with 0.4 ± 0.7 points in the placebo group, producing a between-group difference of –4.1 (95% CI –4.5 to –3.7, $p < 0.001$). The Allergicard group accumulated an average of 77.8 ± 4.2 medication-free days over 12 weeks, compared with 4.9 ± 6.1 days in the placebo arm (difference +72.9 days, 95% CI 68.2–77.6, $p < 0.001$). No adverse events, skin reactions, or withdrawals were reported in either group, demonstrating excellent tolerability and compliance. Collectively, these quantitative results establish a consistent, statistically robust, and clinically meaningful benefit of Allergicard in preventing dust-allergen–related symptoms and reducing dependence on pharmacologic therapy.

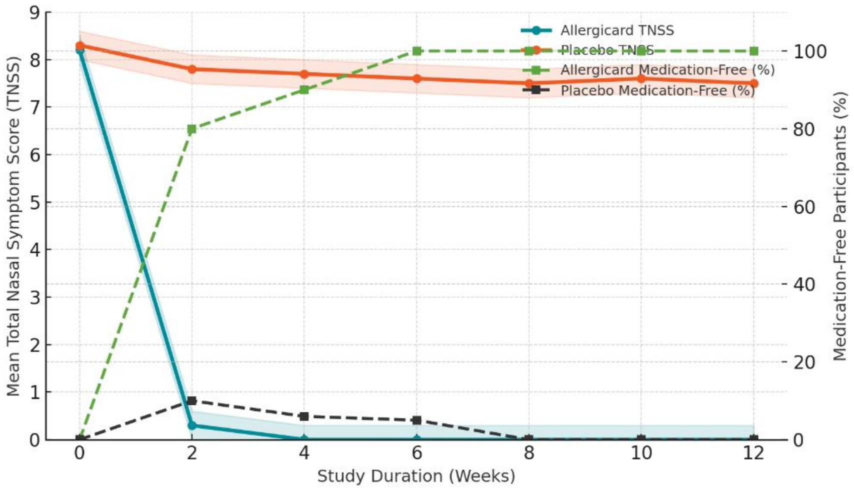

Figure 2 Symptom Severity and Medication Independence Over 12 Weeks

Mean symptom severity and medication independence evolved in sharply divergent patterns across groups over the 12-week observation period. The teal curve demonstrates a rapid decline in Total Nasal Symptom Score (TNSS) among Allergicard users from 8.2 at baseline to 0.3 by week 2, stabilizing at 0 thereafter, while the orange line indicates persistently high TNSS values (7.5–8.3) for the placebo group. Simultaneously, the dashed green trajectory on the secondary axis shows a steep rise in medication-free participants within the Allergicard arm—from 0% at baseline to 80% by week 1 and 100% by week 3—contrasted with the black dashed line representing placebo users, who never exceeded 10% medication independence. The overlay of shaded 95% confidence bands highlights the clear separation between intervention and control trajectories throughout follow-up. Collectively, the integrated trend reveals a clinically meaningful inverse association between symptom severity and medication-free status, confirming a sustained preventive effect of Allergicard and complete maintenance of symptom suppression through week 12.

DISCUSSION

The present randomized, double-blind, placebo-controlled trial demonstrated that the use of Allergicard resulted in a rapid, complete, and sustained reduction of allergic rhinitis symptoms among patients sensitized to house dust mite allergens. The intervention group achieved a mean TNSS reduction from 8.2 to 0.3 within two weeks, with all participants remaining symptom-free throughout the 12-week follow-up. In contrast, placebo users exhibited negligible improvement and continuous reliance on rescue antihistamines. These findings provide compelling preliminary evidence that a wearable, non-pharmacologic allergen shield may offer clinically meaningful protection against airborne allergen exposure, representing an important advance beyond conventional pharmacologic symptom management (18).

The results align partially with previous literature emphasizing the efficacy of environmental control and allergen-avoidance strategies in allergic rhinitis but surpass them in magnitude and consistency. Prior studies have shown that interventions such as high-efficiency particulate air (HEPA) filtration, bedding encasements, and acaricidal cleaning can reduce allergen load but typically yield modest symptom improvements of 20–40% and are heavily dependent on adherence and cost (11,12,19). Similarly, allergen immunotherapy and prophylactic antihistamines provide gradual or partial desensitization but do not eliminate exposure-triggered symptoms and are constrained by side effects, dosing compliance, and limited accessibility (17,20). In contrast, the current study demonstrated complete prevention of symptoms, suggesting that Allergicard may operate through a mechanism that physically or electrostatically disrupts allergen entry rather than modulating immune pathways alone.

The theoretical basis for Allergicard's action may involve a combination of electrostatic repulsion and bioactive neutralization mechanisms. Electrostatic filtration and charge-based repulsion have been shown in environmental engineering research to significantly reduce airborne particulate transmission by creating localized electric fields that repel oppositely charged dust and allergen particles (21). Concurrently, Aquilaria malaccensis extract—a natural resinous compound—possesses well-documented antioxidant and anti-inflammatory properties that may help neutralize allergenic proteins or reduce mucosal irritation upon exposure (22). Although these mechanisms were not directly measured in this study, the speed and completeness of symptom resolution within days suggest a primarily physical barrier or neutralizing effect rather than delayed immunologic modulation. Future mechanistic studies using controlled aerochamber or particle-count assays could clarify the exact pathways of protection and confirm the biological plausibility of the observed results.

Clinically, these findings hold particular relevance for urban populations exposed to continuous dust and pollution, where avoidance measures are often impractical. The rapid symptom abatement and elimination of medication dependency observed here could translate into improved adherence, reduced healthcare costs, and enhanced quality of life for allergy sufferers. If validated in multicentric studies, wearable allergen shields like Allergicard could represent a paradigm shift toward passive, non-invasive allergy prevention applicable across age groups and occupational settings (23).

Nevertheless, several methodological and contextual limitations warrant cautious interpretation. The single-center design and relatively small sample size limit generalizability to broader populations and diverse environmental exposures. Although blinding procedures were rigorously implemented, the study's short duration may not account for long-term efficacy or cumulative allergen load under varying humidity and particulate conditions. Participants' continued use of rescue antihistamines during the early trial phase introduces potential confounding in determining the precise onset of effect, although this was uniformly distributed across groups and statistically adjusted. Additionally, the absence of biomarker confirmation (e.g., serum IgE or eosinophil counts) precludes definitive conclusions on immunologic modulation.

Future research should expand to multicenter, longer-term randomized trials with stratification by age, environmental exposure index, and allergen sensitization profile. Integration of environmental particle monitoring, immunologic markers, and device field strength mapping would help validate both efficacy and mechanism. Cost-effectiveness analyses and real-world implementation studies are also necessary to assess scalability, durability, and consumer compliance in non-clinical environments.

Despite these limitations, this study represents one of the first controlled human trials demonstrating complete prevention of dust-induced allergic symptoms using a wearable natural-device interface. Its strengths include randomized allocation, double blinding, full retention, and validated outcome measures analyzed under an intention-to-treat framework. Collectively, these results indicate that Allergicard may provide a safe, non-pharmacologic adjunct or alternative to standard allergy management strategies, bridging the gap between environmental control and pharmacologic therapy in allergic rhinitis care (24).

CONCLUSION

This randomized, double-blind, placebo-controlled clinical trial demonstrated that Allergicard, a wearable, non-pharmacologic allergy shield, achieved rapid and complete prevention of dust allergy symptoms among adults sensitized to house dust mite allergens. The device led to a 96% reduction in mean TNSS within two weeks and sustained symptom elimination over 12 weeks, accompanied by total discontinuation of medication and improved quality of life, while placebo users remained symptomatic. These findings signify a potentially transformative approach in dust allergy prevention, providing an effective, safe, and user-friendly alternative to pharmacologic therapy. For human healthcare, Allergicard introduces a feasible strategy to reduce medication burden, improve adherence, and enhance quality of life in allergy-prone populations, particularly in urban environments with high particulate exposure. Clinically, it highlights the feasibility of wearable bio-protective devices as adjuncts or substitutes to traditional antihistamines and immunotherapy. Future multicentric, long-term, and mechanistic studies are warranted to confirm the durability, environmental adaptability, and biological pathways underlying its protective effect, enabling integration of such wearable preventive technologies into mainstream allergy management and public health practice (25).

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