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Research article

HLA variants and their association with IgE-Mediated banana allergy: A cross-sectional study

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ABSTRACT

Background: Banana allergy is on the rise in tropical regions. Advances in genomics and candidate gene identification have increased interest in genetic factors in food allergies. However, the genetic basis of IgE-mediated banana allergy is underexplored.
Objective: To characterize HLA variants and their association with IgE-mediated banana allergy.
Methods: This cross-sectional study recruited banana-allergic adults, confirmed by allergology tests, with non-allergic individuals as controls. Genomic DNA extraction and sequencing BAM files for HLA typing were conducted. Allele frequency was calculated using the direct counting method, and odds ratio (OR) with 95 % confidence interval (CI) were determined. Fisher's exact or chi-square tests were used to assess associations with Bonferroni's correction for multiple tests. The allele frequency of the Thai population from The Allele Frequency Net Database was used to compute the allele enrichment ratio (ER).
Results: A total of 59 cases and 64 controls were recruited. HLA genotyping indicated potential associations of HLA-B*15:25 (OR 11.872; p-value 0.027), HLA-C*04:03 (OR 7.636; p-value 0.033), and HLA-DQB1*06:09 (OR 11.558; p-value 0.039) with banana allergy. However, after Bonferroni correction, none of these associations reached statistical significance. Comparing allele frequency with the general population from The Allele Frequency Net Database, our ER

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analysis revealed a higher prevalence in the banana allergy group for B*15:25 (ER 1.849), C*04:03 (ER 1.332), and DQB1*06:09 (ER 6.602) alleles.
Conclusions: This study provides initial genetic insights into banana allergy, suggesting potential links with specific HLA alleles. Despite 12 initially identifying alleles, none were statistically significant after multiple testing correction. Larger studies are needed to detect possible significant correlations.

1. Introduction

Food allergy is a complex immune-mediated disorder characterized by adverse reactions to specific food antigens. It affects a significant proportion of the global population and poses a substantial public health burden [1,2]. The etiology of food allergies is believed to involve a combination of genetic and environmental factors [3,4]. Genetic influences play a crucial role in the development of food allergies, as evidenced by family and twin studies showing a higher concordance rate among first-degree relatives [5].

In light of advances in genomics and the identification of genes linked to allergic disorders, there has been an intensified focus on understanding the genetic factors that influence food allergy susceptibility [6,7]. Such exploration has elucidated specific genes associated with this condition. The study of food allergy genetics serves various objectives. Some researchers aim to understand the evolutionary origins and genetic composition, which can provide insights into potential interventions and the adaptability of food allergies. Furthermore, identifying genes that signal a heightened risk of food allergies offers prospects for preemptive treatments for at-risk individuals. Many biomedical researchers aim to identify genes linked to food allergy variations to understand and target its physiological pathways for potential drug interventions [6]. To this end, they've used experimental designs like family, twin, and cohort studies, and analytical techniques such as linkage analysis, candidate gene studies, genome-wide association studies (GWAS), DNA methylation, and microbiome analysis. The hypothesis-driven research on gene links with food allergy has focused on established immune-related genes, many of which are tied to other allergic conditions [6].

A recent systematic review consolidated findings from both candidate-gene association studies and genome-wide scans regarding various food allergy outcomes. Compelling evidence links food allergy with genetic variants at the filaggrin gene (*FLG*), Human Leukocyte Antigen (*HLA*) gene, and interleukin-13 (*IL13*) gene. Additionally, variants like *SPINK5, SERPINB*, and *C11orf30* show some association and merit further study [4]. Other genetic factors such as copy number variations impacting *CTNNA3* and *RBFOX1*, DNA methylation, and single nucleotide polymorphism association at HLA-DR and DQ loci have also been implicated [7].

The Major Histocompatibility Complex (MHC) is located on chromosome 6p21.3 and is densely populated with immune response-related genes. Among these are the *HLA* genes that code for families of cell-surface proteins. These proteins play a crucial role in recognizing antigens by the adaptive immune system. Remarkably, the HLA region has been linked with a greater number of diseases than any other genomic region [8]. The HLA region's propensity for disease association can be attributed to its pronounced genomic polymorphism, with multiple loci like class I HLA-A, -B, –C and class II -DRB1, -DQB1, -DQA1, -DPB1, and -DPA1 genes showing significant variability. This region also demonstrates notable linkage disequilibrium. Currently, over 26,000 alleles have been identified in HLA genes [9]. The HLA molecules, in their diversity, selectively bind peptides from degraded proteins, facilitating antigen presentation. Upon binding to these peptide-HLA ligands, T lymphocytes, with their unique receptors, undergo a differentiation process that influences the nature of the immune response. The specific peptide-HLA pairing, combined with tissue environmental signals like cytokine recognition, can guide T-cell responses, assisting B lymphocytes in producing antigen-specific immunoglobulins, including Immunoglobulin E (IgE) [10]. A positive association between HLA and food allergies has been reported since 1997 in Italian children with cow's milk allergy [11]. Subsequently, numerous HLA loci have been identified in association with peanut allergy. Notably, HLA DRB108, DRB108/12 tyr 16, DQB104, and DPB10301 exhibited significantly higher frequencies among individuals with peanut allergy [12]. Additionally, HLA-DR7 appears to be significantly linked to the presentation of apple and pollen allergens [13].

Food allergies often involve fruits, which are common allergens. These allergies typically develop in childhood, but can also emerge later in life [14–18]. Bananas are among the top consumed fruits globally, especially in Asia, Latin America, and Africa. Our latest report details the clinical features of banana allergy in adults [15]. However, there have been no prior studies examining immediate-type banana allergy. Therefore, our study aims to investigate the genetic association between HLA variants and banana allergy, which might reveal the risk of developing immediate-type banana allergy.

2. Methods

2.1. Study population

This genetic association analysis was conducted using a cross-sectional approach with a case-control design, in adult patients with banana allergies as part of the Thai Adult Banana Allergy Cohort (TABAC). The details of the TABAC cohort were described in our previous study [15]. This study was approved by the Siriraj Institutional Review Board (SIRB), Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, ThailandCOA no. Si 418/2020. Collection of genetic material began following approval from the institutional review board.

2.2. Sample size estimation

Since there is no previous HLA study in banana allergy, we utilized minor allele frequency (MAF) data extracted from a genome-wide association study of food allergy in the Japanese population, which included shrimp and peach allergy [19]. The authors reported high cross-reactivity among various fruits, including apple, banana, kiwi, orange, and peach. Therefore, in this study, we utilized the MAF of the top HLA types identified from peach allergy: HLA-DRB109:01 and HLA-DQB103:03, corresponding to MAFs in the case group of 0.025 and 0.238, respectively.

For control group, we obtained the MAF data of Thai population from the allele frequency net database [https://www.allelefrequencies.net/], with MAFs of 0.122 and 0.114 for HLA-DRB109:01 and HLA-DQB103:03, respectively. Sample size estimation was conducted by comparing two independent proportions using the reported MAF from Japanese cases and the Thai population for each HLA type separately. The minimum number of samples required to achieve 80 % power at a 0.05 alpha level was calculated to be 112 in total, with an equal number of cases and controls (1:1 ratio).

2.3. Case and control definition

This cross-sectional study included case and control populations selected based on their allergic history and allergology test status. Cases were patients diagnosed with banana allergy, while controls were volunteers without self-reported banana allergy. The case group was randomly sampled of patients from the Thai Adult Banana Allergy Cohort (TABAC). Inclusion criteria for this cohort were detailed in the earlier study [15]. Essentially, these criteria included patients with a history indicative of IgE-mediated reactions after the ingestion of bananas, alongside at least one positive allergology test, which could involve PTP tests using fresh bananas, banana-specific ImmunoCAP, or banana challenge. A compatible history of IgE-mediated a banana allergy was defined by clinical symptoms of mast cell activation, such as oral allergy syndrome, urticaria, angioedema, or anaphylaxis, occurring within a 3-h after -consuming any of variety of banana.

Regarding the control group, HLA typing data was obtained from Genomics Thailand, a collaborative human genome research network in Thailand. This data was sourced from the database using simple random sampling. Participants with no history of allergies to bananas or any other fruits were recruited to establish the control group.

In addition, HLA data from the general Thai population, obtained from the publicly accessible database at allelefrequencies.net, were also utilized. This data was used to compare with cases of banana allergy by calculating the enrichment ratio (ER). Due to data availability, a matching method between the case and control groups was not performed.

2.4. Clinical data collection

Patient demographic information and details of clinical reactions were collected using structured questionnaire interviews. Details of structured questionnaire interviews were demonstrated elsewhere [15]. Allergists conducted these interviews, gathering essential demographic data along with comprehensive histories of banana allergy, other allergic diseases, additional medical conditions, and current medication usage.

2.5. DNA extraction, library preparation, sequencing and HLA typing

We conducted genomic DNA extraction using the Gentra Puregene blood kit from Qiagen, USA, following the manufacturer's instructions. Subsequently, we assessed the DNA's concentration and quality with a Nanodrop ND-1000 spectrophotometer (Thermo Scientific, USA). The DNA samples were diluted to a final concentration of 50 ng/μL using TE low EDTA buffer (comprising 10 mM Tris-HCl and 0.1 mM EDTA). This dilution was necessary for the amplification of all 11 HLA loci (HLA-A, B, C, DRB1, DRB3/4/5, DQB1, DQA1, DPB1, and DPA1) using the AllType™ NGS 11-Loci Amplification Kit (ONE LAMBDA, USA).

Afterwards, we prepared libraries using the Ion Shear Plus Reagent kits and the IonXpress Plus Fragment Library kit (Thermo Scientific, USA). The sequencing was conducted with the Ion S5XL system (Thermo Scientific, USA) according to the manufacturer's protocols. Finally, the sequencing BAM files underwent HLA typing using the TypeStream Visual NGS Analysis Software v3.0.

2.6. Statistical analysis

All statistical analyses were performed using Stata 16 (StataCorp, College Station, TX, USA), and Microsoft Excel (Microsoft Corporation). Baseline and clinical data of cases were collected and presented as descriptive analysis. Numerical data were shown as mean and standard deviation (SD), categorical data were described as frequency and percentage. Allele frequencies of HLA were determined by direct counting followed by dividing the count of a specific allele by the total number of alleles in both cases and controls. Fisher's exact or chi-square tests were used to assess the significance of the association. Bonferroni correction was employed for multiple testing, with statistical significance determined by dividing the p-value by the total number of alleles. Odds ratios (ORs) and 95 % confidence intervals (CIs) were calculated to quantify the strength of the associations. Haldane-Anscombe correction was used for zero allele counts by simply adding 0.5 to all zero cells prior to the calculation.

Furthermore, we computed the allele enrichment ratio for a given HLA allele by dividing the frequency of observed alleles in our samples by their frequency observed in the general Thai population derived from the Allele Frequency Net Database (www.allelfrequencies.net) [20]. This ratio enabled us to determine if our potential risk alleles that were more frequently observed in the

banana allergy group compared to the general population [21].

For sample size calculation, no previous HLA association studies have investigated banana allergy. However, a relevant study by Blanca C et al. found an HLA genotype association with fruit allergy in latex-allergic patients [22]. The proportion of HLA-DQB1*0201 in latex-fruit allergy syndrome patients was 30.8 %, while it was 5.8 % in the control group not allergic to fruit. Using a Case-Control (unmatched, 1:1) design, with a significance level set at 5 % and a test power of 90 %, we calculated a minimum sample size of 37 individuals for both the case and control groups.

3. Result

A visual summary of our study is presented in **Fig. 1**. In the TABAC cohort, 133 patients with histories indicative of IgE-mediated banana allergic reactions were initially identified for eligibility assessment and subsequently enrolled in our cross-sectional study. For HLA genotyping, 60 patients were randomly selected from the cohort. However, a genotyping error in one patient led to the final analysis including only 59 patients in the case group. Additionally, 64 non-banana-allergic controls were recruited, maintaining a 1:1 ratio with the case group for odds ratio (OR) comparison **(Fig. 2).**

Table 1 provides an overview of the demographic and clinical attributes of 59 individuals diagnosed with banana allergies. The average age of the participants was 37.0 ± 7.3 years, with the onset of allergic symptoms occurring at an average age of 34.0 ± 8.78 years. Predominantly, 79.6 % of the cases were females, and a significant proportion (67 %) reported experiencing at least one banana-related allergic episode. The mean banana-specific IgE level was found to be 1.64 ± 2.83 kUA/L. Allergic rhinitis was the most prevalent condition among the cases, affecting 57.6 % of the participants, followed by chronic urticaria (15.25 %) and asthma (5.08 %). 3.38 % of the participants indicated having a first-degree relative diagnosed with a banana allergy. Notably, nearly half (47.45 %) had at least one family member with a diagnosed atopic condition, encompassing allergic rhinitis, asthma, eczema, or food allergies.

The data on allele frequency of 64 non-banana allergic individuals from the Genomics Thailand network and the general Thai population were obtained from the publicly acces-sible database at allelefrequencies.net, were utilized for ORs and ERs calculation re-spectively, and are illustrated in Table 2. Notably, the database does not provide in-formation regarding age, sex, or birthplace.

3.1. Association of HLA alleles and banana allergy

Initially, we assessed the significance of association across all HLA-typed alleles using statistical tests (Exact or Chi-squared tests). The findings revealed a significant association between banana allergy with 12 HLA alleles. Supplementary Tables 1–6 present the frequencies of these alleles, along with the corresponding odds ratios (ORs) and p-value when compared to the control group. Further analysis revealed positive associations in 6 alleles with ORs greater than 1 including HLA-B*15:25 (OR 11.872), HLA-C*04:03 (OR 7.636), DQA1*01:02:01 (OR 3.092), DQA1*01:05:01 (OR 10.493), HLA-DQB1*06:09 (OR 11.558), and DRB5*01:01:01 (OR 2.575). However, after applying the Bonferroni correction, these associations did not reach statistical significance.

Enrichment ratio (ER) analysis compared allele frequencies between the sample group and the Thai population. When comparing the allele frequencies observed in our study with those of the general population sourced from The Allele Frequency Net Database (Supplementary Tables 7–8), our analysis indicated a significantly higher prevalence of the B15:25 allele (ER 1.849), the C04:03 allele (ER 1.332), and the DQB106:09 allele (ER 6.602) within the banana allergy group compared to the broader Thai population.

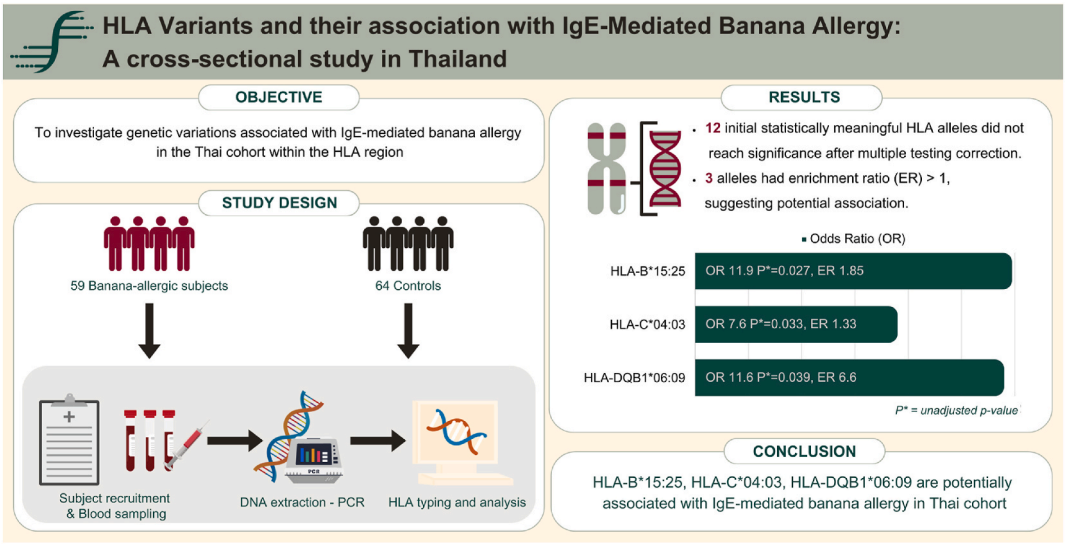

Fig. 1. Graphical abstract.

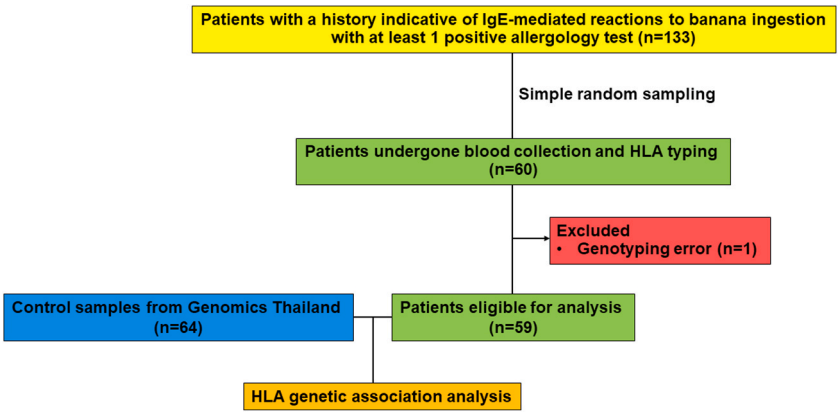

Fig. 2. Flow diagram of study participant.

Table 1
Demographic and clinical characteristics of banana-allergic cases (n = 59).

Demographic factors	Cases
Age, years (mean ± SD)	37.0 ± 7.3
Age of onset of banana allergy (mean ± SD)	34.0 ± 8.78
Sex: Female, n (%)	47 (79.6 %)
At least 1 banana-associated episode in life, n (%)	40 (67 %)
Banana-specific IgE (mean ± SD, kUA/L)	1.64 ± 2.83
Atopic-related disease, n (%)	
Allergic rhinitis	34 (57.6 %)
Asthma	3 (5.08 %)
Atopic dermatitis	2 (3.38 %)
Chronic urticaria	9 (15.25 %)
Contact dermatitis	2 (3.38 %)
Other eczema	2 (3.38 %)
Family history (First-degree relative), n (%)	
Banana allergy	2 (3.38 %)
Food allergies other than banana	7 (11.86 %)
At least 1 atopic disease	28 (47.45 %)

Abbreviation: IgE, immunoglobulin E; kUA/L, kilounits of allergen-specific IgE per liter; SD, standard deviation.

Table 2
The frequency of HLA alleles in banana-allergic cases, non-banana allergic controls, and allele frequency in Thai population.

HLA alleles	Allele frequency		Odds ratio (95 % CI)	p-value*	Adjusted p-value	Allele frequency in the Thai population (n)	Enrichment ratio
	Cases	Controls					
A*02:03:01	0.042	0.123	0.316 (0.087–0.958)	0.034	ns	0.098 (1078)	0.429
B*15:25:01	0.042	0.000	11.872 (NA)	0.027	ns	0.023 (661)	**1.849**
B*40:01:02	0.051	0.156	0.290 (0.091–0.798)	0.010	ns	0.065 (1061)	0.789
C*04:03:01	0.060	0.008	7.636 (0.950–346.911)	0.033	ns	0.045 (612)	**1.332**
DQA1*01:02:01	0.134	0.048	3.092 (1.078–10.060)	0.022	ns	0.219 (929)	0.611
DQA1*01:04	0.018	0.098	0.172 (0.027–1.391)	0.013	ns	0.042 (142)	0.426
DQA1*01:05:01	0.036	0.000	10.493 (NA)	0.048	ns	Not reported in the Thai population	NA
DQB1*06:09:01	0.039	0.000	11.558 (NA)	0.039	ns	0.006 (594)	**6.602**
DRB1*12:01:01G	0.000	0.055	0.067 (NA)	0.015	ns	0.009 (758)	0
DRB1*14:54:01	0.000	0.047	0.079 (NA)	0.03	ns	Not reported in the Thai population	NA
DRB3*01:01	0.000	0.063	0.060 (NA)	0.007	ns	Not reported in the Thai population	NA
DRB5*01:01:01	0.195	0.086	2.575 (1.132–6.142)	0.016	ns	Not reported in the Thai population	NA

Abbreviation: CI, confidence interval; HLA, human leukocyte antigen; NA, not available (due to zero allele frequency); ns, not significant. The *p*-value was estimated by Fisher's exact test. Allele frequency in the Thai population was obtained from 4-digit alleles in the Allele Frequency Net context.

4. Discussion

This study presents an initial exploration into the potential association between genetic factors within the HLA region and banana allergy. Our analysis identified HLA alleles potentially linked to banana allergy at non-multiple correction significance. When examining the association between our sample group and the general Thai population using odds ratio (OR) and enrichment ratio (ER) analysis, the HLA-B*15:25 allele, HLA-C*04:03 allele, and HLA- DQB1*06:09 displayed elevated risk and potential enrichment among the banana allergy population.

In our study, we employed genetic association analysis using statistical tests, OR and ER to determine the relationship between HLA alleles and banana allergy among Thai individuals. We found 12 HLA alleles that showed initial statistical significance; however, none of these remained significant after adjusting for multiple tests. Considering the stringent significant threshold employed and limited functional evidence, these insignificant results cannot be ruled out. Therefore, we further investigate whether these alleles would be relevant to banana allergy. Of these 12 alleles, 6 had an OR greater than 1 which demonstrated strength of association and indicated a higher risk of banana allergy. We then explore whether each genetic variant in our population differs from the general population using enrichment analysis which is described elsewhere [21]. As a result, ER greater than 1 was observed in HLA-B*15:25 (ER 1.849), HLA-C*04:03 (ER 1.332), and HLA-DQB1*06:09 (ER 6.602). This suggests that these particular genetic variants are more prevalent in banana allergy patients than in the general population.

Levine et al. were pioneers in demonstrating the role of HLA class I haplotypes in the IgE response to allergens, specifically from ragweed (*Ambrosia artemisiaefolia*) [23]. Their groundbreaking work set the stage for subsequent studies that identified further HLA-allergen associations. In a recent systematic review [4], HLA gene variants are one of the most reproducible genetic associations with food allergy. The connection between HLA and food allergy was first documented in 1997 [11], and subsequent studies have identified numerous related HLA loci [12,13]. A notable recent study from Canada [12] involving a pediatric cohort demonstrated a marked association between HLA-DQB102 and DQB1*06:03P with peanut allergy. Additionally, in a study with 11,011 Japanese women, researchers identified genetic markers in the HLA-DR/DQ region linked to shrimp and peach allergies. The most strongly associated genetic sequences were HLA-DRB1*04:05-HLA-DQB1*04:01 for shrimp and HLA-DRB1*09:01-HLA-DQB1*03:03 for peach [19]. In addition, a recent study from the Qatar Biobank cohort also demonstrated associations between HLA class II alleles and food sensitization. The study identified both positive and negative associations with 24 HLA class II alleles, indicating that certain variants could function as risk or protective alleles in food allergen sensitization. However, the study did not find significant results for DQB1*06:09, as it did not include banana or fruit allergens in the analysis [24].

Interestingly, our putative associations of HLA-B*15:25 and HLA-C*04:03 which are located in Class I HLA alleles are intriguingly in line with several HLA studies on food allergies. Previous researches has implicated both HLA class I and II alleles in nut allergy development [25]. However, the relationship between Class I HLA alleles and atopy is not as well-defined, given that Class I HLA does not directly influence the recognition of allergenic epitopes during antigen processing and presentation. Only a few studies have reported a heightened frequency of specific Class I HLA alleles in atopic diseases. For instance, Kruszewski et al. [26] highlighted an increased prevalence of HLA-Bw53 in atopic patients, and a 2002 study by Hetherington et al. [27] found that HLA-B57 was significantly more common in white males who had a hypersensitivity reaction to the antiretroviral drug abacavir.

Moreover, compared to other well-established associations of HLA and other traits such as drug hypersensitivity reactions in allopurinol (OR 96.60; 95%CI 24.49–381.00) [28], and carbamazepine (OR 79.84; 95 % CI 28.45–224.06) [29], our magnitude of association was relatively low. Additionally, the previous study of ER in drug hypersensitivity reactions to beta-lactam antibiotics reported that ER $>$ 2 was considered enriched in their context [21]. Comparing to our results, only the HLA-DQB1*06:09 had ER higher than 2. This association is mechanistically plausible given that HLA-DR and HLA-DQ molecules are predominantly found on key antigen-presenting cells, such as B cells, macrophages, and monocytes. These cells are pivotal to allergy development and could explain the HLA genetic region and diverse allergic conditions. Given that these HLA molecules possess distinct molecular polymorphisms within their peptide-binding groove, such variations could modify the binding efficiency of antigen-presenting cells to certain food allergens. These suggest the need for further downstream experiments particularly in functional immunogenetic study, and immune assays of each variant and allergic response.

With the advancment of genotyping techniques, Genome-Wide Association Studies (GWAS) have become a cornerstone in genetic epidemiology and have successfully in identified thousands of genetic variants associated with various allergic conditions. GWAS is advantageous as it is an unbiased approach that scans the entire genome for associations, rather than focusing on previously identified genes or regions. This can lead to the discovery of novel risk loci that may not have been considered in hypothesis-driven studies. GWAS has rapidly developed and yielded numerous successful discoveries that consistently replicate across different ethnic groups [7]. Because GWAS has a standardized approach, results from different studies can be easily combined in a meta-analysis or replicated in independent cohorts. Moreover, GWAS results can be combined to create polygenic risk scores, which might predict individual risk for certain diseases or conditions. Consequently, other genomic variants related to food allergies might have been overlooked in our candidate gene study approach, including potential candidates such as *FLG*, and *IL13*, among others [4].

Our study has certain limitations worth noting. First and foremost is the modest sample size used to explore a complex genetic trait within the HLA region, which may have limited our statistical power and led to non-significant findings. Given that banana allergy is rarely reported, a larger cohort will be essential for future investigations. Additionally, our investigation relied on a candidate gene approach based on a pre-specified hypothesis, which may have inadvertently overlooked other genomic loci relevant to banana fruit allergy. Further research on other genomic regions using the GWAS framework with a well-powered population would be beneficial. Another issue that our study did not address is the involvement of epigenomic and epigenetic factors in food allergy. There are many epigenetic loci associated with food allergy from population-based studies using epigenome-wide investigations targeting candidate

genes. This novel finding highlights several immune pathways involved in food allergy and might add to the understanding of the dramatic increase in food allergy prevalence in the future [30]. Finally, the control group was selected from a database lacking specific data on banana allergy, the rarity of banana allergy in the general population suggests minimal impact on the interpretation of our results. Despite these limitations, our study is the first genetic investigation conducted in patients with allergist diagnosed banana allergy confirmed by objective allergology tests, revealing potential associations with specific HLA. These findings offer valuable insights for future research and may have implications for understanding the pathophysiology of banana allergy and developing primary prevention strategies for food allergies, including banana allergy.

5. Conclusion

In conclusion, our exploration of genetic associations within the HLA region regarding banana allergy has provided intriguing preliminary findings. The potential associations observed between specific HLA alleles and banana allergy, compared with the broader Thai population, highlight the need for further research in this area. Though our results did not reach stringent statistical significance after Bonferroni correction, the trends in HLA allele frequencies suggest a genetic basis that may contribute to the development of banana allergy.

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Institutional review board statement

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Siriraj Institutional Review Board (SIRB), Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand (protocol code 158/2563 (IRB4) and May 20, 2020).

Informed consent statement

Informed consent was obtained from all subjects involved in the study.

Data availability statement

The data associated with our study has been included in the supplementary materials.

CRediT authorship contribution statement

Irin Vichara-anont: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Methodology, Formal analysis, Data curation, Conceptualization. **Lalita Lumkul:** Writing – review & editing, Writing – original draft, Validation, Supervision, Software, Methodology, Investigation. **Settawut Taratikhundej:** Writing – review & editing, Writing – original draft, Visualization, Project administration, Formal analysis, Data curation. **Manop Pithukpakorn:** Writing – review & editing, Writing – original draft, Supervision, Software, Resources, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Ekkapong Roothumnong:** Writing – original draft, Validation, Software, Investigation, Formal analysis. **Chamard Wongsa:** Writing – review & editing, Supervision, Data curation. **Thanachit Krikeerati:** Writing – review & editing, Writing – original draft, Supervision. **Aree Jameekornrak Taweechue:** Writing – review & editing, Project administration, Investigation. **Orathai Theankeaw:** Writing – review & editing, Project administration, Investigation. **Nathachit Limjunyawong:** Writing – review & editing, Writing – original draft, Supervision, Data curation. **Nitat Sookrung:** Writing – review & editing, Supervision. **Torpong Thongngarm:** Writing – review & editing, Conceptualization. **Mongkhon Sompornrattanaphan:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the author(s) used ChatGPT 4 in order to correct and recheck only English grammar. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e32787.

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