SRI VENKATESWARA INTERNSHIP PROGRAM FOR RESEARCH IN ACADEMICS (SRI-VIPRA)

SRI-VIPRA

Project Report of 2024: SVP - 2460

"PREVALENCE OF POLLEN AND FOOD ALLERGIES IN SOUTHEAST ASIA : A META-ANALYSIS STUDY"

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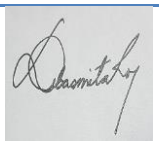
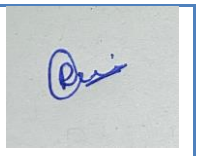
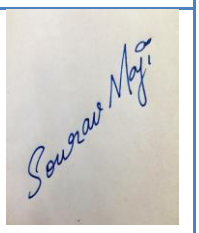
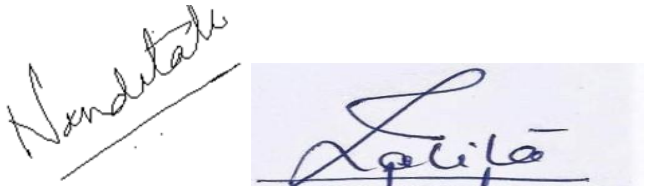
2		DEBASMITA ROY	1322027	B.Sc. (Hons) Biological Science (III Year/ V Sem)	

3		RASHMI	1223030	B.Sc. (Hons) Biochemistry (II Year/ III Sem)	
4		SOURAV MAJI	1223032	B.Sc. (Hons) Biochemistry II Year/ III Sem)	


Signatures of Mentors

Certificate of Originality

This is to certify that the aforementioned students from Sri Venkateswara College have participated in the summer project SVP- **2460** titled "**PREVALENCE OF POLLEN AND FOOD ALLERGIES IN SOUTHEAST ASIA : A META-ANALYSIS STUDY"**.The participants have carried out the research project work under our guidance and supervision during 1st July, 2024 to 30th September 2024. The work carried out is original and carried out in an online/offline/hybrid mode.



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ABSTRACT

The increase in the prevalence of allergies in recent times globally as well as in Asian populations can be attributed to a combination of factors, including globalization, lifestyle changes, urbanization, climate change, and advancements in medical awareness. While the spread of exotic species and global dietary shifts play a significant role in the rise of food and pollen allergies, other factors such as the hygiene hypothesis, air pollution, microbiome diversity, and increased awareness also contribute to this complex issue. Addressing these challenges requires a multifaceted approach, including improving public health policies, promoting healthier diets, and taking action to mitigate the environmental impacts of urbanization and climate change. The present study attempts to extract data from the publications obtained through systematic review mainly regarding the prevalence of food and pollen allergies in the Asian population. The extracted data was subjected to meta analysis using the R software to understand the allergy scenario in this population. The statistical software packages META and GRID were used. The results are presented using Forrest graphs. The conclusions obtained were that the percentage of the Asian population showing food allergy versus pollen allergy was found to be 38.7% and 33.9% respectively. Also, the highest percentage of the population showing allergy to was for the food item - eggs at 25.6% and lowest to was for the food item - meat at 9.4% respectively. Variation with population is lowest for peanut allergy and highest is for soy.

Introduction:

The major food allergies are caused by a comparatively small number of food items. Whereas, pollen allergies had a wide variety of allergens causing the allergy. The foods that have the most number of published reports were included in this analysis. They are milk, egg, soy, wheat, peanut, meat and seafood. Therefore, food items like fruits, vegetables, seeds, nuts, cereals and several other uncommon food items were excluded from this report. Publications having data for overall food and pollen allergy were also considered for the analysis. Publications with data for the Asian population without any gender or age segregation were included. So, papers with data from populations elsewhere except Asia and gender or age segregation were excluded. Assessment of the data was done under a single diagnostic parameter - SPT (single prick test) sensitisation. Other diagnostic parameters like serum IgE levels, data based on questionnaires and others were excluded. This was done due to the lesser availability of publications with data for other diagnostic tests. Further, several publications were also excluded because of undefined data or data which were available in formats that cannot be used for analysis.

Understanding the concept of allergy:

Allergy epidemics are becoming more commonplace worldwide, severely affecting people's quality of life and financial stability (1). Food allergies, certain kinds of asthma, conjunctivitis, rhinitis, urticaria, eczema, angioedema, eosinophilic illnesses, including eosinophilic esophagitis, and medication and insect allergies are manifestations of severe allergic conditions that might prove fatal. Roughly 200–250 million people globally are affected by food-related allergies, while nearly three hundred million individuals worldwide deal with asthma. Drug allergies affect 10% of the population, and 400 million individuals suffer from rhinitis. Furthermore, allergic disorders frequently coexist in the same person, one illness complementing the other. This necessitates a comprehensive strategy for both diagnosis and treatment, as well as increased patient, family physician, and specialist understanding of the fundamental causes (2). Research on the intricate and reciprocal interplay between the immune and neurological systems, which mediates allergic reactions, is still ongoing. Nonetheless, the information we currently possess aids in our comprehension of the molecular processes that underlie the communication that takes place between the skin, gut, and lungs during an allergic reaction (1).

A hypersensitive reaction brought on by particular immunological pathways can be defined as an allergy (3). Exaggerated or inappropriate immune responses to an antigen or allergen are known as hypersensitivity reactions (HR) (4). Allergy-induced inflammation occurs in individuals with allergies when they are repeatedly or persistently exposed to allergens, which are usually naturally occurring harmless compounds found in the environment. This ultimately results in significant problems in the organs' function and persistent changes to the affected organs (5). Coombs and Gell grouped hypersensitivity reactions under four categories: type I, type II, type III and type IV, out of which the first three are referred to as immediate hypersensitivity reactions (IHR) as they manifest within 24 hours after exposure. They are mediated by antibodies such as IgE, IgM, and IgG (4). Allergies are categorized under Type I hypersensitivity response. IgE antibodies which are produced by the immune system in reaction to foreign proteins (allergens) such

pollens, animal dander, dust mites, certain dietary items, and many more, mediates this type of hypersensitivity reactions . The histamine granules that are produced during the reaction and result in inflammation are bound by these antibodies (IgE) to mast cells and basophils. Anaphylactic shock, bronchial asthma, allergic rhinitis, allergic dermatitis, food allergies, and allergic conjunctivitis are among conditions where type I hypersensitivity reactions can be observed (4).

An exaggerated adaptive immune response against non-infectious substances from the environment (allergens), including non-infectious elements of some pathogenic organisms, can be described as an allergy. Allergens fall into two categories. The first class comprises any non-infectious environmental substance that might cause IgE synthesis (thereby sensitizing the individual) so that later re-exposure to that substance induces an allergic reaction. Common allergen sources include latex, some medications, grass and tree pollens, animal dander (skin and fur shedding), house dust mite and their fecal particles, and some foods (such as peanuts, tree nuts, fish, shellfish, milk, and eggs). The second kind is a non-infectious environmental substance that, although it is assumed to happen independently of IgE, can cause an adaptive immune response linked to local inflammation. (for example, allergic contact dermatitis to poison ivy or nickel) (5).

Sensitized individuals have inflammation following exposure to a particular allergen or allergens. An acute reaction, often referred to as an early-phase reaction or a type I immediate hypersensitivity reaction, is caused by a single allergen exposure. This in many instances is followed by a late-phase response. When an allergen is encountered repeatedly or persistently, chronic allergic inflammation and related tissue changes occur. An acute type I hypersensitivity reaction mediated by IgE can happen minutes after being exposed to an allergen. Symptoms could be systemic (anaphylaxis) or localized (hives, urticaria, acute asthma episodes, acute rhinoconjunctivitis in allergic rhinitis, and gastrointestinal symptoms in food allergies). In these reactions, allergen cross links IgE bound to FceRI on mast cells and basophils, causing the release of the cells' various preformed and newly synthesized mediators. Vasodilation, elevated vascular permeability with oedema, and immediate functional alterations in the impacted organs (such bronchoconstriction, mucus secretion in the airways, urticaria, vomiting, and diarrhea) are the outcomes of these events. In addition to facilitating the local recruitment and activation of leukocytes, certain mediators that are produced also aid in the development of late-phase responses. a reaction that usually appears 2–6 hours after exposure to allergens and peaks 6–9 hours later. It usually disappears completely in 1-2 days and is preceded by a clinically noticeable early-phase reaction. Oedema, discomfort, warmth, and erythema are examples of late-phase skin responses (redness). These responses in the lungs are typified by mucus hypersecretion and airway constriction. They show the ongoing generation of mediators by local cells (such as basophils, eosinophils, Th2 cells, and other leukocytes) as well as the local recruitment and activation of these leukocytes. Mediators that trigger late-phase reactions are believed to originate from T cells that identify peptides produced from allergens or from resident mast cells activated by IgE and allergen (these T cells may be recruited to or dwell at sites of allergen challenge). Chronic inflammation brought on by extended or recurrent exposure to particular allergens is usually characterized by significant alterations in the extracellular matrix, as well as changes in the quantity, phenotype, and function of structural cells in

the affected tissues (5). In addition, a high concentration of leukocytes, or innate and adaptive immune cells, are present at the site of inflammation. (5)

Types of allergies

Hypersensitivities ordinarily don't occur at the primary presentation. When a person is uncovered to an allergen for the primary time, the body creates atoms called antibodies against the attacking proteins. This is often called a resistant reaction.

When uncovered to the allergen once more the resistant framework produces expansive sums of antibodies that lead to break down of pole cells that contain chemicals like histamine. This leads to the highlights of hypersensitivities.

This preparation is known as sensitization. Sensitization may take days to a long time. Some of the time sensitization creates as the individual influenced appears indications but never completely creates the hypersensitivity to the allergen. (6)

Symptom Category	Symptoms
Respiratory	- Sneezing - Shortness of breath - Wheezing - Runny nose and eyes
Sinus	- Pain over sinuses (bridge of nose, near eyes, cheeks, temple)
Skin	- Skin rashes (vex rashes, hives) - Swelling of lips or face
Eyes, Mouth, Throat	- Itching (eyes, ears, lips, throat, roof of mouth)
Digestive	- Nausea - Vomiting - Stomach issues (cramps, diarrhea)

Table 1:Showing different symptom categories of hypersensitivity along with the symptoms(6)

Anaphylaxis

When an unfavorably susceptible response is life undermining or severe it is named anaphylaxis or anaphylactic stun. Anaphylaxis includes the full body.

Anaphylaxis includes:

• swelling of the throat and mouth and clogging up of aviation routes driving to trouble breathing, trouble in talking or swallowing

• hasty and tingling somewhere else within the body

• shortcoming and collapse frequently with obviousness due to sudden drop in blood weight

Anaphylaxis requires pressing crisis administration. (6)

Classification of hypersensitivities

Sorts of sensitivity are classified to represent cause, seriousness and conceivable administration and anticipation. These incorporate – • Type I Hypersensitive This is often moreover known as prompt or anaphylactic-type responses. This may be caused due to dust, nourishments and drugs and creepy crawly stings.

• Type II Hypersensitivity This includes particular antibodies called the Immunoglobulin G (IgG) and IgM. There's official to and crushing the cell the counter acting agent is bound on. This sort of response is seen after an organ transplant when the body refuses to see the transplanted organ as its claim.

• Type III Hypersensitivity Usually an Safe complex-mediated reaction. The safe complex is the bound shape of a counter acting agent and an antigen. This leads to a cascade of responses within the body which goes on to crush neighborhood tissues. Cases of this condition incorporate glomerulonephritis and systemic lupus erythematosus (lupus, SLE).

• Type IV Hypersensitivity touchiness Delayed or cell-mediated responses are interceded by extraordinary resistant cells called the T-cell lymphocytes. The T cells take from a number of hours to a number of days to mount an unfavorably susceptible reaction. Illustrations incorporate contact dermatitis such as harm ivy rashes. Common hypersensitivities Hypersensitivities commonly signify Sort I touchiness. This incorporates unfavorably susceptible rhinitis characterized by runny nose, eyes and wheezing. Two major categories are regular unfavorably susceptible rhinitis (SAR) and lasting unfavorably susceptible rhinitis (Standard). Whereas SAR is related with presentation to dust at certain seasons, Standard happens nearly all around the year. Unfavorably susceptible rhinitis influences an assessed 20-40 million individuals in the United States. Other assortments of sort 1 response are nourishment and sedate hypersensitivity and hypersensitivities due to creepy crawly poison. Creepy crawlies which will lead to sensitivities incorporate bees, wasps, yellow coats, ants, hornets etc. Allergic asthma is additionally a sort 1 unfavorably susceptible response. This happens when the allergen is breathed in. Common allergens incorporate dust, creature dander, fungal spores or molds, clean vermin etc. There is serious wheezing, shortness of breath, hack and thick bodily fluid discharges(6).

Classification Of Food Allergy

A food allergy or intolerance may be identified based on the pathophysiological mechanism of the reaction. The phrase "adverse food reaction" refers to any abnormal clinical response linked to the consumption of a food. An adverse physiologic reaction to a food is referred to as food intolerance. It can be caused by the food itself, such as by a toxic contaminant or pharmacologic active component, or it can be caused by the host, such as metabolic disorders, idiosyncratic responses, or psychological disorders. It can also be dose dependent and may not always be repeatable. Most negative responses to food are thought to be caused by food intolerance. An aberrant immune response to a meal that develops in a vulnerable host is referred to as a food allergy These effects are frequently not dosage dependent and are repeatable each time the meal is consumed. Food allergies can be further classified into three categories based on the immunological mechanism involved: a) IgEmediated food allergy reactions, which are best described and are mediated by antibodies belonging to the Immunoglobulin E (IgE); b) cell-mediated food allergy reactions, which occur when an immune system cell component causes the food allergy and primarily affect the gastrointestinal

tract; and c) mixed IgE-cell-mediated food allergy reactions, which occur when both IgE and immune cells are involved in the reaction (Fig. 1) (7)

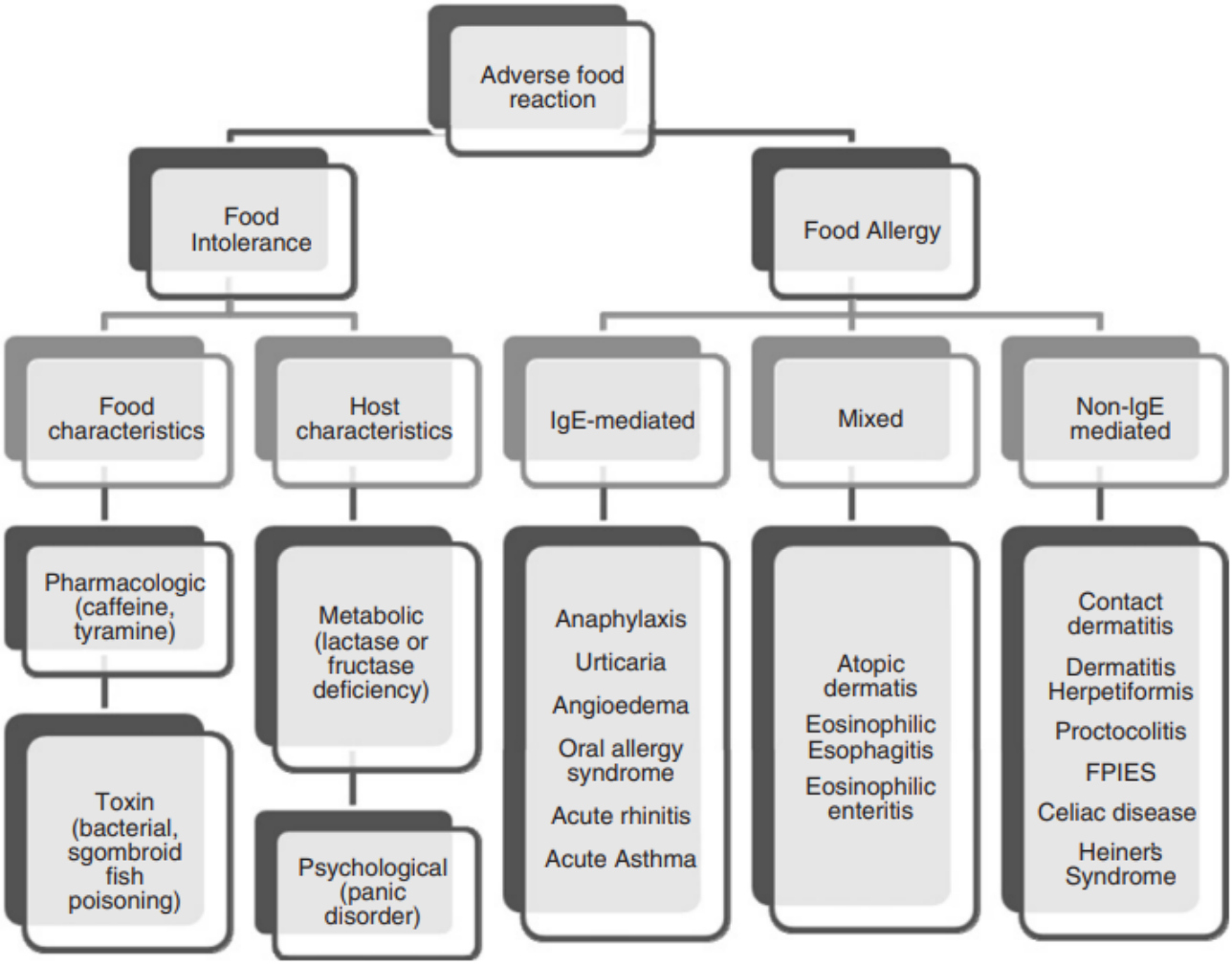

Figure 1: Classification Of Adverse Reactions To Foods(7)

Food allergy

According to the authoritative definition issued in 2010 by an Expert Panel Report sponsored by the National Institute of Allergy and Infectious Diseases (NIAID), food allergy is defined as "an adverse health effect arising from a specific immune response that occurs reproducibly on exposure to a given food" and food intolerance as "nonimmune reactions that include metabolic, toxic, pharmacologic, and undefined mechanisms"

Strict studies of the incidence of food allergies show a tendency towards higher rates of adult-onset cases and greater persistence of paediatric food allergies than previously recognised, at least in westernised nations . The foundation of all forms of food allergy prevention, from elementary to tertiary, is this increasing spectrum, therefore the influence of therapeutic food allergy regimens on nutritional demands will consequently need to be altered in accordance with it . Increasing nutritional challenges for diseases where long-term food elimination is the primary therapeutic strategy are brought about by this evolving

landscape of immediate hypersensitivity reactions to food and increased knowledge of non-IgE- or mixed IgE/non-IgE immunological responses. This highlights the critical role of an appropriate dietary approach that is strictly driven by appropriate diagnostics. (8)

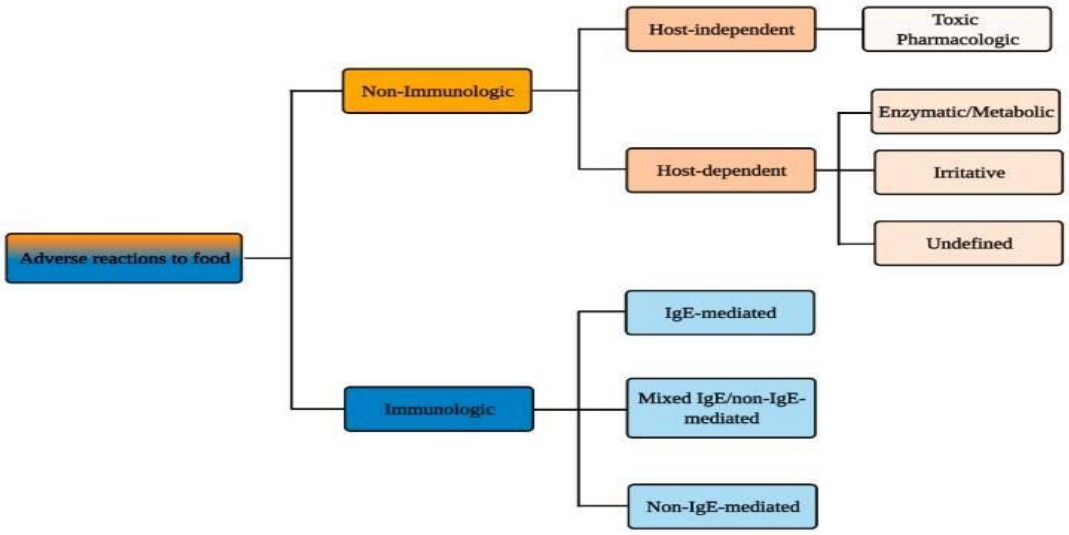

Figure 2:Immunologic vs. non-immunologic adverse reactions to food.(8)

Immunologic Adverse Reactions to Food:

Based on whether IgE-mediated or other immune responses to ingested antigens are involved, adverse immunological reactions to food can be categorised.

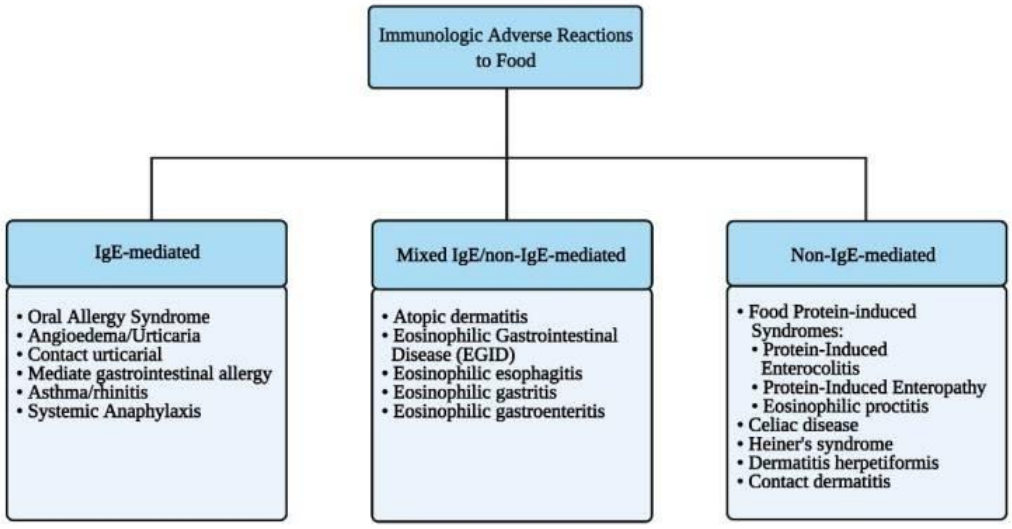

Figure 3:Immunologic adverse reactions to food.(8)

The World Allergy Organisation (WAO) states that an IgE-mediated food allergy is typically characterised by its quick onset, which might occur minutes to hours after eating the allergen. In cases of systemic anaphylaxis, the signs and symptoms may be modest and limited to mucocutaneous manifestations, or they may encompass many systems that are simultaneously impacted (8)

Cutaneous: Urticaria-angioedema/pruritus	Food consumption or contact (food allergies are thought to be the cause of 20% of acute urticarial cases); the proportion of the affected skin can be used to gauge the severity of an IgE-mediated food allergy.
Respiratory: Rhinoconjunctivitis/asthma	Infrequently isolated; often linked to involvement of other organs or apparatus; perhaps brought on by intake of allergens or inhalation of aerosols (as in baker's asthma)
Neurological	Weakness or dizziness, altered mental state, and unconsciousness (usually linked to anaphylaxis)
Cardiovascular	Cardiovascular collapse, hypotension, and tachycardia (usually linked to allergy)

Table 2: Clinical presentations of IgE-mediated food allergy beyond the GI system.(8)

The oral allergy syndrome (OAS) and the symptoms of immediate GI hypersensitivity are the primary clinical manifestations of rapid hypersensitivity reactions to food, which frequently affect the gastrointestinal (GI) tract. Pollen-food syndrome, or OAS, is characterised by minor oedema, erythema of the perioral and oral mucosa, and stinging and tingling feeling of the oral mucosa and/or upper pharynx that happens minutes after consuming certain foods, especially fresh fruits and vegetables.Patients with respiratory allergies (rhinoconjunctivitis, asthma) who have specific IgE directed against pan allergens—proteins with homologous epitopes found in seasonal or perennial aeroallergens, like pollens—and certain foods, mainly fruits and vegetables, are the main causes of this localised reaction. Patients with IgE-mediated sensitivity to ragweed pollen may respond to melons (e.g., watermelon or cantaloupe) and banana, while patients with birch-pollen hay fever may experience OAS symptoms after consuming hazelnut, apple, carrot, and celery. The majority of the time, the signs and symptoms are self-limiting and rarely extend beyond the areas that were directly involved in the first encounter with the offending food. This happens because the implicated epitopes are conformational, meaning that specific IgE can only recognise them when the protein is in its normal form and not when it has been denatured by low pH in the stomach. Due to the destruction of epitopes during cooking, most OAS patients are able to tolerate the triggering meal

when it is consumed. These are significant characteristics that set OAS apart from the initial signs and symptoms of a more widespread allergic reaction, including urticaria or angioedema that arises as a separate cutaneous reaction or as a component of an anaphylactic reaction.

Watery or mucous diarrhoea, cramps, nausea, vomiting, and/or stomach pain are signs of acute GI hypersensitivity. The most frequent symptom and the one with the greatest evidence supporting an immunological and IgEmediated explanation is acute vomiting. A list of additional food-induced allergy symptoms caused by IgE can be found in.

Anaphylaxis is a severe allergic reaction that can be potentially fatal and manifest quickly. IgE and other immunological or non-immunological pathways may mediate it. As per the present NIAID criteria, anaphylaxis is considered highly probable in the following cases:

(i) an acute illness that develops quickly (minutes to several hours); (ii) cutaneous manifestations that involve the respiratory or cardiovascular system (dizziness, weakness, tachycardia, hypotension, syncope); or (iii) an association of two or more cutaneous, respiratory, GI, and cardiovascular manifestations linked to exposure to a likely allergen for the patient.

One of the most frequent causes of anaphylaxis is food. According to the majority of surveys, food-induced reactions can be responsible for up to 81% of instances of anaphylaxis in children and for 30 to 50% of cases in North America, Europe, Asia, and Australia. The most common food triggers in the world include peanuts, tree nuts (walnut, almond, pecan, cashew, hazelnut, Brazil nut, etc.), milk, eggs, sesame seeds, fish and shellfish, wheat, and soy; however, any food has the potential to cause anaphylaxis. It is crucial to emphasise that in certain extremely allergic patients, even very small amounts of food can result in a potentially fatal reaction. These patients may experience symptoms from breathing in cooking fumes from trigger foods (like fish) or from coming into contact with bodily fluids (like saliva or seminal fluid) from people who have consumed the food they are allergic to. (8)

Pollen allergy

Green areas play a crucial role in offering ecosystem services by reducing pollution, air temperature, noise, and soil erosion. While they bring undeniable advantages to humans, improper management could lead to various issues. One of the main problems associated with plants is the allergic reactions caused by pollen grains released during pollination (9). Pollen grains, which are a common allergen in atopic patients, can trigger asthma and rhinitis (10). Pollen contains panallergens that are also found in other allergen sources, leading sensitized individuals to potentially experience food allergies (11). Because of their huge estimate, dust cannot enter the thoracic locales of the respiratory tract but can affect the nasopharyngeal mucous layer. Additionally, submicronic-pollen particles can function as respirable particles, reaching deeper into the upper airways and exacerbating asthma, chronic obstructive pulmonary disease (COPD), and other allergic reactions (9). AR is a common respiratory condition that has a substantial impact on patient quality of life (12). Seasonal allergic rhinitis, also called pollinosis, hay fever, or pollen allergy, occurs due to sensitivity to pollen components (13). Grass pollen allergens primarily come from plants in the Poaceae family, which are widely distributed and produce significant amounts of pollen (13). Patients suffering from pollinosis frequently exhibit sensitivity to pollen allergens originating from various grass species because of the cross-reactivity of IgE antibodies to pollen proteins found in grass pollens (13). The prevalent allergens are as follows:

Birch pollen, which are the minuscule particles discharged by trees in the spring and are effortlessly dispersed by the wind.

During spring, oak trees release pollen into the air, similar to birch trees. While oak pollen is not as allergenic as other types, it remains airborne for an extended period, leading to increased exposure for more individuals and the potential for severe allergic reactions.

Grass pollen is the predominant allergen in the summer, often causing the most challenging symptoms to treat. Allergy tablets and shots are the most effective means of alleviating grass pollen allergic reactions.

Ragweed Pollen is produced by the ragweed plant and is most prevalent in late spring and early autumn. (14)

Pollen can be challenging to avoid for allergy sufferers. Minimizing exposure to pollen is possible by staying indoors on windy days during high pollen seasons, using a mask, and refraining from gardening activities (14). There are medications like antihistamines and decongestants that can help relieve symptoms. In cases where these medications are ineffective, some individuals opt for allergy shots. These shots work by gradually desensitizing the immune system to the allergen, reducing the severity of allergic reactions (14).

The susceptibility to allergies varies from person to person, and the individual's immune system plays a significant role in how they interact with allergens. Genetic factors can also make some individuals more prone to allergies, although this is not the case for everyone, as other factors and probabilities also come into play (15).

Reasons addressing the possible increase in allergic manifestations in recent years

In recent decades, the prevalence of allergies has been on a steady rise, particularly in developed countries. This phenomenon has sparked concern among health professionals and researchers alike, prompting investigations into the underlying causes and potential solutions. The World Allergy Organization (WAO) estimates that 30-40% of the global population is affected by one or more allergic conditions (16). A study published in The Lancet found that the prevalence of food allergies increased by 350% between 1997 and 2011 in children in the United States (17).

The Hygiene Hypothesis - One of the most widely discussed theories explaining the rise in allergies is the "hygiene hypothesis." This theory suggests that reduced exposure to microbes and parasites in early childhood, due to improved hygiene and sanitation, may lead to an underdeveloped immune system that is more prone to allergic reactions (18). In cleaner environments, particularly in developed countries, children are less exposed to infections and microorganisms, which play a crucial role in teaching the immune system how to distinguish between harmful and harmless substances. As a result, the immune system may become more prone to overreacting to allergens such as pollen, dust mites, and certain foods (19).

A study published in the New England Journal of Medicine found that children growing up on farms, exposed to a wider variety of microorganisms, had significantly lower rates of asthma and allergies compared to their urban counterparts (20). This supports the idea that early microbial exposure may play a crucial role in shaping the immune system. Studies have shown that exposure to farm animals and their surroundings can help promote a more balanced immune response, reducing the likelihood of allergic diseases like asthma and eczema (21).

Environmental Factors - Changes in our environment, including increased air pollution and climate change, have been linked to the rising prevalence of allergies. A review in the journal Allergy found that air pollutants can act as adjuvants, enhancing the allergenic potential of common allergens like pollen (22).

Air pollution, particularly from traffic-related sources, is a significant contributor. Pollutants such as diesel exhaust particles, nitrogen dioxide (NO2), and ground-level ozone are known to exacerbate respiratory allergies and asthma (23). Exposure to these pollutants not only irritates the airways but also interacts with pollen, making it more allergenic. For instance, studies have shown that pollen grains exposed to pollutants release more allergenic proteins, increasing the likelihood of allergic reactions (24).

Additionally, urbanization alters the natural environment, reducing green spaces and increasing exposure to synthetic materials and chemicals, many of which are irritants or allergens themselves. The lack of vegetation in urban areas can increase the concentration of airborne pollutants and allergens, further contributing to allergic conditions (16).

Climate Change and Pollen Allergies - Climate change plays a crucial role in increasing the prevalence of pollen allergies. Rising global temperatures and elevated carbon dioxide (CO2) levels contribute to higher pollen production in plants, especially allergenic species like grasses, trees, and weeds. Warmer climates also extend the duration of the pollen season, allowing for prolonged exposure to these allergens. Studies have shown that plants such as ragweed, which produce highly allergenic pollen, thrive in environments with elevated CO2 levels (24). This results in a higher concentration of pollen in the air, which exacerbates allergic reactions and leads to a longer allergy season (16).

Additionally, climate-induced changes in weather patterns, such as more frequent thunderstorms, can cause pollen particles to fragment into smaller pieces. These fragmented particles can penetrate deeper into the respiratory system, increasing the severity of allergic reactions, particularly during storm seasons (23).

Climate change is also extending pollen seasons and increasing the potency of allergens. A study in the Proceedings of the National Academy of Sciences reported that pollen seasons in North America have lengthened by an average of 20 days and pollen concentrations have increased by 21% since 1990 (24).

Dietary Changes - The modern Western diet, characterized by processed foods, reduced fiber intake, and increased use of food additives, has been implicated in the rise of food allergies. A study in Nature Medicine suggested that emulsifiers, commonly used in processed foods, can disrupt the gut microbiome and increase the risk of food allergies (25).

Moreover, changes in infant feeding practices, such as delayed introduction of potential allergens, may have inadvertently contributed to increased food allergies. Recent research, including the LEAP (Learning Early About Peanut Allergy) study, has shown that early introduction of peanuts to high-risk infants significantly reduces the likelihood of developing peanut allergies (26).

Another critical factor influencing the rise in allergies is the gut microbiome, the diverse community of microorganisms that live in the digestive tract and play an essential role in regulating immune function. A growing body of research suggests that changes in the composition and diversity of the microbiome, driven by diet, antibiotic use, and lifestyle, are contributing to the rise in allergic diseases (27).

Modern diets, particularly in developed nations, are often high in processed foods, low in fiber, and rich in sugar and unhealthy fats. These dietary patterns have been linked to a reduction in gut microbiome diversity, which weakens the immune system's ability to differentiate between harmful pathogens and harmless environmental substances like pollen or food proteins (19).

Genetic Factors - While genetics alone cannot explain the rapid increase in allergy prevalence, genetic predisposition plays a role in an individual's susceptibility to allergies. A twin study published in the Journal of Allergy and Clinical Immunology found that genetics account for approximately 50-80% of the risk for allergic diseases (28).
However, the interaction between genes and the environment (epigenetics) is increasingly recognized as a crucial factor. Environmental exposures can alter gene expression without changing the DNA sequence, potentially increasing allergy risk across generations (29).

Introduction of Exotic Species - The global trade and transport of plants have introduced new species to various regions, leading to changes in local ecosystems and pollen profiles.. A study in Japan found that the introduction of ragweed (*Ambrosia artemisiifolia*) has led to an increase in pollen allergies in urban areas (30). Similarly, the spread of London plane trees (Platanus × acerifolia) in Chinese cities has contributed to a rise in plane tree pollen allergies, a condition previously rare in the region (31).

Similarly, the introduction of birch trees to North America has led to increased pollen allergies in regions where these trees were not native (32). This highlights how globalization can alter local allergen profiles and expose populations to new allergenic threats.

Global Food Trade and Changing Diets - The globalization of food trade has led to the widespread availability of foods that were once considered exotic or rare in many parts of the world. This has resulted in increased exposure to potential food allergens. A review in the Journal of Allergy and Clinical Immunology reported that the prevalence of peanut allergies in China has increased significantly in recent years, coinciding with the adoption of a more Westernized diet that includes peanut products (33).
In Japan and South Korea, the increased consumption of dairy products, wheat-based foods, and nuts due to Western dietary influences has been linked to the rising rates of food allergies, especially among children. This is notable because historically, these populations consumed relatively little of these foods (21).

Moreover, the global sharing of food recipes and culinary traditions has led to the incorporation of new ingredients in local cuisines. For example, the popularity of sushi worldwide has increased exposure to fish and shellfish, which are common allergens. A study in the Journal of Investigational Allergology and Clinical Immunology found an increase in fish and shellfish allergies in inland areas of Spain, correlating with increased consumption of these foods (34). As food ingredients and recipes are shared across borders, people in these regions are increasingly exposed to allergens that were not traditionally part of their diet, making food allergies a growing health concern.

Cross-Reactivity and Novel Allergies - Globalization has also led to an increase in cross-reactive allergies. For instance, individuals allergic to birch pollen may develop allergies to certain fruits due to similarities in protein structures. This phenomenon, known as oral allergy syndrome or pollen-food allergy syndrome, has become more common as people are exposed to a wider variety of foods and pollens (35).

In Asian populations, unique patterns of cross-reactivity have been observed. For instance, a study in Japan found that individuals allergic to Japanese cedar pollen often develop cross-reactive allergies to tomatoes and other foods in the Solanaceae family (36). This phenomenon demonstrates how regional allergens can interact with globally available foods to create new allergy patterns.

Furthermore, the introduction of novel foods to different regions can lead to the emergence of new allergies. In addition to the kiwi fruit example mentioned earlier, the growing popularity of quinoa in Asia has led to

reports of quinoa allergies in countries like India and China, where this grain was previously uncommon (37).

The "Asian Paradox" in Allergy Prevalence - Interestingly, some Asian countries have shown a different trend in allergy prevalence compared to Western nations, often referred to as the "Asian Paradox." For example, despite rapid industrialization and urbanization, the prevalence of allergic diseases in some parts of Asia remains lower than in Western countries. A study comparing allergy rates between Hong Kong and mainland China found that children in Hong Kong, which is more Westernized, had higher rates of asthma and allergies than their counterparts in mainland China.

However, this paradox appears to be diminishing as Asian countries continue to adopt Western lifestyles. A longitudinal study in Japan showed that the prevalence of allergic rhinitis and food allergies has been steadily increasing over the past three decades, approaching levels seen in Western countries (38).

The increase in the prevalence of allergies in recent times can be attributed to a combination of factors, including globalization, lifestyle changes, urbanization, climate change, and advancements in medical awareness. While the spread of exotic species and global dietary shifts play a significant role in the rise of food and pollen allergies, other factors such as the hygiene hypothesis, air pollution, microbiome diversity, and increased awareness also contribute to this complex issue. In Asian populations, the shift from traditional to modern lifestyles, rapid urbanization, and climate change have all played a role in the rising rates of allergic conditions. Addressing these challenges requires a multifaceted approach, including improving public health policies, promoting healthier diets, and taking action to mitigate the environmental impacts of urbanization and climate change.

Understanding systematic review and meta-analysis

Systematic review - A comprehensive review of factors influencing Internet skills, use and outcomes of the digital divide was developed through a systematic literature review. This review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) protocol to ensure transparency and reproducibility (39).The first step in a systematic review is to formulate clear and structured questions to solve the problem. After the evaluation questions are developed, changes to the protocol should only be considered if other ways to identify populations, interventions, outcomes, and study designs are identified.Identifying relevant studies requires conducting a systematic search for studies using multiple sources, computer and print, without language restrictions. The selection criteria for the studies should answer the review questions correctly and be determined in advance. Reasons for inclusion or exclusion from studies should be documented.It is important to evaluate the quality of the studies at each stage of the review. The questions developed in the first phase and the study selection criteria should define the minimum level of design. Selected studies should be more rigorously assessed for quality using a comprehensive critical appraisal guide and design-related quality checklists. These assessments help explore heterogeneity, inform decisions about the validity of meta-analyses, assess the strength of conclusions, and provide recommendations for future research.Summarizing evidence involves documenting study characteristics, quality and effects, as well as using statistical methods to explore differences between studies and combine their effects (meta -analysis). Exploring variability and its sources requires prior planning. If a full meta-analysis is not possible, a subgroup meta-analysis can be considered.The interpretation of the findings is to address the issues identified in each of the previous

sections. In addition, the review should assess the risk of publication bias and related biases. It is important to determine whether the generalization can be trusted by detecting heterogeneity. Otherwise, effects found in high-quality studies should be used for conclusions. Each piece of evidence should be graded based on the strengths and weaknesses of the evidence (40).

Meta-analysis **-** Meta-analysis is a quantitative research technique that produces a pooled effect size—a more accurate estimate of the effects of an intervention or exposure—by statistically combining data from several studies that address the same research issue. This method is crucial for getting over the drawbacks of individual research, including sample size or demographic characteristics, and providing a more thorough and trustworthy foundation of data for decision-making. It is employed in the healthcare industry to reconcile discrepancies among studies, enhance statistical power, and compile evidence. Through assessing interventions (such as vaccinations or lifestyle modifications), directing clinical guidelines, and bolstering public health policies, it plays a critical role in disease prevention and control. Heterogeneity, or variability between research, is addressed by using both fixed and random effects models. (41)

There are several steps that are to be followed in Meta-Analysis. They start with formulating the research question. A clearly defined question (PICOS: Participants, Interventions, Comparisons, Outcomes, Study designs) eases the procedure and serves as a guide for the entire process. Literature Search and Study Selection comes forth as the next step. A systematic search of databases (e.g., PubMed, NCBI) identifies relevant studies. Inclusion and exclusion criteria ensures consistency of the relevant publications. This is followed by data extraction wherein information from individual studies, including study design, outcomes, and results, is collected systematically. Statistical Analysis serves as a crucial step, here statistical methods are used to compute pooled effect sizes, often presented as standardized mean differences (SMD), standard error (SE), confidence intervals (CI), and p-values. Fixed-effects models assume the same underlying effect across studies, while random-effects models allow for variability among studies. Heterogeneity is assessed using tests like Cochran's Q and I^2 statistics to determine variability. Substantial heterogeneity suggests the need for a random-effects model. This is followed by the need for visualisation techniques where forest and funnel plots are commonly used. Forest plots show the effect sizes of individual studies along with their confidence intervals, providing a visual summary of the meta-analysis. However, funnel plots are used to detect publication bias, funnel plots illustrate the relationship between study size and effect size, with asymmetry suggesting bias. (41)

Objectives:

1. To perform a systematic review on publications related to food and pollen allergy in the South-East Asian population.Identification of data sources to obtain publications on food and pollen allergy in the South-East Asian population on the basis of set criteria.
2. To perform data extraction from the publications obtained through systematic review and subject it to statistical synthesis (meta-analysis).
3. To establish inclusion and exclusion criteria for setting a baseline as to what publications are to be used in this meta-analysis study. To address potential limitations and gaps that might exist in this research study.

Relevance of this study

The major food allergies are caused by a comparatively small number of food items. Whereas, pollen allergies had a wide variety of allergens causing the allergy. The foods that have the most number of published reports were included in this analysis. They are milk, egg, soy, wheat, peanut, meat and seafood. Therefore, food items like fruits, vegetables, seeds, nuts, cereals and several other uncommon food items were excluded from this report. Publications having data for overall food and pollen allergy were also considered for the analysis. Publications with data for the Asian population without any gender or age segregation were included. So, papers with data from populations elsewhere except Asia and gender or age segregation were excluded. Assessment of the data was done under a single diagnostic parameter - SPT (single prick test) sensitisation. Other diagnostic parameters like serum IgE levels, data based on questionnaires and others were excluded. This was done due to the lesser availability of publications with data for other diagnostic tests. Further, several publications were also excluded because of undefined data or data which were available in formats that cannot be used for analysis.

Methods:

Search strategy:

Guidelines from the Cochrane Handbook for Systematic Reviews were followed closely. Search strategy included several key terms pertaining specifically to allergy and its relevant terminologies. The search technique employed three sets of medical subject heading (MeSH) to locate papers on the prevalence of allergies in databases maintained by the NLM, PubMed, and MEDLINE/PubMed search modes. The first group of terms that were included are ‘prevalence’ and ‘incidence’ and the second group the terms were ‘allergy and immunology’, ‘hypersensitivity’, ‘food/pollen hypersensitivity’, ‘skin tests’. In the third group we included the terms related to food such as ‘dairy products’, ‘milk’, ‘egg’, ‘fish’, ‘shrimps’, ‘crustaceans’, ‘peanut’, ‘hazelnut’, ‘peach’, ‘celery’, ‘soy foods’, ‘walnut’, ‘sesame seed’ and other food items. Articles that fulfilled the condition of having at least 1 term from each series from January 1995 to December 2023 were selected. The search was performed on google scholar, MEDLINE (PubMed), Research gate and NIH. 240 references with their abstracts were obtained through this search. We excluded papers which were found to be duplicates, if the papers had other diagnostic criteria along with SPT sensitisation wherein data was not represented properly. Several other publications were also excluded due to the presence of insufficient data which couldn’t be used for further analysis. We extracted information based on region, total population that underwent SPT, population who had tested positive for SPT and other relaxant information needed for reporting the analysis. We excluded articles if there wasn’t a mention of the initial population who screened for allergy and underwent SPT. All SE’s or 95% CI’s for the range of publications were calculated following the guidelines to ensure uniformity.

Analysis:

Meta-analysis to estimate the prevalence of allergy for overall food and pollen, and for each food item was performed for positive SPT subjects. The analysis was not stratified for age or gender, hence the analysis was done by taking a range of population from infants to adults with no gender separation. The analysis was performed on R, version 4.4.1. The statistical software packages META and GRID were used. The results are presented using Forrest graphs. We analyzed the heterogeneity of the prevalence between studies

for overall food and pollen and each food item with all the studies present under these categories. Estimates performed through META are based on SE and provided approximately 95% CI.

Standardized Mean Deviation:

$$\frac{x - X}{SD}$$

where:
x =Mean

X =Average mean

SD=Standard Deviation

Standard Error:

$$\frac{SD}{\sqrt{n}}$$

where,
SD=Standard Deviation
n=number of SPT positive individuals

Confidence Interval

$$x \pm 1.96 \times \frac{SD}{\sqrt{n}}$$

x = mean
SD= Standard Deviation
n=number of SPT positive individuals

Results

Based on the search strategy about 241 papers were screened and subjected to inclusion and exclusion guidelines. Only papers from Asian populations which had a significant population screened by the standard SPT tests were included. Data that could not be analysed for SMD and SE calculations as well as data that was insufficient was excluded . Based on this 52 papers were taken for metaanalysis. Figure shows the Prisma flowchart that defines the search strategy and the final papers that were taken for analysis. Annexure 1 lists the 51 papers from which data was extracted for meta analysis.

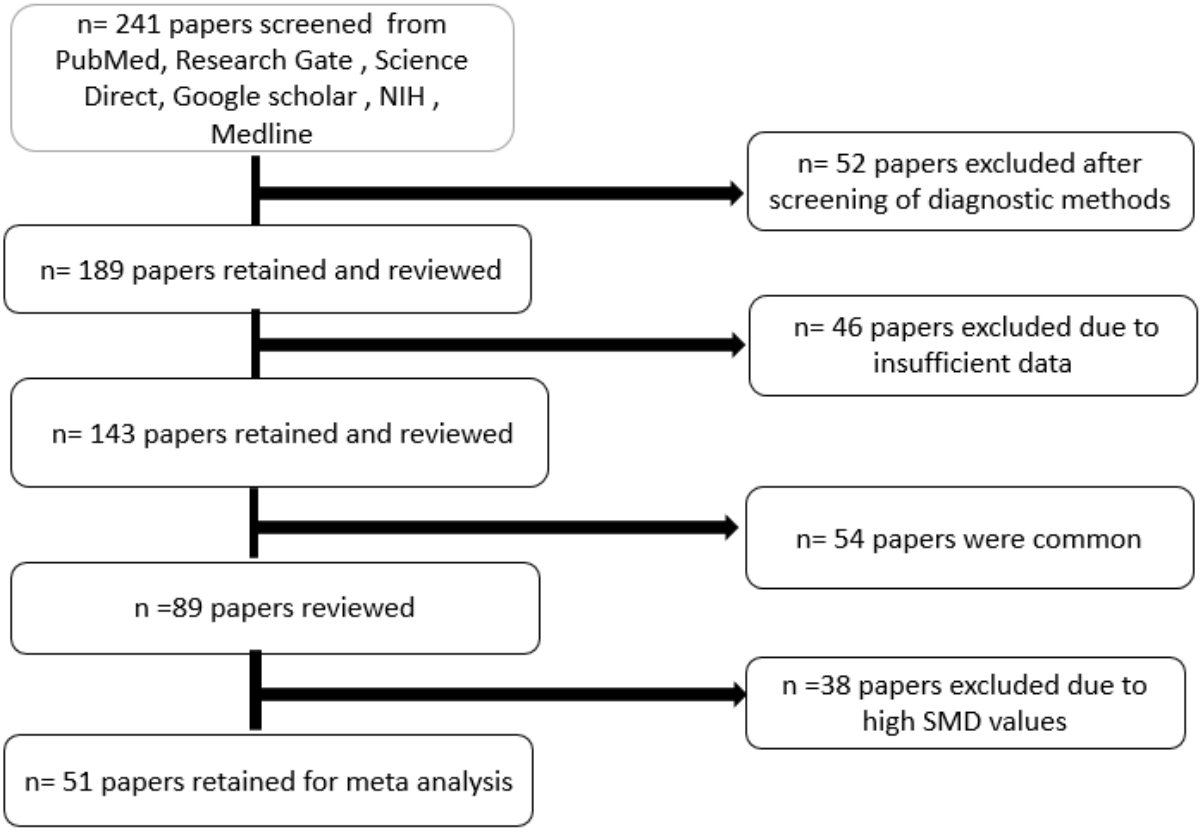

Figure 4 : Prisma Flowchart for the screening of publications during the systematic review.

The following tables list the publications that were screened for calculation of SMD and SE for overall food allergies and pollen allergy in asian population

FAP	Author	Total no of participants (N)	No of SPT positive (n)	Mean	SD	SMD	SE
FAP1	Li Sha et. al, 2019	127	24	0.189	0.07991636253	-2.954878618	0.01631285919
FAP3	F. Orhan et. al, 2009	145	48	0.331	0.06792136998	-1.386060837	0.00980360531
FAP7	Huan Dai et. al, 2019	146	50	0.342	0.06708740567	-1.239325773	0.009487591897
FAP12	Bahri Elmas & Oner Ozdemir, 2017	56	14	0.25	0.1157275125	-1.513409795	0.03092947871
FAP13	Yan Hu et. al, 2010	401	72	0.18	0.04527692569	-5.414306452	0.005335936865
FAP40	Muhammad Inam et. al, 2016	689	270	0.391872279	0.0297089833	-1.119892645	0.001808031146
FAP43	Savitha et. al, 2020	300	92	0.306666667	0.04807401702	-2.464459832	0.005012062745
FAP67	Z A Asha'ari et. al, 2011	90	36	0.4	0.08164965809	-0.3079394512	0.01360827635
FAP68	Kumar et. al, 2006	216	144	0.666666667	0.03928371006	6.148184979	0.003273642505
FAP36	Rong Guo et. al, 2019	8393	2544	0.3	0.00908554730	-13.77387038	0.0001801326924
FAP37	Karmakar et. al 2019	100	30	0.3	0.08366600265	-1.495746742	0.01527525232
FAP56	Thong et. al, 2018	120	77	0.64	0.0547010767	3.927835832	0.006233766234
FAP71	Sripramong et. al, 2023	2678	539	0.2012696	0.01726152	-12.96951548	0.0007435063007
FAP72	A Kongpanichkul et. al, 1998	100	70	0.7	0.05477226	5.018176155	0.006546537215
FAP80	Thong et. al, 2008	74	23	0.31081081	0.09650579	-1.184720014	0.02012284825
FAP81	Hosseini et. al, 2015	313	182	0.58146965	0.03656717	4.275050519	0.002710538977
FAP84	Asha' ari et. al, 2011	90	30	0.3333333	0.08606629	-1.066734152	0.01571348282
FAP88	Kim SR et. al, 2017	134	73	0.5447761	0.05828545	2.052535394	0.006821795933

Table 3.1 -SMD and SE for overall food allergy in Asian Population

PAP	Author	Total no of participants (N)	No of SPT positive (n)	Mean	SD	SMD	SE
PAP3	Supakthanasiri et. al 2014	100	42	0.42	0.07615773106	0.5492632044	0.01175139303
PAP7	Celik *et al*	100	25	0.25	0.08660254038	-1.479972297	0.01732050808
PAP8	Dalkan *et al* 2014	580	66	0.1137931034	0.03908891051	-6.763459346	0.004811512276
PAP9	Elmas et. al 2017	623	71	0.113964687	0.03771216733	-7.005820463	0.004475610848
PAP11	Bharti *et al 2017*	2219	457	0.2059486255	0.01891671191	-9.104158055	0.0008848864599
PAP18	Tanmoy Deb 2020	37	32	0.8648648649	0.06043426966	8.053303316	0.01068337047
PAP19	Manpreet *et al* 2020	159	131	0.8238993711	0.03327989071	13.39337363	0.002907677551
FAP30	Savitha MR et .al 2020	300	171	0.57	0.03785938897	5.06692381	0.002895179835
PAP62	Arbat *et al* 2016	454	99	0.218061674	0.04150097727	-3.857925695	0.004171005153
PAP64	R. A. Hope *et al 1981*	293	11	0.03754266212	0.05731350042	-5.943219241	0.01728067057
PAP22	Kumar *et al* 2020	200	54	0.27	0.06041522987	-1.790431996	0.008221471437
PAP24	Sharma *et al* 2020	140	108	0.7714285714	0.04040610178	9.732668916	0.003888078957
PAP29	Kumar *et al* 2022	200	54	0.27	0.06041522987	-1.790431996	0.008221471437
PAP38	Sam et. al 1999	200	59	0.295	0.05937171044	-1.400824736	0.007729538325
PAP39	Santoso et. al 1999	50	2	0.04	0.1385640646	-2.440527142	0.09797958971
PAP41	A. Kongpanjchkul et. al 1998	100	14	0.14	0.09273618495	-2.568246265	0.02478478796
PAP44	Trinh et. al 2024	423	9	0.02127659574	0.04810163109	-7.419556402	0.01603387703
PAP45	Ho et. al 1996	314	75	0.2388535032	0.04923447399	-2.829640415	0.005685107363
PAP46	Aydin et. al 2010	1552	687	0.4426546392	0.01895031079	3.402861267	0.0007229994523
PAP47	Nadzrah et. al 2016	102	35	0.3431372549	0.08024855659	-0.4365449946	0.01356448181
PAP50	K.L. Liang et. al 2011	419	118	0.2816229117	0.041406567	-2.331669972	0.003811783459
PAP51	Sabit et. al 2021	541	130	0.2402957486	0.0374734472	-3.679234826	0.003286638737
PAP69	Sabit et.al 2020	541	205	0.3789279113	0.03388226022	0.02238784162	0.002366439703
PAP72	L Wan Ishlah et. al *2005*	135	85	0.6296296296	0.05237828009	4.800850059	0.00568122214
PAP77	Hosseini et. al 2014	313	182	0.5814696486	0.03656716659	5.559640162	0.002710538725

Table 3.2 -SMD and SE for overall pollen allergy in Asian Population

Papers reporting food allergies were further screened for specific allergy against the following food items like milk, eggs, soy, wheat, peanut, and meat Tables 4.1-4.6 shows the SMD and SE calculation for this data

FAP	Authors	Total no of participants (N)	No of SPT positive (n)	Mean	SD	SMD	SE
FAP3	F. Orhan, et al. 2009	88	11	0.125	0.0997155044	0.1810941519	0.03006535581
FAP 7	Huan Dai, et al. 2019	50	3	0.047	0.1221897432	-0.4905657689	0.07054628111
FAP13	Yan Hu, et. al. 2010	72	25	0.347	0.09520315121	2.521533076	0.01904063024
FAP16	Bharti Chogtu, et al. 2017	2381	216	0.0907	0.01954026114	-0.8312122945	0.001329546368
FAP43	Mysore RS & Thanuja B 2020	300	9	0.03	0.05686240703	-1.353127828	0.01895413568
FAP92	Jyotshna Mandal, et al. 2009	144	36	0.25	0.07216878365	1.982268336	0.01202813061
FAP36	Rong Guo, et al. 2019	8393	233	0.0278	0.01077016225	-7.348274193	0.0007055767885
FAP37	Karmakar, et al. 2019	100	4	0.04	0.09797958971	-0.6832250012	0.04898979486
FAP46	Muhammad Inam, et al. 2016	689	138	0.2	0.03405026123	2.732956851	0.002898550725
FAP71*	Sripramong, et al. 2023	2678	153	0.057	0.01874336484	-2.664521858	0.001515311236
FAP72	A Kongpanichkul, et al. 1997	100	2	0.02	0.09899494937	-0.878247889	0.07
FAP73	Lee, et al. 2018	95	5	0.0276	0.07326423411	-1.08295823	0.03276476156
FAP75*	Khazaei, et al. 2004	1286	276	0.2146	0.02471187885	4.356524056	0.001487480309
FAP78*	Ahanchian, et al. 2017	371	37	0.1	0.04931969619	-0.14075726	0.008108108108
FAP80*	Thong, et al. 2008	74	1	0.014	0.1174904251	-0.791061103	0.1174904251
FAP81	Hosseini, et al. 2015	313	20	0.217	0.0921713079	1.194058077	0.020610131
FAP85	Yan, et al. 2020	7996	449	0.0562	0.0108688845	-4.66856606	0.0005129343731
FAP88*	Kim SR, et al. 2017	134	1	0.007	0.08337265739	-1.198739592	0.08337265739
FAP89	Kuravi, et al. 2020	603	94	0.161	0.03790792316	1.426031557	0.003909903735

Table 4.1 -SMD and SE for Milk allergy in Asian Population

FAP	Authors	Total no of participants (N)	No of SPT positive (n)	Mean	SD	SMD	SE
FAP3	F. Orhan et al. 2009	88	25	0.284	0.09018736053	-0.1536384912	0.01803747211
FAP7	Huan Dai et al. 2019	50	6	0.1164	0.1309268498	-1.385936118	0.05345066261
FAP40	Muhammad Inam et al. 2016	689	148	0.215	0.03376938883	-2.453590452	0.002775826662
FAP43	Savitha & Basavanagowda 2020	30	16	0.53	0.1247747971	1.860501923	0.03119369928
FAP67	Z A Asha'ari et al. 2011	9	7	0.78	0.1565703858	3.079405773	0.05917804336
FAP92	Jyotshna Mandal et al. 2009	150	56	0.373	0.06462405014	1.162783048	0.00863575195
FAP52	Debarati Dey et al. 2014	5161	1187	0.23	0.01221473139	-5.555279753	0.0003545345478
FAP73	Lee et al. 2018	95	3	0.0165	0.07354760363	-3.825498536	0.04246272876
FAP75*	Khazaei et al. 2004	1286	390	0.3033	0.0232769893	0.2338683036	0.00117867617
FAP80*	Thong et al. 2008	74	2	0.027	0.114610209	-2.363282053	0.08104165596
FAP81	Hosseini et al. 2015	313	14	0.2	0.1069044968	-0.9153614016	0.02857142857
FAP88*	Kim SR et al. 2017	134	1	0.007	0.08337265739	-3.488628756	0.08337265739

Table 4.2 -SMD and SE for Egg allergy in Asian Population

FAP	Author	Total no of participants (N)	No of SPT positive (n)	Mean	SD	SMD	SE
FAP46	Muhammad Inam et al. 2016	689	83	0.12	0.03566916254	-0.5991934146	0.003915199231
FAP22	M. Inam et al. 2016	79	253	0.311	0.02910244954	5.828625266	0.001829655326
FAP43	Savitha & Basavanagowda 2020	30	8	0.26	0.1550806242	0.7649393554	0.0548292805
FAP68	Kumar, et al. 2006	22	7	0.32	0.1763114128	1.013135054	0.06663945023
FAP71*	Sripramong et al. 2023	2678	147	0.055	0.01880349512	-4.593440037	0.001550886138
FAP72	A Kongpanichkul et al. 1998	100	4	0.04	0.09797958971	-1.034631065	0.04898979486
FAP74	Rengganis et al. 2019	100	20	0.2	0.0894427191	0.6554728355	0.02
FAP78*	Ahanchian et al. 2017	371	14	0.037	0.05044870096	-2.068888303	0.01348298247
FAP88*	Kim SR et al. 2017	134	11	0.082	0.08272407586	-0.7177200432	0.02494224734
FAP89	Kuravi et al. 2020	603	42	0.102	0.04669964821	-0.8431054368	0.007205912161

Table 4.3 -SMD and SE for Soy allergy in Asian Population

FAP	Authors	Total no of participants (N)	No of SPT positive (n)	Mean	SD	SMD	SE
FAP42	Z A asha'ari et. al 2011	90	8	0.089	0.1006721163	-1.270154778	0.03559296806
FAP46	Muhammad Inam et.al 2016	689	156	0.23	0.0336935663	0.3897132877	0.002697644284
FAP52	Debarati Dey et. al 2014	5161	1135	0.22	0.012295911	0.2546237119	0.0003649747168
FAP67	Z A Asha'ari et. al 2011	9	8	0.89	0.1106232344	6.084895581	0.03911121962
FAP68	Kumar, et al.2006	22	4	0.18	0.1920937271	-0.19193323	0.09604686356
FAP92	Jyotshna Mandal et. al 2009	138	30	0.2173	0.07529513264	0.005721890445	0.01374694754
FAP71*	Sripramong et. al 2023	2678	236	0.08812546677	0.01845279925	-6.976919922	0.00120117492
FAP73	Lee et. al 2018	95	15	0.08287292818	0.07118286589	-1.882422682	0.01837933694
FAP78*	Ahanchian et. al 2017	371	11	0.02964959569	0.05114195677	-3.660782372	0.01541988015
FAP81	Hosseini et. al 2015	313	11	0.183	0.1165843432	-0.290512161	0.03515150209
FAP84	Asha'ari et al 2011	90	18	0.2	0.09428090416	-0.1789245622	0.02222222222
FAP88*	Kim SR et. al 2017	134	11	0.08208955224	0.08276519781	-1.628457623	0.02495464608
FAP89	Kuravi et. al 2020	603	85	0.153	0.03904612657	-1.635736374	0.004235147056
FAP 93	Manpreet et. al 2019	131	4	0.03	0.08529361055	-2.190892944	0.04264680527

Table 4.4 -SMD and SE for wheat allergy in Asian Population

FAP	Author	Total no of participants (N)	No of SPT positive (n)	Mean	SD	SMD	SE
FAP40	Muhammad Inam et. al 2016	687	79	0.115	0.0358927728	0.09171457492	0.004038252441
FAP67	Z A Asha'ari et. al 2011	90	11	0.122	0.09868038398	0.1042952002	0.02975325526
FAP3	F. Orhan et. al 2009	91	2	0.022	0.103720779	-0.8649000755	0.07334166619
FAP22	M. Inam et. al 2016	812	238	0.293	0.02950224318	6.145020542	0.001912346389
FAP36	Rong Guo et. al 2019	8393	945	0.1126	0.01028283822	0.0867358195	0.0003345007524
FAP37	Karmakar et. al 2019	100	2	0.02	0.09899494937	-0.9263918027	0.07
FAP42	Z A Asha'ari et. al 2011	90	11	0.122	0.09868038398	0.1042952002	0.02975325526
FAP71*	Sripramong et. al 2023	2678	174	0.06497386109	0.01868558029	-2.501086281	0.001416550186
FAP73	Lee et. al 2018	95	7	0.03867403315	0.07287793347	-1.002142528	0.02754526972
FAP74	Rengganis et. al 2019	100	27	0.27	0.08544003745	1.852666444	0.01644294287
FAP78*	Ahanchian et. al 2017	371	15	0.04043126685	0.05085704114	-1.401513756	0.01313123156
FAP80*	Thong et. al 2008	74	2	0.02702702703	0.1146659645	-0.7385023353	0.08108108108
FAP81	Hosseini et. al 2015	313	13	0.14	0.09623688722	0.2939817695	0.02669131011
FAP84	Asha'ari et al 2011	90	23	0.2555555556	0.09094836413	1.581638629	0.01896404486
FAP88*	Kim SR et. al 2017	134	6	0.0447761194	0.08443066044	-0.7927450745	0.03446867279
FAP 93	Manpreet et. al 2018	131	22	0.17	0.08008518192	0.7278736091	0.01707421816

Table 4.5 -SMD and SE for peanut allergy in Asian Population

FAP	Authors	Total no of participants (N)	No of SPT positive (n)	Mean	SD	SMD	SE
FAP3	F. Orhan et. al 2009	90	9	0.1	0.1	0.057	0.03333333333
FAP16	Bharti Chogtu et. al 2017	2219	202	0.09	0.0201356784	4.469678062	0.001416741388
FAP67	Z A Asha'ar et. al 2011	90	3	0.033	0.1031358328	0.3199663891	0.05954550081
FAP92	Jyotshna Mandal et. al 2009	120	32	0.26	0.0775403121	3.353094577	0.01370732012
FAP36	Rong Guo et. al 2019	8393	176	0.02	0.01055289706	1.895214166	0.0007954545455
FAP37	Karmakar et. al 2019	100	2	0.02	0.09899494937	0.2020305089	0.07
FAP73	Lee et. al 2017	95	24	0.132	0.06909413868	1.910437014	0.014103782
FAP75	Khazaei et. al 2003	1286	247	0.192	0.02506154368	7.661140209	0.001594627838
FAP78	Ahanchian et. al 2016	371	3	0.007	0.04813522619	0.1454236441	0.0277908858
FAP84	Asha'ari et al 2010	90	8	0.089	0.1006721163	0.8840581014	0.03559296806

Table 4.6 -SMD and SE for meat allergy in Asian Population

The data in terms of SMD and SE are represented as forrest plots to see the heterogenietiy index (which signifies the variance in the populations scrneed) and the confidence intervals for

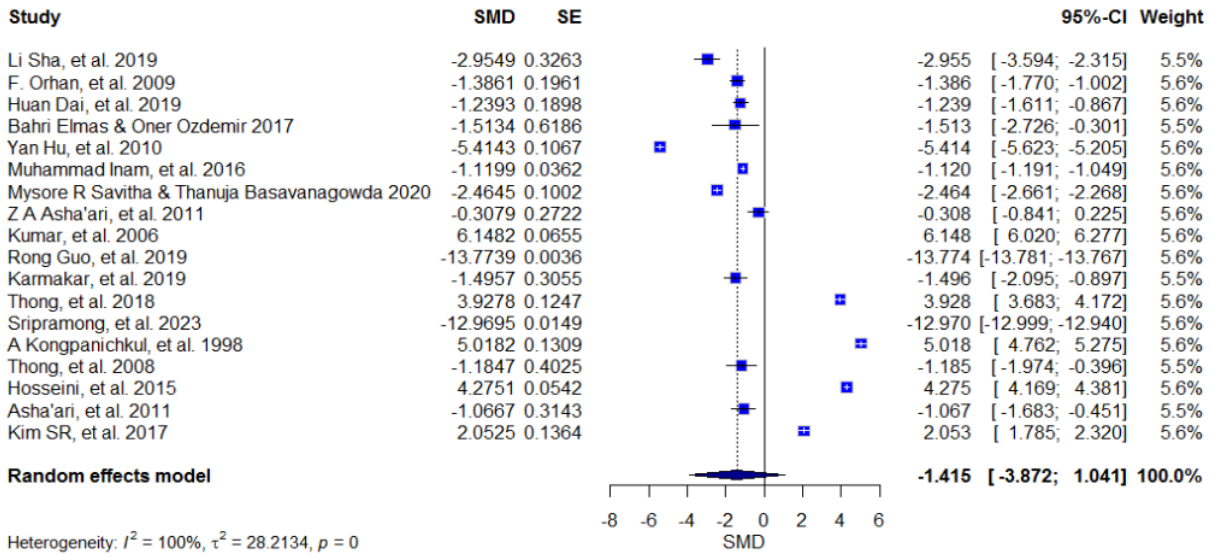

Figure 5 : Forest plot showing the variation in allergy towards food in Asian population

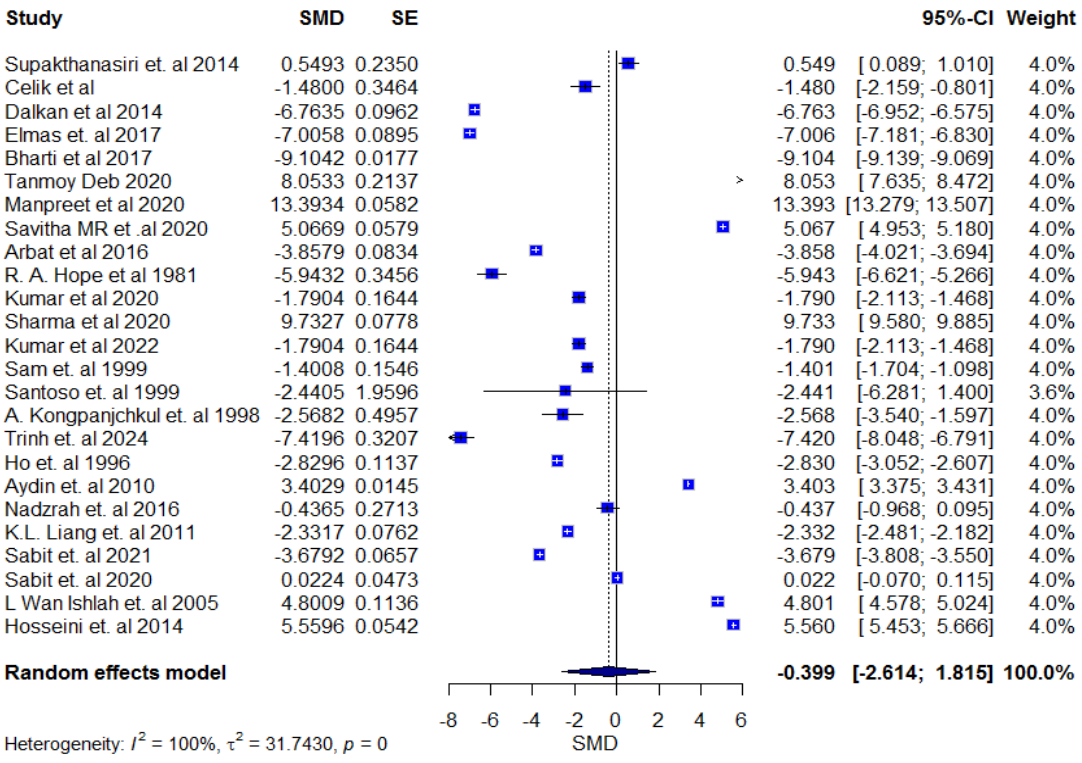

Figure 6 : Forest plot showing the variation in allergy towards pollen in Asian population

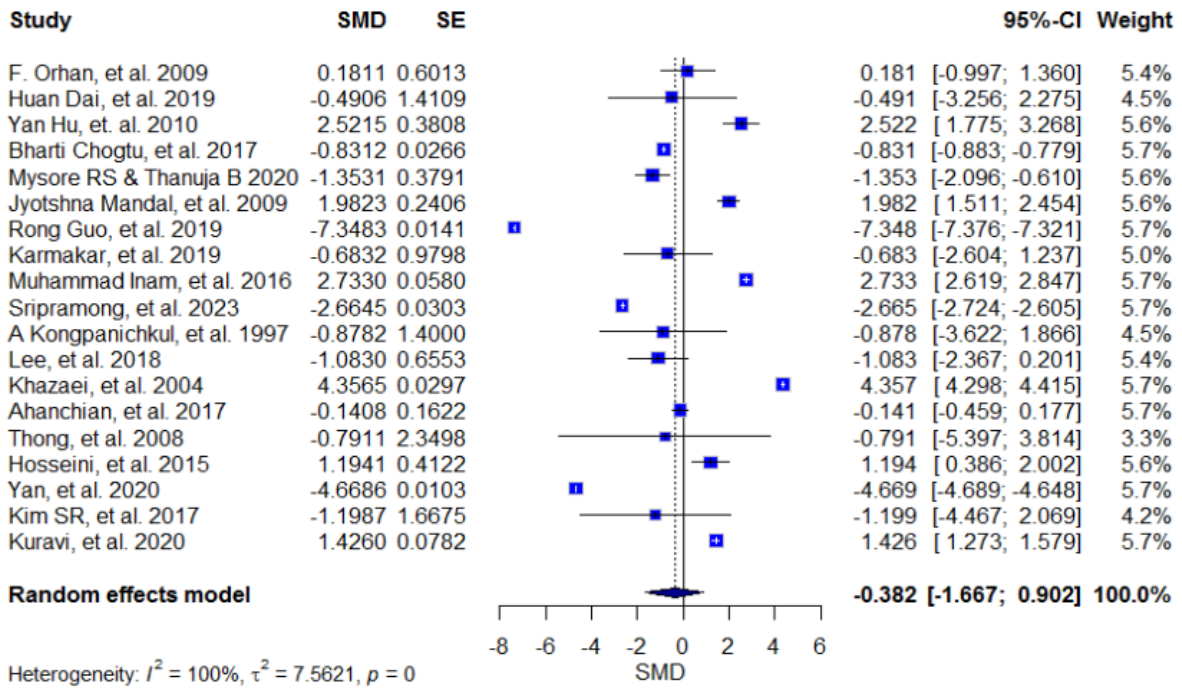

Figure 7 : Forest plot showing the variation in allergy towards specific food item - 'milk' in Asian population

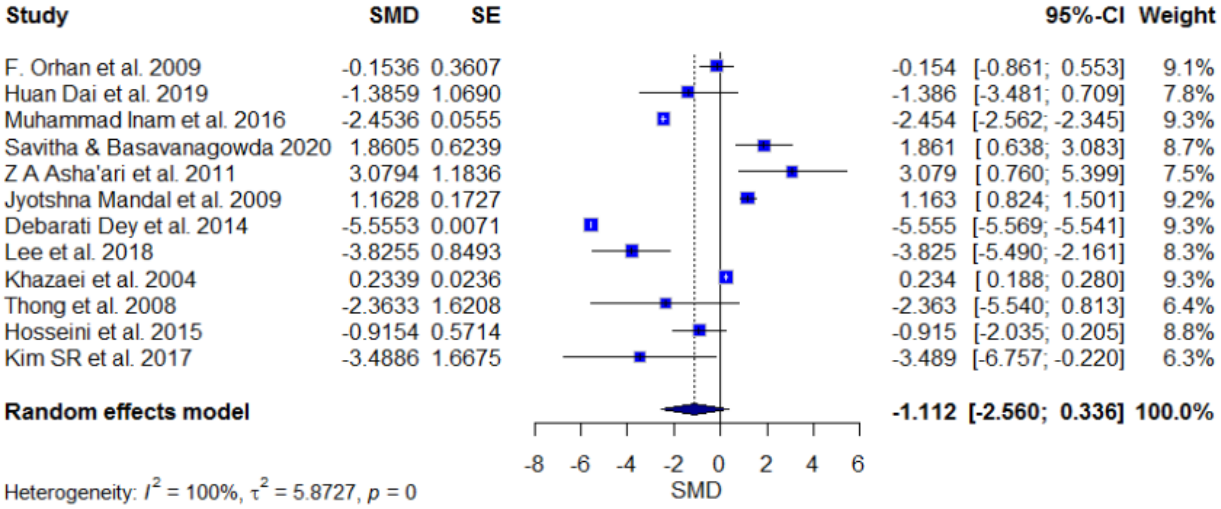

Figure 8 : Forest plot showing the variation in allergy towards specific food item - 'eggs' in Asian population

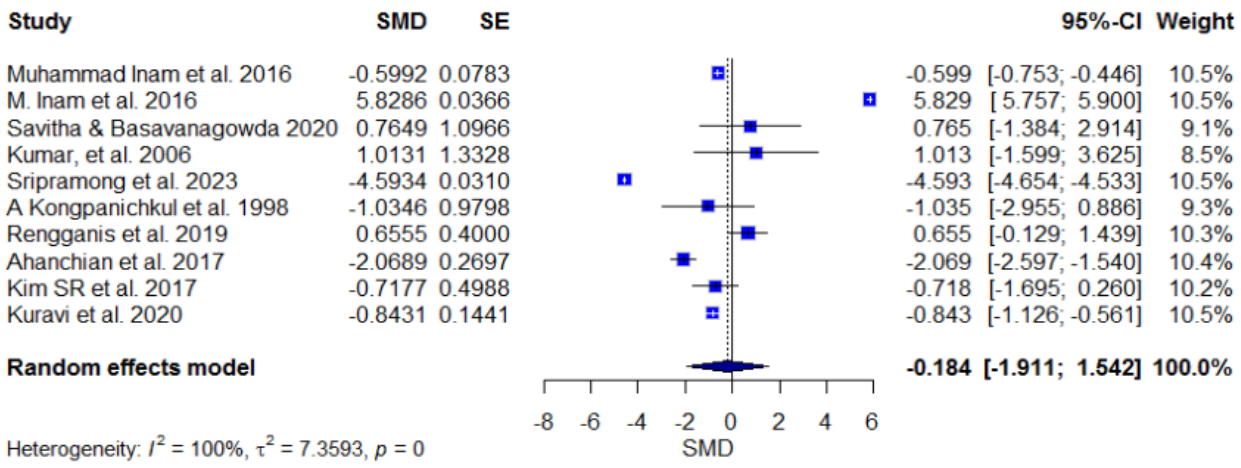

Figure 9 : Forest plot showing the variation in allergy towards specific food item - 'soy' in Asian population

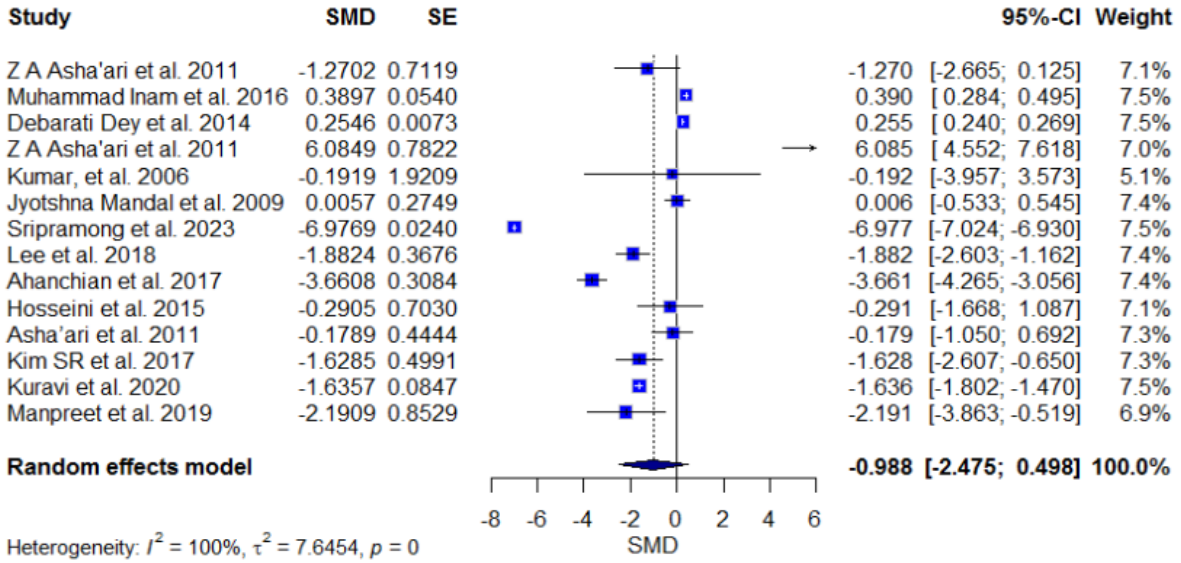

Figure 10 : Forest plot showing the variation in allergy towards specific food item - 'wheat' in Asian population

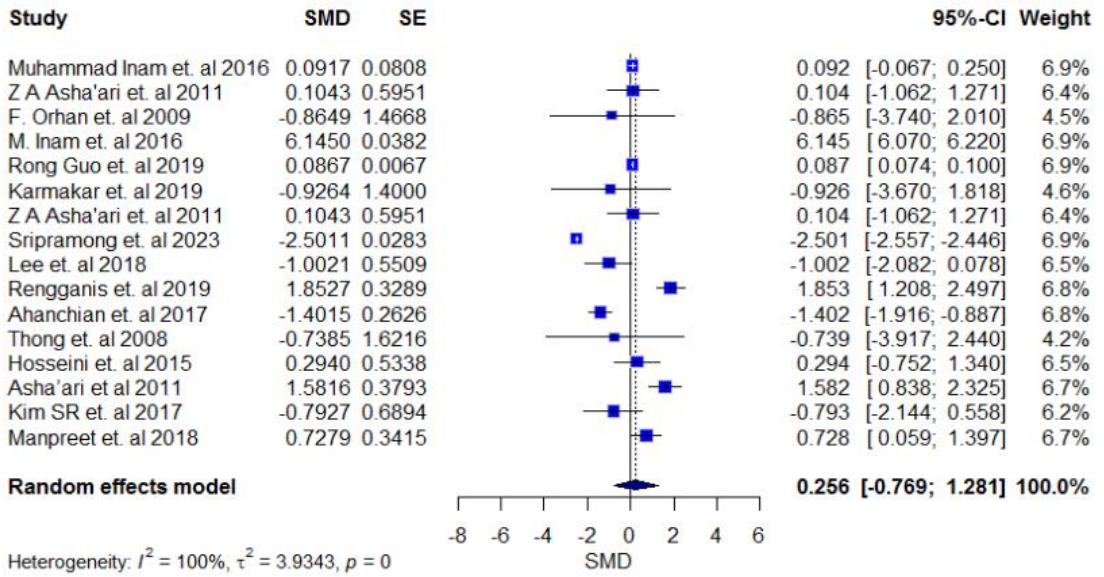

Figure 11 : Forest plot showing the variation in allergy towards specific food item - 'peanut' in Asian population

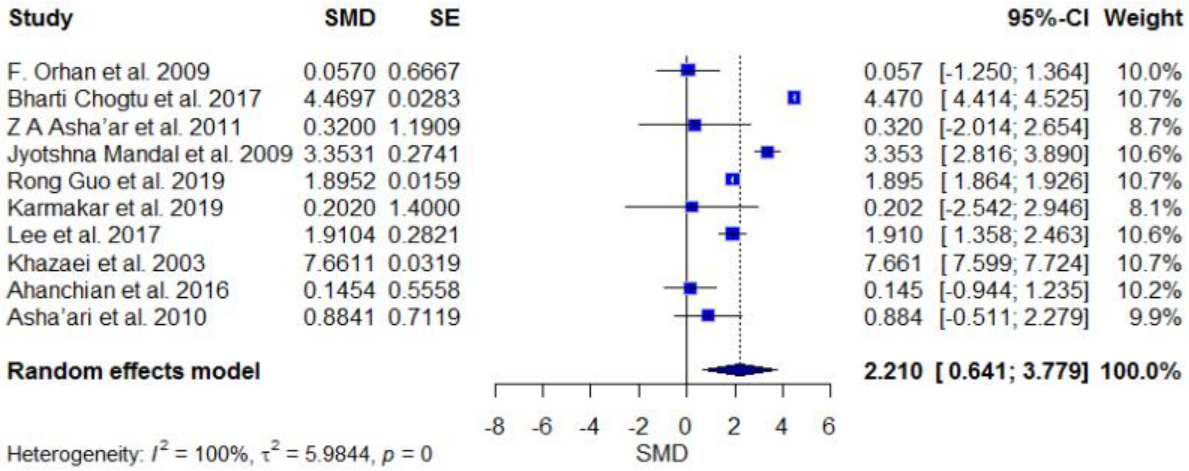

Figure 12 : Forest plot showing the variation in allergy towards specific food item - 'meat' in Asian population

Conclusion:

1. Percentage Asian population showing food allergy versus pollen allergy was found to be 38.7% and 33.9% respectively.
2. Highest percentage of the population showing allergy to was for the food item - eggs at 25.6% and lowest to was for the food item - meat at 9.4% respectively.

3. Variation with population is lowest for peanut allergy and highest variation in the population is for soy.

Reference table:

Reference	Author	Year of publicatio	Place	Sample size
1	Li Sha et. al	2019	China	127
2	F. Orhan et. al	2009	Turkey	145
3	Huan Dai et. al	2019	China	146
4	Bahri Elmas & Oner Ozdemir	2017	Turkey	56
5	Yan Hu et. al	2010	China	401
6	Muhammad Inam et. al	2016	Pakistan	689
7	Mysore R Savitha & Thanuja Basavanagowda	2020	Mysuru, India	300
8	Z A Asha'ari et. al	2011	Malaysia	90
9	KUMAR, *ET AL.*	2006	Delhi, India	216
10	Rong Guo et. al	2019	China	8393
11	Karmakar et. al	2019	India	100
12	Thong et. al	2018	Singapore	120
13	Sripramong et. al	2023	Bangkok, Thailand	2678
14	A Kongpanichkul et. al	1998	Bangkok, Thailand	100
15	Thong et. al	2008	Singapore	74
16	Kim SR et. al	2017	Korea	134
17	Bharti Chogtu, et al.	2017	India	2381
18	Jyotshna Mandal, et al.	2009	Calcutta, India	144
19	Lee, et al.	2018	Suwon, Korea	95
20	Khazaei, et al.	2004	Iran	1286
21	Ahanchian, et al.	2017	Iran	371
22	Yan, et al.	2020	Shanghai, China	7996
23	Kuravi, et al.	2020	Chennai, India	603
24	Debarati Dey et al.	2014	Kolkata, India	5161
25	Rengganis et al.	2019	Jakarta, Indonesia	100
26	Manpreet et. al	2019	Himachal Pradesh, India	131
27	M. Inam et. al	2016	Pakistan	812
28	Supakthanasiri et. al	2014	Singapore	100
29	Celik *et al*	1999	Ankara, Turkey	100
30	Dalkan *et al*	2014	North Cyprus	580

31	Elmas et. al	2017	Turkey	623
32	Bharti *et al*	2017	Karnataka, India	2219
33	Tanmoy Deb	2020	Tripura	37
34	Manpreet *et al*	2020	Himachal Pradesh, India	159
35	Arbat *et al*	2016	India	454
36	R. A. Hope *et al*	1981	Nepal	293
37	Kumar *et al*	2020	Delhi NCR, India	200
38	Sharma *et al*	2020	India	140
39	Kumar *et al*	2022	India	200
40	Sam et. al	1999	Malaysia	200
41	Santoso et. al	1999	Indonesia	50
42	A. Kongpanjchkul et. al	1998	Thailand	100
43	Trinh et. al	2024	Japan	423
44	Ho et. al	1996	Malaysia	314
45	Aydin et. al	2010	Turkey	1552
46	Nadzrah et. al	2016	Malaysia	102
47	K.L. Liang et. al	2011	Taiwan	419
48	Sabit et. al	2021	Philippines	541
49	Sabit et.al	2020	Philippines	541
50	L Wan Ishlah et. al	2005	Malaysia	135
51	Hosseini et. al	2014	Tehran, Iran	313

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