Allergy Bulletin

JULY-SEPTEMBER 2026: AUTUMN EDITION

"A KNOWLEDGE INITIATIVE"



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2014-2018



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2018-2021



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Dr. Mosam Maroo

Presidential Address

IAP Allergy Chapter — Autumn Edition



Dr. Neelam Mohan

President, Indian Academy of Pediatrics (IAP) 2026

A Presidential Perspective: Advancing Child Health and Allergy Care in India

The health and well-being of every child remain the cornerstone of a nation's progress. As pediatricians, we are entrusted not only with treating illnesses but also with nurturing healthier generations through prevention, advocacy, innovation, and compassionate care. It has been a tremendous privilege to serve as the President of the Indian Academy of Pediatrics (IAP), an organization that has consistently championed excellence in child healthcare and professional development across our country.

Throughout my journey as a pediatrician and pediatric gastroenterologist, I have witnessed remarkable advances in pediatric medicine. At the same time, we continue to face evolving health challenges that demand multidisciplinary collaboration and continuous learning. Among these, allergic diseases have emerged as one of the fastest-growing non-communicable disorders affecting children worldwide, including India. Their increasing prevalence calls for greater awareness, timely diagnosis, evidence-based management, and stronger integration into routine pediatric practice.

Indian Academy of Pediatrics has always believed that every pediatric subspecialty contributes significantly to comprehensive child healthcare. Our specialty chapters serve as engines of academic excellence, translating scientific evidence into practical knowledge that benefits children across diverse healthcare settings. I am delighted to see the IAP Allergy Chapter making significant strides in education, research, guideline development, and capacity building for practicing pediatricians.

Today, allergic diseases extend far beyond isolated episodes of rhinitis or eczema. Asthma, food allergy, drug allergy, anaphylaxis, allergic rhinitis, atopic dermatitis, eosinophilic disorders, and environmental allergies significantly influence a child's nutrition, growth, school performance, mental well-being, and overall quality of life. Increasing urbanization, changing lifestyles, environmental pollution, climate change, dietary transitions, and altered microbial exposures have all contributed to this growing burden.

As pediatricians, we must adopt a holistic approach that recognizes the interconnected nature of childhood diseases. My own experience in pediatric gastroenterology has highlighted the close relationship between gastrointestinal disorders and allergic diseases. Conditions such as cow's milk protein allergy, eosinophilic gastrointestinal disorders, food protein-induced enterocolitis syndrome, and functional gastrointestinal disorders often require close collaboration between gastroenterologists, allergists, nutritionists, and primary care pediatricians. Such interdisciplinary care ultimately benefits our young patients and their families.

One of the greatest strengths of the Indian Academy of Pediatrics has been its commitment to continuing medical education. Across the country, IAP has empowered pediatricians through conferences, workshops, webinars, practice guidelines, fellowship programs, and digital learning platforms. These initiatives ensure that children, irrespective of geography, have access to modern, evidence-based pediatric care delivered by well-trained clinicians.

The IAP Allergy Chapter has played an important role in this mission by promoting awareness regarding allergy prevention, rational diagnosis, appropriate use of allergy testing, allergen immunotherapy, and emergency management of anaphylaxis. Equally important has been its emphasis on avoiding overdiagnosis and unnecessary dietary restrictions, ensuring that children receive scientifically sound and patient-centered care.
Looking ahead, I envision pediatric allergy becoming an integral component of routine pediatric practice rather than a niche subspecialty. Every practicing pediatrician should be equipped to recognize common allergic disorders, identify children at risk for severe allergic reactions, provide appropriate first-line management, and refer complex cases when required. Strengthening this continuum of care will improve clinical outcomes and reduce the burden of chronic allergic diseases.

Research must also remain a key priority. India presents unique environmental, genetic, nutritional, and socioeconomic factors that influence allergic diseases differently from many Western populations. Collaborative multicentric research, national disease registries, and indigenous clinical guidelines will help generate robust evidence that reflects our local realities and informs best practices. I am encouraged to see increasing collaboration between clinicians, academic institutions, and international organizations in advancing allergy research within India.

Another area that deserves continued attention is public awareness. Parents today are increasingly exposed to conflicting information through digital media, often leading to misconceptions regarding food allergies, environmental triggers, vaccination, elimination diets, and alternative therapies. Pediatricians have an important responsibility to provide clear, evidence-based guidance and reassure families with empathy and scientific accuracy. Public education campaigns conducted through IAP and its specialty chapters can significantly improve health literacy and reduce unnecessary anxiety among caregivers.

The future of pediatric healthcare lies in collaboration rather than isolated expertise. Whether addressing malnutrition, infectious diseases, allergies, chronic gastrointestinal disorders, adolescent health, or developmental concerns, multidisciplinary teamwork will continue to define high-quality pediatric care. IAP remains committed to fostering such collaborations across specialties, institutions, and regions while maintaining the highest standards of ethics, professionalism, and academic integrity.

As President of Indian Academy of Pediatrics, I take immense pride in witnessing the enthusiasm, dedication, and innovation demonstrated by our members across the country. The collective efforts of our specialty chapters, including the IAP Allergy Chapter, exemplify the spirit of continuous learning and service that defines our Academy. Their initiatives are strengthening pediatric practice, promoting evidence-based medicine, and ultimately improving the lives of countless children.



I congratulate the Editorial Board of IAP Allergy Bulletin for providing an excellent platform to disseminate knowledge, share clinical experiences, and stimulate academic dialogue. Such initiatives play an invaluable role in connecting clinicians, researchers, and trainees while encouraging lifelong learning.

As we move forward, let us reaffirm our shared commitment to ensuring that every child in India receives timely, equitable, and high-quality healthcare. By embracing innovation, strengthening academic collaboration, advancing research, and promoting comprehensive allergy care, we can build a healthier future for our children.

Together, let us continue to uphold the ideals of the Indian Academy of Pediatrics and work towards a future where every child has the opportunity to thrive, free from preventable illness and empowered by the highest standards of pediatric care.





Chairperson's Address

IAP Allergy Chapter — Autumn Edition



Dr. Uppin Narayan Reddy

Chairperson, IAP Pediatric Allergy and Applied Immunology Chapter

Dear Friends and Colleagues,

It gives me great pleasure to greet you through the Autumn Edition of our Bulletin, which is reaching your hands even as PEDALLERCON 2026 is getting under way at the J N Tata Auditorium, IISc, Bengaluru.

The 14th National Conference on Pediatric Allergy and Applied Immunology with Environmental Issues is bringing together national and international faculty who are generously sharing their knowledge and experience with us over these two days. The theme is a timely one — the child with allergic disease is no longer someone we can treat in isolation from the air she breathes, the season she is born into, or the city that is shaping her early years.

What I am particularly delighted by is the emphasis this year on hands-on learning. Four workshops are running alongside the scientific programme:

- **Food Allergy Workshop**
- **Pulmonary Function Test (PFT) Workshop**
- **Skin Prick Test (SPT) and Other Diagnostics Workshop**
- **Allergy Immunotherapy Workshop**

These are the skills our specialty is built on, and I would urge every delegate — particularly our younger colleagues — to make the fullest use of them. Knowledge that is practised in the hands travels back to the clinic far better than knowledge that is only heard.

My warmest congratulations go to the Pediatric Allergy and Applied Immunology Chapter, Karnataka, which is hosting this conference, and to the organising committee whose quiet, unglamorous work over many months is bearing fruit before us today. I also record our appreciation of the Divecha Centre for Climate Change, the IAP Karnataka State Branch, the IAP Karnataka State Environment Chapter and IAP Bengaluru, whose association is lending this meeting both breadth and scientific weight. Details of the programme are available at **www.pedallercon.com.**

As we turn towards October, our homes will be filling with light, sound and the smell of things frying. Whether you are keeping Durga Puja or Diwali, or simply enjoying the good company that both bring, may the season be a restful and joyful one for you and your families. May the festival of lights bring health and prosperity to every home, and may the learning we are gathering here help a few more children breathe easier in the year ahead.

With warm regards,

Dr U Narayan Reddy

Chairperson, IAP-PAAI

ALLERGY BULLETIN

IAP Allergy and Applied Immunology Chapter

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Advisor

Dr Uppin Narayan Reddy

Dr Saibal Moitra

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Dr Mitesh Kakkad

Dr Soundarya M

Dr Rama Rajyam

Dr Nayan Mani Deka

Secretary's Desk – IAPAAI Chapter

IAP Allergy Chapter — Autumn Edition



Dr. Sowmya Arudi Nagarajan
Honorary Secretary (2026–2027)
IAP Allergy and Applied Immunology Chapter

Dear Esteemed Colleagues and Members,

Warm greetings from the **IAP Allergy and Applied Immunology Chapter.**

As we gather for **PedAllercon 2026**, it is heartening to witness the remarkable growth of our specialty and the collective commitment of our members towards advancing allergy care and education across the country. This edition of the Allergy Bulletin reflects our shared vision of fostering academic excellence, collaboration, and advocacy in pediatric allergy.

A significant milestone this year has been the recognition by the **Central Indian Academy of Pediatrics (CIAP)** of **11th October as National Allergy Day.** This landmark initiative acknowledges the growing burden of allergic diseases in children and the urgent need for greater public awareness and professional education. Our Chapter is committed to observing National Allergy Day every year through a series of educational outreach activities, public awareness campaigns, webinars, school and community engagement programmes, and collaborative initiatives with IAP State Chapters. We invite every member to actively participate in making this a nationwide movement to improve allergy awareness and early diagnosis.

Our flagship **Friday Webinar Series** reached a significant milestone this year with the successful completion of **50 academic webinars**. Initiated under the visionary leadership of **Dr. Jose Ouseph** during his tenure as Chairperson of the IAP Allergy & Applied Immunology Chapter and coordinated by **Dr. Neeraj Gupta**, the series has become a cornerstone of continuous allergy education for pediatricians across India. The golden jubilee webinar was honoured by the presence of **Prof. Mario Morais-Almeida, President of the World Allergy Organization (WAO),** as the invited speaker—an occasion that celebrated not only fifty successful webinars but also the Chapter's unwavering commitment to academic excellence and global collaboration.

14th Pedallercon 2026 marks another defining moment for our Chapter as we have the privilege of welcoming the leadership of the European Academy of Allergy and Clinical Immunology (EAACI). The participation of **Prof. Mohammed Shamji, EAACI President, Dr. Maria Torres, Immediate Past President, EAACI and Prof. Jolanta Walusiak-Skorupa Vice-President Education & Specialty EAACI,** reflects the growing international partnerships that our Chapter has nurtured. Their presence enriches our scientific programme and strengthens opportunities for collaborative research, education, and guideline development between India and Europe.

We are equally delighted by the enthusiastic response to our **Allergy India Dialogues**, which continues to serve as a vibrant platform connecting IAP State Allergy Chapters through case-based discussions, expert interactions, and region-specific academic exchanges. This initiative has fostered meaningful collaborations and will continue to expand in the coming year, bringing high-quality allergy education closer to pediatricians across India.

As we celebrate another successful year, I extend my sincere gratitude to our President, Executive Committee members, faculty, authors, reviewers, and every Chapter member whose dedication has strengthened our collective mission. Together, we are building a future where every child has access to evidence-based allergy care and every pediatrician is empowered with the knowledge to deliver it.

I wish you an enriching academic experience at Pedallercon 2026 in Bengaluru, India and thank you for your continued support of the IAP Allergy and Applied Immunology Chapter.



From the Editor's Pen



Dear Colleagues,
As autumn settles over the country in a haze of festive lights and cool morning air, it brings me great joy to present this Autumn Edition of the Allergy Bulletin — a season that, fittingly, mirrors the energy and warmth of our own growing community of allergists and paediatricians.

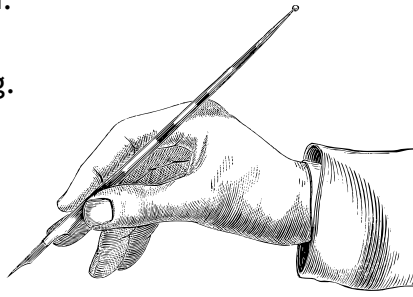
This has been a landmark year for our Chapter. The Central IAP's recognition of 11th October as National Allergy Day is a moment we have long worked towards, and one that I hope will translate into real momentum — more screening, more awareness among parents, and more confidence among general paediatricians in recognising and managing allergic disease early. Our Friday Webinar Series, too, reached a quiet but significant triumph this year, completing fifty sessions of continuous academic exchange, culminating in a golden jubilee lecture graced by Prof. Mario Morais-Almeida, President of the World Allergy Organization. That such global stalwarts continue to engage with our Chapter is a testament to how far Indian paediatric allergy has travelled — and how much further we intend to go.

This edition arrives at a particularly special juncture, released at the 14th PedAllercon in Bangalore, where we are honoured to welcome leadership from the European Academy of Allergy and Clinical Immunology alongside our own outstanding faculty. It is this spirit of collaboration — between institutions, between countries, and between generations of clinicians — that has always animated the Allergy Bulletin.

Within these pages, you will find a rich and deliberately wide-ranging selection: a comprehensive update on drug allergy, a practical primer on Component Resolved Diagnostics written with our younger colleagues in mind, insights on eosinophilic oesophagitis and the evolving microbiome story, and the latest GINA 2026 updates on MART therapy and immunotherapy. Each article has been chosen not merely to inform, but to be genuinely usable at the bedside on Monday morning.

None of this would be possible without the tireless contributions of our authors, the guidance of Dr Neeraj Gupta as our Founder Editor, and the quiet, steady support of our editorial board. To every reader who takes the time to write in, critique, or simply share how an article changed their practice — thank you. This bulletin exists because of you, and it is for you that we keep building it, edition after edition.

Wishing you all a festive season filled with warmth, and a year ahead rich in learning.



With warm regards,
Dr Sanjukta De
Editor, Allergy Bulletin IAP Allergy and Applied Immunology Chapter

Message from the Organising Chairperson

Dear Colleagues and Friends,

It gives me immense pleasure to welcome you to the **Winter 2026 Edition of the Allergy Bulletin**, released in conjunction with **PedAllercon 2026**, the Annual National Conference of the IAP Allergy and Applied Immunology Chapter.

This year's conference brings together distinguished national and international experts, clinicians, researchers, educators, and young pediatricians with a common goal—to advance the science and practice of pediatric allergy and improve the lives of children affected by allergic diseases.

The theme of PedAllercon 2026 reflects one of the most pressing challenges of our time: the growing impact of environmental change on childhood allergies. As the prevalence of asthma, allergic rhinitis, eczema, food allergy, and anaphylaxis continues to rise, it is imperative that we look beyond treatment and embrace prevention, innovation, research, and collaborative learning. The conference has been thoughtfully designed to provide practical, evidence-based updates while fostering meaningful dialogue on emerging concepts such as precision allergy, One Health, and planetary health.

The **Allergy Bulletin** has become an important academic voice of our Chapter, serving as a platform for sharing knowledge, inspiring discussion, and promoting excellence in allergy care. This special edition features contributions from renowned experts who have shaped the field through their clinical expertise, research, and leadership. I am confident that readers will find its contents both informative and relevant to their daily practice.

I extend my sincere gratitude to the Executive Committee of the IAP Allergy and Applied Immunology Chapter, our scientific committee, faculty, authors, reviewers, industry partners, and the dedicated organizing team whose collective efforts have made PedAllercon 2026 a reality.

I wish all our delegates an enriching academic experience, meaningful interactions, and a memorable stay in Bengaluru. May this conference strengthen our commitment to advancing pediatric allergy care and inspire us to build a healthier future for every child.

With warm regards,
Dr. A. R. Somashekhar
Professor of Pediatrics
Ramaiah Medical College, Bengaluru, India.
Organising Chairperson, PedAllercon 2026

Dr. H. Paramesh

Childhood Allergies in a Changing Environment: A Call to Protect the Health of Future Generations



Dr. H. Paramesh



Dr. Sowmya Nagarajan

Over the past three decades, we have witnessed an unmistakable rise in allergic diseases among children. Asthma, allergic rhinitis, eczema, food allergy, and anaphylaxis are no longer uncommon conditions confined to a few urban centres; they have become major public health concerns affecting children across India causing significant psycho socio economic burden. While advances in diagnostics and therapeutics have significantly improved patient care, one question continues to challenge us: **Why are childhood allergies increasing so rapidly?**

The answer extends far beyond genetics. Our genes have changed little over the past few decades, but our environment has changed dramatically. **Genetics deals the hand, Environment plays it.**

Children today are growing up in an era of unprecedented environmental change. Air pollution, climate change, rapid urbanization, biodiversity loss, altered dietary patterns, reduced exposure to natural ecosystems, increasing indoor pollution, and changing lifestyles have collectively reshaped the environment in which a child's immune system develops. These environmental factors interact with genetic susceptibility during early life, influencing immune maturation and increasing the risk of allergic diseases. **Air pollution has impact from womb to tomb.**

Air pollution remains one of the most important and preventable contributors. Fine particulate matter, vehicle emissions including sound pollution, industrial pollutants, biomass smoke, and tobacco smoke damage the developing airways, impair epithelial barriers, and amplify allergic inflammation. Climate change further compounds this challenge by extending pollen seasons, increasing aeroallergen concentrations, promoting fungal growth, and intensifying extreme weather events that adversely affect respiratory health.
Light pollution severely disrupts the biological cycles (Circadian rhythm) of humans, plants, insects, nocturnal animals and migratory birds. The airway diameter is directly controlled by circadian rhythm and exacerbate asthma and other allergic diseases.

Equally concerning is the gradual loss of children's interaction with nature. Modern lifestyles have reduced opportunities for outdoor play and exposure to diverse natural environments. Emerging evidence suggests that contact with green spaces and environmental biodiversity contributes to healthy immune development causing heat islands, while excessive urbanization and reduced microbial diversity may predispose children to allergic diseases.

The burden is particularly significant in low- and middle-income countries such as India, where rapid economic growth often coexists with environmental degradation. Protecting children from allergies therefore requires us to look beyond prescriptions and inhalers. It calls for a broader vision that integrates healthcare, environmental stewardship, public policy, and community engagement.

It is also important to know that planetary health and human health are interrelated. Our planet is 4600 million years old, our environment is a mixture of living and non living elements which supply our resources to live, assimilate the waste we generate and helps to sustenance of lives for good health for all especially our future generations.

The greed in human species spoiled our **Pancha Bhutas (elements) Air, Water, Sky, Fire and Earth** which has an impact on our children health directly or indirectly through agriculture, water resources, indentation of our coastal areas, destroying our cryosphere and loss of glaciers of our planet.

As per 6th IPCC report on impact of air pollution and climate change on our planet observed about a dozen items like:

1. Extinction of species is thousand times higher than before
2. Marine health waves are 34% more frequent and 17% longer since 1925
3. The fire season is extended and there is dissolved potential burnable areas
4. Wildfire in Arctic Tundra increased by 9 times between 1996-2015
5. Amazon rain forest basin transformed into grassland 4 times
6. Extinction of land and sea species is 54% in this century
7. The loss of green turtles is 59%
8. There will be 75% reduction of freshwater fishes by 2075
9. There is 15% loss of permafrost by 2100 and release of 67 billion tons of carbon further increasing the global warming
10. There will be death of Coral reefs by 70-90%
11. There will be reduction of simmer ice by 25%
12. Our ecosystem will be near or beyond their ability to adopt

diseases. We must advocate for cleaner air, healthier schools, smoke-free homes, sustainable urban planning, and policies that prioritize children's environmental health. Prevention begins before symptoms appear, and the environments we create today will determine the health of future generations.

The concept of **One Health** reminds us that human health is inseparable from the health of animals and the ecosystems we share. Similarly, the emerging framework of Planetary Health recognizes that safeguarding the Earth's natural systems is fundamental to protecting human wellbeing. These are no longer abstract concepts—they are central to the future of pediatric healthcare.

Every child deserves the opportunity to breathe clean air, grow in a healthy environment, and achieve their fullest potential. Addressing the environmental determinants of allergic diseases is not merely an academic challenge; it is a moral imperative.

As we continue to advance the science of allergy, let us also become champions of healthier environments. The future of pediatric allergy lies not only in better medicines and precision diagnostics, but also in creating a cleaner, greener, and healthier world for every child.

"The health of our children ultimately depends on the health of the environment we leave behind."

Nature has provided everyone's need not for his greed.

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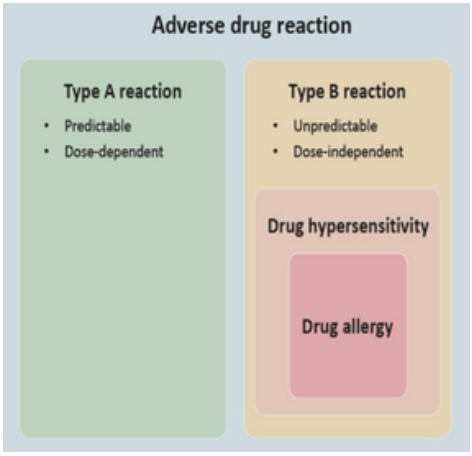
Article: Drug Allergy

MECHANISM AND TESTING

Dr Mona Kulpati

Most drug reactions are not allergic in nature
rather are perceived as drug allergy

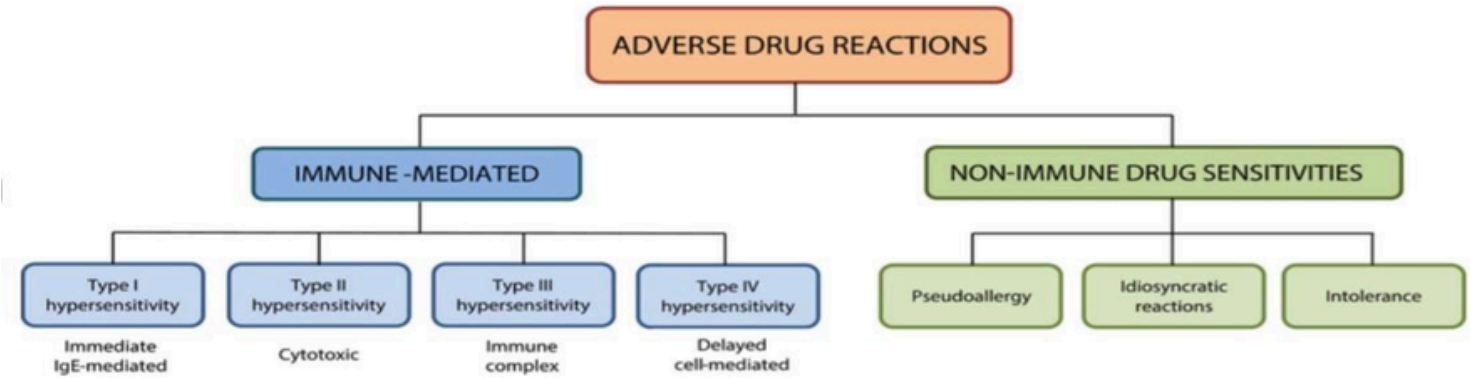


The burden of drug allergy is primarily contributed by antibiotics
which account for > 50% drug allergy labels,
beta lactams, sulphonamides being most common
Others being NSAIDS, antiepileptics, radiocontrast media

Drug allergy is a small subset of Adverse Drug Reaction (ADR)

Drug allergy is essentially a Type B adverse drug reaction which is unpredictable, dose independent, not related to pharmacologic effect, occurs in genetically predisposed patients

Type A adverse drug reaction is predictable, dose dependent, related to pharmacologic property of drug, with no unique susceptibility



Mechanisms of drug hypersensitivity:

Immune mediated mechanism:

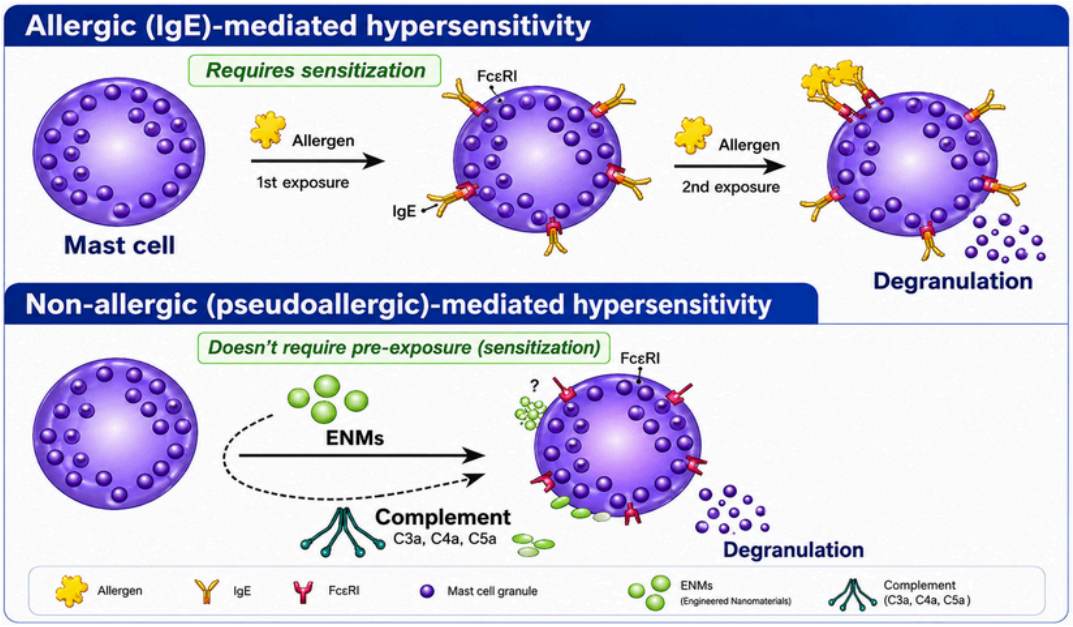
Only 6-10 % of all drug reactions are immunologically mediated
Drug allergy is an immunologically mediated response to a pharmaceutical and/or excipient agent in a sensitized person

IgE mediated allergy	Non-IgE mediated/ delayed
Onset of symptoms within minutes To a few hours (upto 6 hours)	72 hours to weeks
appears and recedes quickly	appears slowly & lasts longer
urticaria, pruritic rash, angioedema, respiratory symptoms, pain abdomen	SCAR, liver/renal dysfunction, deranged blood count
anaphylaxis	no anaphylaxis, systemic / skin features debilitating
type 1 DHR penicillin allergy	type 2 DHR -drug induced hemolytic anaemia type 3 DHR – serum sickness type 4 DHR – Severe Cutaneous Adverse Reactions SCAR*

*SCAR Life threatening drug hypersensitivity reaction which primarily attack skin and mucous membrane during active phase , often leave permanent physical and functional scars
eg Steven Johnson Syndrome(SJS), Toxic Epidermal Necrosis(TEN) , Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS)

Non immune mediated mechanism or Non allergic drug hypersensitivity :

- direct mast cell activation. e.g. vancomycin infusion reaction
- enzyme pathway alteration e.g. Aspirin exacerbated respiratory disease AERD
- direct mast cell activation e.g. reaction to radiocontrast media



- Drug intolerance (ADR type B)- undesirable pharmacologic effect at low dose e.g. aspirin induced asthma
- Drug idiosyncrasy (ADR type B)- unexpected, unpredictable, unrelated to pharmacologic effect e.g. Quinidine induced tinnitus

Not drug allergy - Adverse drug reaction ADR type A :

- Side effect – known effect, therapeutically undesirable, pharmacologically unavoidable, can occur in normal dose in anyone e.g. tachycardia with epinephrine injection
- Drug overdose pharmacologic effect related to concentration of drug, occurs quickly. e.g. respiratory distress with sedatives
- Drug toxicity – pharmacologic effect with even normal dose e.g. Drug induced liver injury (DILI) with ATT
- Secondary effect – related to primary pharmacologic action of drug e.g. Herxheimer reaction in treatment of syphilis with penicillin

Risk factors for drug hypersensitivity

- Previous / repetitive exposure
- Chemical property of drug -haptenation, molecular weight
- Age 20-50 years
- Gender – women
- Route – parenteral or topical
- Dose- high and intermittent dose
- Atopy
- Genetic susceptibility

Testing for drug hypersensitivity:

Strictly to be done under guidance of an experienced allergist/ immunologist

When to test :

- presence of drug hypersensitivity and absence of structurally unrelated, effective alternative drug
- Benefit outweighs the risk
- H/O severe drug hypersensitivity reaction for other drugs

When not to test :

- When no temporal association established between drug intake and onset of symptoms
- In case of severe life threatening/ debilitating reactions

Skin prick test (SPT)

To demonstrate drug specific IgE
It is a rapid , reliable , sensitive test with high negative predictive value , provided non irritant concentration of drug and good technique is used

Validated for penicillin, few antibiotics , NM blocking agents, latex, local anaesthetics

Positive SPT = presence of drug specific IgE
Positive predictive value (PPV) < 60% for penicillin

Negative SPT = does rule out presence of specific IgE
Negative predictive value (NPV) 97-99% for penicillin

Intradermal (ID) skin testing

Consider if strong history and negative SPT
SPT and delayed intradermal testing together will increase diagnostic sensitivity to > 60 %

Penicillin G(commercially available) is used , major antigenic determinant Penicilloyl moiety (Prepen) is not available in India

PPV – 40-50% for penicillin
NPV -87% for penicillin

Limitation of SPT and ID testing for drugs

- Lack standardized test concentrations availability
- Non immunogenic skin irritation due to high concentration , giving false positive results
- No predictive value for delayed type or non IgE mediated reactions

Drug provocation test (DPT) – gold standard

Drug challenge is the diagnostic criterion standard for exclusion of an immediate reaction and is recommended after allergy evaluation when the patient is deemed unlikely to be allergic to given drug

DPT is recommended only where indicated eg

- Low risk patient - weak / unverified history with positive SPT
- (Amoxicillin is the penicillin most commonly used for drug challenges because it has both immunologically significant core penicillin structure and potentially significant R group side chain)
- Strong history with negative SPT
  - (DPT co relates very well with negative SPT for specific medication eg penicillin)
- To exclude cross reactivity
- In case of perioperative reaction
- to find safe alternative to highly suspected/ proven allergen

Patch testing

For type 4 hypersensitivity reaction - T cell mediated mechanism /delayed drug reaction

A patch test for penicillin and other suspect penicillins/beta lactams is performed using a concentration of 5%-10 % penicillin in petroleum

Specificity 90-100%
Sensitivity less than 50%

In vitro tests

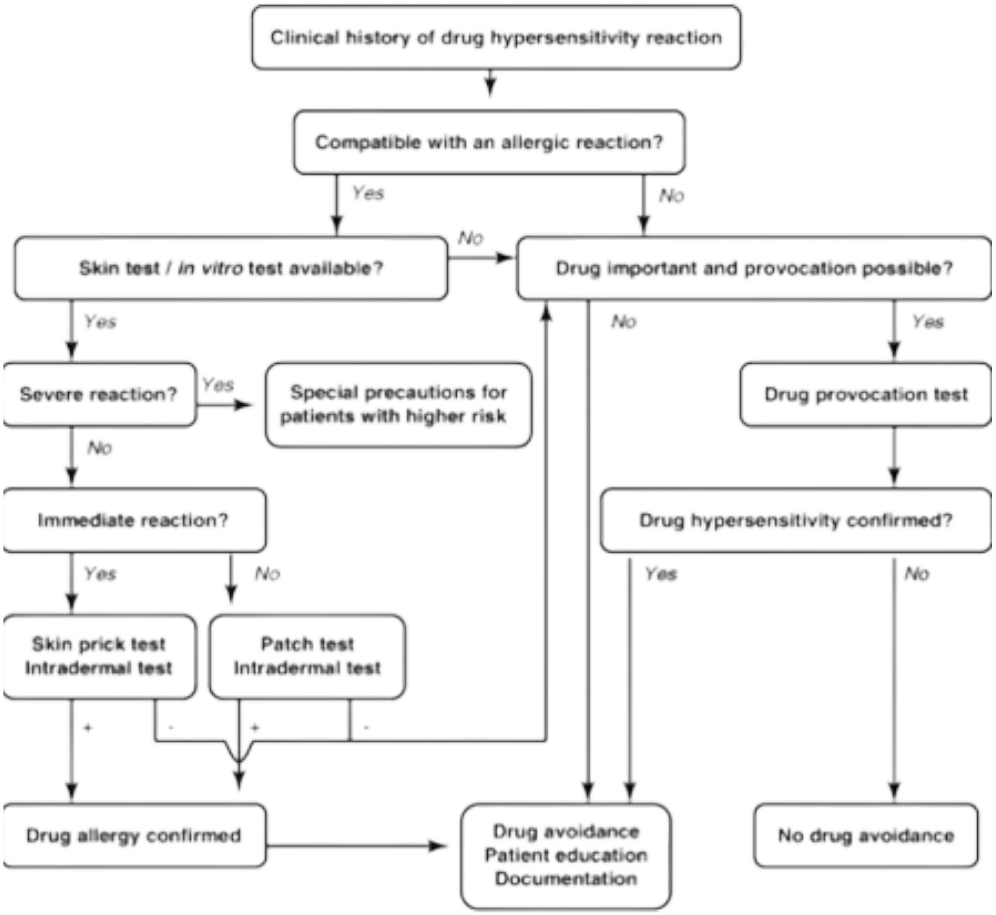
- Serum specific IgE assay (Immunocap)
    - low sensitivity (45-50% for penicillin) and specificity
- Basophil activation test – test not validated, research tool
    - Sensitivity about 50%, specificity 60%
- Relevant epitopes – not available
- Serum tryptase – not specific, however useful in IgE mediated anaphylaxis
    - Measure acute level of serum tryptase within 2 hours of acute event
    - Measure baseline level of serum tryptase after 24 hours of acute event
    - Acute level >120% of Baseline level +2 ng/ml
    - International diagnostic tool to objectively prove

Genetic testing

HLA based genetic screening available for select drugs

- HLA B*57:01 for Abacavir
- HLA B* 58:01 for allopurinol
- HLA B* 15:02, HLA B*13:01 for carbamazepine

Algorithm for testing of drug allergy:



Penicillin allergy Delabeling

Delabeling – removing the LABEL and reinstituting the drug

Penicillin is the most commonly reported drug allergy -about 10% of population .However, only about 1% are actually allergic
Patients with penicillin allergy label are at risk for suboptimal treatment with antibiotics, poorer clinical outcomes , drug resistant infections and adverse drug events

Low risk patients can safely undergo direct oral challenge with full dose (amoxicillin 50mg/kg) without prior skin testing

Low risk patient definition:

- 5-18 years – H/O benign cutaneous only reaction more than 1 year ago
- >18 years- H/O benign cutaneous only reaction more than 10 years ago
- Benign rash occurring more than one year ago, any other benign somatic symptoms or an unknown history

Desensitization

Induction of temporary tolerance by altering patients immune response

Patient continues to be allergic , immune system is conditioned to tolerate the drug because the medication is important for patient
eg , aspirin desensitization for Aspirin exacerbated respiratory diseases (AERD)

Indications:

- Strong H/O IgE mediated reaction
- No alternative drug for patients condition
- Medication needs to be continued for effective clinical management

Key messages:

- Drug allergy is adverse drug reaction mediated by adaptive immune mechanism
- Overdiagnosis of drug hypersensitivity (especially to antibiotics) is a major contributing factor to development of drug resistance
- Detailed chronology of drug intake and appearance of symptoms , presence of concomitant medication or infection , underlying hepatic or renal disease , route /dose of drug ,family history is crucial
- Drug allergy testing is for limited indications
- Drug provocation test is gold standard for diagnosis of drug allergy
- Delabel in low risk patients
- No scientific evidence for routine test dose before full dose administration of parenteral medication , intravenous or intradermal (even in atopic individual)

Article: Drug in Focus



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Atopic dermatitis (AD) is the commonest chronic inflammatory skin disease of childhood. In most children, emollients, topical corticosteroids and calcineurin inhibitors suffice; in a substantial minority, disease remains uncontrolled, with relentless itch, disturbed sleep, recurrent infection and a heavy burden on the whole family. For these children, conventional systemic immunosuppressants — cyclosporine, methotrexate, azathioprine — offer limited and monitoring-intensive relief, and practice guidelines have generally considered such agents inappropriate for the youngest patients. Dupilumab has changed this landscape.

Mechanism

Dupilumab is a fully human monoclonal antibody directed against the alpha subunit of the interleukin-4 receptor. Because this subunit is shared by the type I and type II receptor complexes, blockade abrogates signalling of both IL-4 and IL-13 — the central effector cytokines of type 2 inflammation. The result is targeted modulation of barrier dysfunction, itch and the inflammatory infiltrate, rather than broad immunosuppression.

Indication and dosing

It is approved for moderate-to-severe AD from six months of age, given subcutaneously with weight-tiered dosing. In the youngest group, 200 mg is used for 5 to <15 kg and 300 mg for 15 to <30 kg, four-weekly. In children 6–11 years the regimen is 100–200 mg two-weekly by weight or 300 mg four-weekly, and in adolescents 200 mg two-weekly below 60 kg and 300 mg two-weekly at or above 60 kg, with loading doses at initiation. Topical corticosteroids may be continued as required.

Evidence

The adolescent pivotal trial (LIBERTY AD ADOL) and the 6–11 year trial with concomitant topical corticosteroids both showed marked improvement in EASI, pruritus, sleep and quality of life. LIBERTY AD PRESCHOOL extended this to infants and preschoolers, and open-label extensions confirm durability of response over two to three years.

Pros and cons at a glance

Pros	Cons
Targeted IL-4/IL-13 blockade — no broad immunosuppression	High cost; limited reimbursement and access in India
Licensed from 6 months of age	Indefinite subcutaneous therapy; relapse common on stopping
Rapid relief of itch, sleep loss and quality-of-life burden	Conjunctivitis; head-and-neck erythema in a subset
No routine laboratory monitoring needed	Injection-site reactions
Concurrent benefit in asthma, CRSwNP, eosinophilic oesophagitis	Live vaccines to be avoided during therapy
Steroid-sparing; no increase in serious infection	Response not universal

Practice points

Reserve for genuinely refractory, quality-of-life-limiting disease after adherence and topical technique have been reviewed. Complete the live vaccine schedule before starting where feasible. Counsel families about conjunctivitis and the long-term nature of therapy. Cost remains the principal barrier in our setting, and a documented severity score at baseline strengthens the case for support.

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Article: Eosinophilic Esophagitis

The Evolving Allergy–Gastroenterology Interface



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A changing paradigm from "diet versus drugs" to personalized care

Eosinophilic esophagitis (EoE) has emerged as an increasingly recognized chronic, immune-mediated disease of the esophagus. Once considered an uncommon condition, EoE is now diagnosed with increasing frequency, partly because of greater awareness of its varied clinical presentations and pediatricians, gastroenterologists, allergists and primary-care physicians, together with improved access to endoscopy and biopsy, has resulted in a substantial increase in its recognition and diagnosis.

For allergists, EoE represents an important intersection between **type 2 inflammation, food antigen exposure and atopic disease.** However, an important distinction is that EoE should not be regarded simply as an IgE-mediated food allergy.

When should we suspect EoE?

The presentation varies considerably with age.
Infants and young children may present with:

- Feeding difficulty or food refusal
- Vomiting or regurgitation
- Prolonged mealtimes
- Selective eating or restricted food repertoire
- Difficulty progressing to textured foods
- Poor weight gain

Older children and adolescents may develop:

- Dysphagia, especially to solids
- Food impaction
- Chest or abdominal discomfort
- Excessive chewing
- Drinking liquids to help swallow food
- Avoidance of certain food textures

Adults most commonly present with solid-food dysphagia and recurrent food impaction.
An important clinical clue is **adaptive eating behavior.** Patients may unconsciously modify their eating habits—cutting food into small pieces, chewing excessively or drinking water with every bite—and therefore may not volunteer dysphagia unless specifically asked.

EoE and the Atopic Spectrum

EoE frequently coexists with other atopic conditions, including:
Atopic dermatitis | Asthma | Allergic rhinitis | IgE-mediated food allergy
This strong association supports the role of type 2 immune pathways in EoE. IL-4, IL-5 and IL-13 signaling, epithelial-derived cytokines and eosinophilic inflammation contribute to disease development.

However, the immunology of EoE is more complex than classical immediate IgE-mediated food allergy.

A key message for allergists
EoE is food-antigen driven in many patients, but it is not simply an IgE-mediated food allergy of the esophagus.

This distinction is clinically important because conventional allergy tests do not reliably identify the foods responsible for EoE.

Diagnosis: Symptoms + Histology + Clinical Context

Current diagnostic criteria require:

- Symptoms of esophageal dysfunction
- ≥15 eosinophils/high-power field on esophageal biopsy
- Appropriate clinical assessment to exclude alternative causes of esophageal eosinophilia

A prior mandatory PPI trial is no longer required to establish the diagnosis.
Endoscopy should be systematically assessed using the **EoE Endoscopic Reference Score (EREFS):**
Edema
Rings
Exudates
Furrows
Stricture

Importantly, the esophagus may appear normal endoscopically despite active disease. Therefore, **biopsy remains essential when clinical suspicion is high.**
The 2025 ACG guideline recommends obtaining multiple biopsies from different esophageal levels, with at least six targeted biopsies generally recommended. [1]

The Allergy Testing Dilemma

One of the most frequently encountered misconceptions is that skin-prick testing or serum-specific IgE testing can identify the foods responsible for EoE.
Although these tests may identify **IgE sensitization,** they do not reliably predict which foods are driving EoE inflammation.
Therefore:
❌ **Avoid**
“Positive skin test → eliminate that food to treat EoE.”
✅ **Prefer**

Empiric dietary elimination + objective assessment of response.
The current ACG guideline does **not recommend allergy-test-directed elimination diets** for EoE. [1,2]
This is an important area where allergists can provide valuable counseling and prevent unnecessary food restriction.

Diet Versus Drugs: The Debate Is Changing

Historically, EoE management was often framed as:
“Should we use an elimination diet or medication?”
The modern approach is more nuanced.
Dietary therapy, PPI therapy and swallowed topical corticosteroids are all evidence-based treatment options.
The choice should be based on:

- Age
- Disease severity and phenotype
- Nutritional status
- Atopic comorbidities
- Patient/family preference
- Treatment burden
- Adherence
- Availability of dietitian support

Thus, the current paradigm is:
Not “diet versus drugs”, but “the right treatment for the right patient.”

Dietary Therapy: Start Less, Add More

The traditional six-food elimination diet **(6-FED)** removes:
Milk + Wheat + Egg + Soy + Nuts + Fish/Shellfish
Although effective, extensive elimination can impose a significant nutritional, social and psychological burden.

The current approach increasingly favors **step-up dietary elimination.**
1-FED
Usually elimination of **milk** alone.
2-FED
Usually elimination of **milk** + **wheat.**
If remission is not achieved, further dietary restriction can be considered.
This approach aims to achieve disease control while minimizing unnecessary dietary restriction. [1]

Why this matters in children
Unnecessary elimination can contribute to:

- Nutritional deficiencies
- Inadequate calorie intake
- Impaired growth
- Feeding difficulties
- Reduced quality of life

Dietitian involvement is therefore particularly important in pediatric EoE.

Pharmacological Options

Proton-Pump Inhibitors

PPIs are now considered a therapeutic option in EoE rather than simply a diagnostic test to distinguish EoE from GERD.

A proportion of patients achieve clinical and histological remission with PPI therapy.

Their effects may involve mechanisms beyond acid suppression, including modulation of inflammatory pathways.

Swallowed Topical Corticosteroids

Swallowed topical corticosteroids are established anti-inflammatory therapy for EoE.
Commonly used agents include:

- **Budesonide**
- **Fluticasone**

The objective is prolonged contact of the medication with the esophageal mucosa.

Patients should be counseled regarding appropriate administration and avoiding food/drink immediately after dosing to maximize mucosal exposure.

Dupilumab: A Major Therapeutic Advance

The introduction of **dupilumab**, targeting IL-4 receptor alpha and thereby inhibiting IL-4/IL-13 signaling, represents a major advance in EoE therapy.

It is particularly relevant for patients with:

- **Persistent disease despite conventional therapy**
- **PPI non-response or intolerance**
- **Moderate-to-severe disease**
- **Significant concomitant atopic disease**

Dupilumab has demonstrated improvements in both **histological inflammation and clinical outcomes** and has expanded the therapeutic options available for patients with EoE. [3,4]

What About Esophageal Dilatation?

Long-standing EoE can result in esophageal remodeling and fibrostenosis.

Patients may develop:
Rings → Narrow-caliber esophagus → Strictures → Food impaction

Endoscopic dilatation can provide significant improvement in dysphagia.

However:
Dilatation treats the mechanical consequence of EoE; it does not treat the underlying inflammatory disease.

Therefore, anti-inflammatory treatment should generally accompany dilatation when active EoE is present. [1]

Maintenance Therapy: Control Today, Prevent Tomorrow

EoE is a chronic disease with a high likelihood of recurrence after withdrawal of effective treatment.
Once remission is achieved, maintenance therapy is recommended.

The long-term goals are to:

- Maintain histological remission
- Control symptoms
- Prevent food impaction
- Prevent fibrostenotic remodeling
- Reduce stricture formation
- Preserve quality of life

This makes EoE fundamentally different from a short-lived episode of esophageal inflammation.

A Practical EoE Treatment Algorithm

Suspect EoE
↓
Upper GI endoscopy + multiple esophageal biopsies
↓
Symptoms + ≥15 eos/hpf → establish diagnosis
↓
Select initial therapy through shared decision-making
Dietary therapy
1-FED / 2-FED → step-up if required
OR
Pharmacological therapy
PPI
± swallowed topical corticosteroid
↓
Reassess objectively
Symptoms + EREFS + histology
↓

If inadequate response
Optimize/adapt therapy
→ alternative dietary strategy
→ topical steroid/PPI
→ consider **dupilumab** in appropriate patients
↓
Fibrostenotic disease
Esophageal dilatation + anti-inflammatory therapy
↓
Remission
Continue maintenance therapy + long-term monitoring

KEY TAKEAWAYS

- **EoE is increasingly recognized**

Think of EoE in patients with unexplained dysphagia, food impaction or persistent feeding problems.

- **EoE is not simply IgE-mediated food allergy**

Atopy is common, but conventional allergy testing does not reliably identify EoE trigger foods.

- **Start with a pragmatic diet**

Less restrictive approaches such as 1-FED or 2-FED can reduce nutritional and lifestyle burden.

- **Medication is equally important**

PPIs and swallowed topical corticosteroids are established therapeutic options.

- **Dupilumab has changed the landscape**

Biologic therapy provides an important option for selected patients with persistent disease and/or significant type 2 comorbidities.

- **Symptoms are not enough**

Treatment response requires clinical + endoscopic + histological assessment.

- **Treat inflammation and fibrosis**

Dilatation can improve narrowing but does not replace anti-inflammatory treatment.

- **Think long term**

EoE requires maintenance therapy and ongoing monitoring.

Conclusion

Eosinophilic esophagitis has moved from an under-recognized disorder to an important chronic disease at the allergy–gastroenterology interface.

The therapeutic conversation is evolving from “diet versus drugs” toward individualized, shared decision-making. Dietary elimination remains an effective option, but less restrictive approaches can minimize treatment burden. PPIs and swallowed topical corticosteroids provide established pharmacological choices, while dupilumab has expanded the therapeutic landscape for selected patients.

For allergists, the central message is clear: EoE is closely linked to the atopic spectrum, but it is not simply an IgE-mediated food allergy.

The modern goal is not merely to make the patient feel better. It is to achieve and maintain clinical, endoscopic and histological disease control while minimizing nutritional and treatment burden.

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Article: Microbiome

Maternal Diet, Prenatal Exposures and theGut Microbiome's role in the Development of Childhood Allergy: A NarrativeReview



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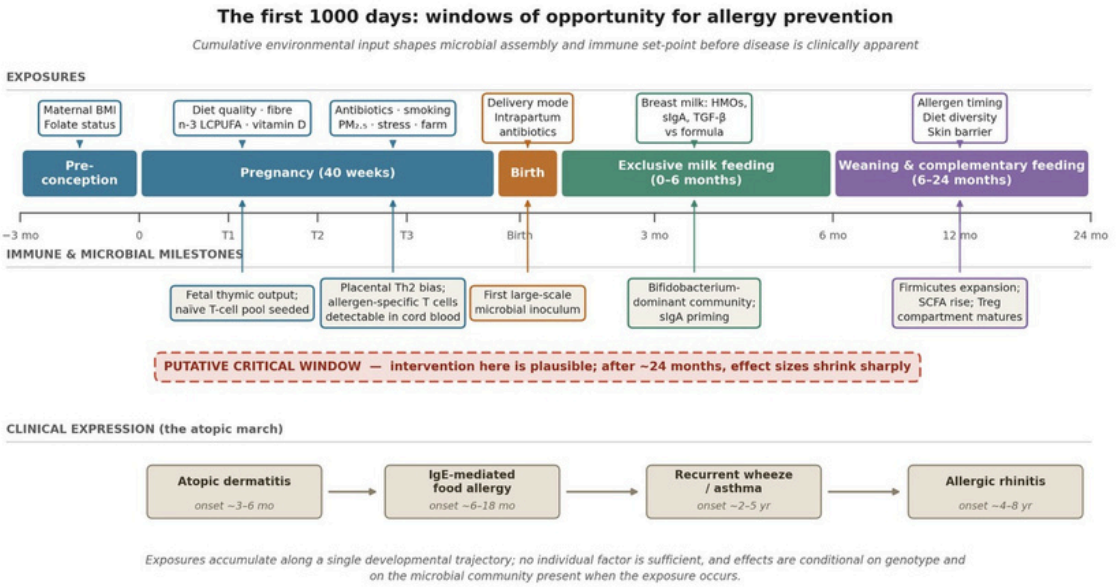
1. Introduction

Within three generations allergic disease has moved from clinical curiosity to the commonest chronic disorder of childhood in high-income countries, and it is now rising steeply in urbanising low- and middle-income settings.[1,2] The pace of that change excludes any explanation resting on alteration of the human genome. Attention has therefore turned to the environment, and in particular to the narrow developmental interval in which the immune system acquires its operating characteristics.

Strachan's observation that hay fever was less common in children with many older siblings[3] was the first quantitative statement of this idea. Its original framing — that infection protects against atopy — has not survived contact with the evidence, and has been superseded by the "old friends" and biodiversity formulations, which hold that it is chronic, low-grade exposure to a diverse commensal and environmental microbiota, rather than acute infection, that instructs immune regulation.[4,5] Reframed in this way, the hygiene hypothesis becomes a hypothesis about microbial ecology, and the gut microbiome becomes its principal mechanistic substrate.[6]

That substrate is assembled during a period that overlaps almost exactly with the window in which the developmental origins of health and disease (DOHaD) framework predicts the greatest environmental sensitivity.[7] Germ-free and gnotobiotic models show that the regulatory architecture of the mucosal immune system is established during a defined early window and that reconstitution after it has closed does not fully restore normal function.[8] In humans the equivalent window appears to open before conception and to close, for most allergic outcomes, somewhere around the second birthday (**Figure 1**).

Figure1:



This review examines three streams of evidence that converge on that window. The first concerns maternal diet: what a pregnant and lactating woman eats, and what that does to her own microbiota and metabolome. The second concerns non-dietary prenatal exposures, from farm environments to antibiotics and particulate air pollution. The third concerns the assembly of the infant's own gut microbiome, and the increasingly reproducible finding that its trajectory diverges in children who will later develop allergic disease, months before any clinical manifestation. Our aim is neither to advocate a preventive regimen nor to catalogue every reported association, but to establish which parts of this literature are strong enough to change practice, which are strong enough only to change research priorities, and where the two are being confused.

2. Scope and approach

MEDLINE, Embase and the Cochrane Central Register of Controlled Trials were searched from inception to the date of writing, combining terms for maternal nutrition, pregnancy, prenatal exposure, gut microbiota, short-chain fatty acids and dysbiosis with terms for atopic dermatitis, food allergy, asthma, wheeze, allergic rhinitis and sensitisation. Reference lists of retrieved systematic reviews and guidelines were hand-searched. This is a narrative rather than a systematic review; studies were selected on the basis of design quality, sample size, length of follow-up and mechanistic informativeness, with priority given to randomised controlled trials, prospective birth cohorts with objectively defined outcomes, and pooled analyses. Preclinical work is cited where it clarifies a causal pathway that human data can only describe. Two constraints on interpretation apply throughout and are not repeated at every turn: outcome definitions vary substantially between cohorts, and the great majority of the non-randomised literature is vulnerable to confounding by indication, reverse causation and shared genetic liability between mother and child.

3. The developmental window

3.1 Immune ontogeny before birth

The foetal immune compartment is not simply an immature version of the adult one; it is functionally distinct and biased. Successful pregnancy requires that the maternal–foetal interface suppress type 1 effector responses, and the resulting cytokine environment is relatively type 2 polarised.[9] Every neonate therefore begins life with a Th2-skewed default that must be actively counter-regulated after birth. In children who do not become atopic, allergen- specific responses in the first year shift towards a type 1 and regulatory profile; in those who do, the neonatal type 2 bias persists and consolidates.[10] Allergy, on this reading, is less an acquired aberration than a failure to exit a physiological starting state.

Two features of foetal immunity make that exit susceptible to environmental influence. First, the foetal thymus generates regulatory T cells with unusual efficiency, and foetal T cells exposed to non- inherited maternal alloantigen preferentially adopt a regulatory rather than an effector phenotype.[11] The machinery of tolerance is therefore already engaged, and already responsive to signals crossing the placenta, well before birth. Second, the maternal microbiota influences that machinery. In gnotobiotic dams colonised transiently during gestation only, the offspring (themselves germ-free at birth) show altered intestinal innate lymphoid cell and mononuclear cell programmes, mediated by maternally transferred microbial molecules and antibody.[12] Microbial exposure of the mother is thus transduced to the foetus without any microbial transfer at all, which reframes the maternal gut as an endocrine organ acting on foetal immunity.[13]

3.2 A cumulative rather than a single-hit model

No single prenatal exposure identified to date accounts for more than a small fraction of population-attributable risk, and this has repeatedly been mistaken for evidence that the exposures do not matter. A more defensible reading is that the system is cumulative and probabilistic. Each exposure shifts a distribution slightly; effects are conditional on genotype, on the microbial community present when the exposure occurs, and on other concurrent exposures; and clinical disease emerges when enough shifts accumulate in the same direction. This has direct methodological consequences. Trials of single nutrients in unselected populations are, on this model, close to the least informative design available, and their frequently null results should be interpreted accordingly rather than taken as refutation of the underlying biology.

4. Maternal diet during pregnancy and lactation

4.1 Dietary patterns and dietary diversity

Table 1 summarises the maternal dietary exposures considered below. Single-nutrient analyses have generally proved less informative than analyses of whole dietary patterns, which better capture the correlated intakes and the microbial substrate that a real diet delivers. In a Spanish birth cohort, high maternal adherence to a Mediterranean pattern during pregnancy was associated with lower persistent wheeze and lower atopy in the offspring at 6.5 years, with the association surviving adjustment for maternal atopy, smoking and social class.[14] The European Academy of Allergy and Clinical Immunology (EAACI) systematic review of dietary factors in pregnancy reached a similar conclusion: pattern-level measures showed more consistent inverse associations with childhood atopic outcomes than any individual nutrient.[15] A maternal diet index developed in the Healthy Start cohort, weighting foods by their observed relation to offspring outcomes, was associated with atopic dermatitis, food allergy and wheeze in the first years of life.[16]

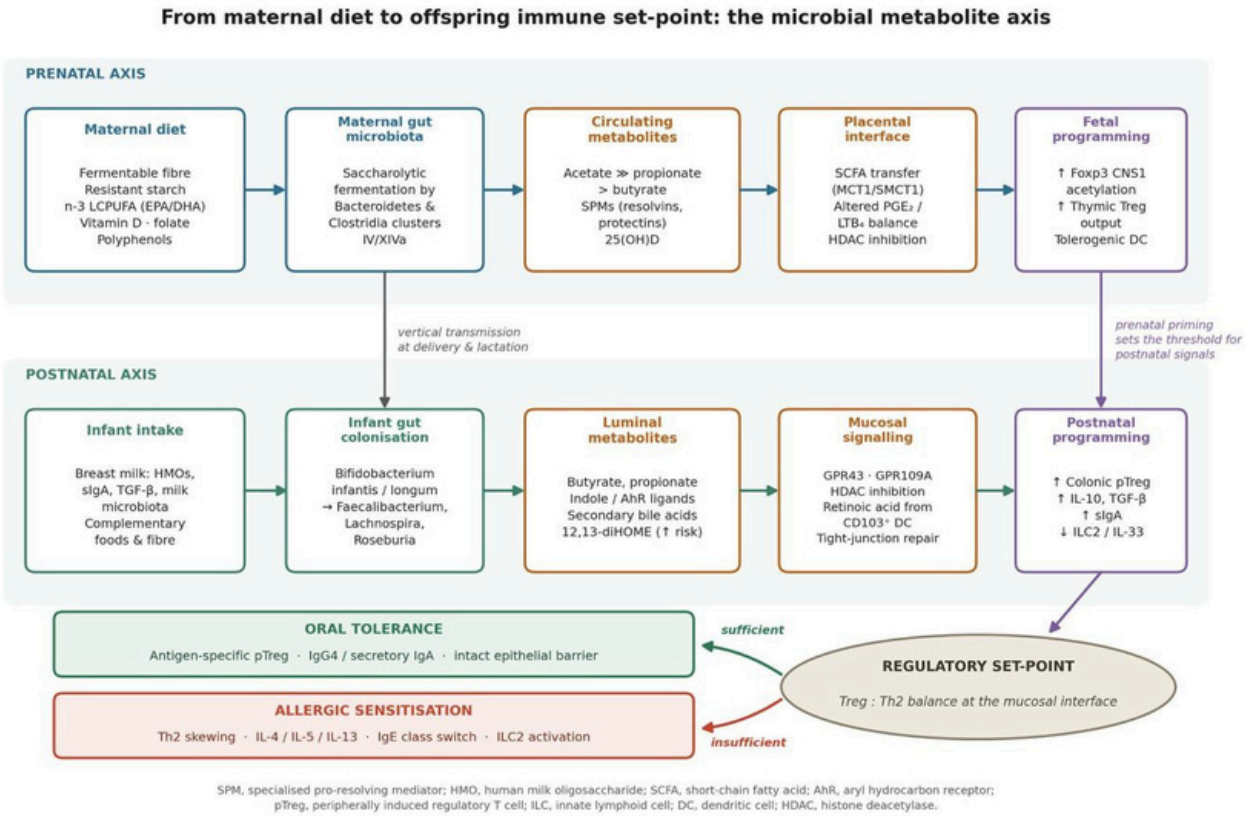
These findings are not unanimous. In Project Viva, no maternal dietary pattern during pregnancy was associated with recurrent wheeze in mid-childhood.[17] The discrepancy is instructive rather than fatal: cohorts differ in the range of dietary variation they contain, and a protective pattern cannot be detected in a population where almost nobody eats it. Diet diversity has emerged as a partially separable construct, and an EAACI position paper has argued that the number and variety of foods consumed (rather than the presence of any particular one)may be the operative exposure, plausibly because diversity of substrate drives diversity of the fermenting community.[18]

4.2 Fermentable fibre and short-chain fatty acids

Of all dietary components, fermentable fibre has the clearest mechanistic path from maternal plate to offspring immune phenotype. Fibre reaching the colon undisturbed is fermented by saccharolytic commensals to acetate, propionate and butyrate. These short-chain fatty acids (SCFAs) act both as ligands for G-protein-coupled receptors, principally GPR43 and GPR109A, and as inhibitors of histone deacetylase, and through the latter promote acetylation at the *Foxp3* locus and expansion of regulatory T cells.[19-21]

In mice, a high-fibre diet or dietary acetate alone alters the gut microbiota, increases circulating SCFA and attenuates allergic airway inflammation; a low-fibre diet has the opposite effect.[22] Critically, the effect is transmissible across generations: acetate given to pregnant dams protects the offspring, an effect associated with increased acetylation at the *Foxp3* promoter in foetal tissue and with suppression of an asthma-related gene expression signature.[23] Maternal high-fibre feeding during pregnancy and lactation likewise expands regulatory T cells in the offspring.[24] This is the strongest available demonstration that a maternal dietary input can, through a microbial intermediary, alter the immunological set-point of a child (**Figure 2**).

Figure2:



Human data are correspondingly sparse but concordant in direction. In the PASTURE cohort, higher faecal butyrate and propionate concentrations at one year were associated with lower subsequent atopic sensitisation, food allergy and asthma, with the strongest associations for the highest tertile of butyrate.[25] No randomised trial has yet tested maternal fibre or prebiotic supplementation in pregnancy against allergic outcomes in the child, which is arguably the single most conspicuous gap in this literature.

4.3 Long-chain n-3 polyunsaturated fatty acids

The rationale for n-3 long-chain polyunsaturated fatty acid (LCPUFA) supplementation is that eicosapentaenoic and docosahexaenoic acids compete with arachidonic acid, reducing production of pro-inflammatory eicosanoids and generating specialised pro-resolving mediators. Early work showed that fish oil in pregnancy modifies neonatal allergen-specific cytokine responses.[26]

The most convincing clinical evidence comes from COPSAC2010, in which 2.4 g/day of n-3 LCPUFA from the twenty-fourth week of pregnancy reduced persistent wheeze or asthma in the offspring by age five (hazard ratio 0.69, 95% confidence interval 0.49–0.97), with an absolute risk reduction of approximately seven percentage points.[27] The effect was concentrated in women whose baseline blood levels of EPA and DHA were in the lowest third and in carriers of a minor allele at the FADS locus in those least able to synthesise these fatty acids endogenously. This is a substantive finding about who benefits, not a subgroup artefact, and it predicts that trials in populations with adequate baseline intake will be null.

That prediction is broadly borne out. An Australian trial found no reduction in overall allergic disease in the first year, although IgE-associated eczema was less frequent in the supplemented group.[28] Meta-analyses report modest reductions in sensitisation to egg and in atopic eczema but inconsistent effects on asthma and rhinitis.[29,30] Long-term follow-up of a Danish trial found lower rates of allergic asthma in adult offspring, suggesting effects that only become visible with follow-up far longer than most trials sustain.[31]

4.4 Vitamin D

Observational data linking higher maternal vitamin D intake or status to lower childhood wheeze are extensive,[32] and vitamin D has plausible actions on dendritic cell maturation, regulatory T-cell induction and antimicrobial peptide expression. Two adequately powered randomised trials reported concurrently. VDAART, using 4400 IU/day from 10–18 weeks, found a reduction in asthma or recurrent wheeze at three years that did not reach conventional significance (odds ratio 0.80, 95% CI 0.58–1.11).[33] COPSAC2010, using 2400 IU/day from week 24, similarly reported a non-significant reduction in persistent wheeze (hazard ratio 0.76, 95% CI 0.52–1.12).[34] A pre-specified combined analysis of both trials yielded an odds ratio of 0.74 (95% CI 0.57–0.96), with the effect greatest in women with the highest baseline 25- hydroxyvitamin D concentrations, implying that a sufficient total dose rather than correction of deficiency alone may be required.[35]

A systematic review commissioned for guideline development concluded that the evidence remains insufficient to recommend vitamin D supplementation specifically for allergy prevention, while noting that supplementation to correct deficiency is warranted on other grounds.[36] The distinction matters clinically: in populations with widespread hypovitaminosis D, including much of urban South Asia, the case for supplementation rests on skeletal and obstetric outcomes, and any allergy benefit should be regarded as a possible secondary gain rather than the indication.

4.5 Antioxidant micronutrients

Lower maternal vitamin E intake has been associated with childhood wheeze and asthma in cohort studies,[37] and a systematic review found reasonably consistent inverse associations for vitamin E, zinc and selenium.[38] None has been tested in an adequately powered randomised trial with clinical allergic endpoints, and the pattern of findings is equally compatible with vitamin E acting as a marker of overall dietary quality. This body of work is best regarded as hypothesis-generating.

4.6 One-carbon metabolism and methyl donors

Folate is the one nutrient in this field for which a plausible mechanism predicts harm rather than benefit. Methyl-donor availability influences DNA methylation, and in a murine model, in utero supplementation with methyl donors enhanced allergic airway disease in the offspring through hypermethylation of genes including

Runx3, with the phenotype transmissible to a subsequent generation.[39] Cohort data are less alarming but not reassuring: folic acid supplementation in late pregnancy has been associated with a small increase in lower respiratory tract infection and wheeze in early childhood, with no association for supplementation in the periconceptional period.[40]

The practical implication is narrow and should not be overstated. Periconceptional folic acid at recommended doses prevents neural tube defects, and nothing in this literature justifies withholding it. What the data do question is high-dose supplementation continued through the third trimester without an indication, which is common practice in some settings.

4.7 Maternal allergen intake and the failure of avoidance

Maternal avoidance of common allergenic foods during pregnancy and lactation was widely advised in the 1990s and 2000s. A Cochrane review found no evidence that it prevents atopic disease in the child, and some evidence that it impairs maternal and foetal nutrition.[41] The reverse is if anything more likely: in Project Viva, higher maternal peanut, milk and wheat intake during pregnancy was associated with lower odds of the corresponding allergy and of asthma in mid-childhood.[42] Current EAACI guidance explicitly recommends against maternal exclusion diets for allergy prevention.[43] Despite this, avoidance advice remains in circulation, and correcting it is one of the few unambiguous actions available to a clinician counselling a pregnant woman.

4.8 Maternal obesity and the metabolic milieu

Maternal pre-pregnancy obesity and excessive gestational weight gain are associated with childhood asthma and wheeze in meta-analysis, with a modest effect that is at least partly independent of childhood body mass index.[44] Candidate pathways include systemic low-grade inflammation, altered adipokine signalling, a shifted maternal microbiota and lower dietary quality. Disentangling these from the confounding structure (obesity, diet quality and socioeconomic position are tightly correlated) is not currently possible with observational data.

5. Non-dietary prenatal exposures

5.1 Microbial richness and the farm effect

The most reproducible environmental association in allergy epidemiology is the protection conferred by growing up on a traditional farm. Endotoxin concentration in a child's mattress dust is inversely related to hay fever, atopic sensitisation and asthma,[45] and the diversity of bacterial and fungal taxa in domestic dust is inversely related to asthma independently of endotoxin.[46] The comparison of Amish and Hutterite children (genetically similar populations with markedly different farming practice) is close to a natural experiment: Amish children, exposed to single-family dairy farms, had a fraction of the asthma prevalence of Hutterite children on industrialised communal farms, with corresponding differences in innate immune gene expression, and Amish house dust conferred protection when instilled into mice through a MyD88- and Trif- dependent pathway.[47]

The protective effect is at least partly mediated through the gut. In the PASTURE cohort, farm exposure accelerated maturation of the gut microbiome during the first year, and this accelerated maturation statistically mediated a substantial share of the protection against asthma, with faecal butyrate on the causal path.[48] Farm exposure, dietary fibre and SCFA production are therefore not three separate protective factors but three entry points to one pathway.

5.2 Tobacco smoke and ambient air pollution

Prenatal maternal smoking increases wheeze and asthma in the offspring, with meta-analytic estimates around a 40 per cent relative increase for wheeze in early childhood, and effects on lung function that are detectable at birth.[49] This is among the few associations in the field with high certainty and a clear dose– response relationship.

Traffic-related air pollution shows a similar pattern with somewhat lower certainty. Meta- analysis of birth cohorts links early-life exposure to particulate matter and nitrogen dioxide with asthma and sensitisation.[50] Proposed mechanisms include oxidative stress at the airway epithelium, adjuvant effects of diesel exhaust particles on IgE responses, and epigenetic modification of immune genes.[51] Given ambient PM2.5 concentrations in many South Asian cities, this exposure is likely to carry a far larger population-attributable fraction there than in the European cohorts from which most estimates derive; a point returned to below.

5.3 Antenatal antibiotics and acid suppression

Registry studies consistently link maternal antibiotic use in pregnancy to childhood asthma, typically with hazard ratios between 1.2 and 1.4 and a dose–response relationship by number of courses.[52] Whether this is causal is genuinely uncertain. A study designed specifically to address confounding found that the association was substantially attenuated by comparison with siblings and with maternal antibiotic use before pregnancy, indicating that a large part of it reflects shared familial propensity to infection and to prescription rather than a microbiome- mediated effect.[53] This is a useful corrective to the tendency to read every antibiotic association as microbial causation.

Acid-suppressive medication is a more recent concern. In a large cohort of infants, prescription of histamine-2 receptor antagonists or proton pump inhibitors in the first six months was associated with subsequent food allergy, medication allergy, anaphylaxis, allergic rhinitis and asthma, with a dose–response relationship.[54] Elevated gastric pH permits intact protein to reach the small intestine and alters the upper gastrointestinal microbiota, both plausible mechanisms; confounding by indication, particularly by gastro- oesophageal reflux as an early marker of atopic disease, is a serious alternative explanation.

5.4 Psychosocial stress

Prenatal maternal stress has been associated with childhood wheeze and atopic dermatitis, with candidate pathways through cortisol-mediated effects on foetal immune development and through stress-induced changes in the maternal microbiota.[55] Exposure measurement in this area is heterogeneous and the literature is small; the association is plausible but not established(**Figure 3**).

Figure3:



6. Assembly of the infant gut microbiome

6.1 Does colonisation begin before birth?

A report that the placenta harbours a low-biomass microbiome[56] generated a substantial literature on prenatal colonisation. It has not withstood scrutiny. A large study using extensive contamination controls found no evidence of a placental microbiome, detecting only reagent contaminants and, in a minority of samples, recognised pathogens such as Streptococcus agalactiae.[57] A subsequent multi-author analysis concluded that the reported foetal and placental microbiota are explicable by contamination introduced during sampling, extraction and sequencing, and set out the controls that low-biomass studies require.[58]

The current position is therefore that the healthy foetus is essentially sterile, and that maternal microbial influence in utero is mediated by metabolites, microbial molecular patterns and antibody rather than by live organisms.[12] This does not weaken the case for prenatal programming; it specifies its mechanism more precisely, and it is the reason maternal diet can matter before birth at all.

6.2 Delivery and the first inoculum

Birth delivers the first large microbial inoculum. Vaginally born infants acquire communities resembling the maternal vaginal and faecal microbiota, dominated by Lactobacillus, Prevotella and, shortly afterwards, Bacteroides and Bifidobacterium; infants born by caesarean section acquire communities resembling maternal skin, dominated by Staphylococcus, Corynebacterium and Propionibacterium, with delayed Bacteroides acquisition.[59] Meta- analysis links caesarean delivery to modest increases in food allergy, allergic rhinitis and asthma, with pooled estimates around 1.2.[60]

Two qualifications matter. First, almost all caesarean deliveries involve intrapartum antibiotic prophylaxis, and the independent contribution of the antibiotic has not been separated from that of the delivery route. Second, the compositional deviation is partly reversible: infants born by caesarean section who received Lactobacillus rhamnosus GG in early life showed partial normalisation of composition and function, though whether this translates into altered allergic outcomes is unknown.[61]

6.3 Human milk as a microbial substrate

Human milk shapes the infant microbiome more powerfully than any other postnatal exposure. Beyond immunoglobulin A, lactoferrin and transforming growth factor beta, it contains human milk oligosaccharides (HMOs);a family of more than 150 structurally distinct glycans that are indigestible by the infant and function as selective substrate for *Bifidobacterium longum* subsp. *infantis* and related taxa.[62] HMO composition varies between women, principally with secretor and Lewis blood group status, and in the CHILD cohort the overall HMO profile, rather than any single oligosaccharide, was associated with food sensitisation at one year.[63] Breastfeeding is thus not a single exposure but a variable one, which may explain why studies of breastfeeding duration and allergic outcomes have been so inconsistent.

6.4 Weaning and dietary diversity

Introduction of solid food triggers the largest reconfiguration of the infant microbiome, from a milk-adapted *Bifidobacterium*-dominated community to an adult-like community dominated by Clostridia capable of fermenting complex plant polysaccharides, with a corresponding rise in butyrate.[64]

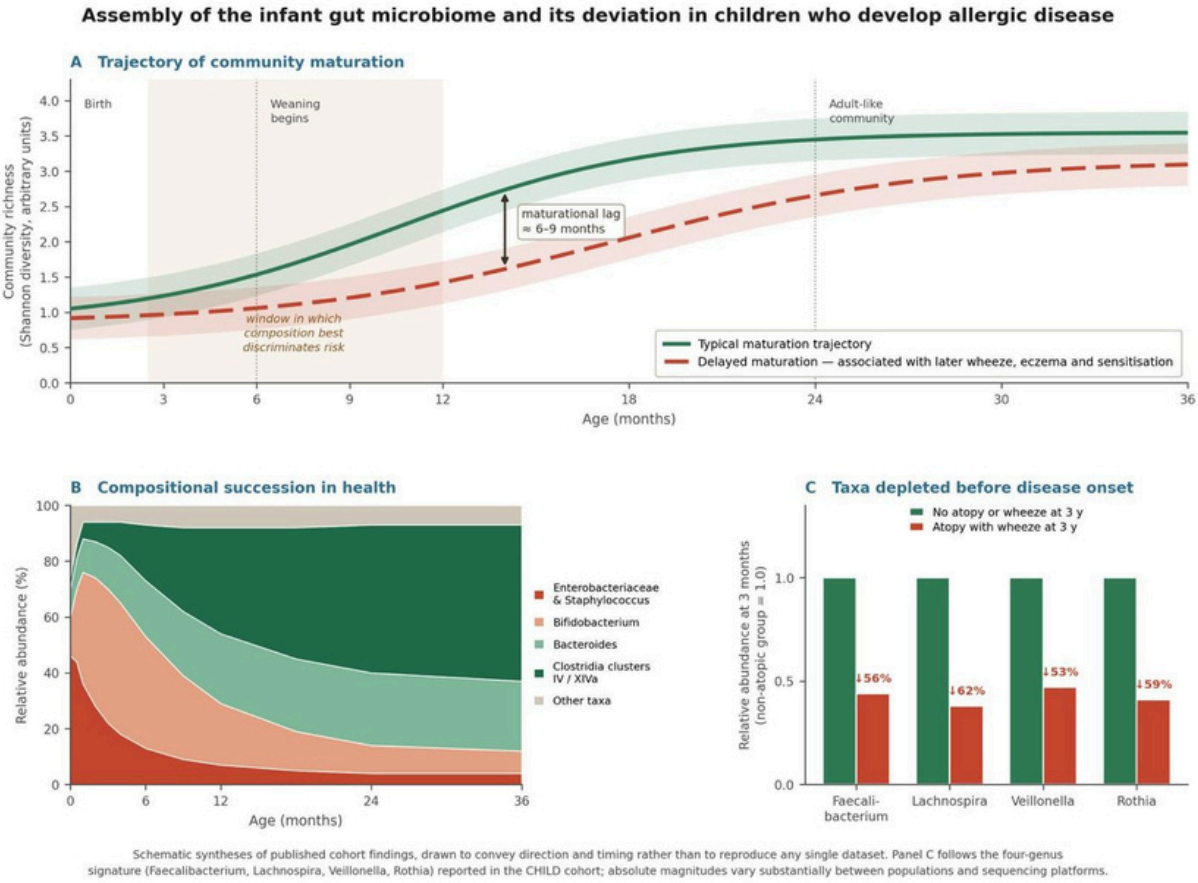
Cessation of breastfeeding, rather than introduction of solids as such, appears to be the principal driver of this transition. Geographical comparisons show that the adult configuration reached at the end of this process differs markedly between populations, with higher diversity and greater representation of *Prevotella* in non-industrialised settings.[65]

The variety of foods introduced in the first year is inversely associated with atopic dermatitis, food allergy and asthma, with each additional food group in the first year associated with a reduction in subsequent allergic outcomes in the PASTURE cohort.[66] Reverse causation is an obvious concern (infants with early eczema have narrower diets because of parental caution) but the association persisted after excluding children with early symptoms.

6.5 Microbial signatures that precede disease

The most important development in this field has been the demonstration that microbial deviation precedes rather than follows clinical allergy. In the CHILD cohort, infants who went on to develop atopy with wheeze at three years showed, at three months of age, relative depletion of four genera (Faecalibacterium, Lachnospira, Veillonella and Rothia)together with reduced faecal acetate and dysregulated enterohepatic metabolites; the difference was much attenuated by one year (Figure 4).

Figure4:



Inoculation of germ-free mice with these four taxa reduced airway inflammation in their offspring.[67] In a US cohort, a neonatal faecal community state characterised by depletion of *Bifidobacterium*, *Akkermansia* and *Faecalibacterium* and enrichment of certain fungi was associated with multi-sensitised atopy at two years and with a lower proportion of regulatory T cells in ex vivo assays.[68]

COPSAC2010 refined this from taxa to trajectory: it was not composition at any single time point but the rate of maturation between one and twelve months that predicted asthma at six years, and only in children of asthmatic mothers.[69] This gene–environment interaction structure is likely to be general[70], and it explains why unstratified analyses in this field so often return null results. Mechanistic work has begun to identify specific effectors: elevated neonatal faecal concentrations of the bacterially produced lipid 12,13- diHOME were associated with asthma risk and, in mice, reduced pulmonary regulatory T cells.[71]

In food allergy specifically, infants with cow's milk allergy who achieved tolerance by eight years had distinctive gut communities at three to six months, enriched in Clostridia and Firmicutes.[72] Transfer of faecal microbiota from healthy, but not from cow's-milk-allergic, nfants protected germ-free mice from anaphylactic responses to ß-lactoglobulin, and the effect was attributable to a specific Clostridial species acting through the ileal epithelium.[73]

A defined consortium of Clostridiales or Bacteroidales suppressed food allergy in mice through a MyD88- and RORγt-dependent regulatory T-cell pathway.[74] These experiments come closer to establishing causation than anything available from human observation.

6.6 Beyond bacteria

Attention to the bacterial community has outpaced work on other kingdoms. The infant gut mycobiome is associated with environmental determinants including antibiotic exposure and pet ownership, and specific fungal profiles have been linked to later atopic wheeze.[75] The virome remains almost unexamined in this context. Given that fungal and bacterial communities interact, and that Candida overgrowth follows antibiotic exposure, this is a plausible site of unmeasured effect.

7. Mechanisms linking diet, microbiota and tolerance

7.1 Short-chain fatty acids and regulatory T cells

The SCFA pathway is the best characterised route from dietary substrate to immune phenotype. Butyrate and propionate induce differentiation of colonic peripherally induced regulatory T cells; butyrate acts predominantly through histone deacetylase inhibition, enhancing acetylation of histone enhancing acetylation of histone H3 at the Foxp3 promoter and conserved non-coding sequence regions, while propionate acts substantially through GPR43.[19-21] SCFAs additionally act on epithelial cells to reinforce tight junctions, on dendritic cells to promote a tolerogenic phenotype with retinoic acid production, and on bone marrow precursors to alter the functional programming of dendritic cells that subsequently populate the lung.[22] The last of these explains how a gut-derived metabolite can influence airway immunity.

7.2 Other microbial metabolites

Microbial tryptophan metabolites act as aryl hydrocarbon receptor ligands, supporting intraepithelial lymphocyte and group 3 innate lymphoid cell function and thereby epithelial integrity. Secondary bile acids generated by bacterial 7α-dehydroxylation act on farnesoid X and TGR5 receptors with immunomodulatory consequences. Not all microbial products are protective: 12,13-diHOME, a bacterially produced oxylipin elevated in the neonatal stool of infants at high asthma risk, reduces regulatory T-cell numbers in the lung.[71] The framing of dysbiosis as loss of beneficial taxa is therefore incomplete; gain of specific metabolic capacity matters too.

7.3 Barrier function and the route of first exposure

The dual allergen exposure hypothesis holds that the immunological outcome of encountering a food protein depends on the route: cutaneous exposure through inflamed, barrier-defective skin favours sensitisation, whereas oral exposure favours tolerance.[76] This single idea reconciles the epidemiology of peanut allergy with the failure of avoidance strategies, and it predicted the results of the early introduction trials. The broader epithelial barrier hypothesis extends the argument beyond food, proposing that detergents, surfactants, microplastics, ozone and particulate matter damage epithelial barriers at skin, gut and airway, permitting allergen and microbial translocation and driving type 2 inflammation.[77] On this account the microbiome and the barrier are not competing explanations: barrier damage alters the microbial community, and microbial metabolites are required to repair the barrier.

7.4 Persistence and epigenetic memory

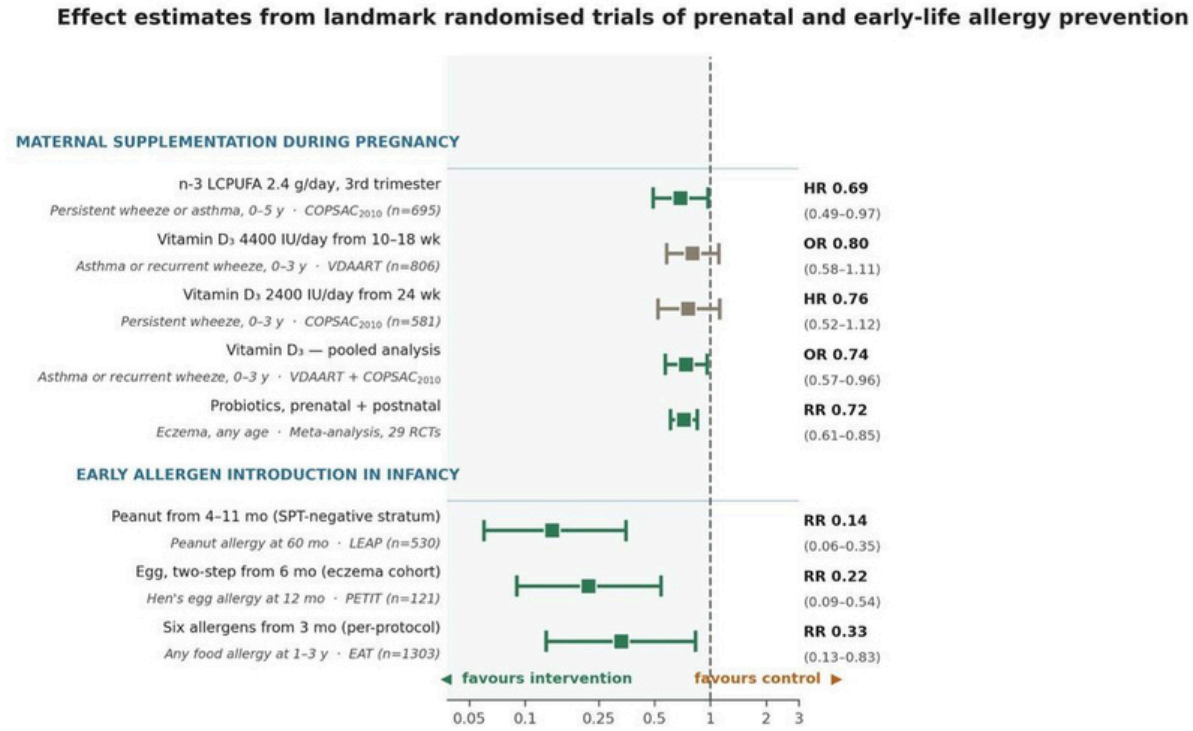
Why an exposure during a window of a few months should have effects measurable years later requires explanation. Three mechanisms are plausible and not mutually exclusive: stable modification of the microbial community itself, which is self-perpetuating once established; epigenetic marking of immune cell lineages, as demonstrated for Foxp3 acetylation after maternal acetate exposure[23] and for Runx3 methylation after methyl donor supplementation[39]; and durable functional reprogramming of innate cells, including the invariant natural killer T cell accumulation seen in germ-free mice that cannot be reversed by colonisation after the neonatal period.[78] Early-life microbial diversity also determines long-term serum IgE set-point in mice through a mechanism operating specifically in the neonatal window.[79]

8. What intervention trials show

8.1 Maternal supplementation

Taken together, randomised trials of maternal supplementation during pregnancy have produced smaller and less consistent effects than the observational literature implied (Figure 5).

Figure5:



Green intervals exclude the null. Estimates are as reported in the primary publications, except LEAP and EAT, which are calculated from published event counts.
HR, hazard ratio; OR, odds ratio; RR, risk ratio; SPT, skin prick test. Trials differ in population risk, comparator and outcome definition, so estimates are not formally poolable.

The n-3 LCPUFA and vitamin D trials each yielded point estimates favouring intervention with confidence intervals that either just include or just exclude the null, and in both cases the effect was concentrated in a subgroup defined by baseline status. The most reasonable interpretation is that these are real but conditional effects, detectable only where the deficit being corrected is present. Trials that enrol unselected populations will continue to dilute them.

8.2 Probiotics, prebiotics and synbiotics

The first trial of perinatal probiotics reported halved atopic eczema at two years with Lactobacillus rhamnosus GG given to mothers before delivery and to infants for six months.[80] Subsequent trials have been heterogeneous in strain, dose, timing and outcome, and the aggregate picture is narrower than the initial result suggested. Meta-analysis indicates a reduction in eczema when probiotics are given both prenatally and postnatally, with no convincing effect on asthma, wheeze, food allergy or sensitisation.[81] The World Allergy Organization–McMaster guideline panel issued a conditional recommendation, based on very low certainty evidence, for probiotic use in pregnant women, breastfeeding mothers and infants at high risk of allergy, on the basis of the eczema benefit alone.[82] A parallel panel made a conditional recommendation for prebiotic supplementation in non-exclusively breastfed infants.[83]

Two trials deserve individual mention. Lactobacillus rhamnosus HN001 given from 35 weeks of gestation to six months postpartum reduced eczema through to eleven years of age, one of very few demonstrations of persistence beyond early childhood.[84] Conversely, several trials of L. rhamnosus GG in different populations have been null, and meta-analysis restricted to that strain suggests the benefit is not robust across settings.[85] Strain specificity, and the possibility that the effect depends on the recipient's existing community, are the most likely explanations. In children with established cow's milk allergy, formula supplemented with L. rhamnosus GG expanded butyrate-producing strains and was associated with tolerance acquisition, suggesting the mechanism is community modification rather than any direct action of the administered organism.[86]

8.3 Early allergen introduction

The clearest interventional evidence in allergy prevention concerns the infant rather than the mother. LEAP randomised infants aged 4 to 11 months with severe eczema or egg allergy to peanut consumption or avoidance; at 60 months, peanut allergy occurred in 13.7 per cent of the avoidance group and 1.9 per cent of the consumption group among those with a negative baseline skin prick test, and in 35.3 versus 10.6 per cent among those with an initially positive test.[87] PETIT, using stepwise introduction of heated egg from six months in Japanese infants with eczema, was stopped early for efficacy, with egg allergy at twelve months in 37.7 per cent of the placebo group versus 8.3 per cent of the intervention group.[88] EAT, which introduced six allergenic foods from three months in exclusively breastfed infants from the general population, was null in intention-to-treat analysis but showed significant benefit per protocol, with adherence to the early introduction regimen proving difficult in practice.[89]

The magnitude of these effects (relative risk reductions of 70 to 85 per cent) is an order of magnitude larger than anything achieved by maternal supplementation. This asymmetry is the central practical finding of the field and is reflected in current guidelines from EAACI and from the American and Canadian societies, both of which recommend introduction of peanut and egg in the first year and neither of which recommends any maternal dietary restriction or supplementation for allergy prevention.[43,90]

8.4 Microbiome restoration

Direct restoration strategies remain investigational. Vaginal seeding of caesarean-born infants partially restores initial composition but has no outcome data and carries a real risk of transmitting pathogens; professional bodies advise against it outside research.

Supplementation with *B. longum* subsp. *infantis* in breastfed infants produces a marked and persistent shift towards a bifidobacteria-dominated community, and represents a rational approach given the substrate specificity of HMOs, but allergic outcome data are not yet available. Defined bacterial consortia, informed by the murine work on *Clostridiales*,[74] are in early clinical development. The general lesson from the probiotic literature(that administering an organism is not the same as changing a community)should temper expectations.[91,92]

9. Translating the evidence to practice

The gap between what this literature supports and what is often advised is wide, and in both directions. Four statements can be made with reasonable confidence.

First, maternal exclusion diets during pregnancy or lactation should be actively discouraged. They do not prevent allergy, they compromise maternal nutrition, and the advice persists in clinical practice long after its withdrawal from guidelines.[41,43]

Second, a varied, high-quality maternal diet containing fish, fruit, vegetables, whole grains and dairy should be recommended — but as general antenatal nutritional advice with an additional plausible benefit, not as an allergy prevention intervention with a defined effect size. The distinction protects the clinician from overpromising and the woman from a sense of culpability if her child develops allergy anyway.

Third, allergenic solids should be introduced in the first year, from around four to six months, alongside continued breastfeeding, and not delayed. In infants with severe eczema or established egg allergy (the LEAP population) assessment before peanut introduction is appropriate where specialist services exist; where they do not, the risk calculus favours introduction at home over indefinite delay.[43,87,90]

Fourth, avoidable exposures with reasonably established harm should be addressed: tobacco smoke without qualification, and antibiotic and acid-suppressive prescribing in pregnancy and infancy where the indication is weak. These are defensible on general grounds irrespective of the strength of the allergy-specific evidence.

Conversely, several practices in current circulation are not supported: routine probiotic supplementation for the general population, vitamin D specifically for allergy prevention, hydrolysed formula for allergy prevention in infants at risk, and delayed introduction of any food.

10. Considerations for India and comparable settings

Almost all of the evidence reviewed here derives from cohorts in Europe, North America and Australasia, and its transferability to South Asia cannot be assumed. Several features of the Indian context alter the calculation.[93]

The exposure distributions differ substantially. Ambient PM2.5 concentrations in many Indian cities exceed those in the European cohorts from which pollution estimates derive by an order of magnitude, so the population-attributable fraction for air pollution is likely to be considerably larger, and correspondingly larger relative to nutritional factors. Household biomass fuel use adds an exposure barely represented in the source literature. Conversely, dietary fibre intake in traditional Indian diets is typically higher than in the European populations in which the fibre hypothesis was formulated, and the relevant exposure contrast may be between traditional and transitioning urban diets rather than between adequate and inadequate intake in absolute terms. Vitamin D deficiency is highly prevalent despite abundant sunlight, so the VDAART finding that benefit was concentrated in women with higher baseline concentrations has awkward implications for a population in which few would qualify.

Caesarean section rates in urban private-sector obstetric practice are among the highest in the world, and antibiotic use in pregnancy and infancy is extensive and frequently without a bacterial indication. Both are potentially modifiable at a scale that no nutritional intervention could match. Breastfeeding initiation and duration remain comparatively favourable, which is an asset.

Finally, the food allergen profile differs. Peanut allergy prevalence appears lower than in Western populations despite high peanut consumption, which is consistent with the early introduction hypothesis; chickpea, lentil and other legumes, and buffalo milk, feature in Indian clinical practice in ways that Western guidance does not address. Extrapolating an introduction schedule built around peanut, egg and cow's milk to this setting requires local data that do not currently exist. Establishing well- characterised birth cohorts with microbiome sampling in South Asia is, in our view, a higher priority than replicating supplementation trials.

11. Limitations of the evidence base

Several structural weaknesses recur throughout this literature and constrain what can be concluded.

Dietary exposure is measured almost entirely by food frequency questionnaire, with substantial measurement error that biases associations towards the null and makes dose–response estimation unreliable. Biomarker-based studies are rare. Outcome definitions are heterogeneous: parent-reported wheeze, physician-diagnosed asthma and objectively confirmed food allergy are not interchangeable, yet are frequently pooled. Follow-up is usually short relative to the natural history of allergic disease, so trials of prenatal interventions routinely report at ages before asthma and rhinitis are reliably diagnosable.

Microbiome studies carry their own limitations. Most rely on 16S ribosomal RNA amplicon sequencing, which resolves to genus at best and gives no functional information; faecal samples are an imperfect proxy for mucosa-associated communities; and the placental microbiome episode demonstrates how readily low-biomass contamination is mistaken for signal.[58] Cross-sectional association between community composition and disease cannot establish direction, and only a small number of studies have sampled sufficiently early to demonstrate temporal precedence.[67-69]

Confounding remains the dominant threat. Maternal diet, education, income, urbanicity, antibiotic exposure and mode of delivery are correlated with each other and with atopic family history. Sibling-comparison and Mendelian randomisation designs, which offer some purchase on this, have been applied only sporadically and where they have been, as in the antenatal antibiotic literature, they have generally attenuated the association substantially.[53] Finally, publication bias and selective outcome reporting are likely to be material in a field with many small trials and many measurable outcomes.

12. Future directions

Progress will require trials designed around the biology rather than around nutrient categories. Four features seem essential.

Stratification by baseline status. Both the n-3 LCPUFA and vitamin D trials found their effects concentrated in a definable subgroup. Future trials should enrol on the basis of measured deficiency or genotype rather than population risk category, accepting the smaller and more expensive samples that this implies.

Multi-component intervention. If the model is cumulative, single-agent trials are underpowered by construction. A trial combining dietary pattern modification, targeted supplementation and early allergen introduction, with factorial or sequential design, would be more informative than several more single- nutrient trials.

Mechanistic intermediates as co-primary outcomes. Measuring faecal and circulating SCFA, microbial community maturation and regulatory T-cell frequency alongside clinical endpoints would allow a null clinical result to be interpreted; distinguishing an intervention that failed to engage the mechanism from a mechanism that does not produce the outcome.

Adequate duration. Asthma and allergic rhinitis cannot be assessed at three years. The Danish fish oil follow-up, which found effects in adult offspring not apparent earlier,[31] indicates what is being missed by conventional trial horizons.

Beyond design, the field needs shotgun metagenomic and metabolomic characterisation rather than amplicon sequencing alone; systematic study of the mycobiome and virome; and birth cohorts in low- and middle- income settings, where both the exposure distributions and the disease trajectories differ from those in which the current evidence base was assembled.

13. Conclusion

Maternal diet, prenatal exposures and the infant gut microbiome are not three separate influences on childhood allergy but three views of a single system, linked by microbial metabolism of dietary substrate and by the developmental sensitivity of the neonatal immune compartment. The mechanistic case for this system is strong and is supported by experimental work that human studies cannot provide. The interventional case is much weaker: maternal supplementation trials have produced modest, conditional and inconsistent effects, and the only reliably large effects in allergy prevention have come from intervening in the infant, at the point of first antigen encounter, rather than in the mother during pregnancy.

That asymmetry should be stated plainly to patients and to policymakers. It does not mean that prenatal nutrition is irrelevant; it means that its effect is distributed, conditional and small at the individual level while potentially substantial at the population level; the characteristic signature of an exposure that should be addressed through diet quality and environmental policy rather than through supplements prescribed in clinic. The most useful thing a clinician can currently do with this literature is to stop recommending avoidance, start recommending timely introduction, and treat maternal nutrition as one part of general antenatal care rather than as an allergy intervention in its own right.

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Article

Paediatric Asthma: Updates from GINA 2026

Part 1 — MART therapy | Part 2 — Allergen immunotherapy



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The 2026 GINA Strategy Report, released in May 2026, carries two messages of particular consequence for the paediatric allergist: the anti-inflammatory reliever has finally acquired paediatric evidence, and allergen immunotherapy in children remains a promise still awaiting definitive proof.

Part 1: MART and its application in paediatric asthma

The founding principle is unchanged — no child aged six years or above should be treated with a short-acting beta-2 agonist (SABA) alone. Every such child requires inhaled corticosteroid (ICS)-containing treatment. For children aged 6–11 years, maintenance-and-reliever therapy (MART) with ICS-formoterol remains an evidence-based option at Steps 3 and 4, where a single formoterol-containing inhaler serves as both scheduled controller and reliever.[1]

The strongest new evidence comes from the CARE trial, the first randomised comparison of ICS-formoterol reliever against SABA reliever in children under twelve. Among 5–15 year olds previously using salbutamol alone, as-needed budesonide-formoterol reduced asthma attacks from 0.41 to 0.23 per child per year — a 45% relative reduction — with no adverse effect on growth velocity.[2] Earlier paediatric work had already shown that a MART regimen prolongs time to first exacerbation compared with higher-dose ICS.[3]

GINA 2026 has also simplified AIR/MART dosing instructions and replaced the rigid “maximum dose” concept with a safety prompt: the family should seek medical review if more than a specified number of inhalations are needed within 24 hours. For children aged 6–11 on budesonide-formoterol MART, the daily ceiling remains 48 mcg of metered formoterol (36 mcg delivered), roughly eight inhalations. ICS-formoterol has additionally been endorsed as an alternative to SABA for mild exacerbations in this age group presenting to primary care or the emergency department — a practical opportunity to initiate anti-inflammatory reliever therapy before discharge.[1]

Two caveats deserve emphasis:

- Only formoterol-containing combinations may be used for MART. ICS-salmeterol, ICS-vilanterol and ICS-SABA cannot be substituted.
- An evidence gap persists at Step 2 in 6–11 year olds, where it is still unknown whether as-needed ICS-formoterol matches daily ICS with as-needed SABA.[1]

Part 2: Immunotherapy and its role in paediatric asthma

GINA's position remains deliberately conservative. House dust mite (HDM) sublingual immunotherapy (SLIT) is suggested as add-on therapy for adults and adolescents with HDM-driven asthma inadequately controlled on ICS, provided FEV_1 exceeds 70% predicted. For subcutaneous immunotherapy, GINA advises weighing modest benefit against cost, inconvenience and a three-to-five-year commitment, and never initiating until asthma is well controlled.[1]

The pivotal paediatric trial, MT-11, randomised 533 children aged 5–17 years with inadequately controlled HDM allergic asthma to SQ HDM SLIT-tablet or placebo for 24–30 months. The rate ratio for clinically relevant exacerbations was 0.89 (95% CI 0.60–1.31) — directionally favourable but not superior to placebo, an outcome attributed largely to pandemic-era exacerbation rates far below those anticipated. Reassuringly, adverse events were predominantly mild local reactions, with no anaphylaxis and no adrenaline use.[4] The companion MT-12 trial in 5–11 year olds met its rhinitis endpoints and showed secondary improvement in asthma symptoms and medication use.[5] EAACI, applying a lower evidence threshold, conditionally recommends HDM subcutaneous immunotherapy in children and SLIT drops as add-on therapy in controlled HDM-driven asthma.[6]

The practical message: immunotherapy is an adjunct, never a substitute for ICS. Select children on demonstrated clinical relevance of sensitisation rather than on a skin-prick panel alone, ensure control before dosing, and counsel families for a minimum three-year course. In our own humid, mite-dominant eastern Indian setting, the greatest unmet need is well-conducted local data.

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Article: Patient Information Leaflet



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Allergy and Diwali: Celebrate Safely, Breathe Easily

Diwali is a festival of lights, celebration and togetherness. But for some children and adults with asthma, allergic rhinitis, eczema or other allergies, the festival can bring unexpected health problems.
Firecracker smoke, dust, fragrances, incense, colours and changes in routine can trigger respiratory and allergic symptoms. With a few simple precautions, people with allergies can enjoy Diwali more safely.

Why can Diwali trigger allergies?

Diwali celebrations may increase exposure to several potential irritants and allergens:

1. Firecracker smoke and air pollution

Firecrackers release **fine particulate matter (PM2.5), sulphur dioxide, nitrogen oxides and other irritating chemicals** into the air.
These can cause:

- Cough
- Wheezing
- Breathlessness
- Chest tightness
- Throat irritation
- Burning or watering of the eyes
- Worsening of asthma and allergic rhinitis



Important: Smoke from firecrackers is primarily an **airway irritant,** rather than a conventional allergen. It can nevertheless worsen symptoms in people with allergic airway disease.

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2. Dust and particulate matter

Cleaning homes before Diwali can disturb accumulated house dust, dust mites, mould spores and other particles.
This may trigger:

- Sneezing
- Runny or blocked nose
- Itchy nose and eyes
- Cough and wheezing

Tip: Prefer damp dusting and vacuum cleaning rather than sweeping, which can make dust airborne.



3. Incense sticks, dhoop and scented candles



Incense smoke and strong fragrances can irritate sensitive airways and may worsen:

- Asthma
- Allergic rhinitis
- Headache
- Eye irritation

Children with asthma should ideally **avoid prolonged exposure to incense smoke,** especially in poorly ventilated rooms.

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4. Perfumes, essential oils and fragrances



Diwali often means increased use of:

- Perfumes
- Room fresheners
- Scented candles
- Essential oils
- Fragranced cosmetics

Strong fragrances may trigger symptoms in sensitive individuals even when they are **not allergic to the fragrance itself.**

What about skin allergies?

Diwali can also cause skin problems.

Exposure to:

- Cosmetics and makeup
- Mehendi
- Fragrances
- Soaps and detergents
- Artificial jewellery
- Certain paints and colours

may cause **irritant or allergic contact dermatitis.**

Look out for:

- Itching
- Redness
- Burning
- Swelling
- Dry or cracked skin
- Blisters or rashes

Avoid applying new cosmetic products immediately before an important celebration. If a product has caused a reaction previously, avoid it.

What about firecrackers?

For people with asthma or significant respiratory allergies, **avoiding firecracker smoke is the safest choice.**
If you are around firecrackers:

✅ *Stay as far away from the smoke as possible*
✅ *Choose an open, well-ventilated area*
✅ *Avoid areas where crackers are being burst continuously*
✅ *Keep children with asthma indoors when outdoor air quality is poor*
✅ *Follow the child's prescribed asthma action plan*
✅ *Keep prescribed reliever medication readily accessible*

❌ *Do not deliberately inhale smoke*
❌ *Do not allow children to play around burning crackers*
❌ *Do not stop regular controller asthma medication during Diwali*

If my child has asthma, should we stop the inhaler during Diwali?

No.

- Children should continue their **regular prescribed asthma controller medication** during Diwali.
- Stopping controller treatment because the child feels well can increase the risk of an asthma exacerbation when exposed to smoke and air pollution.
- Keep the child's **reliever inhaler and spacer** easily accessible.
- If your child has a written asthma action plan, follow it.

How can I protect my child with allergic rhinitis?

Before Diwali

- Continue prescribed allergy medicines.
- Keep the child's room as dust-free as possible.
- Avoid unnecessary fragrances.
- Wash bedding regularly.
- Use damp cleaning methods.

During Diwali

- Minimise exposure to smoke.
- Keep windows closed when outdoor pollution is particularly high.
- Avoid prolonged outdoor activity during periods of heavy smoke.
- Consider a properly fitted mask for older children when appropriate.

After Diwali

- Bathe and change clothes after prolonged outdoor exposure.
- Wash the face and hands.
- Continue prescribed treatment if symptoms persist.



Can sweets cause allergy?

Yes—but not every reaction to food is an allergy.

Common food allergens that may occasionally be present in festive foods include:

🥜 Peanuts
🌰 Tree nuts
🥛 Milk
🌾 Wheat
🌱 Sesame
🥚 Egg

People with a **known food allergy should carefully check ingredients and avoid foods containing their specific allergen.**

Be particularly careful with:

- Sweets from unfamiliar sources
- Mixed dry-fruit preparations
- Chocolates
- Bakery products
- Foods with uncertain ingredients

If you have a prescribed **epinephrine/adrenaline auto-injector,** keep it with you during c**elebrations.**

Recognising a serious allergic reaction

Seek urgent medical attention if a person develops:

- Difficulty breathing
- Wheezing or noisy breathing
- Swelling of the tongue or throat
- Difficulty swallowing
- Dizziness or fainting
- Sudden severe weakness
- Widespread hives associated with breathing or circulation problems

These may indicate **anaphylaxis**, a potentially life-threatening allergic reaction.

For someone with a known risk of anaphylaxis, **adrenaline/epinephrine is the first-line emergency treatment.** Do not delay its use while waiting for antihistamines to work.



A safer Diwali for children with allergies

The Diwali Allergy Checklist

□ Continue prescribed asthma/allergy medication
□ Keep the reliever inhaler and spacer accessible
□ Avoid firecracker smoke
□ Reduce exposure to incense and strong fragrances
□ Damp-dust the house
□ Avoid known skin allergens
□ Check food ingredients carefully
□ Keep emergency medication available if prescribed
□ Know the warning signs of severe allergy
□ Seek medical care early if breathing difficulty develops

Remember

Diwali is about celebrating together—not about exposing ourselves to smoke.

For children and adults with asthma or allergies, the safest celebration is one **that minimises smoke, dust, fragrances and known allergens while continuing prescribed treatment.**

Light a diya, not the airways! 🪔💙

Patient message:
Celebrate the festival of lights without letting smoke and allergens dim your health.



Article: TEST IN FOCUS

Component Resolved Diagnostics: Use & Abuse

A practical guide for the young paediatrician



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1. The basis of the test

The idea in one line: an allergen source is not a single thing — it is a bag of proteins — and CRD tests the individual proteins rather than the whole bag.

Think of peanut as a lunchbox. Conventional testing tells you the child reacts to "the lunchbox". CRD opens it and tells you which item inside the IgE is actually binding to. That matters, because some proteins in the bag predict anaphylaxis and others predict nothing more than an itchy mouth.

How the names work

This puts off most trainees, but the rule is simple:

First three letters of the genus + first letter of the species + a number in order of discovery.
So **Ara h 2** = *Arachis hypogaea* = **peanut,** and it was the second peanut allergen to be characterised. Once you know this, the report no longer looks like code.

Prefix	Allergen source
Ara h	Peanut (*Arachis hypogaea*)
Gly m	Soy (*Glycine max*)
Cor a	Hazelnut (*Corylus avellana*)
Ana o	Cashew (*Anacardium occidentale*)
Jug r	Walnut (*Juglans regia*)
Gal d	Hen's egg (*Gallus domesticus*)
Bos d	Cow's milk (*Bos domesticus*)
Tri a	Wheat (*Triticum aestivum*)
Pru p	Peach (*Prunus persica*)
Der p	House dust mite (*Dermatophagoides pteronyssinus*)
Bet v	Birch (*Betula verrucosa*)
Phl p	Timothy grass (*Phleum pratense*)
Pen m	Shrimp (*Penaeus monodon*)
Api m / Ves v	Honeybee (*Apis mellifera*) / Wasp (*Vespula vulgaris*)

The one concept to carry home

Proteins behave according to the family they belong to, not according to the food they came from.

Protein family	Examples	Behaviour	What it usually means
Storage proteins	**Ara h 2, Ara h 6** (peanut), Cor a 9/14, Ana o 3	Survive cooking and stomach acid	**Genuine allergy; systemic risk**
nsLTP	Pru p 3, Ara h 9, Tri a 14	Tough, heat-stable	Genuine; can be systemic
PR-10	**Ara h 8**, Cor a 1, Gly m 4	Fragile — destroyed by cooking and digestion	**Usually mild; oral symptoms only**
Profilins	Bet v 2, Phl p 12	Fragile; present in almost every plant	Cross-reactivity; rarely clinical
CCD (sugar chains)	MUXF3	Sticky sugar groups shared by many plant and insect proteins	**Almost always clinically irrelevant**

Pointer for trainees: stable protein = trouble; fragile protein = usually tolerable. If a protein cannot survive a frying pan, it usually cannot survive the stomach either.

2. How it differs from the tests you already use

- **SPT and extract-based sIgE use a crude mixture.** The extract may under-represent the very protein that matters (classically Ara h 2 and ω-5 gliadin in wheat) and may yield a positive result purely because of a harmless panallergen or sugar group.
- **CRD does not tell you whether the child is allergic.** It tells you what the IgE is directed against — and therefore how concerned to be.
- **Oral food challenge remains the gold standard.** CRD helps you decide whom to challenge, not whether to challenge at all.
- **Formats:** singleplex (ImmunoCAP — one component at a time, quantitative, kUA/L) versus multiplex microarray (ISAC, ALEX2 — hundreds of components at once, semi-quantitative, minimal sample volume).

Pointer: SPT and extract sIgE answer “which food?” CRD answers “which molecule, and how dangerous?”

3. Lab and logistic support needed

- **Sample:** 0.5–1 mL of serum for singleplex; 30–100 µL for microarray. Avoid haemolytic or lipaemic samples.
- No antihistamine stoppage, no skin needed — a real advantage in severe eczema, dermographism, small infants, and any child in whom SPT is considered unsafe.
- **Platform:** An accredited immunoassay laboratory with proper calibration and quality control. In India, this usually means a referral laboratory in a metro, cold-chain transport, and a turnaround of several days.
- **Cost:** several times that of an SPT. Multiplex arrays are expensive and almost always paid for out of pocket.
- **The hidden requirement:** Somebody who can read it. A CRD report handed to an untrained clinician creates more avoidance than it removes.

4. Advantages

- **Risk stratification.** Ara h 2 / Ara h 6 positive → genuine peanut allergy with systemic risk. Ara h 8 alone → usually mild oral symptoms.
- **Untangles the polysensitised child.** The child with fifteen positive tests and no symptoms is usually a profilin or CCD story.
- **Predicts tolerance of baked food.** High Gal d 1 (ovomucoid) → likely to react even to baked egg. High Bos d 8 (casein) → likely to react to baked milk and to have persistent allergy.
- **Guides immunotherapy.** Sensitisation to major mite allergens (Der p 1, 2, 23) predicts better response to AIT; a profilin- or CCD-only child is a poor candidate. In venom allergy, Api m 1 versus Ves v 1 / Ves v 5 decides which venom to use.
- **Identifies specific syndromes.** Tri a 19 (ω-5 gliadin) in wheat-dependent exercise-induced anaphylaxis; alpha-gal in delayed reactions to mammalian meat.
- **Practical gains.** Fewer challenges, fewer needless elimination diets, one safe blood draw, and — not a small thing — a clearer conversation with an anxious parent.

5. Disadvantages

- **Cost and access.** Not reimbursed, and not available in most district settings.
- **It still measures sensitisation, not disease.** A positive component in a child who eats the food happily is not an allergy.
- **Multiplex panels invite abuse.** Test 300 allergens, and you will find positives you never asked about — each one generating anxiety and unnecessary avoidance.
- **Cut-offs are borrowed.** Nearly all published decision points are derived from European and North American cohorts. Birch is irrelevant here; PR-10 sensitisation is uncommon, and mite, grass and weed pollen, and LTP dominate. Applying a European Ara h 2 cut-off to a child in Kolkata is not evidence-based. This is the single largest gap in Indian practice.
- **No role at all** in non-IgE-mediated disease — FPIES, most eosinophilic oesophagitis, delayed reactions.

6. Report interpretation

Read every report by asking three questions, in order:

1. Is this a **genuine marker** or a **cross-reactivity marker?**
2. Does it **fit the history** the mother gave me?
3. Will it **change what I do on Monday?**

If the answer to the third question is no, the test should probably not have been sent.

Source	Suggests genuine / higher risk	Suggests cross-reactivity / lower risk
Peanut	Ara h 1, 2, 3, 6	Ara h 8, CCD
Hazelnut	Cor a 9, Cor a 14	Cor a 1
Cashew	Ana o 3	—
Egg	Gal d 1 (ovomucoid)	Gal d 2 alone
Milk	Bos d 8 (casein)	α-lactalbumin / ß-lactoglobulin alone
Wheat	Tri a 19, Tri a 14	Profilin
Shrimp	Pen m 1 with a matching history	Pen m 1 from mite or cockroach cross-reactivity
Dust mite	Der p 1, Der p 2, Der p 23	Der p 10 (tropomyosin)

Two rules that are easy to forget
- The level predicts the **probability of a reaction**, not the severity of the next reaction.
- A negative CRD with a convincing history **does not exclude** allergy. Believe the history and proceed to challenge.

Three quick cases

- *A 4-year-old with urticaria and vomiting minutes after peanut exposure; Ara h 2 strongly positive.* → Genuine, high-risk. Strict avoidance, adrenaline auto-injector, no challenge.
- *An 8-year-old with atopic dermatitis and twelve positive food tests, eating all of them daily without symptoms; CRD shows only CCD and profilin.* → Cross-reactive noise. Return the foods to the diet. This is the child for whom CRD helps most.
- *A 3-year-old with an egg allergy whose mother asks about birthday cake; Gal d is 1 low.* → A supervised baked-egg challenge is reasonable.

7. Other issues — where "use" becomes "abuse"

Five questions before you order CRD

1. Is there a **documented clinical history**, not just a parental suspicion?
2. Do I have a **specific question** — challenge or not, baked food or not, which venom, which AIT?
3. Will the result **change management?**
4. Has the family been **counselled about cost,** and about what a positive result does and does not mean?
5. Am I the person who will **explain the report** to them?

Common abuses, to name plainly

- Using CRD as a **screening panel** in an asymptomatic child, or as a shortcut in a child with eczema alone.
- Sending a 300-allergen array because it is "more complete".
- **IgG / IgG4 "food intolerance" panels —** these are not CRD, have no diagnostic role, and should be actively discouraged.
- Direct-to-consumer testing arriving in your clinic already interpreted by the internet.

The correct sequence remains: history → SPT / extract sIgE → CRD only for a selected question → challenge where needed.

Repeat testing has a place — roughly annually for milk and egg allergy, to time re-challenge.
What we owe our own patients: Indian component-sensitisation profiles and locally derived decision points. Until we generate them, every cut-off we quote is borrowed.

Suggested reading

[To be finalised by the authors]

• Matricardi PM, Kleine-Tebbe J, Hoffmann HJ, et al. EAACI Molecular Allergology User's Guide. Pediatr Allergy Immunol. 2016;27(Suppl 23):1–250.
• Ansotegui IJ, Melioli G, Canonica GW, et al. IgE allergy diagnostics and other relevant tests in allergy: a WAO position paper. World Allergy Organ J. 2020;13(2):100080.
• Santos AF, Riggioni C, Agache I, et al. EAACI guidelines on the diagnosis of IgE-mediated food allergy. Allergy. 2023;78(12):3057–3076.
• WHO/IUIS Allergen Nomenclature Sub-Committee. www.allergen.org

Article: National Allergy Day – 11th October

Building an Allergy-Aware and Allergy-Ready India



Dr. Neeraj Gupta
Immediate Past Chairperson, IAP Allergy & Applied Immunology Chapter

The burden of allergic diseases in India has increased substantially over the past two decades, affecting children and adults and impacting quality of life, education, nutrition and healthcare utilization. Yet, awareness about allergy recognition, appropriate diagnosis, prevention and emergency preparedness remains limited.

Recognizing this growing need, the **IAP Allergy & Applied Immunology Chapter** conceived **National Allergy Day** with the vision of creating a nationwide movement towards greater allergy awareness, early recognition, evidence-based care and prevention.

We are delighted that **11th October has now been formally adopted by the Central Indian Academy of Pediatrics (CIAP) as National Allergy Day.** This marks an important milestone in bringing allergic diseases into greater focus as a public health priority in India.

National Allergy Day is more than a commemorative day—it is an opportunity to **educate families, empower healthcare professionals, strengthen preparedness in schools and communities, and promote evidence-based allergy care.**

STAMP OUT ALLERGY

National Allergy Day will continue under the Chapter's flagship initiative:

S - Spot allergy symptoms early
T - Test appropriately when clinically indicated
A - Act promptly with evidence-based management
M - Monitor allergic diseases regularly
P - Prevent future allergic reactions through education and preparedness

Through **STAMP OUT ALLERGY,** the Chapter aims to improve allergy literacy, dispel myths, discourage inappropriate testing, strengthen recognition and management of anaphylaxis, and promote allergy-safe environments.

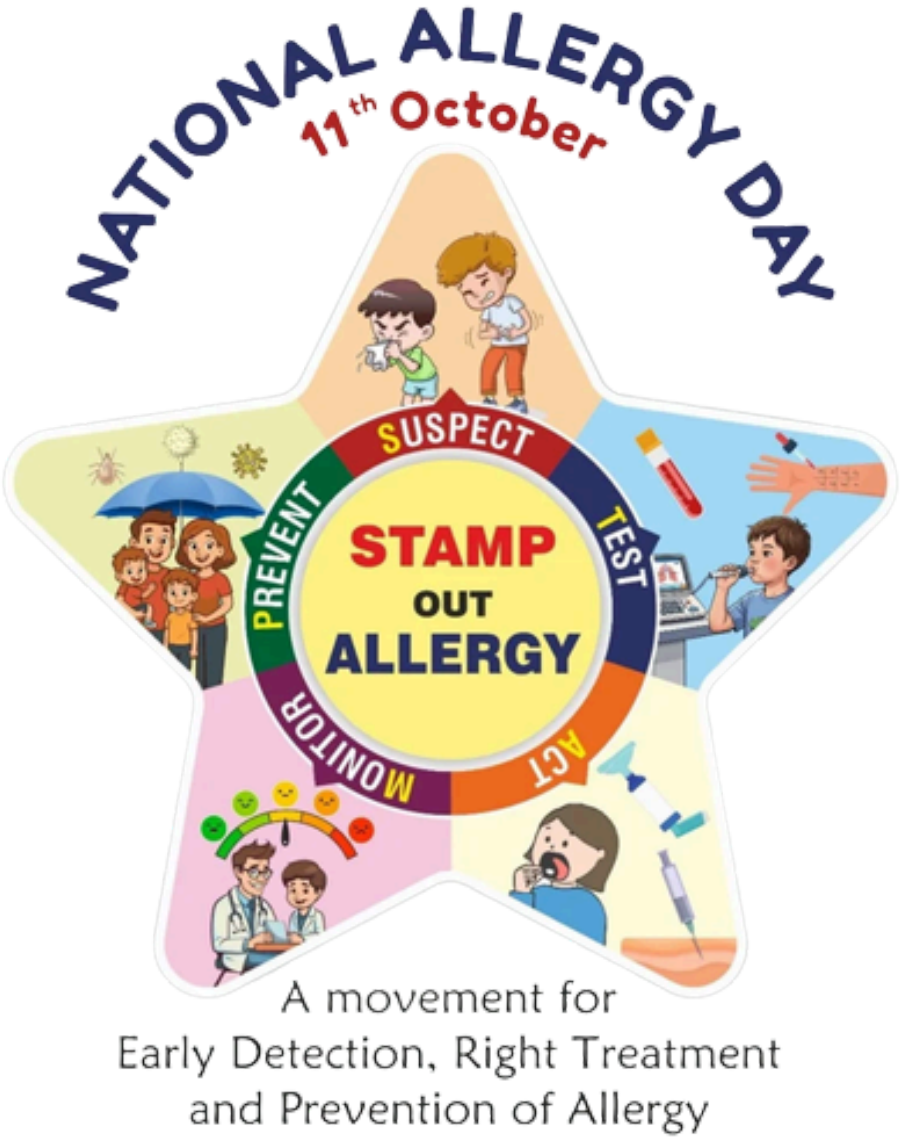
National Allergy Day 2026

To mark the first National Allergy Day following its formal adoption by CIAP, the **IAP Allergy & Applied Immunology Chapter** will undertake a series of educational and community-focused activities leading up to **11th October 2026.** These initiatives will engage healthcare professionals, schools, families and the wider community, with an emphasis on practical allergy awareness and preparedness.

We invite all **IAP members, healthcare professionals, educational institutions, families and community partners** to join this national movement.

National Allergy Day is a shared commitment to creating allergy-aware schools, **allergy-ready families and informed communities.**

Together, let us STAMP OUT ALLERGY and build an allergy-aware, allergy-ready India.



ALLERGY ACTIVITIES

A round-up of recent conferences, symposia and outreach from across the Chapter

Pediatric Allergy Asthma Symposium 2026 — Gujarat

The Pediatric Allergy Asthma Symposium 2026 was held on 23rd August 2026 at Madhubhan Resort & Spa, organised by the Department of Pediatrics, Pramukhswami Medical College, Bhaikaka University, Karamsad, together with the Gujarat IAP Allergy & Applied Immunology Chapter. The symposium brought together clinicians for an academic day dedicated to childhood asthma and allergic airway disease.



Left to right: Dr Mitesh Kakkad ("Asthma Management in Children Above 6 Years"), Dr Sowmya Nagarajan and Dr Neeraj Gupta addressing delegates at the Symposium.

"Building awareness of evidence-based asthma care in children above six years — one symposium at a time."

The day's sessions covered practical, ground-level approaches to diagnosing and managing persistent asthma in the school-age child, reflecting the Chapter's continued commitment to translating international guidelines into everyday clinical practice across the region.

IFS Officers' Workshop — Climate Change: Science, Policy & Adaptive Management

A workshop for Indian Forest Service (IFS) officers on “Climate Change: Science, Policy and Adaptive Management” was conducted from 17th to 21st August 2026 at the Divecha Centre for Climate Change (DCCC), Indian Institute of Science (IISc), Bengaluru, under the guidance of Dr H Paramesh. The programme underlined the growing links between environmental change and paediatric respiratory and allergic disease, connecting policy-level climate action with clinical practice.



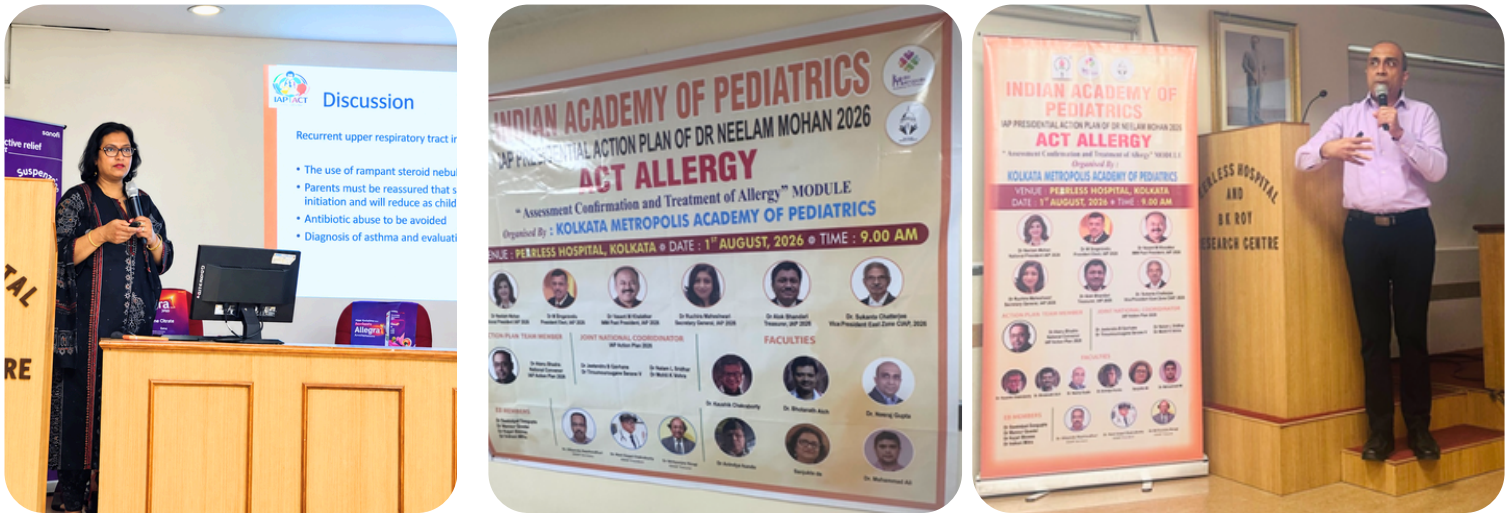
The ceremonial lamp-lighting at the inauguration of the IFS officers' workshop on climate change, DCCC, IISc Bengaluru.

“From forest policy to the paediatric clinic — climate change is a shared airway health challenge.”

Led by Dr H Paramesh, the workshop brought together Indian Forest Service officers to examine the science and policy dimensions of climate change and adaptive management — a timely reminder that environmental stewardship and paediatric allergy and respiratory health are closely intertwined.

ACT Allergy Module — Asthmacon Pre-Conference Workshop

The “ACT Allergy” module — Assessment, Confirmation and Treatment of Allergy, under the IAP Presidential Action Plan of Dr Neelam Mohan 2026 — was held on 1st August 2026 at Peerless Hospital, Kolkata, organised by the Kolkata Metropolis Academy of Pediatrics. The workshop was conducted as the pre-conference programme for Asthmacon, drawing faculty and delegates for a focused, hands-on session on allergy assessment and management in children.

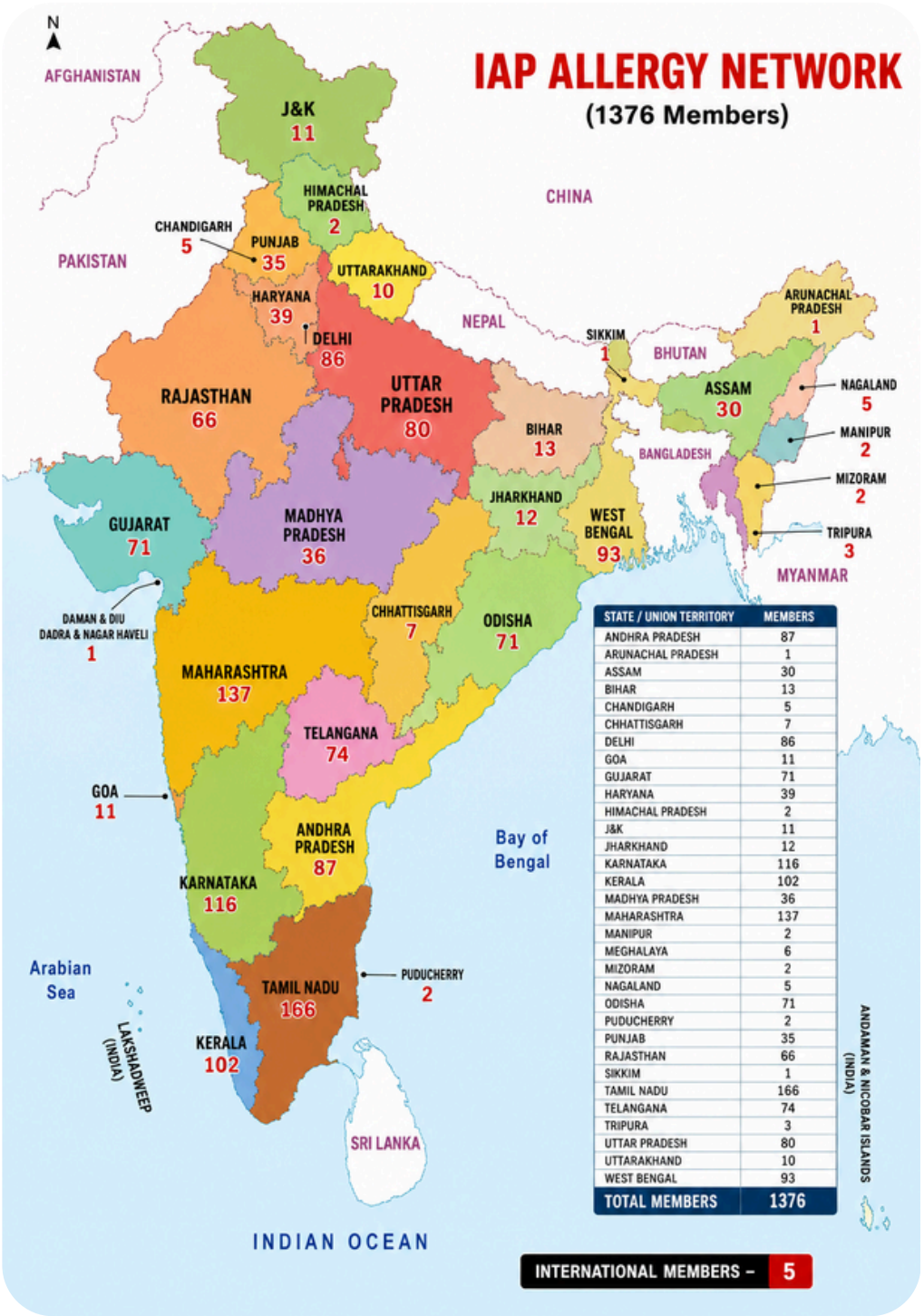


Faculty presenting at the ACT Allergy module, with the event banner listing the IAP Action Plan team and faculty, at Peerless Hospital and B K Roy Research Centre, Kolkata.

“Assessment, confirmation and treatment — taking allergy care back to first principles for every practising paediatrician.”

Discussions covered the judicious use of steroid nebulisation, avoidance of antibiotic overuse, and a structured approach to diagnosing and evaluating asthma in children — reinforcing practical, first-principles allergy care ahead of the main Asthmacon conference.

Membership Snapshot



STATE / UNION TERRITORY	MEMBERS
ANDHRA PRADESH	87
ARUNACHAL PRADESH	1
ASSAM	30
BIHAR	13
CHANDIGARH	5
CHHATTISGARH	7
DELHI	86
GOA	11
GUJARAT	71
HARYANA	39
HIMACHAL PRADESH	2
J&K	11
JHARKHAND	12
KARNATAKA	116
KERALA	102
MADHYA PRADESH	36
MAHARASHTRA	137
MANIPUR	2
MEGHALAYA	6
MIZORAM	2
NAGALAND	5
ODISHA	71
PUDUCHERRY	2
PUNJAB	35
RAJASTHAN	66
SIKKIM	1
TAMIL NADU	166
TELANGANA	74
TRIPURA	3
UTTAR PRADESH	80
UTTARAKHAND	10
WEST BENGAL	93
TOTAL MEMBERS	1376

Wish to join our membership?

ENROLL NOW

Happy Diwali

About Us

IAP Allergy and Applied Immunology Chapter (IAPPAAI) aims to promote awareness, education, research, and best practices in the field of allergy and asthma. It also works to enhance access to care, provide support to healthcare professionals, and advocate for policies that benefit patients with these conditions.

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Don’t Miss This Opportunity!

Join the IAP Allergy & Applied Immunology Chapter today and enhance your knowledge and expertise in the field of allergy and immunology.
For more information, contact us now!

Upgrade Your Expertise..!

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