Bienvenue au cours d'allergologie ALLER2A

- Cours d'Allergologie et Immunologie Clinique
- Étudiants de la région Auvergne Rhone-Alpes
- DES, FST, DESC, Capacité, DUFRAL, FMC (masterclass)
- 2 aspects:
  - 7 modules de 2 jours de cours visio; 1 module /mois de décembre à juin
  - Support e-learning des items sur AllergoLyon https://allergolyon.fr/
- Contact: ext-barbara.gil@chu-lyon.fr

- **Merci de fermer vos micros et caméras**
- Poser les questions via l'onglet discussion

Les 7 modules de cours visio

0 Immunologie de l’allergie

1 Allergologie générale

2 Pédiatrie

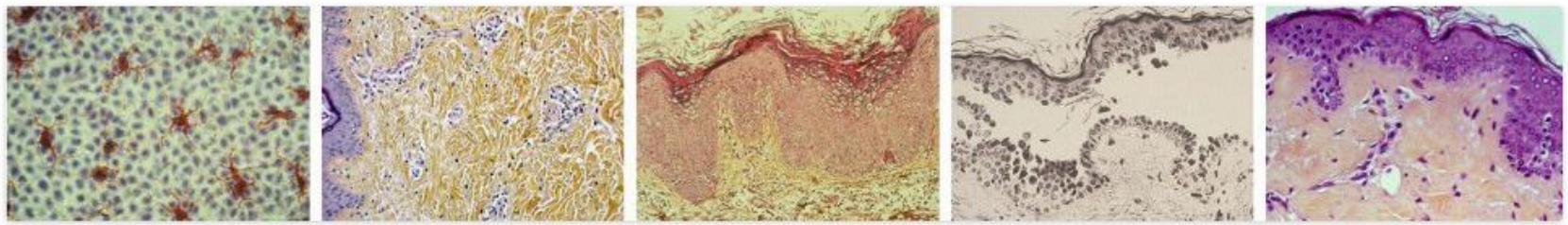
3 Pneumologie / ORL / Ophtalmologie

4 Dermatologie

5 Médicaments

6 Aliments / Hyménoptères / Professionnel

AllergoLyon



Présentation

Bienvenue sur le site du département d'Allergologie et Immunologie Clinique du CHU de Lyon (Centre Hospitalier Lyon Sud).

Sur ce site, vous pourrez découvrir nos activités :

- Les enseignements en Allergologie et Immunologie Clinique
- Nos thématiques de recherche
- Nos activités cliniques
- Les informations à destination des patients et des soignants
- L'éducation thérapeutique
- Nos actualités

RECHERCHE

Rechercher

– Cours Allergologie 2022-23

– Items Enseignement Allergologie et Immunologie

PROGRAMME ALLERGOLOGIE 2022-2023

Programme .pdf

Module 0 : Immunologie de l'Allergie
Jeudi 15 et Vendredi 16 décembre 2022
(M.VOCANSON/A.NOSBAUM)

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• 9h à 10h30 : M. VOCANSON – Le système immunitaire en action
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• 10h30 à 12h : J-F. NICOLAS – Les hypersensibilités allergiques et non allergiques
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• 15h à 16h30 : M. TAUBER – Mécanismes de l'immunothérapie spécifique d'allergène

Module 1 : Allergologie Générale
Jeudi 12 et Vendredi 13 janvier 2023
(C.DZVIGA)

Jeudi 12 janvier 2023
• 9h à 12h : C. PONVERT
Introduction à l'allergie et aux maladies allergiques
Allergie immédiate (Hypersensibilité de type I)
• 13h à 15h : S. VIEL – Biologie de l'allergie
• 15h à 17h : L. BELLANGE– Les tests cutanés en pratique (Prick/IDR/Patch)

Vendredi 13 janvier 2023
• 9h à 10h : P. PRALONG – Antihistaminiques

Items Enseignement Allergologie et Immunologie

Enseignement théorique Allergologie AURA (Auvergne Rhone-Alpes)
(stages réalisés pendant DES, FST, DESC, Capacité, DUFRAL et Master Class)

L'enseignement « Allergologie AURA » est théorique (cours réalisés tout au long de l'année) et pratique (stages réalisés pendant le DES, DESC et FST).

L'enseignement théorique concerne les 10 spécialités de l'allergologie (dénommées 1 à 10 ci-dessous) plus un socle immunologie indispensable à la compréhension des maladies allergiques.

Pour chacune des spécialités nous avons réuni les meilleurs articles qui permettent d'avoir des bases de connaissances qui seront développés lors des cours ; Nous conseillons aux étudiants de parcourir ces articles avant le cours.

Présentation et Items des modules, document en format .pdf

Les 10 articles indispensables (.pdf)

0. Immunologie
1. Allergologie Pédiatrique
2. Allergologie générale
3. Dermatologie
4. Pneumologie
5. Hyménoptères
6. Allergie alimentaire
7. Médicaments
8. ORL
9. Ophtalmologie
10. Allergologie professionnelle

0.1 Introduction générale au système immunitaire, chapitre 1 ASSIM

0.2 Structure et organisation générale du système immunitaire, chapitre 2 ASSIM

0.3 An introduction to immunology and immunopathology 2018

0.4 Classification of hypersensitivity reactions 2019

0.5 Cellules présentatrices d'antigènes et allergie

0.5.1 Les cellules dendritiques, chapitre 8 ASSIM

0.5.2 Antigen Processing and Presentation 2016

0.5.3 Dendritic Cells and Their Role in Allergy 2017

0.6 Immunité de type 2 et allergie

0.6.1 Type 2 Innate Lymphoïd Cells in Allergic Disease 2013

0.6.2 Th2, allergy and group 2 innate lymphoid cells 2013

0.6.3 Innate and Adaptive Type 2 Immunity in Lung Allergic Inflammation 2017

0.7 Lymphocytes Th1, Th2, Treg, Th17 et allergie

0.7.1 Origine, différenciation et répertoire lymphocytaire T, chapitre 9 ASSIM

0.7.2 Immunité adaptative : activation et polarisation des lymphocytes T, chapitre 11 ASSIM

0.7.3 The 3 major types of innate and adaptive cell-mediated effector immunity 2014

0.7.4 Programming dendritic cells to induce TH2 and tolerogenic responses 2010

0.8 Lymphocyte B dans l'allergie

0.8.1 Lymphocytes B : diversité, ontogénèse, différenciation et activation, chapitre 10 ASSIM

0.8.2 B cell responses, cell interaction dynamics and decisions 2019

0.8.3 Non-classical B cell memory of allergic IgE responses 2019

0.9 Cytokines et chimiokines

0.9.1 Cytokines et chimiokines chapitre 19 ASSIM

0.9.2 Chemokine receptors in allergic diseases 2016

0.9.3 Divergent chemokine receptor expression and the consequence for human IgG4 B cell responses 2020

0.10 Immunoglobulines

0.10.1 Les immunoglobulines : structure et fonctions, chapitre 6 ASSIM

0.10.2 Structure and dynamics of IgE–receptor interactions: FcεRI and CD23/FcεRII

0.10.3 The production and regulation of IgE by the immune system 2014

0.11 Bases génétiques de l'atopie et des maladies allergiques

0.11.1 Les prédispositions génétiques dans l'allergie alimentaire 2017

0.11.2 The role of filaggrin in atopic dermatitis and allergic disease 2020

0.11.3 Discovering susceptibility genes for asthma and allergy 2008

0.11.4 The immunogenetics of asthma and eczema: a new focus on the epithelium 2004

Classification of hypersensitivity reactions

Melanie C. Dispenza, M.D., Ph.D.

ABSTRACT

As the primary defense against pathogens, the immune system uses numerous strategies to ensure optimal protection for the host. When immune responses go awry, however, they can cause great damage. "Hypersensitivity" is a broad term used to describe an excessive and/or pathogenic immune response to either foreign or self antigens. Gell and Coombs were the first to categorize hypersensitivity reactions into 4 types according to pathophysiology, but more recent insights into the mechanisms of these disorders have since modified the original classification system. This review describes the immune mechanisms involved in each of the modern Gell-Coombs categories.

(Allergy Asthma Proc 40:470–473, 2019; doi: 10.2500/aap.2019.40.4274)

In 1963, Gell and Coombs[1] created the foundation for classifying hypersensitivity reactions by dividing them into four distinct groups according to mechanisms of tissue injury: type I (immediate or immunoglobulin E [IgE] mediated), type II (cytotoxic or IgG/IgM mediated), type III (immune complex mediated), and type IV (delayed type or T-cell mediated). This classification system has since then been expanded to include subtypes of types II and IV, with the intent to better reflect the immunopathology of diseases (Table 1).[2,3] In clinical practice, however, the hypersensitivity categories can overlap, and patients may display a constellation of symptoms from multiple types of hypersensitivity reactions at the same time. Syndromes that do not fit into the single Gell-Coombs classification categories are often termed mixed drug reactions, and, clinically, it may be more practical to classify these reactions by the organ systems affected. In addition, it is increasingly recognized that many medications, e.g., penicillins, are capable of inducing many types of reactions.[4]

Antigens of all types and sizes may cause hypersensitivity reactions. Large-molecular-weight protein antigens, whether self or foreign, can be processed and recognized directly by T-cell receptors or immunoglobulins. Many biologic drugs are recombinant antibodies which can form immune complexes and/or bind to Fc receptors on leukocytes.[5] In contrast, small low-molecular-weight compounds (<1000 Daltons) cannot be directly processed or presented by antigen-presenting cells, and thus they cannot elicit immune responses by themselves. However, they may stimulate immune responses in other ways. In the hapten model, small molecules (*e.g.*, penicillin, molecular weight ~300 Daltons) covalently bind to soluble serum proteins (*e.g.*, albumin) or cellular proteins to create new epitopes, which can then be antigenic. Some drugs act as prohaptens, meaning that they are inert in their native form, but their metabolites can haptenize, as is the case with sulfamethoxazole.[6] Finally, the p-i concept (pharmacologic interaction with immune receptors) proposes that some drugs that lack hapten characteristics may bind reversibly (noncovalently) to T-cell receptors or human leukocyte antigen (HLA) molecules directly, possibly in a nonspecific or cross-reactive fashion.[7] This model may help to explain why some HLA alleles render patients prone to delayed hypersensitivity reactions due to certain medications. For example, an estimated 55% of HLA-B*57:01 carriers will develop a hypersensitivity reaction from abacavir.[7] Thus, small-molecular-weight compounds can cause all types of hypersensitivity reactions.[5]

From the Division of Allergy and Immunology, Department of Medicine, Northwestern University Feinberg School of Medicine, Chicago, Illinois

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HYPERSENSITIVITY TYPES

Type I

Type I or immediate hypersensitivity is mediated by IgE specific for allergens. An overview of allergens is provided in this issue.[8] Sensitization to allergens occurs when T-helper (Th) type 2 cells and their mediators drive isotype switching in B cells to produce IgE antibodies. A large portion of IgE remains bound to the high-affinity IgE receptor FcεRI on the surface of mast cells and basophils. On re-exposure, allergen cross-links specific IgE on these cells, which causes release of

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which activate macrophages to secrete cytokines such as interferon γ and TNF-α. A prototypical example of this type is contact dermatitis, which can occur to various substances, including poison ivy, oak, and sumac (members of the *Toxicodendron* genus). Type IVb reactions involve the production of IL-4, IL-5, and IL-13 by Th2 cells to induce eosinophilic inflammation and IgE production from B cells. For example, in patients with Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS syndrome) drug-sensitized Th2 cells promote eosinophilic survival, activation, and tissue migration to cause multiorgan injury. In addition, type IVb reactions may be involved in the late-phase inflammation of atopic disorders, such as asthma or allergic rhinitis. Type IVc is primarily mediated by cytotoxic CD8+ T cells, which directly kill targeted cells by using a number of mediators, including perforin, granulysin, and granzyme B. Type IVc reactions seem to be the underlying mechanism of tissue damage in Stevens-Johnson syndrome and toxic epidermal necrolysis, in which activated CD8+ T cells induce apoptosis and/or necrosis of keratinocytes. Type IVd responses cause tissue damage when T-cell–derived CXCL-8 (also known as IL-8) recruits neutrophils into tissues to create sterile neutrophilic inflammation, as is the case with acute generalized exanthematous pustulosis.

IMMUNOLOGY

- Type I reactions are mediated by allergen-specific IgE bound to the high-affinity FcεRI receptor on mast cells and basophils. On allergen cross-linking of these receptors, mediators (e.g. histamine) are released, which result in urticaria, angioedema, and/or anaphylaxis.
- Type II reactions are characterized by IgG/IgM binding to self antigens on the surface of cells, which can cause tissue damage by activating phagocytosis, complement-directed cytotoxicity, and/or antibody-dependent cell-mediated cytotoxicity.
- Type III reactions are mediated by immune complexes of IgG/IgM and antigens, which deposit into tissues to cause direct organ damage.
- Type IV reactions are delayed responses that are mediated by T cells and are further divided into four subtypes.

CLINICAL PEARLS

- Some medications (such as penicillins) can cause all types of hypersensitivity reactions.
- There are no known therapies that can effectively prevent IgE-mediated reactions. Medications such as H_1 antihistamines and corticosteroids only mitigate the effects of some (but not all) mediators.

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