Bangkok
Joint Congress
APAAACI
APAPARI
11 - 14 October
2018

Joint Congress of

Asia Pacific Association of Allergy, Asthma and Clinical Immunology & Asia Pacific Association of Pediatric Allergy, Respirology and Immunology

11 - 14 October 2018

Centara Grand & Bangkok Convention Centre at CentralWorld

Novel Therapies, Prevention and Integrated Action: Towards Improved Patient Care

PROGRAM BOOK

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Symposium #12
APSID Session 3: Innate Immunity Deficiency
10.30-12.00 (Meeting Room 4) Lotus 1-2

Chairpersons: Amir Hamzah Dato' Abdul Latiff (Malaysia) and Tassalalpa Daengsuwan (Thailand)

10.30-10.55 Chronic Granulomatous Disease, a venue of infection and inflammation
Reinhard Seger (Switzerland)

10.55-11.20 Mendelian susceptibility to mycobacterial infection as in born errors of the innate immunity
Amir Hamzah Dato' Abdul Latiff (Malaysia)

11.20-11.45 Gain or loss - A story of STATs
Pamela Lee (HK)

11.45-12.00 The price to pay for universal BCG vaccination – could we avoid paying?
Panel Discussion

Free Paper 3
Rhinitis, Sinusitis and Immunotherapy
10.30-12.00 (Meeting Room 5) Lotus 11

Chairpersons: Orathai Piboonpocanun (Thailand), Mongkol Lao-Araya (Thailand)

10.30-10.45 Comparative metagenomic evaluation of nasal microbiota in infants with rhinitis in their first year of life – A Pilot Study
Gaik Chin Yap (Singapore)

10.45-11.00 Expression and mechanism of TLR2, TLR4 and NF-κB in the nasal mucosa of children with allergic rhinitis
Huasong Zeng (China)

11.00-11.15 Profound differences regarding T cell and IgG reactivity to house dust mite allergen molecules and peptides in sensitized and non-sensitized subjects
Huey-jy Huang (Austria)

11.15-11.30 Subcutaneous immunotherapy with house dust mite allergen extract-based Alutard SQ 510 induces an incomplete protective IgG response: A real life study
Azahara Rodriguez Dominguez (Austria)

11.30-11.45 Nasal mucosal brushing as a diagnostic method for house dust nasal allergy
Aneeza Hamizan (Australia)

11.45-12.00 CORRELATION BETWEEN TLR2 AND TLR4 WITH IL-5 ON CHRONIC RHINOSINUSITIS
Eryati Darwin (Indonesia)

Symposium #13 - Advocacy and Education in Allergy
12.00-13.30 Plenary Hall

Chairpersons: Ruby Pawankar (Japan) and Kanika Piromrat (Thailand)

12.00-12.20 Asthma and Allergy Program: Korean experience
Yoon-Seok Chang (South Korea)

12.20-12.40 National Allergy Strategy: Australian experience
Richard Loh (Australia)

12.40-13.00 Atopic dermatitis education and advocacy
Sooyoung Lee (South Korea)

13.00-13.20 Food allergy education to families and school environment
Pakit Vichyanond (Thailand)

13.20-13.30 Q&A

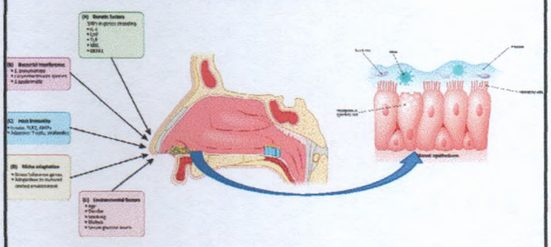
CORRELATION BETWEEN TLR2 AND TLR4 WITH IL-5 ON CHRONIC RHINOSINUSITIS

Eryati Darwin, Bestari Jaka Budiman and Dwitya Elvira
Faculty of Medicine Andalas University, Padang-Indonesia
October, 2018

Introduction

- Chronic rhinosinusitis (CRS) is an inflammatory disease of the sinonasal mucosa that persists for at least 12 weeks
- Associated with many factors that disrupt an immune function of the nasal mucosa
- The nose, paranasal sinuses, and associated lymphoid tissues play important roles in homeostasis and immunity, and CRS significantly impairs these normal functions.

The nasal mucosa



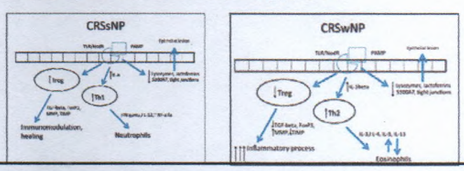
- The nose is the respiratory system's first line of defense.
- The surface of nasal cavities are lined by epithelium → the nasal mucous membrane
- Protect against inhaled pathogens : heats and humidifies 12,000 liters of air every day.

Chronic Rhinosinusitis (CRS)

- Inflammation
- Heterogeneous and multifactorial disease with unknown etiology
- Genetic and environmental factors include allergens, toxins, and microbial agents implicated in etiology of CRS.
- Resulting low quality of life, reduced workplace productivity, and serious medical treatment costs.

Classification of CRS

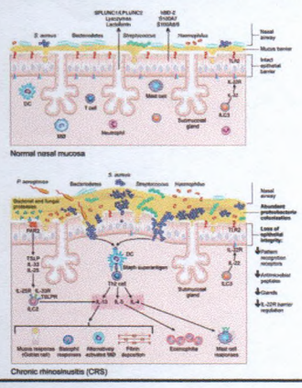
- Phenotype:
  - CRSwNP is characterized by the presence of polyps and an eosinophilic inflammatory infiltrate
  - CRSsNP is characterized by noneosinophilic inflammation associated with neutrophil accumulation, tissue remodeling, and fibrosis.
- Further subtype:
  - clinical criteria
  - severity
  - histopathologic features
  - variability of tissue markers: albumin, IgE, and IL-5



Etiology and pathogenesis of CSR

- fungal hypothesis
- superantigen hypothesis
- biofilm hypothesis
- microbiome hypothesis, which emphasize key environmental factors
- eicosanoid hypothesis
- immune barrier hypothesis, which describe specific host factors

A Model for changes in the nasal microbiome and mucosal immune response in CRSwNP

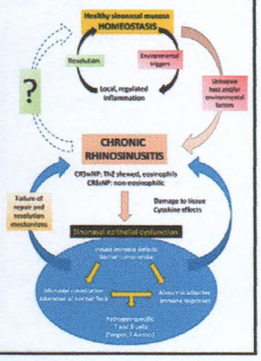


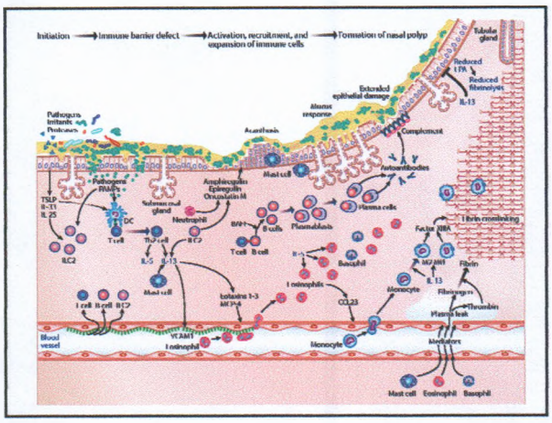
A Model for changes in the nasal microbiome and mucosal immune response in CRSwNP

- The changes that occur in the microbiome in patients with CRSwNP, including increased S. aureus abundance
- decreased Bacteriodetes and decreased diversity
- These changes, along with loss of epithelial integrity, decreased pattern recognition molecules, decreased mucosal glands and decreased antimicrobial peptide production in the nasal polyp and sinus tissue
- → can potentially provide an environment that promotes invasion of microorganisms across the mucosal barrier.
- Enterotoxins produced by S. aureus can act as superantigens and promote Th-2 inflammation, resulting in production of cytokines such as IL-13, IL-4 and IL-5 that further recruit and activate inflammatory cells such as eosinophils, mast cells, basophils and alternatively activated macrophages.
- Bacterial and fungal proteases can induce production of thymic stromal lymphopoietin (TSLP)

Pathogenesis of CRS

- Microbial (fungi, staphylococcal enterotoxin, and biofilms) have been implicated as inflammatory stimuli, along with airborne irritants and allergens
- Defects in innate immunity have gained increased attention as contributors to the chronic inflammatory state.
- A combination of host susceptibility and environmental exposure is widely believed to underlie CRS, although direct evidence is lacking.





Aim of Study

to define the role of TLRs and IL-5 on CRS

Methods

- Cross Sectional study
- Nasal tissues: obtained from 12 patients with CRS were diagnosed by European Position Paper on Rhinosinusitis
- Controls: obtained from nasal tissues of non-CRS patients which are conducted septoplasty or rhinoplasty
- Tissues: collect during surgery.
- Paraffin block stained with immunohistochemical methods, using Mab anti-TLR2, TLR4 and IL-5.
- Approved by Research Ethic Committee of Faculty of Medicine Andalas University

Results

Table 1: Characteristic of subjects

CHARACTERISTIC	CRS (n=12)	CONTROL (n=12)	p
Gender			
- Man	7 (58,3%)	5 (41,7%)	0,41
- Woman	5 (41,7%	7 (58,3%)	
Age	37,58±8,59	34±13,83	0,45
CRS and bacteriologis			
- CRS with polyp (CRSwNP)	7 (58,3%)		
Gram positive bacteria	4 (57%)		
Gram negative bacteria	3 (43%)		
- CRS without polyp (CRSsNP)	5 (41,7%)		
Gram positive bacteria	3 (60%)		
Gram negative bacteria	2 (40%)		

- Cho et al,2016: CRSsNP is more prevalent than chronic rhinosinusitis with nasal polyps CRSwNP
- The results on bacterial diversity in CRS are varied

Table 2 : Percentage of TLR2 ,TLR4 and IL-5 expression on CRS and Control groups

VARIABLE (mean±SD)	CRS (n=12)	CONTROL (n=12)	p
TLR2	84,08±1,45	79,91±1,87	0,549
TLR4	92,91±1,08	90,91±1,142	0,645
IL5	79,16±13,49	88,41±18,87	0,180

TLRs recognize / detect a broad range of human pathogens (pathogen-associated molecular pattern molecules)
TLR2 recognize gram-positive bacteria
TLR4 recognize exogenous molecules from gram-negative bacteria (e.g., LPS)
TLR2 and TLR4 is also involved in the recognition of endogenous molecules released by injured tissues and necrotic cells (damage-associated molecular pattern molecules)
Most of control group were septal deviation→ epithelial shadding→ air flow irritation
IL-5: Type 2 cytokines, control the inflammation in eosinophilic CRSwNP.

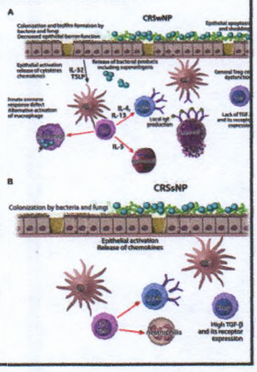
Table 2 :Correlation between TLR2 and TLR4 with IL-5

	IL-5	p
TLR2	R=0,487	0,109 (=weak)
TLR4	R=0,321	0,309 (=moderate)

- TLRs play a role in early innate immune response to invading pathogens
- TLR2 and TLR4 have clear specificity for different microbial ligands, the actual mechanism of TLR activation is still unclear.
- IL-5 stimulates B cell growth and increases immunoglobulin secretion. It is also a key mediator in eosinophil activation
- Zhang et al (2017):
- inflammatory signatures of CRS vary around the world, In Asia: less eosinophilic and more neutrophilic inflammation compared with Europe and North America.
- in the Western world about 80% of nasal polyps carry a type 2 signature, this might be between 20% and 60% in China and Korea or Thailand, respectively.

Pathomechanisms of CRS. A. CRSwNP

- TH2-type with general lack of regulatory T (Treg) cell function, IL-5 induces eosinophilia, and IL-4 and IL-13 induce local IgE production.
- Activated macrophage subset contributes to the inflammation.
- The activation of epithelium colonized by bacteria and fungi leads to release of proinflammatory chemokines and cytokines with increased thymic stromal lymphopoietin (TSLP) and IL-32 levels.
- Activated epithelial cells die, with apoptosis resulting in a compromised epithelial barrier. B. CRSsNP.
- Instead of a TH2-skewed T-cell response, a TH1 or a mixed TH0 response predominates, neutrophilia is often associated, and expression of TGF-β and its receptors is increased. DC, Dendritic cell.



Conclusion

- Chronic rhinosinusitis (CRS) is probably not caused by microorganisms, but more related to allergy

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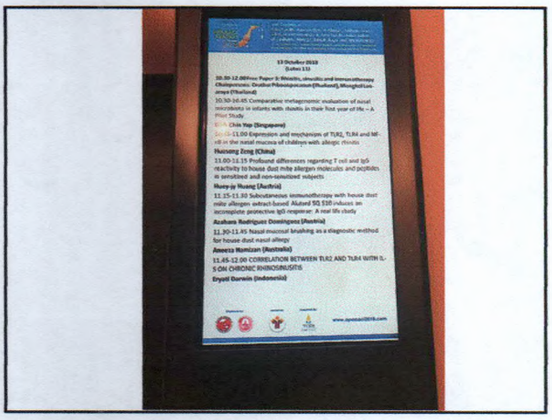
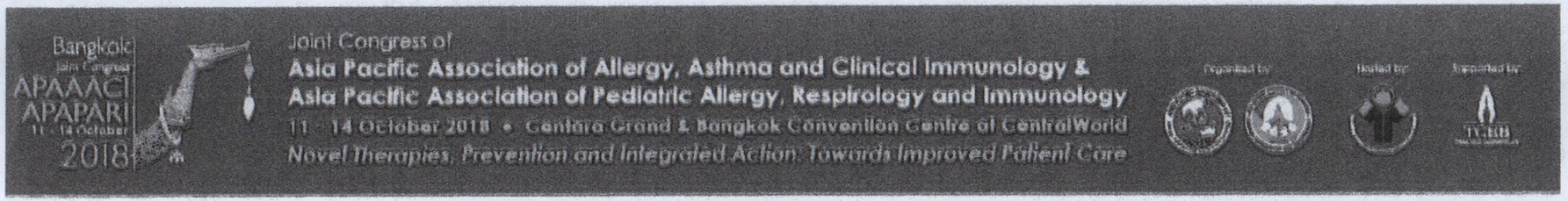
Thank you


13 October 2018
(Lobby 1/2)
Huosong Zeng (China)
Huey-Ju Huang (Austria)
Azahara Rodriguez Dominguez (Austria)
Ameera Mamzan (Australia)
Eryati Darwin (Indonesia)


Thank you



CERTIFICATE OF ORAL PRESENTATION

Presented to

Prof. Eryati Darwin

for the presented paper entitled

"CORRELATION BETWEEN TLR2 AND TLR4 WITH IL-5 ON CHRONIC RHINOSINUSITIS"

at the Joint Congress of
Asia Pacific Association of Allergy, Asthma and Clinical Immunology &
Asia Pacific Association of Pediatric Allergy, Respirology and Immunology
11 - 14 October 2018
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Prof. Kiat Ruxrungtham, MD
Chairperson, Local Organizing Committee

Prof. Pakit Vichyanond, MD
Chairperson, Scientific Committee
APAAACI & APAPARI 2018

APAAACI 30th ANNIVERSARY
2019 APAAACI INTERNATIONAL CONFERENCE
2019 CSA ANNUAL SCIENTIFIC MEETING

Memorable history, Glorious present, and Splendid future: Current to emerging therapies for better patient care.

PROGRAM

北京

5-7 SEPTEMBER, 2019
BEIJING, CHINA

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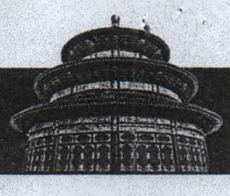
PROGRAM - APAAACI2019

5-Sept, 2019			CNCC 307A
09:00-17:00	APAAACI Allergy training school		
Chair: Su Duan (China)			
09:00-10:00	Asthma in young children	Hugo Van Bever	Singapore
10:00-11:00	Rhinitis	Yuan Zhang	China
11:00-12:00	Food allergy	Bee Wah Lee	Singapore
13:00-14:00	Diagnostic tools in chronic urticaria	Amir Latiff	Malaysia
14:00-15:00	Vaccines and allergies	Iris Rengganis	Indonesia
15:00-16:00	Immunotherapy	Jiu-Yao Wang	Taiwan, China
16:00-17:00	Anaphylaxis	Ruby Pawankar	Japan
			CNCC 306B
10:00-12:00	Molecular allergy workshop		
10:00-10:45	International consensus on molecular allergology	Ruby Pawankar	Japan
10:45-11:30	Importance of molecular allergology in diagnosis of allergic diseases	Jiu-Yao Wang	Taiwan, China
11:30-12:00	Free discussion on practical aspects		
			CNCC 311B
13:30-17:40	Junior member forum		
Chairs: Sze Yin Agnes Leung (HK, China), Jing Li (China)			
13:30-13:40	Opening remarks	Ruby Pawankar	Japan
13:40-14:10	Ins and Outs of conducting good clinical trials	David Fleischer	US
14:10-14:40	The path to precision medicine	Alessandro Fiocchi	Italy
14:40-15:10	Is asthma a western disease? Lessons from China: Exposure to environmental micro-organisms in the regulation in development of allergic asthma	Jing Li	China
15:10-15:30	Coffee break		
Chairs: Lei Cheng (China), Jie Shao (China)			
15:30-16:00	How to tackle allergic rhinitis in Asia?	Soumya Subhash	India
16:00-16:30	Children asthma action plan in China	Kunling Shen	China
16:30-16:40	Coffee break		
16:40-17:00	Epithelia cell derived cytokines: A new asthma endotype	Wei Tang	China
17:00-17:20	Insect venom allergy in China	Kai Guan	China
17:20-17:40	Increasing prevalence of allergic rhinitis in China	Yuan Zhang	China
			CNCC Ball room A
18:00-19:30	Welcome reception		



6-Sept, 2019			CNCC Great Hall B
08:00-08:15	Opening ceremony		
08:15-08:45	30th Anniversary APAAACI ceremony		
08:45-09:25	APAAACI Keynote lectures		
Chairs: Hee Bom Moon (Korea), Yoon-Seok Chang (Korea)			
08:45-09:05	Chronic rhinosinusitis with nasal polyps in Asia	Luo Zhang	China
09:05-09:25	Allergies in Asia pacific: a growing burden in a changing environment: call to action	Ruby Pawankar	Japan
09:25-10:25	EAAS Symposium		
Chairs: Motohiro Ebisawa (Japan) , Ho Joo Yoon (Korea), Lianglu Wang (China)			
09:25-09:45	Periostin, an emerging biomarker for allergic diseases	Kenji Izuhara	Japan
09:45-10:05	Severe asthma and asthma-COPD overlap syndrome: perceptions and real life	Sang Heon Kim	Korea
10:05-10:25	Chinese guidelines for the management of allergic rhinitis	Lei Cheng	China
10:25-10:40	Coffee break		
10:40-11:40	CSA Keynote Lectures (in Chinese)		
Chairs: Xueyan Wang (China), Yinshi Guo (China), Zheng Liu (China)			
10:40-11:00	Recurrent urticaria and anaphylaxis	Yin Jia	China
11:00-11:20	The clinical application and development of allergen molecular diagnosis	Lianglu Wang	China
11:20-11:40	Chronic nasal disease research in China	Luo Zhang	China
11:40-11:55	MSD Symposium (in Chinese)		
11:40-11:55	Research progress on chronic rhinitis in China	Luo Zhang	China
11:55-12:10	Thermo Fisher Symposium (in Chinese)		
Chair: Lianglu Wang (China)			
11:55-12:10	Accurate diagnosis, accurate disease management	Jie Shao	China
12:10-12:25	Xian Janssen symposium		
Chair: Luo Zhang (China)			
12:10-12:25	Tackle with the impact from environment to nasal inflammation —— current practice and emerging evidences	Lei Cheng	China

INTERNATIONAL CONGRESS OF ASIA PACIFIC ASSOCIATION OF ALLERGY, ASTHMA AND CLINICAL IMMUNOLOGY
ANNUAL MEETING OF CHINESE SOCIETY OF ALLERGY

5-7 SEPTEMBER, 2019
BEIJING, CHINA

Certificate of Attendance

This is to certify that

ERYATI DARWIN

as a Speaker in the

APAAACI 30th Anniversary
2019 Apaaaci International Conference
2019 CSA Annual Scientific Meeting

5-7 September, 2019 Beijing, China

Ruby Pawankar, MD, PhD
President, APAAACI
Co-Chair
APAAACI 2019 Joint CSA 2019

Luo Zhang, MD, PhD
Immediate Past President, CSA
Co-Chair
APAAACI 2019 Joint CSA 2019

Lianglu Wang
President, Chinese Society of Allergy
Co-Chair, LOC
APAAACI 2019 Joint CSA 2019

Xueyan Wang
Vice President, Chinese Society of Allergy
President, Beijing Society of Allergy
Co-Chair, LOC
APAAACI 2019 Joint CSA 2019