

出國報告(出國類別：開會)

參加2018 第十一屆國際自體免疫學術大會 心得報告

服務機關：國防醫學院小兒學科

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派赴國家：葡萄牙 里斯本

報告日期：一零七年六月三日

出國時間：一零七年五月十四日至五月二十二日

摘要：

此次參加第十一屆年會 International Congress on Autoimmunity，於 2018 年 5 月 16 日~20 日在葡萄牙 里斯本舉行，因自體免疫疾病原因的複雜與治療的難度有很多的挑戰，因此大會主席 Yehuda Shoenfeld 強調多方位的研究才能就其全貌就用期在 1980 年代就出的概念為:MOSAIC OF AUTOIMMUNITY 來進一步陳述，會中也有諸多學者甚至專題來研討:MOSAIC OF AUTOIMMUNITY ；另外自體免疫與腸道微生物群(Microbiome) (MICROBIOME IN AUTOIMMUNE DISEASES)之關聯性也是本次大會的另一重點，會議及 CME 課程內容主要是探討自體免疫疾病致病機轉的新發現與相關新的治療的新策略運用，以及目前新發展的免疫調節治療方式。在這五天的會議哩，大會依據不同領域及不同研究類別共分了四十一個研究主題(附件一)，提供給全世界自體免疫基礎臨床專家學者來提出其研究或研發成果。其中職在 5/18 提出電子壁報論文報告，並與知名的國外學者有良好的互動，因是電子壁報，可以隨時點閱，並有多人觀看並獲得許多肯定，更讓他們認識台灣研究的質與量，有助於基礎與臨床研究的結合，落實基礎研究及轉譯醫學，期能未來產學合作的進一步契機。在與國際知名學者良好的互動下，將可建立未來跨國合作的先機。

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本文

目的

參加會議的主要目的是希望了解現今世界自體免疫疾病研發的最新趨勢以及發展包括基礎的最新狀況與免疫學之新研究概況與臨床疾病之新的治療與診斷趨勢與方式；並與國際尖端研發人才接觸界以擴展國際視野，並建立未來國際交流契機。

另外針對現行國際上自體免疫臨床新的診斷方式與治療經驗的進一步了解，並將在回國後規劃未來我們在此方面的研發與新的研究方向，繼而開創出之我們的研究成果與特色。

(大會網址:

<https://autoimmunity.kenes.com/2018/congress-information/discover-lisbon#.WxaWskiFPcs>)

過程

國防醫學院醫學系小兒學科專任教師兼微生物免疫學科主任陳錫洲教授於 2018 年 5 月 16 日至 20 日在葡萄牙里斯本參加 11th International Congress on Autoimmunity 年會。

以下是在會議結束後撰寫的回國報告: 2018 年第 11 屆國際自體免疫大會(11th International Congress on Autoimmunity)在葡萄牙里斯本舉辦，里斯本是葡萄牙是的首都和最大都市，建成可以回溯到第十二世紀，與倫敦、巴黎、羅馬等同為西歐歷史最悠久的城市。。此年會參加人數多達二千人五百以上，來自超過七十個國家地區與會，五天的議程超過五百場的大小演講與海報論文報告，盛況空前。

大會於五月十六日正式開始，首先課程第一天上午有免疫學課程 (Academy Autoimmunity-update course of autoimmunity)其一天也有 Basic and translational Immunology for autoimmunologist 及 上午會議行程主要是探討疫苗佐劑與自體免疫知相關研究，如下圖:



另外 HPV 疫苗似乎有較高致死之風險，但此結果仍值得更多研究及分析，否則可能有對

Increased risk of death in association with the HPV vaccination

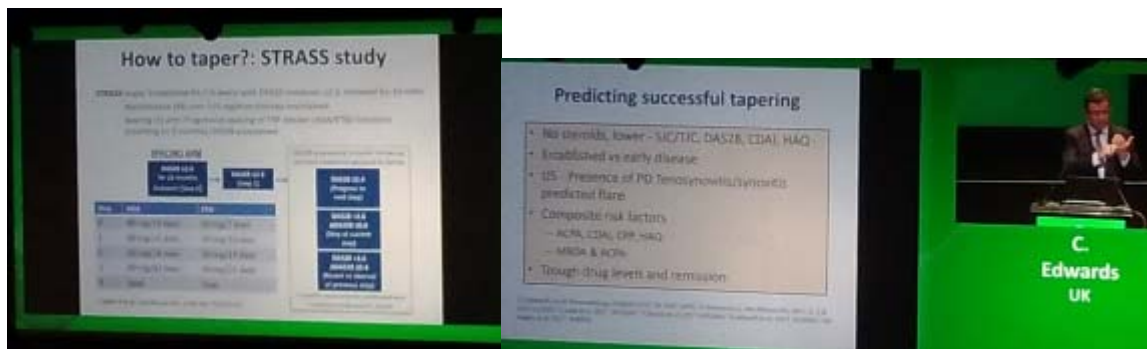
Table 2. 2004 Common and Other Selected HPV-Adverse Events Following Introduction in the United States, Reported to VAERS June 1, 2006, Through December 31, 2010

AEFI	Serious Adverse Events	Non-serious Events	HPV Status	Total No.	Reporting Rate ^a
Myocardial infarction	37 (5)	540 (86)	122 (2)	700	0.5
Stroke	41 (5)	1702 (86)	128 (2)	1871	0.5
Thrombosis	56 (8)	1427 (86)	149 (2)	1632	0.5
Nausea	119 (1)	3942 (86)	363 (2)	4664	0.5
Fatigue	193 (1)	3778 (86)	605 (2)	4576	0.5
Hypersensitivity reaction ^b	47 (5)	379 (86)	302 (2)	728	0.5
Myositis	22 (4)	252 (86)	301 (2)	575	0.5
Vertigo	33 (4)	17 (1)	15 (0)	65	0.5
Autoimmune disorder	18 (1)	22 (4)	31 (0)	71	0.5
Other adverse events	23 (1)	11 (2)	11 (0)	45	0.5
Unknown	8 (1)	26 (1)	10 (0)	44	0.5
Total	49 (1)	3778 (86)	605 (2)	4432	0.5
Myocardial infarction	37 (5)	540 (86)	122 (2)	700	0.5
Stroke	41 (5)	1702 (86)	128 (2)	1871	0.5
Thrombosis	56 (8)	1427 (86)	149 (2)	1632	0.5
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JAMA, August 19, 2014

街種者摻生心理之壓力。

五月十七日上午有 Plenary session:有德國 Schuppan 教授 演講 The gut and Autoimmunity 涵蓋 Microbiota 參與之免疫調節機轉與未來運用之可行性，以及英國 Edwards 教授演講:How to withdraw biological therapy 提到如何降階使用生物性治療以減少併發症之產生 (如下圖):

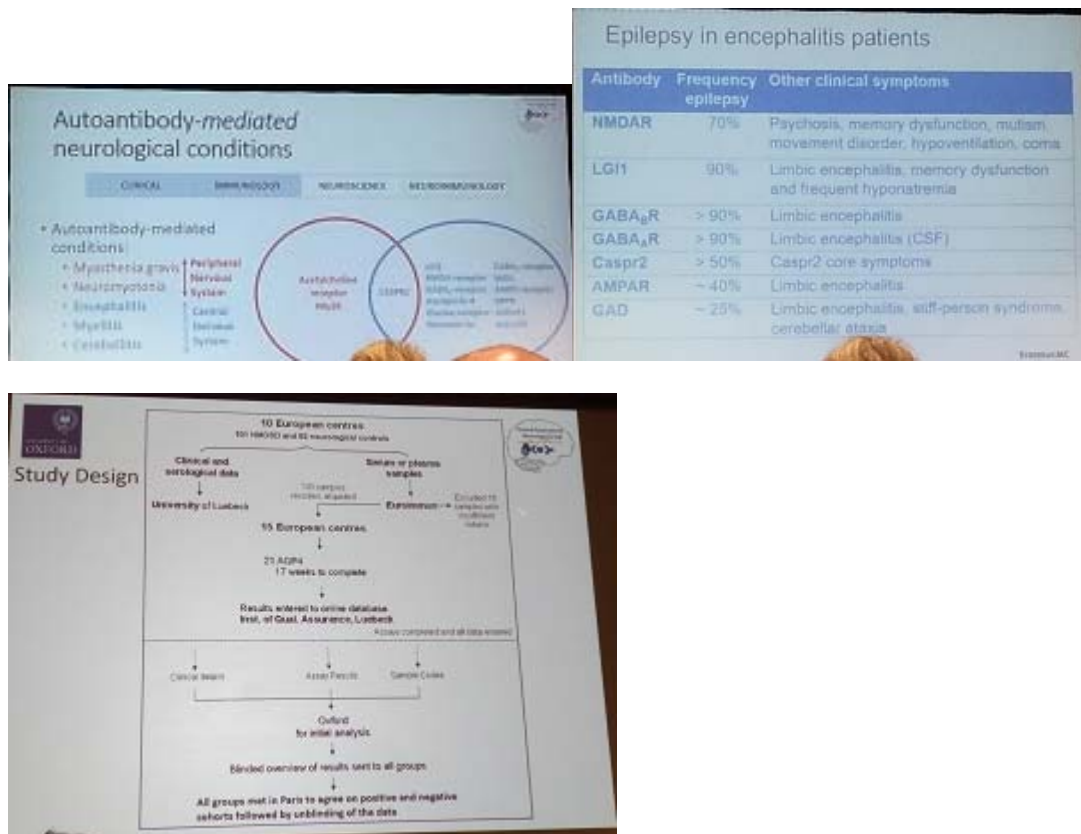


接著有 Autoimmune Autonomic Nerve Diseases and Channelopathies: immunotherapy with IVIG and beyond: (如下圖)

首先由 英國學者 Prof Irani 介紹” autoimmune Channelopathies, The role of Autoantibodies” ；探討自體免疫神經系統與自體抗體的致病相關機轉及其日漸重要的腳色，著重在 NMDAR autoimmune encephalitis 之診斷依據與建議之治療方式，能知道這疾病之特性，盡早適當免疫調節治療。接著由英國的 Leite 教授由臨床經驗與免疫治療的方向討論其在 Autoimmune channelopathies 的運用。

之後由荷蘭學者 Prof. Titeler 論述如何執行與設計實驗並評估成效在 autoimmune epilepsy

在 最後由 Prof Schofield (USA)探討免疫球蛋白治療的安全性及功效在 Refractory autoimmune dysautonomias 的角色 ，整個議程設計，深入淺出且相當新的研究；而其他場次探討自體抗體與疾病的診斷及臨床表現的發現，也非常精彩。

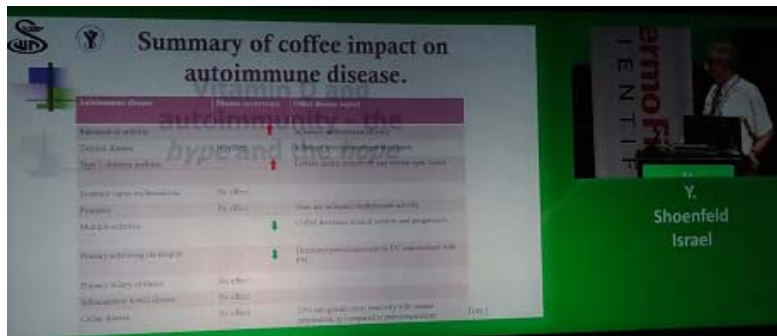


下午場次主題為 Neuronal Autoimmune Diseases; 首先由英國學者 Prof Waters 介紹 Demyelinating disease 相關的自體免疫疾病；另外俄羅斯學者 Korneva 發表的基礎與臨床之研究: Brain Cells Reactions to Antigens and Possible Afferent Pathways from immune system to CNS 非常新穎精彩，獲益良多。

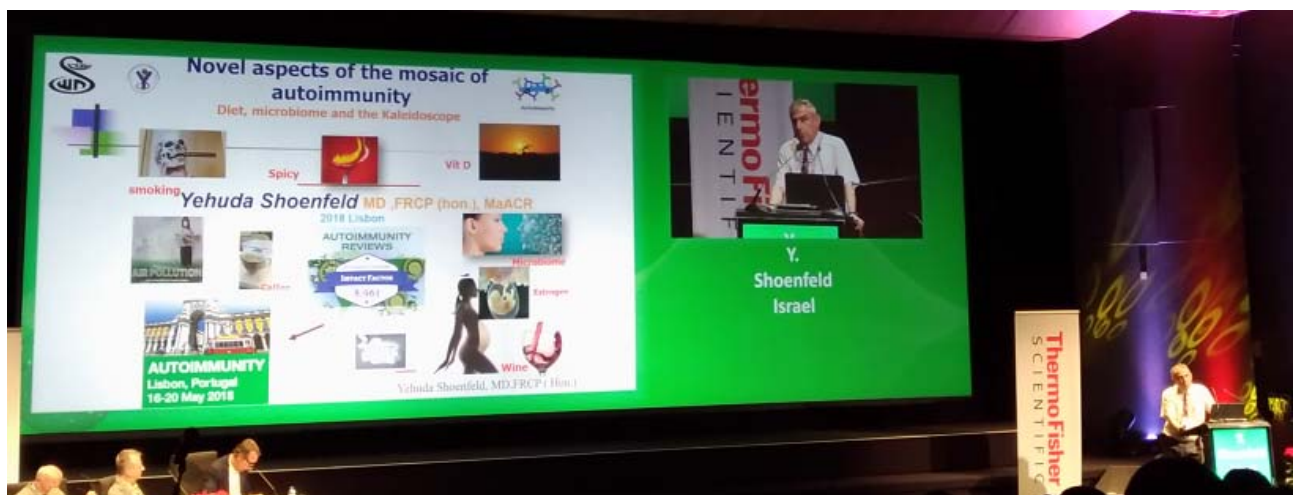
另外由瑞典學者 Olofsson 發展出 Autoantibody profiling of Amyotrophic Lateral sclerosis plasma，在 ALS 疾病 serum autoantibodies 的偵測也已研發出來。

緊接著第三天在 5 月 18 日上午場的大會 Plenary Session 演講首先德國 Prof Kronke 獲得大會研究獎並發表演講主題為: regulatory Checkpoints Controlling the transition between Autoimmunity and Inflammation，為多發性硬化症的基礎與臨床的最新發展，依序由以色列 D. Karussis (Israel)教授介紹 DISEASE OF THE DAY: MULTIPLE SCLEROSIS 包含目前最新分類，免疫治病機制如 IL-21, mTOR 之參與，提供未來診治研發之基礎 ；

接著由大會主席以色列 Prof Shoefled 演講 Mosaic of autoimmunity (1980-2020): etiology, Nutrition, Novel Therapy and Prediction. 從 Mosaic 概念在巴零年代提出，發展至今明確瞭解了自體免疫成因之複雜度，涵蓋了營養、生活習慣、感染、基因背景等，至今成為顯學，大會主席引經據典微微到來，甚是精采。(如下圖)



大會主席對自體免疫疾病在二十年前提出 Mosaic of autoimmunity 之概念，現在並受到討論，也給予肯定。



下午有精彩海報論文:可以了解世界各國近來的研究趨勢(如下圖左,德國馬德堡大學學者之論文海報)利用免疫球蛋白合併鋅劑可以明顯改善自體免疫小鼠腦脊髓炎的嚴重度,次為相對較為新穎之概念,為其免疫機轉並為深刻探討,應是我們未來可以切入研究之處(如下圖):

Combined treatment with intravenous immunoglobulin (IVIg) and zinc aspartate ameliorates experimental autoimmune encephalomyelitis (EAE)

Dirk Reinhold¹, Diana Straubel¹, Karina Guttek¹, Annegret Reinhold¹, and Kurt Grüngreiff¹

¹Institute of Molecular and Clinical Immunology, Otto-von-Guericke-University Magdeburg, Germany,
²Hahneemannstrasse 14, Magdeburg, Germany

Summary

Intravenous immunoglobulin (IVIg) application is widely used in replacement therapy of primary and secondary immunodeficiency disorders and in approved autoimmune indications. In addition, IVIg is in clinical testing or used off-label for other autoimmune indications.

The basic element zinc is shown to play a regulatory role in the maintenance of immune functions. Based on several experimental data, a therapeutic zinc supplementation is under consideration as one possible option to treat T cell-mediated autoimmunity.

The aim of the present study was to investigate the influence of IVIg (Octagam[®], 18 mg/kg), zinc aspartate (Zincovit[®], 38 mg/kg) and the combined application of both preparations in the animal model of Multiple Sclerosis, the Experimental Autoimmune Encephalomyelitis (EAE). Multiple Sclerosis is shown to be an inflammatory autoimmune disorder with a clear T cell-mediated immune pathogenesis.

We observed that a therapeutic intraperitoneal application of zinc aspartate, given from day 15 to day 18, significantly reduced clinical signs during the relapsing remitting phase of EAE in R.A.1 mice. In contrast, the IVIg preparation was capable of diminishing the severity of EAE only after prophylactic application from day 1 to day 15, but not in a therapeutic manner.

Importantly, zinc aspartate increased the therapeutic effect of IVIg administration in the course of the EAE disease. The combined application of both IVIg and zinc aspartate prophylactically significantly reduced the severity of the disease during the acute and the relapsing remitting phase of the EAE.

Taken together, the data suggest that combined administration of IVIg and zinc aspartate may have beneficial effects on T cell-mediated autoimmune diseases, like MS. Further prospective studies should verify the possibility of controlled immunosuppressive IVIg and zinc therapies for such diseases.

Introduction

Intravenous immunoglobulins (IVIg)

- purified IgG from 1,000 - 10,000 blood donors
- extracted IgG from blood plasma
- used for IgG replacement in disease (IgG) deficiencies
- used for therapy in different autoimmune diseases
- several mechanisms of action are described
- IVIg preparations contain high amounts of bound TGF- β 1

Zinc

- is an essential trace element, required for different physiological processes
- cofactor of >300 enzymes
- regulation of apoptosis and protection against free radicals
- special importance for the immune system (affects innate and adaptive immunity)
- zinc deficiency leads to immune dysfunction and secondary immunodeficiency
- low-dose zinc supplementation induces immunostimulation
- high-dose zinc supplementation leads to immunosuppression [2,3]

Multiple Sclerosis (MS)

- most frequent demyelinating disease of the CNS; prevalence: 0.5%
- mean age of onset: 28 - 33 years
- symptoms: parietal, altered sensation, cognitive and emotional disturbances
- CNS infiltration of immune cells; T cell-mediated autoimmune pathogenesis
- autoimmune target: myelin oligodendrocytes
- animal model: experimental autoimmune encephalomyelitis (EAE)

Aim

The aim of the present study was to investigate the influence of IVIg (Octagam[®], 18 mg/kg), zinc aspartate (Zincovit[®], 38 mg/kg) or the combined application of both preparations in the animal model of Multiple Sclerosis, the Experimental Autoimmune Encephalomyelitis (EAE).

Methods

- EAE was induced in female SJL/J mice with proteolipin protein (PLP) peptide PLP_{(139-151)}}
- mice were treated with IVIg (Octagam[®], 18 mg/kg), zinc aspartate (Zincovit[®], 38 mg/kg) or both preparations in a therapeutic manner between day 17 and 18 or day 11 and 18
- mice were examined daily (EAE score)
- for histological analysis mice were sacrificed at day 28 post-immunization. Spinal cords were removed, fixed in 4% paraformaldehyde, embedded in paraffin and stained with hematoxylin and eosin

Results

IVIg reduces clinical signs of EAE only in a prophylactic manner

$p < 0.05$, $* p < 0.05$

Combined, therapeutic application of IVIg and zinc aspartate reduces clinical signs of EAE

$p < 0.05$, $* p < 0.05$

IVIg treatment day 11-18, $p < 0.05$, $* p < 0.05$

Zinc aspartate treatment day 11-18, $p < 0.05$, $* p < 0.05$

IVIg treatment day 17-18, $p < 0.05$, $* p < 0.05$

Zinc aspartate treatment day 17-18, $p < 0.05$, $* p < 0.05$

Histological analysis confirms clinical data

$p < 0.05$, $* p < 0.05$, histological analysis of spinal cord sections at day 28

Conclusion

- IVIg reduces the severity of the EAE only in a prophylactic, but not in a therapeutic manner
- Therapeutic application of zinc aspartate, given from day 11 to day 18, significantly reduces clinical signs of EAE
- Combined application of both IVIg and zinc aspartate significantly reduces the severity of the disease during the acute and the relapsing remitting phase of the EAE

Thus, combined administration of IVIg and zinc aspartate may have beneficial effects on T cell-mediated autoimmune diseases, like MS. Further prospective studies should verify the possibility of controlled immunosuppressive IVIg and zinc therapies for such diseases.

References:

- 1) J. Koenig et al., *Lancet* 377, 704-05 (2010)
- 2) S. Sauer et al., *Bioessays* 35, 508-19 (2013)
- 3) C. Schuster et al., *Bioessays* 37, 249-62 (2015)

本人則發表電子海報論文(如下圖)，提出由 DC 與上毅公司合作研發之一剛獲得專利的棉毅調節單株抗體，除了可以緩解自體免疫小鼠腦脊髓炎的疾病嚴重度亦提出，對發炎性細胞素有免顯抑制作用，獲得國外學者之肯定與相當迴響:

響:



11th International Congress on

Autoimmunity

Lisbon, Portugal, 16-20 May 2018



Department of Paediatrics, Tri-Service General Hospital,
Department of Microbiology and Immunology, National Defense Medical College, Taipei, Taiwan

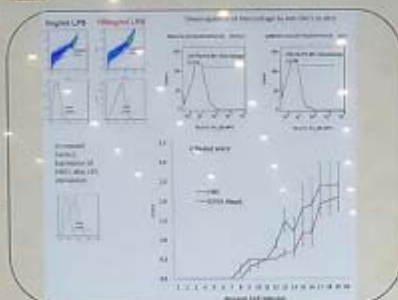


Figure 1: Surface ENO1 overexpression after LPS stimulation and Anti-ENO1 down regulation on macrophage and ameliorate disease of EAE in C57BL/6 mice

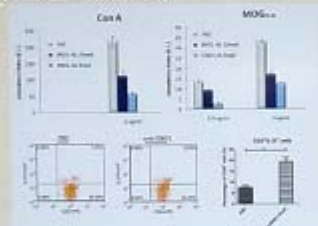


Figure 2: Antigen-Specific stimulation and upregulation of Tr1 (CD4+IL10+) cell in anti-ENO1 treated EAE group

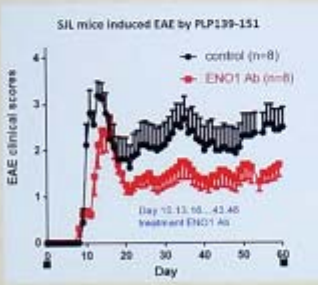


Figure 3: Antigen-Specific stimulation and upregulation of Tr1 (CD4+IL10+) cell in anti-ENO1 treated EAE group

ACKNOWLEDGEMENTS

ENO1 blocking antibody (ENO1 Ab) generated by DCB Co. Tw and ENO1 Ab was transferred technically to HuiLife Biotechnology Co and renamed as HuiLife 001 which is a kind gift and supported by HuiLife Co in this study. We thanks Dr Gu-Jun Lin and PhD graduate students Jing-Wen Chen, Chung-Hsi Jien, and Master student Chia-Ling Hsieh for their efforts on this project.

REFERENCES

Alta-ecolase specific antibodies and method of use in immune disease. Patent: WO 2014/095461 A1, US 9,382,331 B2.
Regulation of plasminogen receptor expression on human macrophages and monocyte cell lines. *The Journal of cell biology* 1990; 111(4):1673-1680.
Ecolase and arctin are novel myosin autoantigens in multiple sclerosis. *Journal of clinical immunology* 2007; 27(4):384-395.
Metalloin ectolase (arctin) is a novel autoantigen in multiple sclerosis: its expression and expression characteristics in active inflammation

ABSTRACT

- Ecolase-II(ENO1) as a plasminogen receptor has been detected on the surface of hematopoietic cells such as monocytes, T cells and B cells, neuronal cells, and endothelial cells.
- Currently available information on plasminogen receptors, particularly their mechanisms of action and their roles in inflammatory, autoimmune and malignant disease had been addressed.
- Serum ENO1 levels were higher in neurologic disease may be of use in predicting outcome in patients with acute neurological disease e.g. hypoxia, ischemia, traumatic brain injuries and Alzheimer's disease (AD).
- However, the exact immunopathologic role of ENO1 had not been proved in multiple sclerosis (MS).

METHODS

Mice: All available animals are proved by IACUC(NDMC, Taiwan). We had imported SJL/J mice from Jackson Lab and maintain in our animal center. EAE induction in C57BL/6 mice with MOG₃₅₋₅₅ and in SJL/J mice with PLP₁₃₉₋₁₅₁.

Flow cytometry analysis: Prepared splenocytes (1×10^6 cells) in 0.1 ml of PBS were incubated on ice and stained with the following marker-specific antibodies (0.5 μ g of antibody/ 10^6 cells) and intracellular cytokine detection was also done.

Antigen-specific proliferation: Splenocyte cell suspensions were isolated from MOG₃₅₋₅₅-immunized mice treated with or without ENO1 Ab.

Real-time PCR: The expression of mRNA for candidate cytokines and, was normalized to that of HPRT.

RESULTS

- We first proved that over expression of ENO1 from cytoplasm to cell surface of RAW247 mice phage cell line by LPS stimulation (Fig 1)
- We demonstrated that ENO1 blocking antibody (ENO1 Ab) repressed macrophage activation in vitro (Fig 1)
- From in vivo study, we examined ENO1 Ab ameliorated disease severity of MOG-EAE (Fig 1)
- We disclosed less activation of splenocyte from ENO1 Ab treated mice either ConA stimulation or antigen-specific stimulation of EAE (Fig 2)
- Moreover, Tr1 (CD4+IL10+) cells are significantly increased in splenocytes of ENO1 Ab treated mice of EAE (Fig 2)
- We test further in relapse-remission experimental model of multiple sclerosis with SJL mice and we revealed ENO1 Ab preserved long term effects of disease amelioration in EAE via SJL-PLP model (Fig 3)

CONCLUSIONS

- We revealed the therapeutic role of ENO1 Ab in either MOG-EAE or PLP-EAE respectively.
- And only ENO1 Ab group showed significant upregulation of Tr1 (CD4+IL10+ T cells) and only mild elevation of Treg (unpublished data).
- We thus also provide the theoretical basis of ENO1 Ab

海報現場(如下圖):

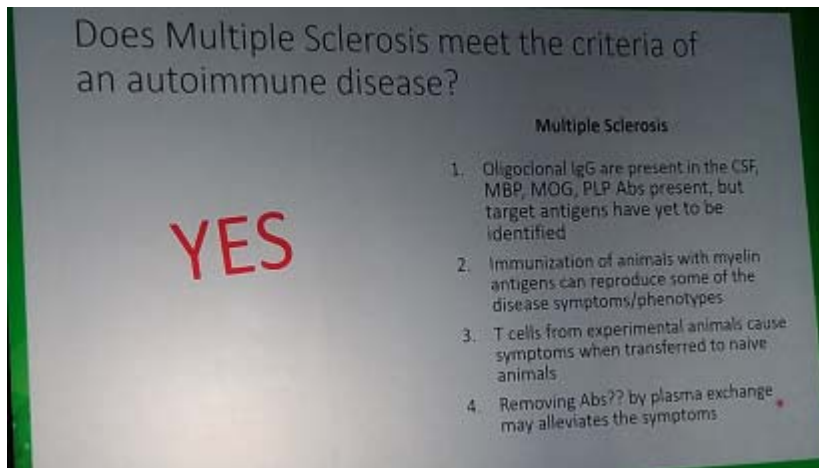
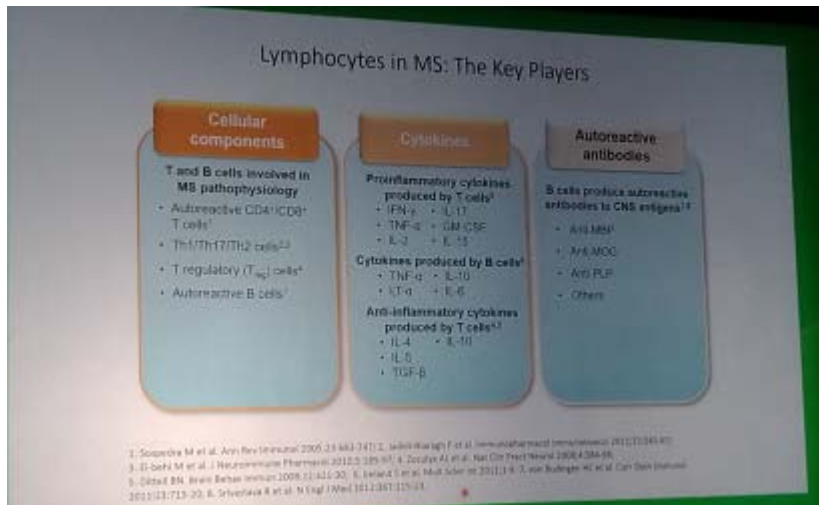


在 5 月 19 日為第四天的上午議程主要為多發性硬化症的研究，先由以色列 Achiron 教授從臨床症狀到基礎研究的介紹(如下圖)，

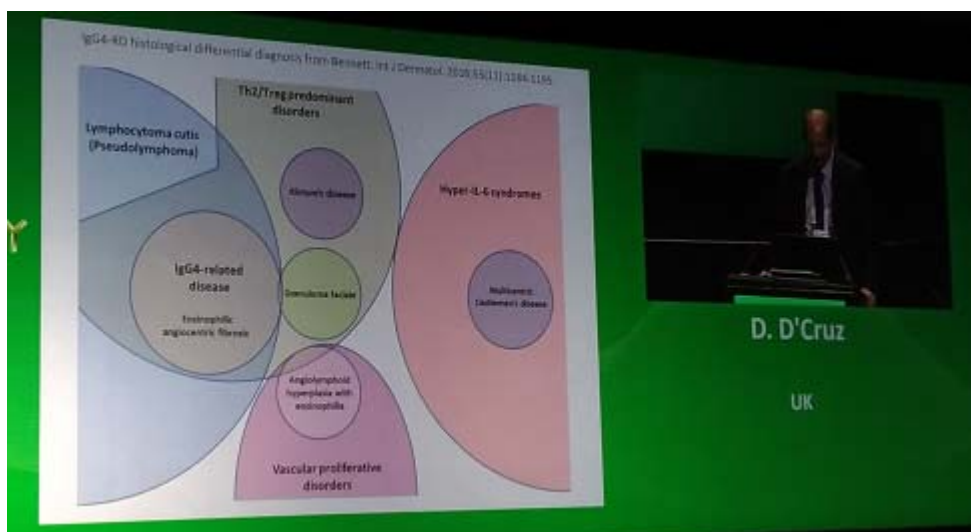


並經由反覆辯證與佐證來認，如免疫系招參與包括 T 淋巴細胞與 B 淋巴細胞；細胞素

(TNF-a, IL-17, GM-CSF)與疾病嚴重度之關係及自體抗原或自體抗體在病人身上可以偵測 (如 MBP、MOG、PLP 等)，因此定多發性硬化症為自體免疫系統之疾病(如下圖)



英國學者 Cruz 提到 Ig4 related diseases 參與 Th2/Treg 之調控 涵蓋許多風溼性疾疾病及血管發炎等免疫系統之疾病是相當新的概念，(見下圖)



最後由以色列學者 Dimitrios Karussis 教授發表 Immune Constitution Therapy in multiple sclerosis，治療經驗相當豐富與卓著，



包括幹細胞移植的最新進展及其經驗，值得學習。

此外荷蘭學者 Prof. Maarten Titulaer, MD, PhD (Erasmus University Medical Center)，在自體免疫腦炎的研究頗為深入且其實驗室已經發展並開發許多自體免疫腦炎的抗體發展包含已經確認之自體抗體與自體腦炎及副腫瘤症候群之關聯性已經有很好之成果，此在台灣雖然有許多醫學中心研究，但其實驗室整合許多歐洲國家的資料形成大數據之模式，可值得台灣借鏡。(見下圖)

	Well characterized	Autoimmune encephalitis
Antigen target	Intra-neuronal	Neuronal cell membrane or synaptic proteins
Common Antibodies	anti-HU anti-Yo anti-Ri anti-Ampiphysin anti-MA2 and 1 anti-CV2	anti-NMDAR anti-AMPA anti-LGI1 anti-CASPR2
Pathogenic mechanism	-Mediated by Cytotoxic T-cells. -Antibodies are thought to be markers	Antibodies have a direct pathogenic effect
Laboratory	Serum and CSF	Serum and CSF
Associated tumor	High association. SCLC	Less association with cancer

五月二十日為第五天也是大會最後一天議程，內容亦相當豐富包括；

聖彼得堡大學之自體免疫論壇講座，總共十二個專題，針對人文與科學的連貫演講，包括介紹聖彼得堡大學傑出科學家，該大學並有九位諾貝爾得主，其他甚至有大學部學生參與研究並上台口頭發表論文成果，值得我們生醫與醫學院大學生來借鏡。大會最後由大會主席做 closing remark 並期待下次在希臘雅典，第 12 屆國際自體免疫大會二年後能

見到國際學者更新穎與更有效之診治在人類自體免疫疾病上，就畫下圓滿句點。

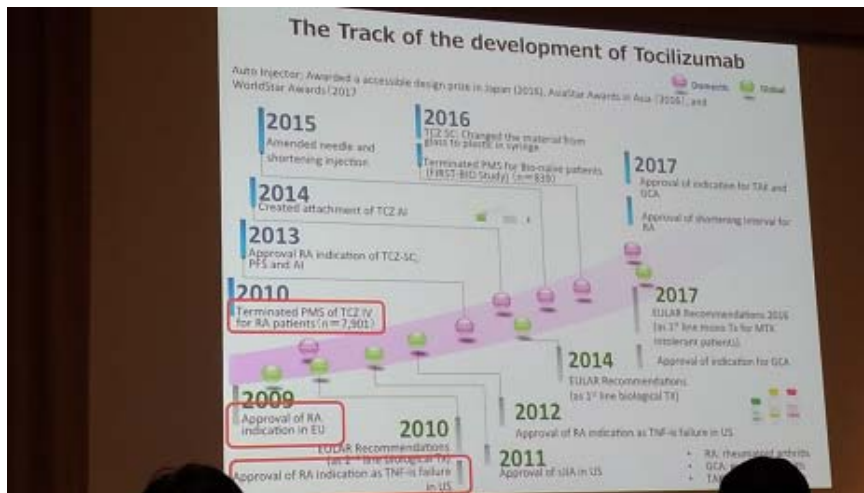


本人在參加聖彼得堡大學之自體免疫論壇講座，並認識聖彼得堡大學免疫學大師 Prof Churilov (如下圖一)，並合照留影甚感榮幸；



心得

本次會議的國際學者，還蓋層面相當廣、層次亦相當高深，多能從基礎的分子免疫機轉研究的探討與實驗室的新的診斷方式的開發然後運用到臨床的發展皆有其階段與步驟的鋪陳，呈現各國甚至跨國的研發成果，如 tocilizumab(It is a humanized monoclonal antibody against IL-6R)開發歷程及之後臨床運用到風濕性關節炎甚至幼年型風濕性關節炎等，之後推展到國際大規模的認證，足為借鏡 (如下圖)；



大會主席(Professor Yehuda Shoenfeld)演講提到的 the mosaic of autoimmunity 更是種新的概念如環境、感染、基因、賀爾蒙、免疫缺損等，皆有部分參與發生自體免疫疾病的相關因素，也讓我們對自體免疫疾病有了更進一步之了解，也回顧從 1989 年首先提出此概念，至今已獲得大家的肯定及認證。大會代表性的國際知名雜誌 Autoimmunity，近幾年，其論文影響力節節上升。

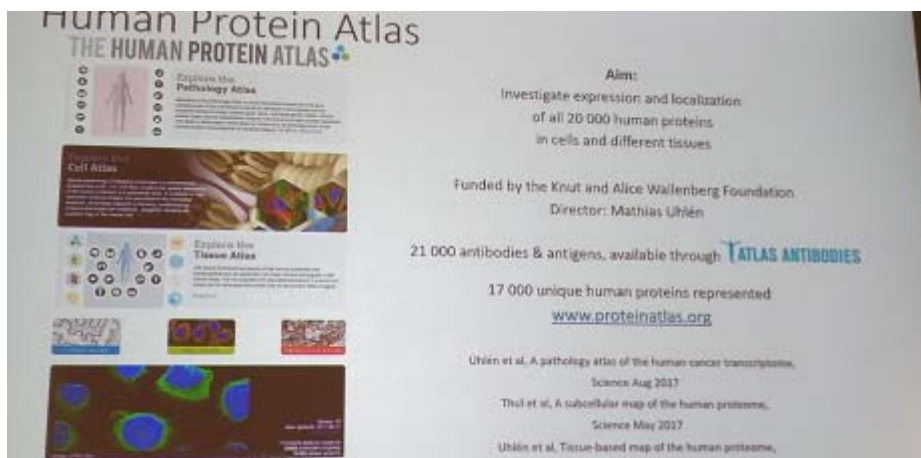
在此會議中，全球來自不同領域的專家學者特別是許多歐美知名國際學者的參與，除了有精采的演講另外還有口頭報告論文及 e-poster 與豐富的壁報論文展示，提出相當有建設性的意見，讓職獲益良多，不虛此行。

在這五天緊湊的議程，除了擴展國際視野更認識許多國際學者有人，對目前研究的新進展更感到國際研發競爭的激烈，藉此參與此會希望能對未來研究有更新的突破與發展。

特別要感謝科部補助，使申請人有此一難得的機會前往葡萄牙里斯本參與此重要的國際會議；藉由參與此次會議，能對於現今免疫生技、新型診斷工具的開發與治療的進展，有進一步的了解，包括許多因免疫製劑如免疫球蛋白、許多新式的單免疫調節單株抗體的成功研發，藉由了解免疫調節機轉進而證實在治療自體疾病的療效，讓人瞭解研究之路的漫長，與其成果對造福人類健康影響之深遠。

建議事項:

目前台灣在自體免疫疾病研究，轉譯(translation medicine)醫學部分，已經逐漸在發展，但成果仍有限。這次國際自體免疫學大會，發現亞洲參與的人數有增加特別是日本，可見這個國際會議的影響力。另外本次大會對疾病的分類有許多學者有特別的見解，如 Ig4 related disease, 甚至我們了解一般古典解剖學以器官來分門別類，這次有學者提及 Human protein atlas 之分類 (如下圖)，屬於大膽且新穎的學說甚至可能是未來的重要學門，值得持續關注



此外大會對各種不同免疫疾病的相關機轉，本次亦加入環境與基因及營養等的影響，確實更為宏觀。參與之學者不論臨床與基礎研究學者皆相當多，且許多皆為結合兩者的重要成果與創新發現，發特別歐美之間合作之頻繁且跨國研究風氣相當風行，這是值得學習的研究規劃與前瞻思維。

附件一：投稿摘要的研究主題與領域：

1. ANTIPHOSPHOLIPID SYNDROME TOWARDS THE THIRD DECADE
2. CANCER AND AUTOIMMUNITY
3. CELIAC DISEASE AND AUTOIMMUNITY
4. CENTRAL NERVOUS SYSTEM AND AUTOIMMUNITY: EYE, SMELL AND PSYCHIATRY
5. CITRULLINATION AND AUTOIMMUNITY: ANTI-CCP
6. COMPLEMENT IN AUTOIMMUNITY
7. DIAGNOSTIC MEASUREMENTS IN AUTOIMMUNITY
8. ENVIRONMENT, VACCINATION, ADJUVANTS (ASIA SYNDROME), AND AUTOIMMUNITY
9. EXPERIMENTAL AUTOIMMUNE MODELS
10. GASTRO INTESTINAL AUTOIMMUNITY
11. GENETICS AND EPIGENETICS IN AUTOIMMUNITY
12. HELMINTH EGGS AND MODULATION OF AUTOIMMUNE DISEASES (HYGIENE THEORY)
13. HEMATOLOGICAL DISEASE AND AUTOIMMUNITY
14. IL17, TH17 AND AUTOIMMUNITY
15. IMMUNOMODULATION

16. INFECTION AND AUTOIMMUNITY:MICROBIOME, INFECTOME AND INTERACTOME
17. INNATE AUTOIMMUNITY, TLRs AND AUTOIMMUNITY
18. IVIG AND NATURAL AUTOANTIBODIES
19. LIVER AND AUTOIMMUNITY
20. MECHANISM AND ETIOLOGY OF AUTOIMMUNE DISEASES
21. MIND-BODY INTERACTION IN AUTOIMMUNITY
22. MULTIPLEX DIAGNOSTIC TECHNIQUES AND CHIPS
23. NEW CYTOKINES AND ANTI-CYTOKINES
24. NOVEL ASPECTS OF THERAPIES – GLUCOCORTICOIDs, BIOSIMILARs, CANNABIS, MUSIC
25. NOVEL THERAPEUTIC TARGETS IN AUTOIMMUNITY
26. PEPTIDES AND AUTOIMMUNE DISEASES:DIAGNOSTICS AND THERAPY
27. PRECISION (PERSONALIZED) MEDICINE IN AUTOIMMUNITY
28. POLYMYOSITIS, MYASTHENIA GRAVIS AND MULTIPLE SCLEROSIS
29. PREDICTION, MONITORING AND PREVENTION
30. PREGNANCY, SEX HORMONES AND AUTOIMMUNITY
31. PROTECTIVE ANTIBODIES
32. PULMONARY ARTERIAL HYPERTENSION AND SYSTEMIC SCLEROSIS
33. RITUXIMAB AND B-CELL DEPLETION THERAPY

34. SLE, SJÖGREN'S DISEASE
35. STANDARDIZATION OF DIAGNOSTICS
36. STEM CELL THERAPY IN AUTOIMMUNE DISEASES
37. T CELLS AND BREGULATORY CELLS (TREG, BREG) - TOLERANCE
38. THYROID AUTOIMMUNITY
39. TRANSLATIONAL IMMUNOLOGY:FROM BENCH TO THE PATIENT
40. TYPE 1 DIABETES MELLITUS
41. VASCULITIDES, HORMONES AND AUTOIMMUNITY