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SCIENTIFIC PROGRAM & E-POSTER DISCUSSIONS

ACKNOWLEDGEMENTS & INDUSTRY SUPPORT

WELCOME MESSAGE

Dear Friends,

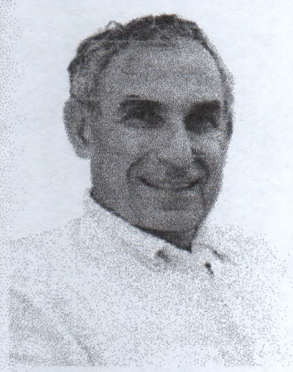
The International Congress on Autoimmunity has reached a historical moment: in April 2016 more than 2000 of the world's autoimmunologists have gathered for the 10th time to exchange knowledge about the more than 80 autoimmune diseases.

The meeting point this time is the artistic city of Leipzig, Germany, known for its long tradition in trade fairs and its compelling selection of museums, musical events and other cultural offerings.

Our loyal participants are already familiar with the high level of medical science that awaits them at the International Congresses on Autoimmunity and our newcomers will be impressed by the diversity of excellent sessions offered on a variety of topics, ranging from basic research to novel diagnostic and treatment methods of autoimmune diseases. This year's Congress introduces a variety of hot subjects for the first time: from spicy food and cannabis to obesity, smoking, the microbiome, novel peptides and revolutionary therapies.

The International Congress on Autoimmunity is the biggest multidisciplinary congress that discusses all aspects of the related diseases under one roof, offering courses and lectures by some of the world's most distinguished experts. At the same time, the Congress prides itself on providing a stage for young upcoming talents to present their research to a first-rate audience.

Join us and enjoy the inspiring atmosphere of medical science among old and new colleagues; share, learn and network to build the future of autoimmunology at the 10th International Congress on Autoimmunity!



Sincerely,

Yehuda Shoenfeld, MD, FRCP, MaACR
Congress President

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Serum level of Interleukin-17 (IL-17) and transforming Growth Factor-β (TGF-β) on Grave's disease patients

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Abstract

Grave's disease is an organ-specific autoimmune disease, characterized by excessive autoantibody due to breakdown in tolerance to thyroid-specific autoantigens. The impairment occurs in adaptive immune response, particularly the imbalance of Th1, Th2, and Th17 cells. Th17, a novel class of CD4+ T cells that secrete IL-17, were differentiated from naïve T cells by cytokines including TGF-β . To determine the role of IL-17 and TGF-β in Grave's disease patients, we studied the serum level of IL-17 and TGF-β in Grave's disease patients and to compare with those of normal control subjects . In the present study, the level of IL-17 and TGF-β was investigated in peripheral blood from 30 patients with Grave's disease and 27 healthy individuals by ELISA. In Graves' disease patients, the levels of IL-17 (14,43 ± 2,15 ρg/mL) were significantly higher than controls (7,07 ± 1,45 ρg/mL). The level of TGF-β were significantly higher in Graves' disease patients (10,44 ± 3,19) than control (4,95 ± 1,35 ρg/mL). Furthermore, in Graves' disease patients, we detected a positive correlation between free TRAb and IL-17 levels (r2 = 0.81, P<0.00). The elevated serum IL-17 and TGF-β of Graves disease patients reflect the activation of Th1 and Th2 cells which may be consisten with chronic inflammation processes of thyroid gland

Keywords: Grave's disease, IL-17, TGF-β

Introduction

Graves' disease (GD) is an autoimmune thyroid disease (AITD) characterized by excessive autoantibody formation by the lymphocyte B cells, and the proliferation of thyroid cells, as well as hyperthyroidism. The autoantibodies will bind to Thyroid Stimulating Hormone receptors (TSHR) and enhance the production of thyroid hormone. The pathogenesis of AITD is still unclear and both environmental and genetic factors tend to play critical roles. At present, many studies focus on the roles of cytokines and its associated with the abnormal immune response and inflammatory process of these diseases.

Inflammatory cytokines and chemokines appear to be centrally involved in the pathogenesis of these disease. CD4+ T helper (Th) lymphocytes are traditionally classified into Th1 and Th2 subsets on the basis of different cytokine secretion. In autoimmunity, a strong maybe deregulated Th1 response is often found. However, there is compelling evidence for a third effector CD4+ Th pathway in autoimmunity. These so-called Th17 T cells produce IL-17A and IL-17F, two cytokines not produced by either Th1 or Th2 CD4+ T cel. IL-17 is an inflammatory cytokine mainly secreted by Th17 cells and the causative role of Th17 cells has been proposed in many autoimmune diseases. A combination of transforming growth factor (TGF) and IL-6, together with IL-23 leads to generation of this CD4+T cell subtype.

Material and Methods

Thirty of Graves disease patients were diagnosed according to the clinical diagnostic criteria of Auto Immune Thyroid Disease, had hyperthyroidism, goiter and positive thyroid-stimulating hormone receptor antibody (TRAb). Blood samples from 27 healthy individuals matched for age and gender were collected at the Blood Transfusion Unit used as controls. The level of IL-17 and TGF-β were measured by ELISA. The study was approved by the Research Ethics Committee of the Faculty of Medicine Andalas University

Results

In Graves' disease patients, the levels of IL-17 (14,43 ± 2,15 ρg/mL) were significantly higher than controls (7,07 ± 1,45 ρg/mL). The level of TGF-β were significantly higher in Graves' disease patients (10,44 ± 3,19) than control (4,95 ± 1,35 ρg/mL). Furthermore, in Graves' disease patients, we detected a positive correlation between free TRAb (5,63± 3,72IU/ml) and IL-17 levels (r = 0.81, P<0.05).

Table: The level of IL-17 and TGF-β in Graves disease patients and control

Group	n	IL-17 (ρg/mL)	TGF- β (ρg/mL)	p
Graves	30	14,43 ± 2,15	10,44 ± 3,19	<0,05
Control	27	7,07 ± 1,45	4,95 ± 1,35	<0,05

Discussion

In healthy homeostatic conditions, the levels of IL-17A in human sera are undetectable, however, the levels are markedly increased in inflammatory disease such as psoriasis, systemic lupus erythematosus (SLE), multiple sclerosis (MS), and rheumatoid arthritis. In our study, overproduction of IL-17 promotes inflammation by mobilizes, recruits and activates different cells to increase inflammation. TGF-β in the presence of IL-6 drives the differentiation of T helper 17 (Th17) cells, which can promote further inflammation and augment autoimmune conditions. The high level of TGF-β may regulate and induce of peripheral tolerance. TRAb found to be related to the level of 17 IL-17, may be indirectly involved in Ab production by enhancing production of B cell activator by other immune cells. While TGF-β enabled activated CD8(+) T cells to inhibit antibody production by blocking the induction of this response. TGF-beta could have an equally important role in the generation of regulatory T cells.

Conclusion

IL-17 play a role in chronic inflammation associated with autoimmune diseases and antibody production. The elevated serum IL-17 and TGF-β of Graves disease patients reflect the activation of Th1 and Th2 cells which may be consisten with chronic inflammation

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Station
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E-POSTER STATION 7


Station
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CME/CPD Certificate

This is to certify that

Eryati Darwin

(first, last name, degree)

participated in the

10th International Congress on Autoimmunity (Autoimmunity 2016)

Leipzig, Germany
April 6-10, 2016

As speaker in Short Oral Presentation

Yehuda Shoenfeld, MD, FRCP, MaACR
Congress President

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