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成绩

专业能力（国际医学杂志上发表 SCI 论文）

- ▶ Lin Y, Zou Q, Li H (2009) Tipping the balance: anti-tumour necrosis factor alpha therapy may damage cerebral nerve reservation. *Med Hypotheses* (IF=1.41) 73: 958-960.
- ▶ Lin Y, Zou Q, Li H (2010) Localization of cerebral functional deficits in patients with non-neuropsychiatric systemic lupus erythematosus. *Human Brain Mapping* (IF=6.25) DOI: 10.1002/hbm.21158 (In Press).

英语能力（国家级奖励）

- ▶ 2008 年 CCTV 杯全国英语演讲大赛全国优胜奖、重庆市一等奖
- ▶ 2006 年全国大学生英语竞赛全国特等奖

教育和工作经历

- ▶ 医学学士学位（第三军医大学，2002/9/1 – 2007/6/20）
- ▶ RIC International 签约译员（2010 – 今）
- ▶ Logos 集团签约译员（2010 – 今）

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- ▶ **GE** 新型 CT 和 MRI 扫描机操作员说明手册（英译中）
- ▶ **PHILIPS** 新型 3T MRI 扫描机说明手册（英译中）
- ▶ **Medela** 产品推荐和教育大会，重庆-2009年9月（交替传译）
- ▶ **Mortara Instrument** 心电图机测试报告和说明手册（英译中）
- ▶ **Olympus Chemistry Analyzer AU680** 化学分析仪手册（英译中）
- ▶ **Olympus CEA, AFP** 等试剂盒说明书（英译中）
- ▶ **Reckitt Benckiser DADA** 片剂测试报告和商业推广手册（英译中）
- ▶ **Pfizer VIAGRA** 包装盒文字说明和片剂标签（英译中）
- ▶ **Bard Peripheral Vascular PTA** 球囊扩张导管说明手册（英译中）
- ▶ **Perceptive Informatics** X光、CT 和 MRI 研究中心指南（英译中）

Localization of Cerebral Functional Deficits in Patients With Non-Neuropsychiatric Systemic Lupus Erythematosus

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Abstract: Neuropsychiatric systemic lupus erythematosus (NP-SLE) is a common complication of systemic lupus erythematosus (SLE), and clinical interventions are of only limited efficacy despite relatively high prevalence. Such complications have been studied extensively, but the pathoetiology of NP-SLE has not yet been elucidated. Diagnosis of NP-SLE focuses primarily on psychological manifestations, and the underlying mechanisms leading to neuropsychiatric complications remain unknown. To address potential changes in brain function before NP-SLE development, we used resting-state functional magnetic resonance imaging (MRI) to compare regional brain activity in SLE patients versus matched controls. We report that regional activity in cerebellum and in areas of the default mode network are attenuated in patients with SLE, and moreover individual alterations in cerebellar activity correlated positively with the disease activity index. These findings provide direct evidence that significant alteration of brain function, resembling that observed in patients with NP-SLE, is already present in SLE patients without neuropsychiatric complications, highlighting the need for early evaluation and intervention in SLE patients. Furthermore, the disease activity rating correlated with regional functional alterations in the cerebellum, suggesting that the cerebellum could play a role in the pathogenesis of NP-SLE. *Hum Brain Mapp* 00:000–000, 2010. © 2010 Wiley-Liss, Inc.

Keywords: systemic lupus erythematosus; cerebral functional deficits; non-neuropsychiatric systemic lupus erythematosus; resting state functional magnetic resonance; regional homogeneity

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by multisystem involvement and diverse clinical manifestations [Rahman and Isenberg, 2008]. Neuropsychiatric systemic lupus erythematosus (NP-SLE) is a common complication of SLE and has been studied extensively, but the pathoetiology of NP-SLE has not been elucidated. Possible damage mechanisms include vasculopathy, autoantibodies, and inflammatory mediators, but these have not yet been confirmed [Boumpas et al., 1995]. Currently, the diagnosis of NP-SLE is based on case categorization into one of 19 neurological and

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Received for publication 25 March 2010; Revised 18 July 2010;

Accepted 2 August 2010

DOI: 10.1002/hbm.21158

View this article online at wileyonlinelibrary.com.

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Contents lists available at ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

Tipping the balance: Anti-tumour necrosis factor alpha therapy may damage cerebral nerve reservation

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ARTICLE INFO

Article history:

Received 9 June 2009

Accepted 9 June 2009

SUMMARY

Anti-tumour necrosis factor alpha therapy has transformed the treatment of certain inflammatory diseases including rheumatoid arthritis, inflammatory bowel disease and ankylosing spondylitis, but onset of demyelinating events associated with multiple sclerosis as an adverse event was continuously reported, and such adverse events were only viewed as occasional. Multiple sclerosis is an autoimmune demyelinating disorder affecting central nervous system, with varied clinical manifestations of cognitive, visual and motor network disorder. Recently, there is increasing evidence from functional magnetic resonance that cortical reorganization, a property that allows the central nervous system to adapt itself to various brain insults, which was viewed as to limit the clinical expression of tissue damage in patients with multiple sclerosis. In light of the mentioned above, we hypothesis that cerebral tissue damage may existed in a broader aspects of patients treated with anti-tumour necrosis factor therapy, but its clinical manifestations from brain lesions were compensated by cortical reorganization. In other words, cerebral nerve reservation may be damaged by the therapy. If confirmed, the hypothesis may lead to a safety concern of the therapy, and an insight of the pathophysiology of both multiple sclerosis and certain inflammatory diseases.

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Background

Anti-tumour necrosis factor alpha (anti-TNF- α) therapy is a newly developed cytokine therapy with satisfied response on certain inflammatory diseases including rheumatoid arthritis, inflammatory bowel disease and ankylosing spondylitis; meanwhile, adverse events including demyelinating events associated with multiple sclerosis (MS) has been continuously reported [1–4], although the occurrence of such events seems occasional, due to its symptoms significantly interfere the life quality of patients, the induction of MS by anti-TNF- α therapy has drawn great concerns [5–7].

Cognitive impairment has been noticed as an early manifestation of MS since the 80s last century, clinical trials confirmed that nearly half of the patients in very early stage MS were of cognitive impairment [8–12], the diagnostic tests of MS gradually developed from expanded disability status scale (EDSS) [13] concerning physical fitness, to multiple sclerosis functional composite (MSFC) [14] with cognitive test and was well accepted. However, the pathophysiology and diagnosis of MS still requires further study.

Development of magnetic resonance, particularly the application of functional magnetic resonance image (fMRI) in scientific research has provided non-conventional tools to the insight of the

brain, determining the exact region activated during a certain task. fMRI investigations of the visual, cognitive and motor networks in patients with MS have shown an altered recruitment of regions normally devoted to the performance of a given task and/or the recruitment of additional areas in comparison to healthy individuals [15–17], suggesting cortical reorganization existed in patients with MS, such property enables the central nervous system (CNS) to adapt itself to various brain insults. Based upon evidences from fMRI experiments, different researchers proposed the same theory that clinical manifestation of MS may be limited by cortical reorganization [18–20].

Anti-TNF- α therapy and MS

Immunological mechanism

The mechanism of how anti-TNF- α therapy induced demyelinating events remains to be clearly elucidated. Systemic administration of anti-TNF- α may promote the function of antigen presenting cell, reduce T-cell receptor signalling, lead to an impaired apoptosis of potentially autoreactive T-cells, and induce the production of proinflammatory cytokines including IFN- γ , which is known to activate MS [21–24]. Prolonged contact with anti-TNF- α could increase the activation and survival of potentially autoreactive peripheral T-cells. Such cells could penetrate the CNS and cause demyelination [25].

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中央电视台英语频道 外语教学与研究出版社
 2008年10月18日


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