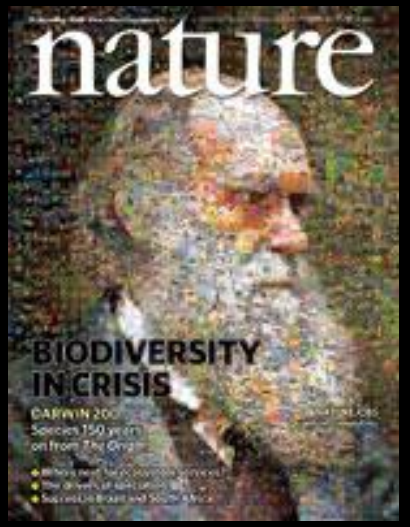
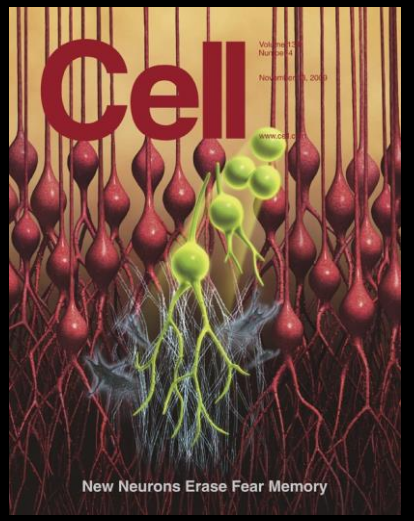


SCI期刊投稿策略與技巧

陳春榮
台中榮民總醫院教學研究部

The NEW ENGLAND JOURNAL of MEDICINE



學術研究成果呈現項目

學術期刊　　專利　　技術移轉　　專書

“近年來投稿學術期刊已成為一個發表研究成果的重要管道”

如何撰寫高品質之期刊論文
提升學術研究效率及論文寫作
學術期刊投稿策略與技巧
如何投稿SCI國外學術期刊
期刊投稿經驗分享

希望藉由經驗的累積與分享，經驗傳承，熟悉研究方法與技巧，進而達到提昇成果發表的目的。

學術期刊領域分類

Science Citation Index (SCI)

收錄科學技術類期刊

Engineering Index (EI)

Social Science Citation Index (SSCI)

Arts & Humanities Citation Index (A&HCI)

非SCI

正式論文，簡報型論文，病例報告，致編輯信，綜合評論等

Impact factor

The frequency of “average article” in a Journal has been cited in a year.

Dividing the number of citations to articles published in the two previous years by the total number of articles Published in the two previous years.

A Journal’s relative importance within the same field.

學術研究成果努力追求目標

以SCI論文而言

質	量
高Impact Factor 高Citation	篇數多

第一/通訊作者
共同作者

SCI學術研究成果衡量影響面

學校或研究機構聲譽
研究學者績效評量
研究計畫申請執行能力評量
學術課程
工作機會
職務升遷
薪資
等

研究人員近五年內研究成果統計及獲獎勵情形（表 A）

1. 五年內曾懷孕生產或請育嬰假者，得填寫近七年內研究成果統計及獲獎情形，但須附證明文件。
2. 五年內曾服國民義務役者，得加上實際服役時間延長選填研究成果及獲獎情形，但須附證明文件。

(修正：2008/12/03)

研究人員姓名	
任職機關系所	

※本表所填寫下列各項數量資料均應以研究人員個人資料表所填列之資料為依據。

(一)請填寫五年內(2004.1.1 迄今)已發表或已被接受發表之研究論文數量

研究成果 / 作者序	SCI、SSCI、EI 期刊論文（包括填表說明六(一)之 1.所列四類論文：正式論文、簡報型論文、病例報告、綜合評論）			其他學術期刊論文（左列 3 類以外之期刊論文）
	SCI 論文	SSCI 論文	EI 論文	
第一作者論文篇數				
非第一作者之通訊作者論文篇數				
非第一或通訊作者之其他序位作者論文篇數				
總篇(件)數（以上三項總和）				

SCI 、SSCI、EI 之期刊論文資料，可就近至各大學圖書館、國科會科技政策研究與資訊中心等查閱或上網檢索。上述 SCI、SSCI 及 EI 期刊資料以 2007 年版本為準。

(二)請填寫五年內(2004.1.1 迄今)已獲得或已刊登之下列研究成果數量

成果名稱	專利	技轉	研討會論文摘要	專書或專書章節	其他
（件、冊、章、篇）數					

(三)請填寫五年內(2004.1.1 迄今)獲得獎勵情形

年　　度	請選填下列獎項名稱：傑出獎、吳大猷獎、其他獎(請填獲獎名稱)
1. 93 年(93.1.1~93.12.31)	
2. 94 年(94.1.1~94.12.31)	
3. 95 年(94.1.1~95.12.31)	
4. 96 年(95.1.1~96.12.31)	
5. 97 年(97.1.1~迄今)	

國科會生物處
研究成果統計及獲獎情形

國科會AB表及RPI分數

表A

研究人員近五年內研究表現指數（RPI）統計（表 B）

(修正：2008/12/03)

姓　　名：	
機關系所：	
研究年資：（請打✓）	□滿 5 年以上(選最佳 10 篇)；□滿 4 年(選最佳 7 篇)；□滿 3 年(選最佳 5 篇)；□未滿 3 年(選最佳 3 篇) 1. 五年內曾生產或請育嬰假者，得於七年內選擇上列所須論文篇數，研究年資則扣除 2 年後，於上列四項年資中勾選年資，並請附上生產或請育嬰假證明文件。 2. 五年內曾服國民義務役者，得加上實際服役時間延長選擇上列所須論文篇數，研究年資則為扣除實際服役時間，於上列四項年資中勾選您的年資，並請附上服國民義務役證明文件。

序號	成果類別代碼（參看填表說明之三）	五年內(2004.1.1 迄今)代表性研究成果名稱 ★1. 學術論文必須填寫所有作者(按期刊所刊登之原排序)、著作名稱、期刊名稱、年份、卷期、起迄頁數。 ★2. 專利必須填寫專利名稱、發明人、證書號碼、國別、專利期限。 ★3. 技術移轉必須填寫技術名稱、技轉金額及對象、年份。 ★4. 刊登雜誌分類排名以 2007 年版本之 SCI 及 SSCI 資料為準。	論文性質分數 (C)	刊登雜誌分類分數 (J)	作者排名分數 (A)	分數 (CxJxA)
1						
2						
3						
4						
5						
6						
7						
8						
9						
10						
積分（以上各項研究成果分數之總和）						
研究表現指數(RPI)【(積分 x100)/ 指標上限滿分】						

★註 1. 已被接受但未出刊之論文須附接受函或相關證明文件，技術移轉須附上合約書，專利須附上專利證書，採相同貢獻作者計分者須附該論文註明「相同貢獻作者」部份之電子檔，五年內曾生產或請育嬰假或曾服國民義務役而延長選取研究成果著作期限者請附證明文件，前述文件請掃描附於本表之後一併上傳，未附者將不採計。

★註 2. 申請人填寫本表之資料經核對結果，若填寫不實將予更正，無法辨識者將取消計分；蓄意造假者，其申請案不予通過外，並送本會學術倫理審議委員會按情節輕重程度議處。

表B

研究表現指數
RPI
(Research Performance Index)

評比論文篇數

不同研究年資之「M值」及「指標上限滿分」

研究年資	M值	指標上限滿分
滿五年及五年以上	10	750 (10x75)
滿四年但未滿五年	7	525 (7x75)
滿三年但未滿四年	5	375 (5x75)
未滿三年	3	225 (3x75)

分母

研究成果計分

（一）學術期刊論文之計分：每篇論文依下列方式填入論文性質分類、刊登雜誌分類排名及作者排名等三項之加權分數後，求其乘積(CxJxA)即為該篇論文之歸類計分。

1.論文性質分類加權分數(C)

論文性質分類	加權分數(C)
正式論文（Full Article）	3 分
簡報型論文	2 分
病例報告	1 分
綜合評論（Review article）；一年一篇為限	2 分

註 1.技術報告或 DNA、RNA 及 amino acid 序列登錄，均不計分。

註 2.碩、博士論文、未發表於學術期刊之論文或研究報告、科普性、評論他人或自己論文、或回覆其他評論者之意見或疑問等而非發表自己研究成果數據之文章、學會年會或研討會摘要、以及專書或其章節，均不能視為上表所列各項論文。

2. 學術論文刊登雜誌分類排名加權分數(J)：

2-1. 國外 SCI、SSCI 期刊排名百分比 （期刊排名/該領域期刊總數；以 2007 年版 JCR 資料為準）	加權分數(J)
IF≥5	IF
排名≦20.00%	5 分
20.00%<排名≦40.00%	4 分
40.00%<排名≦60.00%	3 分
60.00%<排名≦80.00%	2 分
排名 80.00%以後	1 分
2-2. 國內 SCI 期刊	參看附表 1
2-3. EI 期刊 （以 2007 年版 Publications in Engineering 收錄資料為準）	1 分
2-4. 其它國內外非 SCI、SSCI、EI 學術性雜誌	0.5 分

3. 作者排名加權分數(A)

作者序	加權分數(A)
第 1 作者或通信作者	5.0 分
第 2 作者	3.0 分
第 3 作者	1.0 分
第 4 作者或以後之作者	0.5 分

相同貢獻作者 (Equal Contribution) 採計相同貢獻作者計分者，須附該論文註明「相同貢獻作者」部份之影本。	1. 有 2-3 位作者相同貢獻，相同貢獻作者均以其排序之加權分數計分。 2. 有 4-6 位作者相同貢獻，相同貢獻作者均以其排序之加權分數 60%計分。 3. 有 7 位及以上作者相同貢獻，相同貢獻作者均以其排序之加權分數 20%計分。 4. 相同貢獻之作者均視為同一排序，其後一位作者之排序則以其在所有作者中之序位計算加權分數；以上計分若未達 0.5 分者均以 0.5 分計分。

C

J

A

C x J x A
分子

SCI論文

五年內專利或技術移轉之加權分數（2005 1.1.以後獲得之專利或簽約之技術移轉）

No.	類　　別	加權分數 (C)	(J)	(A)
1	國內新型或新式樣專利	15	1	1
2	國外新型或新式樣專利	20	1	1
3	國內發明專利	40	1	1
4	國外發明專利	50	1	1
5	技轉金台幣 20 萬元以下之技術移轉	50	1	1
6	技轉金台幣 20-50 萬元(含 20 萬元)之技術移轉	60	1	1
7	技轉金台幣 50~100 萬元(含 50 萬元)之技術移轉	75	1	1
8	技轉金台幣 100 萬元以上(含 100 萬元)之技術移轉	90	1	1

註 1 同一項發明獲多個國家(多處)專利者仍視為一件專利，選其最高之分數計分；已有發明之新型改良不視為另一件專利；專利且有技轉者採其一或較高之技轉分數計分，不能分為兩次計分。

註 2 同一專利或技術移轉之所有共同發明人或技術共同所有權人（立合約人）2 人以內者各以本表所列加權分數 100%計分，3 人者各以本表所列加權分數之 90%計分，4 人者各以本表所列加權分數之 80%計分，5 人或 5 人以上者各以本表所列加權分數之 70%計分。

註 3 技轉金額以技轉合約所載為準，以同一專利或同一技術之技轉累計總金額計算。

註 4 非真正技術移轉產生之技轉金，如研究計畫之先期技轉金等，不能視為上表所列之技轉金項目。

附表 1. 國內 SCI 期刊、國科會生醫科學雜誌之排名加權分數(J)

No.	期刊名稱	出版單位	SCI 期刊 (J)	加權分數
1	**生醫科學雜誌** (Journal of Biomedical Science)	國科會	SCI	4.0
2	台灣醫學會雜誌 (Journal of the Formosan Medical Association)	中華民國台灣醫學會	SCI	3.0
3	中國化學會誌 (Journal of the Chinese Chemical Society)	中國化學會	SCI	3.0
4	動物研究學刊 (Zoological Studies)	中央研究院生物多樣性研究中心	SCI	3.0
5	中國生理學雜誌 (Chinese Journal of Physiology)	中國生理學會	SCI	2.0
6	Botanical Studies	中央研究院植物暨微生物學研究所 中央研究院生物多樣性研究中心	SCI	4.0
7	藥物食品分析 (Journal of Food and Drug Analysis)	行政院衛生署藥物食品檢驗局	SCI	3.0
8	中華醫學會雜誌 (Chinese Medical Journal-Taipei)	中華醫學會	SCI(expanded)	1.5
9	中華皮膚科醫學雜誌 (Dermatologica Sinica)	台灣皮膚科醫學會	SCI(expanded)	1.5

※ 上表所列國內 SCI 雜誌依據 2008 JCR 資料。

學術期刊選擇

Impact factor
領域排名
同領域參考主流
發行量/期數
接受率/退稿率
刊出時間
費用
投稿方式

Aims and Scope
Format

Relevant topics
Criteria
Matched Journal

Special Issue

Journal homepage
Content
Author instruction


JCR-Web 4.5 Welcome - Microsoft Internet Explorer
ISI Web of Knowledge℠
Journal Citation Reports®
Information for New Users
Select a JCR edition and year:
JCR Science Edition
2007
Select an option:
View a group of journals by
Subject Category
Search for a specific journal
View all journals
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This product is best viewed in 800x600 or higher resolution
NOTICES
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Year
Category
Specific Journal

資料庫

JCR-Web 4.5 Journal Information - Microsoft Internet Explorer
檔案(F) 編輯(E) 檢視(V) 我的最愛(A) 工具(T) 說明(H)
上一頁 搜尋 我的最愛
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移至 Go PDF 連結

ISI Web of Knowledge℠

Journal Citation Reports®

2007 JCR Science Edition

Journal: JOURNAL OF NEUROCHEMISTRY

Mark	Journal Title	ISSN	Total Cites	Impact Factor	5-Year Impact Factor	Immediacy Index	Citable Items	Cited Half-life	Citing Half-life
☐	J NEUROCHEM	0022-3042	34937	4.451	4.561	0.656	717	7.1	6.8

Cited Journal | Citing Journal | Source Data | Journal Self Cites

CITED JOURNAL DATA | CITING JOURNAL DATA | IMPACT FACTOR TREND | RELATED JOURNALS

Journal Information

Full Journal Title: JOURNAL OF NEUROCHEMISTRY
ISO Abbrev. Title: J. Neurochem.
JCR Abbrev. Title: J NEUROCHEM
ISSN: 0022-3042
Issues/Year: 18
Language: ENGLISH
Journal Country/Territory: ENGLAND
Publisher: BLACKWELL PUBLISHING
Publisher Address: 9600 GARSINGTON RD, OXFORD OX4 2ZG, OXON, ENGLAND
Subject Categories: BIOCHEMISTRY & MOLECULAR BIOLOGY — SCOPE NOTE | VIEW JOURNAL SUMMARY LIST | VIEW CATEGORY DATA
NEUROSCIENCES — SCOPE NOTE | VIEW JOURNAL SUMMARY LIST | VIEW CATEGORY DATA

Journal Rank in Categories: JOURNAL RANKING

Eigenfactor™ Metrics
Eigenfactor™ Score
0.10408
Article Influence™ Score
1.465

Additional Links
TCVGH Holdings GO

Journal Impact Factor

Cites in 2007 to items published in: 2006 = 2596 Number of items published in: 2006 = 642
2005 = 3043 2005 = 625
Sum: 5639 Sum: 1267

Calculation: Cites to recent items 5639 = **4.451**
Number of recent items 1267

5-Year Journal Impact Factor

Cites in {2007} to items published in: 2006 = 2596 Number of items published in: 2006 = 642

網際網路
開始 收件匣 - Outlook ... 台中榮民總醫院 ... 台中榮總圖書館... JCR-Web 4.5 Jour... Microsoft PowerPo... 下午 02:01

JCR-Web 4.5 Journal Summary List - Microsoft Internet Explorer
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Journal Citation Reports®

36/211=17.06%

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2007 JCR Science Edition

Journal Summary List

Journal Title Changes

Journals from: subject categories NEUROSCIENCES VIEW CATEGORY SUMMARY LIST

Sorted by: Impact Factor SORT AGAIN

Journals 21 - 40 (of 211)

[1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10]

Page 2 of 11

MARK ALL UPDATE MARKED LIST

Ranking is based on your journal and sort selections.

Mark	Rank	Abbreviated Journal Title (linked to journal information)	ISSN	JCR Data						*Eigenfactor*™ Metrics	
				Total Cites	Impact Factor	5-Year Impact Factor	Immediacy Index	Articles	Cited Half-life	*Eigenfactor*™ Score	*Article Influence*™ Score
☐	21	NEUROSCIENTIST	1073-8584	1782	5.796	5.711	0.660	53	3.8	0.01317	2.303
☐	22	HIPPOCAMPUS	1050-9631	5028	5.745	5.128	0.655	110	6.3	0.02162	2.048
☐	23	SLEEP MED REV	1087-0792	1225	5.705	5.342	1.656	32	4.4	0.00671	1.862
☐	24	NEUROBIOL AGING	0197-4580	8208	5.607	5.999	1.278	194	5.4	0.03064	1.859
☐	25	CURR OPIN NEUROL	1350-7540	3085	5.550	4.807	0.568	88	4.5	0.01649	1.730
☐	26	NEUROIMAGE	1053-8119	26201	5.457	6.825	0.900	677	4.3	0.15479	2.459
☐	27	GLIA	0894-1491	7165	5.380	5.128	1.065	155	5.4	0.02793	1.599
☐	28	PAIN	0304-3959	21465	5.249	5.552	1.156	231	8.1	0.04935	1.546
☐	29	NEUROTOX RES	1029-8428	1206	5.234		0.442	43	3.1	0.00409	
☐	30	J CEREBR BLOOD F MET	0271-678X	10393	5.147	5.327	1.435	177	7.2	0.02959	1.699
☐	31	J COGNITIVE NEUROSCI	0898-929X	8848	4.997	6.515	0.543	164	6.0	0.04457	2.870
☐	32	INT J NEUROPSYCHOPH	1461-1457	1745	4.895	4.826	1.013	80	3.5	0.00857	1.335
☐	33	J NEUROPATH EXP NEUR	0022-3069	7328	4.718	5.137	1.090	111	7.5	0.02309	1.773
☐	34	BRAIN BEHAV IMMUN	0889-1591	2290	4.659	4.480	1.564	110	4.5	0.00859	1.207
☐	35	J PHYSIOL-LONDON	0022-3751	43833	4.580	4.658	1.116	628	9.2	0.11769	1.654
☐	36	J NEUROCHEM	0022-3042	34937	4.451	4.561	0.656	717	7.1	0.10408	1.465
☐	37	BIPOLAR DISORD	1398-5647	2365	4.442	4.860	0.640	111	3.8	0.01255	1.428

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     - Revision requested
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Rejection
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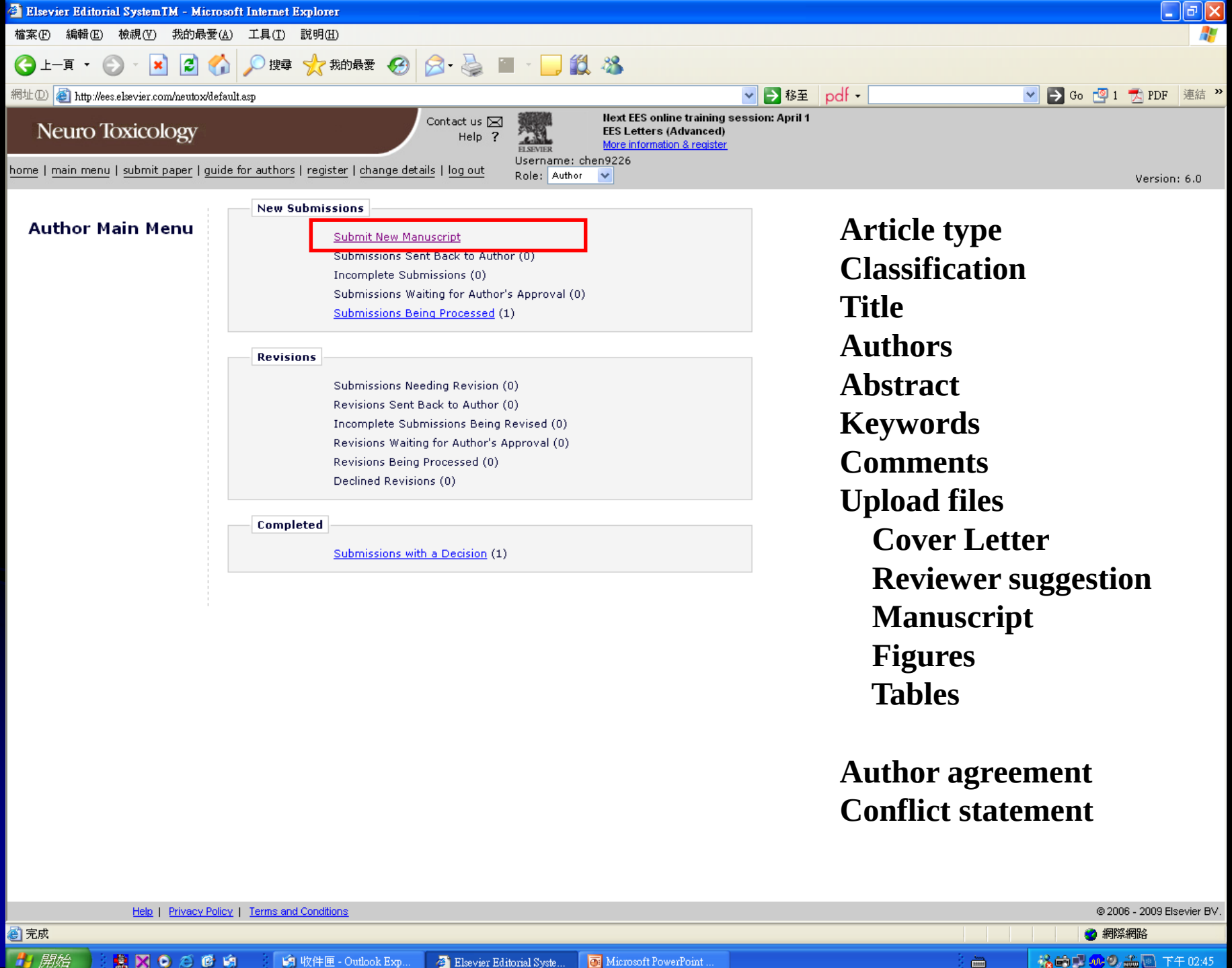

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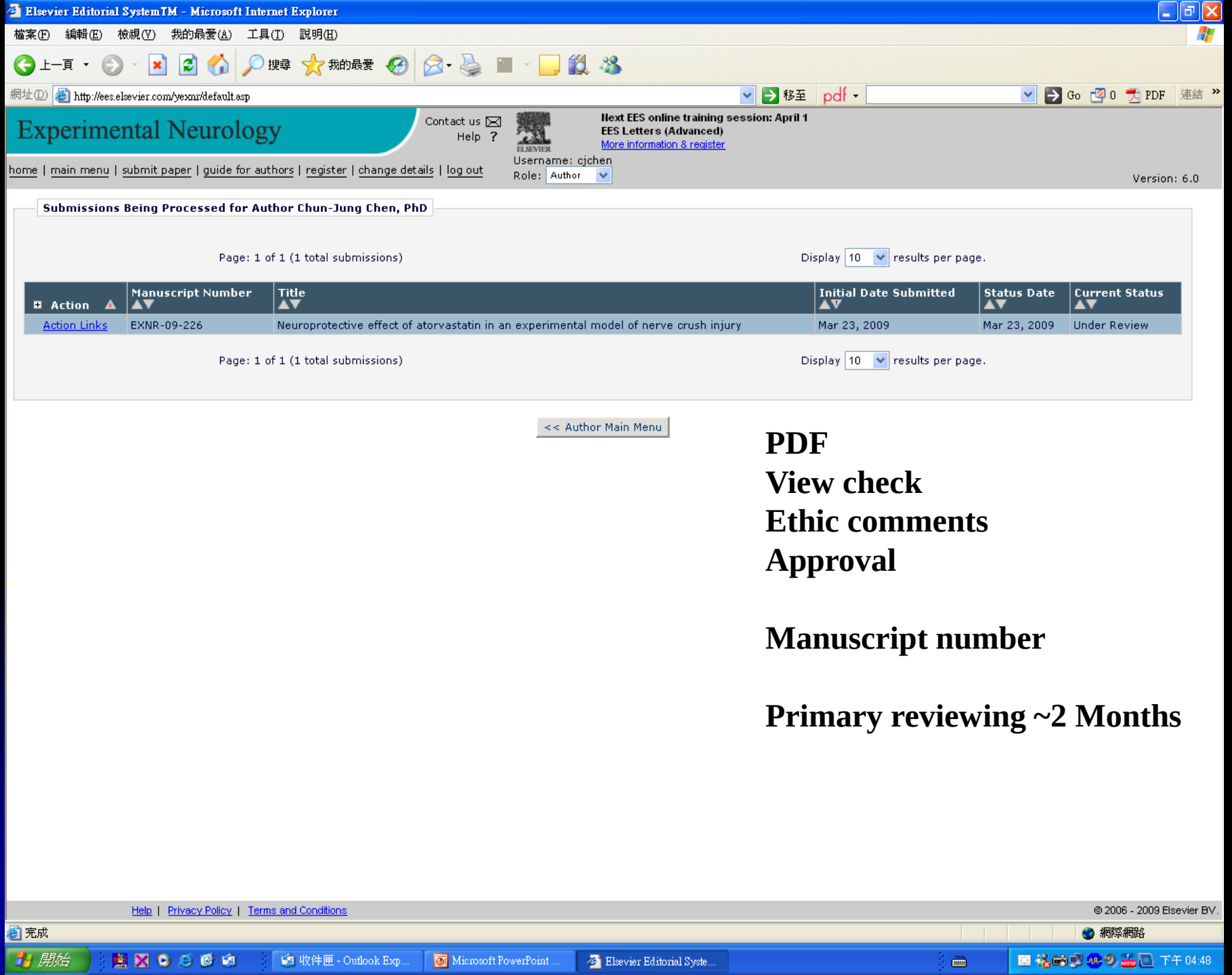

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05-Dec-2009

Dear Dr C.J. Chen:

The above manuscript, entitled "The mechanisms of action of TIANHUA on anti-tumor activity in Lung Cancer cells" with Professor Ko as contact author has been submitted to Pharmaceutical Biology.

I would be grateful if you would kindly agree to act as a reviewer for this paper. The abstract appears at the end of this letter, along with the names of the authors.

Please let me know as soon as possible if you will be able to accept my invitation to review. To do this please either click the appropriate link below to automatically register your reply with our online manuscript submission and review system, or e-mail me with your reply.

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Sincerely,

With best regards from Andrew Marston.

MANUSCRIPT DETAILS

TITLE: The mechanisms of action of TIANHUA on anti-tumor activity in Lung Cancer cells

AUTHORS: Li, Chien-Te; Lin, Ching-Hsiung; Kao, Te-Yu; Wu, Ming-Fang; Yeh, Chin-Shui; Yeh, Kun-Tu; Ko, Jiunn-Liang

ABSTRACT: Context: TIANHUA (TH-R) is extracted from Trichosanthes kirilowii Maxim containing trichosanthin, a traditional Chinese medicine, which has been locally reported to have good anticancer effects in vivo in both animal and human models. However, there have been several reports on trichosanthin has an anticancer effect in apoptosis.
Objective: To investigate other anticancer effects of TH-R, various tumorigenesis parameters were verified.
Materials and methods: Telomerase activity, anti-apoptosis, anti-migration and immunomodulatory activity were estimated by TRAP, flow cytometry, Boyden chamber assay and ELISA assay, respectively.
Results: In our studies, we are the first to find that TH-R had a cytotoxic effect on lung cancer cells in MTS assays; it could change the cell cycle distribution of human lung cancer cells (A549 cell line) and

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Protective effect of rofecoxib and nimesulide against intra-striatal quinolinic acid induced behavioral, oxidative stress and mitochondrial dysfunctions in rats

Original Submission
Chun-Jung Chen, Ph.D. (Reviewer 1)

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Anti-inflammatory characterization of quercetin in activated microglia
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Your manuscript has been examined by the editors and two qualified reviewer. We are pleased to inform you that they find the manuscript worthy of publication if revised in accordance with the enclosed comments.

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Yours sincerely,
Janet Holmén
Managing Editor
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COMMENTS FROM THE SCIENTIFIC EDITORS:

The manuscript is interesting but, in addition to the comments by the reviewers, attention should be paid to the following points:

1. Change the title to better describe the main findings of the study.

2. When presenting effects of quercetin on various factors (e.g. percentage inhibition of something), give the concentration of quercetin which caused the effect in question; this should be checked throughout the Results section.

3. Results, page 9, when you measure phosphorylation of signaling molecules (e.g. ERK, JNK, P38...) as a marker of their activation, change the wording accordingly (i.e. change activity / activation / inactivation to phospohorylation / reduced phosphorylation) to describe the phenomenon which was actually measured.

4. Explain what are lipid drafts.

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(4) We encourage you to test the relevance of your key words by using them for a database search and comparing the results with the topic of your own paper.

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Reviewer #1: **In this manuscript, authors characterize quercetin's anti-inflammatory profile in activated BV-2 microglia. Quercetin was found to down-regulate activation of p38, ERK, Akt, Scr, JAK-2, Tyk2, STAT-1 and NF-<kappa>B along with its inhibitory effect on NO production and iNOS expression in cells treated with LPS and IFN<gamma>. In addition, quercetin was found to suppress free radical generation in LPS/IFN<gamma> treated cells as compared to cells treated with stimuli only. The accumulation of lipid rafts was disrupted by quercetin.**

Few minor points:

1. In Materials and Methods; Cell culture: Please check how the origin of the cells was written in Chen CJ et al. 2004 (cell line was kindly donated by..). In my opinion, it should be stated here in similar manner, and the reference is then unnecessary.

2. In Materials and Methods; NO determination: Chen CJ et al. 2004 is not the correct reference for the Griess reaction. Please correct this. In addition, Griess reaction measures nitrite, not nitrate (unless reduction step is included), please correct this in Materials and Methods, page 5.

3. In Materials and Methods; Isolation of RNA..: Please check also here the use of Chen CJ et al. 2004 as a reference - is the method reported where indicated?

4. In Materials and Methods; Preparation of nuclear..: Please consider if you could omit some of the text when you refer to Chen CJ et al. 2004

5. Figure legends, Figure 5. (B) at the end of the text should propably not be there.

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Strength
Specific comments

Reviewer #2: **The submitted manuscript describes the modulatory effect of quercetin against neuroinflammation. Materials and Methods section is complete and methods are well described. Experiments are clear cut. Results are interesting above all from a speculative point of view. Results are adequately discussed in the light of most recent papers on this matter.**

Journal	IF
Journal of Virology	5.308
Biochemical Pharmacology	4.838
Journal of Neurochemistry	4.500
Journal of Nutritional Biochemistry	4.352
Diabetes Obesity and Metabolism	4.259
Experimental Neurology	3.974
Neurosurgery	3.398
European Journal of Neuroscience	3.385
Neurochemistry International	3.228
Journal of General Virology	3.092
European Journal of Pharmacology	2.787
Biochemical and Biophysical Research Communications	2.648
Life Science	2.583
NeuroToxicology	2.409
Neurochemical Research	2.260
Neuroscience Letters	2.200
NeuroReport	1.904
American Journal of Chinese Medicine	1.058

Journal Reviewer

Neuroscience Letters
Journal of Neurochemistry
Brain Research
Glia
Journal of Neuroscience Research
Acta Pharmacologica Sinica
European Journal of Neuroscience
Experimental Neurology
Transactions of the Royal Society of Tropical Medicine and Hygiene
PHARMACOLOGICAL REPORTS
Neurochemical Research
Chemical Research in Toxicology
Neurochemistry International
BMC Neurology
Brain Research Bulletin
Cell Biology International
Neuroscience
Journal of Medical Virology
Planta Medica
Life Sciences
NeuroToxicology
Pharmaceutical Biology

學術期刊論文撰寫流程及注意事項

整理實驗結果、數據繪圖
蒐集研討相關文獻
擬定論文訴求主軸重點
依結果選定適切期刊
依期刊格式列出大綱
撰寫稿件
擬定題目及作者
論文稿件修改
撰寫cover letter

Title page
Abstract and key words
Introduction
Materials and Methods
Results
Discussion
Conclusion
Acknowledgment
Reference
Figure legend
Tables
Figures

成果內容部份

實驗數據的完整性
新穎性
技術創新
觀念想法創新
實驗設計邏輯
實驗結果闡述、論文撰寫

Neuroprotective effect of atorvastatin in an experimental model of nerve crush injury

實驗條件與臨床的差異、原因、相關實驗的差異、原因

Statins have therapeutic benefit for the management of several disorders. A short-term course of high-dose statins pre-treatment seems to have a neuroprotective effect. However, the molecular basis underlying their neuroprotective action remains unclear. We investigated whether a short-term course of high-dose atorvastatin pre-treatment has neuroprotective effects on biochemical, functional, electrophysiological, and morphological alterations occurring during injury-induced degeneration/regeneration through a rat model of sciatic nerve crush injury. Atorvastatin or saline was given orally to rats for 7 days before injury. Motor function recovery, compound muscle action potential, nerve conduction latency, and axonal integrity showed significant improvement in the atorvastatin-treated group. Crush injury disrupted nerve integrity, produced oxidative stress, induced inflammatory changes, and caused cell apoptosis, and these detrimental alterations were attenuated by atorvastatin. After injury, parameters of restorative potential were further upregulated in groups receiving atorvastatin, including cell proliferation and viability and elevated expression of neurofilament, growth-associated protein-43, myelin basic protein, ciliary neurotrophic factor, and collagen. The progression of degeneration/regeneration after crush injury was accompanied by elevated activity of extracellular signal-regulated kinase (ERK), c-Jun N-terminal kinase (JNK), Akt, signal transducer and activator transcription-1 (STAT1), Smad2/3, NF-κB, and AP-1. Intriguingly, the suppression of ERK, AKT, STAT1, and NF-κB and the elevated activation of JNK, Smad2/3, and AP-1 were both associated with the neuroprotective action of atorvastatin. These findings suggest that a short-term course of high-dose atorvastatin pre-treatment can protect against sciatic nerve crush injury through directly altering primary insult and/or modifying intracellular or extracellular environment making it favorable for regeneration.

Conclusion

In summary, our findings indicate that pre-administration of atorvastatin led to improved functional, biochemical, electrophysiological, and morphological outcomes in a rat model of sciatic nerve crush injury. Although we did not fully establish a direct causal relationship between signaling molecules, transcription factors, and gene expression profiles, this improvement was associated with attenuated activity of cytotoxic molecules and stimulated activity of cytoprotective molecules, suggesting that the neuroprotective effect of atorvastatin following sciatic nerve crush injury may be attributable, in part, to a dampening of damaging potential and strengthening of regenerating potential. That is, rather than directly altering primary insult, atorvastatin might modify intracellular or extracellular environment making it favorable for regeneration after peripheral nerve crush injury. The findings imply that hypercholesterolemia patients receiving statin treatment might suffer less severity from peripheral nerve injury. However, the clinical relevance (such as dosage and treatment course) for therapeutic intervention against peripheral nerve injury by atorvastatin warrants further investigation.

過度闡訴結論

Protective effect of Labisia pumila on stress induced behavioral, biochemical and immunological alterations.

Abstract

The aim of the present study was to investigate the stress busting potential of *Labisia pumila* aqueous extract (LPPM/A003) using different models of stress. Pretreatment of experimental animals with LPPM/A003 caused increase in the swimming endurance time, hypoxia time and also showed the recovery of physical stress induced depletion of neuromuscular coordination and scopolamine induced memory deficit. LPPM/A003 at graded doses reversed the chronic restraint stress(RST) induced depletion of CD4+ and CD8+ T lymphocytes, NK cell population and corresponding cytokines expression and down regulated the stress induced increase in plasma corticosterone, a major stress hormone. In addition, LPPM/A003 reversed the chronic stress induced increase in adrenal gland weight, serum alanine aminotransferase (ALT), alkaline phosphatase (ALP) and hepatic Lipid peroxidation (LP) levels and augmented the RST induced decrease in hepatic Glutathione (GSH), thymus and spleen weight.

For Peer

訴求重點、機制、差異、結論

Abstract

Objective: To study the effect of application of Buflomedil on the pathological and functional repair of crush nerve injuries and also the expression of VEGF. **Methods:** Rat sciatic nerves were crushed by pincers to establish the model of crush injury. All of the 400 sprague dawley (SD) rats were randomly divided into: control; saline; saline + VEGF-antibody; Buflomedil; and Buflomedil + VEGF antibody groups. The SPSS 11.5 software was used for statistical analysis. The expression of VEGF in dorsal root ganglia (DRGs), following crush injury to sciatic nerves, was studied by reverse transcribed-polymerase chain reaction (RT-PCR), immunohistochemistry. The effects of Buflomedil on expression of VEGF, repair of neural pathology, and recovery of neural function were also evaluated. **Results**: We found that VEGF messenger ribonucleic acid (mRNA) was significantly increased in Buflomedil and Buflomedil + VEGF-antibody groups, compared to the saline and saline + VEGF antibody groups. The number of VEGF-positive neurons was significantly increased in the Buflomedil group, compared to the saline, saline + VEGF antibody, and Buflomedil + VEGF antibody groups. Besides, addition of this drug also caused less pathological changes in DRGs, better improvement of nerve conduction velocities (NCVs) of sciatic nerves, and more increase of toe spaces of right hind limbs of rats. **Conclusions:** The vasoactive agent Buflomedil may decrease the pathological lesion of peripheral nerves and improve the rehabilitation of the neural function, which may relate to upregulation of the expression of VEGF, following crush injury to the peripheral nerves.

For Peer Review

主軸標的分子介紹
實驗設計邏輯
檢體分析標的

Graptopetalum paraguayense E. Walther, a traditional Chinese herbal medicine, possesses several biological/pharmacological activities including hepatoprotective, anti-oxidant, and anti-inflammatory. The aim of this study was to evaluate the effect of *Graptopetalum paraguayense* E. Walther extracts on inflammation-associated brain injury and neuroinflammation. Water (GWE), 50% alcohol (GE50) extracts of *Graptopetalum paraguayense* E. Walther, and extracts obtained from further extraction of GE50 with ethyl acetate (GEE) were used in this study. Focal cerebral ischemia/reperfusion injured rat was used as a model to evaluate the neuroprotective and anti-inflammatory effects of *Graptopetalum paraguayense* E. Walther extracts. Its anti-inflammatory mechanism was further investigated on lipopolysaccharide (LPS)/interferon-γ (IFN-γ)-activated BV-2 microglial cells. Oral administration of GEE, but not GWE or GE50, for 2 weeks protected animals against ischemic brain injury The neuroprotective effect of GEE was accompanied by decreased caspase-3 activity, malondialdehyde content, and inducible nitric oxide synthase (iNOS) expression. GEE decreased H2O2- and LPS/IFN-γ-induced free radical generation and LPS/IFN-γ-induced iNOS expression in BV-2 microglial cells. Mechanistic study revealed that the neuroactive effects of GEE were markedly associated with anti-oxidative potential, activation of serine/threonine and tyrosine phosphatase, and down-regulation of extracellular signal-regulated kinase (ERK), c-Jun N-terminal kinase (JNK), p38, Akt, Src, Janus kinase-1 (Jak1), Tyk2, signal transducer and activator of transcription-1 (STAT1), and NF-κB. Furthermore, the neuroprotective and anti-inflammatory effects of *Graptopetalum paraguayense* E. Walther extracts were positively paralleled to their polyphenolic contents such as gallic acid, genistin, daidzin, and quercetin. These results suggest that *Graptopetalum paraguayense* E. Walther extracts protected against ischemic brain injury and the neuroprotective effect might be associated with their ability to scavenge free radicals and attenuate NO production in microglia.

The nonregenerative capability of the injured adult brain has been challenged in recent years and neural plasticity has been observed experimentally in both global and focal brain ischemia in animal models. Whether neuro-genesis increases in response to brain lesios or stem cells can be used for transplantation are the potential questions to be answered. Here, we performed the middle cerebral artery occlusion (MCAO) operation at rodent model and try to rescue neurons surrounding insult from the ischemic damage using acute transplantation of neural stem cells (NSCs). The infarct volumes and brain atrophy were obvious diminished at grafted NSCs treatment. The behavior recovery of grafted NSCs treatment significantly improved compared with vehicle control. Furthermore, we detected the inflammation related moleculars such as COX-2 and IL-1β and found that the grfated NSCs treatment after ischemic stroke could repressed the expression of inflammation moleculars. We also detected the protein level of heat shock protein 27 (HSP27) as a protective protein against apoptosis. The results showed that the group of grafted NSCs treatment induced the protein level of HSP27 and down-regulated activity of caspase-3 compared with vehicle control. Our results demonstrate that transplanted NSCs provide benefit in behavioral function recovery after MCAO in rats and more neuroprotection or less inflammatory destruction. These data reveal the another essential explain of cellular transplantation therapy in damage recovery from ischemic stroke and offer new therapeutic possibilities.

Anti-allergic Flavonoids, Baicalin and Isoorientin, from *Scutellaria baicalensis* and *Phyllostachys edulis*

The goal of this study was to screen plants for anti-allergic effects by evaluating the amounts of histamine and leukotrienes released after guinea pig lung mast cell activation. Bioassay-guided fractionation led to two flavonoids, baicalin from *Scutellaria baicalenesis* and isoorientin from *Phyllastachys edulis*. Based on these compounds, we developed two standardized extracts, SSBE from *S. baicalensis* and SPEE from *P. edulis*. SSBE or SPEE remarkably inhibited histamine and leukotrienes release in a dose-dependent manner. We also prepared Uniflavon™ by combining these extracts at a 1:2 ratio; the resulting combination had a stronger activity than either extract alone. These data suggest that Uniflavon™ in low dose than in dose of each standardized extract alone may be utilized as a therapeutic agent not only for allergic diseases but also for immune diseases related to mast cells, including rheumatoid arthritis and osteoarthritis, in further studies.

論訴重點一致性

謝謝聆聽

祝您成功！

意 見 交 流