

出國報告（出國類別：國際會議）

第 17 屆世界基礎及臨床藥理學會議
17th World Congress of Basic and
Clinical Pharmacology

服務機關：神經科學研究所

姓名職稱：詹銘煥、教授

派赴國家：南非

出國期間：7. 12-18, 2014

報告日期：7, 28, 2014

國立政治大學邁向頂尖大學計畫

出國成果報告書（格式）

計畫編號 ¹		執行單位 ²	神經科學所
出國人員	詹銘煥	出國日期	2014 年 7 月 12 日至 2014 年 7 月 18 日，共 7 日
出國地點 ³	南非、開普敦	出國經費 ⁴	

摘要：

參加南非、開普敦所舉辦 2014 第十七屆世界基礎及臨床藥理學(17th World Congress of Basic and Clinical Pharmacology)學術會議。會議期間為 7 月 13 至 18 日，2014 年。學會主題為結合基礎及臨床藥理學會議，所發表及討論的研究論文範圍涵蓋廣泛，包含肺結核病新藥理處理，阿茲海默症新藥研究及開發，天然藥物對神經，代謝與血管之作用機制，單株抗體對腫瘤之療效，後生調控藥物代謝酵素及傳送，以及肥胖與糖尿病之新藥標靶等等議題。每日大會皆安排各項主題的大會演講、討論會與壁報論文等。整體而言，此次藥理學會議內容豐富，與會者學習新知，收穫良多。



本文：

會議及參訪目的：

參與國際學術會議並發表近期研究成果論文，同時進行學術交流，同儕討論，並學習新知以增進研究廣度及國際觀。基於學院推動學術國際化的目標，為加強基礎自然科學的教學與研究，欲達學術交流國際化之目標，出席較大型國際學術會議並發表論文是最基本且有效的途徑。

會議及參訪過程：

大會開幕第一場演講，由 Prof. Robert J Lefkowitz 為諾貝爾獎得主，主題為 G protein coupled receptors，介紹 G 蛋白及受體的研究歷程及歷史，以及 G 蛋白訊息調控機制，講演精彩且多方面談到研究的挑戰及契機。

參加「阿茲海默症新藥研究及開發藥物」研討會，這些藥物具備許多作用點，相關研究資料提供未來對阿茲海默症良好治療之應用。研討會中為「腦部疾病中鈣離子訊息傳導」之研究，結果顯示調控鈣離子訊息也可作為阿茲海默症治療的標的。

¹ 單位出國案如有 1 案以上，計畫編號請以頂大計畫辦公室核給之單位計畫編號 + 「-XX（單位自編 2 位數出國案序號）」型式為之。如僅有 1 案，則以頂大計畫單位編號為之即可。

² 執行單位係指頂大計畫單位編號對應之單位。

³ 出國地點請寫前往之國家之大學、機關組織或會議名稱。

⁴ 出國經費指的是實際核銷金額，單位以元計。

” 訊息傳導之蛋白質磷酸化”及” 訊息傳導之醫療”，顯示在細胞功能中蛋白質磷酸化之重要角色。許多酵素及受體可因”磷酸化及去磷酸化”而轉換成”開與關”。所以 Tau 蛋白質磷酸化也許可為阿茲海默症治療的另一手段。另外，Rho-kinase (ROCK)磷酸化也可作為新穎方式治療乳癌。

由 Dr. Stephen Stahl 主講” 精神藥理學_ Mechanism of Action of the Atypical Antipsychotics in Psychosis and Mood Disorders: The Pines, the dones, two pips and a rip”，內容活潑生動有趣，大家熱烈討論並交換意見。中午是我個人壁報論文說明時間，題目為: The protective and therapeutic effects of cortex Magnoliae on neuronal damage and behavioral deficits induced by neurotoxin. 與來自各地學者專家相互詢問討論，同時也給予寶貴的意見，提供未來研究之重要策略。“Phosphodiesterase 4 inhibitors as novel anti-inflammatory drugs”. Phosphodiesterase 4 抑制劑將可作為抗發炎藥及新穎藥物得以治療氣喘及慢性呼吸道阻塞症。

” Targeting the TRPA1 channel 對疼痛之治療” TRPA1 在疼痛、麻醉、體溫調控、對細菌內毒素之神經感覺、化學性治療引起周邊神經病變及偏頭痛過敏性的角色皆有描述。

總結，參加此次國際學術會議相當成功，獲益良多。

心得及建議：

參加國際性會議是促進國內研究學者與國際交流的機會，應多加鼓勵。多日來大家於會議室中分享彼此研究成果，積極參與下更加深學者相互熟識，提供更直接學習合作機會及較新的研究模式，另外更多機會進行未來學術交流，對自己未來的研究方向有所啟發，頗有收穫。此次有數位台灣學者被選為口頭報告，已較上一屆 WCP 多，且講演內容精彩，美中不足，仍未能有擔任主持人或 organize a session，增加台灣研究學者在國際會議上之能見度仍是必須努力的方向。

附錄：

1. 研討會議程: (附件一)。
2. 壁報論文發表及標題 (附件二)。
3. 壁報論文摘要及內容(附件三)。
4. 壁報會場(附件四)。

研討會議程: (附件一)

Sunday 13 July 2014

TRACK 1	TRACK 2	TRACK 3	TRACK 4	TRACK 5	TRACK 6	TRACK 7
			09.00 – 12.30 (CPD=3.5) Training of Medicines development and regulation in Emerging Countries			
			13.30 – 16.30 (CPD = 3.5) PharfA Symposium1: Control of Complementary Medicines			
18.30 – 19.30 (CPD=1) G-Protein coupled receptors Presenter: Robert Lefkowitz						

Monday 14 July 2014

TRACK 1	TRACK 2	TRACK 3	TRACK 4	TRACK 5	TRACK 6	TRACK 7
10.30 – 12.00 (CPD=1.5) Pharmacology of novel tuberculosis regimens	10.30 – 12.00 (CPD=1.5) New drug research and development for Alzheimers disease	10.30 – 12.00 (CPD=1.5) Natural Products: neuro-metabolic vascular mechanisms	10.30 – 12.00 (CPD=1.5) Therapeutic monoclonal antibodies in oncology	10.30 – 12.00 (CPD=1.5) What every pharmacist should know about children	10.30 – 12.00 (CPD=1.5) Epigenetic regulation of drug metabolizing enzymes & transporters	10.30 – 12.00 (CPD=1.5) New drug targets for obesity and diabetes
13.30 – 15.00 (CPD=1.5) Antibiotic resistance	13.30 – 15.00 (CPD=1.5) New targets for Stress	13.30 – 15.00 (CPD=1.5) Therapeutic targets for treating or preventing insulin resistance and cardiometabolic complications	13.30 – 15.00 (CPD=1.5) Targeted small molecule therapy in oncology	13.30 – 15.00 (CPD=1.5) Communicating with the public and the policy community	13.30 – 15.00 (CPD=1.5) Orphan G protein coupled receptors – What are the new ligand and new drug targets	13.30 – 15.00 (CPD=1.5) Drug interactions: genotype to bedside

15.30 – 17.00 (CPD=1.5) Novel therapeutic strategies in HIV	15.30 – 17.00 (CPD=1.5) Calcium signalling in brain diseases	15.30 – 17.00 (CPD=1.5) Advances in reproductive pharmacology	15.30 – 17.00 (CPD=1.5) Targeting B cell signalling in oncology	15.30 – 17.00 (CPD=1.5) 2020 vision for Pharmacology Education		15.30 – 17.00 (CPD=1.5) Advances in clinical pharmacology
17.30 – 19.30 (CPD=2) PharfA symposium 2: Strategies for medicines development in constraint economies						

Tuesday 15 July 2014

TRACK 1	TRACK 2	TRACK 3	TRACK 4	TRACK 5	TRACK 6	
10.30 – 12.00 (CPD=1.5) New drugs for neglected infectious diseases	10.30 – 12.00 (CPD=1.5) Protein S-nitrosylation as a therapeutic target	10.30 – 12.00 (CPD=1.5) Obesity: Basic and clinical pathophysiology and pharmacology	10.30 – 12.00 (CPD=1.5) TGFB in radiation biology and therapy	10.30 – 12.00 (CPD=1.5) IUPHAR natural products section	10.30 – 12.00 (CPD=1.5) Innovations in drug therapies – the future is now	
13.30 – 15.00 (CPD=1.5) Pharmacogenetics in infectious diseases	13.30 – 15.00 (CPD=1.5) Optimizing anti-epilepsy drug discovery	13.30 – 15.00 (CPD=1.5) Renin-angiotensin system pharmacology revisited	13.30 – 15.00 (CPD=1.5) DNA repair and topoisomerase inhibitors in oncology	13.30 – 15.00 (CPD=1.5) Internet based solutions to alleviate shortages of pharmacology faculty in developing countries	13.30 – 15.00 (CPD=1.5) Plants and animal toxins as sources of new therapeutic drugs	

15.30 – 17.00 (CPD=1.5) Recent breakthroughs in malaria treatment	15.30 – 17.00 (CPD=1.5) The addictive brain through different receptor subtypes	15.30 – 17.00 (CPD=1.5) Aspirin the wonder drug	15.30 – 17.00 (CPD=1.5) Sarcoma genetics and targeted therapeutics	15.30 – 17.00 (CPD=1.5) Regulatory challenges in herbal and traditional medicines	15.30 – 17.00 (CPD=1.5) NC-IUPHAR and the IUPHAR/BPS guide to pharmacology	
17.00 – 18.00 (CPD=1) Adventures in allostery: From function to structure (IUPHAR Analytical Pharmacology lecture)						

Wednesday 16 July 2014

TRACK 1	TRACK 2	TRACK 3	TRACK 4	TRACK 5	TRACK 6	TRACK 7
10.30 – 12.00 (CPD=1.5) Novel drug targets in oncology	10.30 – 12.00 (CPD=1.5) Rethinking Mood Therapeutics – novel pharmacological approaches for anxiety and depression	10.30 – 12.00 (CPD=1.5) Manipulation of gut microbiome as a treatment strategy for gastrointestinal and metabolic disorders	10.30 – 12.00 (CPD=1.5) Update in geriatric pharmacology. Optimal prescribing in older patients	10.30 – 12.00 (CPD=1.5) Managed introduction of new medicines	10.30 – 12.00 (CPD=1.5) Evolution, sport and modern diseases	
13.30 – 15.00 (CPD=1.5) Vaccine development	13.30 – 15.00 (CPD=1.5) PDE4 inhibitors as novel anti-inflammatory drugs	13.30 – 15.00 (CPD=1.5) Neuroendocrine regulation of gastrointestinal protection: Central and peripheral pathways	13.30 – 15.00 (CPD=1.5) Controversies in essential medicines		13.30 – 15.00 (CPD=1.5) Advances in signal transduction & ion channels	
15.30 – 17.00 (CPD=1.5) Inflammation, Allergy: New therapeutic Avenues	15.30 – 17.00 (CPD=1.5) New approaches for non-neuronal brain diseases	15.30 – 17.00 (CPD=1.5) Lipid modifying strategies. Needs beyond statins	15.30 – 17.00 (CPD=1.5) Problems with OTC analgesics	15.30 – 17.00 (CPD=1.5) Global PGx-based personalized medicine		

				17.00 – 19.00 (CPD=2) Scientific Capacity/Capability Development		
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Thursday 17 July 2014


TRACK 1	TRACK 2	TRACK 3	TRACK 4	TRACK 5	TRACK 6
10.30 – 12.00 (CPD=1.5) Glucocorticoids: new insights into mechanism of action	10.30 – 12.00 (CPD=1.5) Targeting the TRPA 1 channel for pain treatment	10.30 – 12.00 (CPD=1.5) Hydrogen sulphide in GI health and disease	10.30 – 12.00 (CPD=1.5) Hot topics in Pharmacoeconomics	10.30 – 12.00 (CPD=1.5) Using clinical toxicology studies to improve biomarkers and regulatory decisions	10.30 – 12.00 (CPD=1.5) The role of drug transporters in inter-individual variations in drug response
13.30 – 15.00 (CPD=1.5) Immunopharmacology of the systemic inflammatory response syndrome	13.30 – 15.00 (CPD=1.5) The nitric oxide cGMP pathway in neuropsychiatric illness: An update	13.30 – 15.00 (CPD=1.5) Advances in GI pharmacology: New approaches to upper and lower GI ulcers and inflammation	13.30 – 15.00 (CPD=1.5) Can academic drug discovery deliver in rare diseases?	13.30 – 15.00 (CPD=1.5) IUTOX session in nanomedicines	13.30 – 15.00 (CPD=1.5) Understanding drug induced liver Injury

15.30 – 17.00 (CPD=1.5) Immunobiologicals and chronic inflammatory diseases	15.30 – 17.00 (CPD=1.5) Nitric oxide research reveals new ideas in pharmacology	15.30 – 17.00 (CPD=1.5) Endothelium dependent control of vascular tone	15.30 – 17.00 (CPD=1.5) Pharmacoepidemiology :at the cutting edge	15.30 – 17.00 (CPD=1.5) The changing face of Paediatric drug development	15.30 – 17.00 (CPD=1.5) Understanding and Predicting the Extent of Drug-Drug Interactions
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Friday 18 July 2014

TRACK 1	TRACK 2	TRACK 3	TRACK 4	TRACK 5	TRACK 6	TRACK 7
10.30 – 11.30 (CPD=1) Global HIV clinical pharmacology capacity building and implementation research	10.30 – 11.30 (CPD=1) Combination medications as novel treatments for stimulant Addiction	10.30 – 11.30 (CPD=1) New challenges for the treatment of diabetes mellitus	10.30 – 11.30 (CPD=1) Epigenetic mechanisms in cell- and drug based heart failure therapies	10.30 – 11.30 (CPD=1) GRIP – Global training programmes in paediatric pharmacology	10.30 – 11.30 (CPD=1) Innovative methods for assessing drug toxicity and efficacy	

壁報論文發表及標題 (附件二)



The Protective and Therapeutic Effects of Cortex Magnoliae on Neuronal Damage and Abnormal Behaviors Induced by Neurotoxin

Hsiao-Yu Liao¹, Pei-Wen Chu¹ and Ming-Huan Chan¹
1. Institute of Neuroscience, National Chengchi University, Taipei, Taiwan

Abstract
Cortex Magnoliae, the bark of *Magnolia officinalis*, has been prescribed in the traditional herbal medicine to treat a variety of mental disorders including depression. The main constituents of cortex Magnoliae contain the biphenyl compounds such as honokiol and magnolol. Both biphenyl compounds were shown to have the neuronal protective effect which is related to the anti-oxidation, anti-inflammation, and anti-excitatory toxicity. Thus, it was proposed that cortex Magnoliae may act as the potential therapeutic agent for the treatment of neurodegenerative disorders such as Parkinson's disease (PD). The aim of the present study was to examine whether cortex Magnoliae exhibits the neuroprotective and therapeutic action against the neuronal toxicity and behavioral deficits in learning, memory, and motor function induced by neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). Our results showed that MPTP and cortex Magnoliae did not affect mouse coordination and balance in beam walking test. However, cortex Magnoliae can improve the cognitive impairments determined by novel-location recognition task (NLRT) and novel-object recognition task (NORT) in MPTP-induced PD mouse model. Additional, cortex Magnoliae can restore MPTP-induced loss of dopaminergic neurons in striatum. Therefore, the preliminary results suggest that cortex Magnoliae may be a novel candidate for the treatment of Parkinson's disease in the future. The pharmacological mechanism of cortex Magnoliae in PD treatment needs further study.

Materials and Methods

- Male ICR mice (30 g-35 g) were administered with MPTP (25 mg/kg, i.p.) once daily for 5 consecutive days to induce neurotoxicity and behavioral impairment.
- In co-treatment group, mice were orally administrated with cortex Magnoliae (100 or 300 mg/kg) 1 hour before MPTP injection for 5 days and then followed by oral administration of cortex Magnoliae alone for consecutive 14 days.
- In post-treatment group, mice were orally administered with cortex Magnoliae (100 or 300 mg/kg) for consecutive 14 days after the final injection of MPTP. Mice in control group were injected with saline (0.9%, i.p.) and orally administrated with corn oil.

Fig 1. Protocol of agent administration to mice

Fig 5. Effects of co-treatment (A) and post-treatment of cortex Magnoliae (B) on MPTP-induced impairment of recognition memory after first MPTP injection 7 and 8 days. Values are the mean ± S.E.M. Data were statistically analyzed by one-way ANOVA and post-hoc Student-Newman-Keuls test. **P<0.01, ***P<0.001 as compared with control group. **P<0.01, ***P<0.001 as compared with MPTP treatment. STM: Short-term memory, LTM: Long-term memory.

Results

> Behavioral experiments:

Fig 2. Protocol of agent administration to mice for behavioral tests.

Fig 6. Effects of co-treatment (A) and post-treatment of cortex Magnoliae (B) on MPTP-induced impairment of recognition memory after first MPTP injection 15 and 16 days. Values are the mean ± S.E.M. Data were statistically analyzed by one-way ANOVA and post-hoc Student-Newman-Keuls test. **P<0.01, ***P<0.001 as compared with control group. **P<0.01, ***P<0.001 as compared with MPTP treatment. STM: Short-term memory, LTM: Long-term memory.

> Immunohistochemistry:

Fig 7. Effects of co-treatment or post-treatment of cortex Magnoliae on MPTP-induced impairment of recognition memory after last orally Cortex Magnoliae 7 days. Values are the mean ± S.E.M. Data were statistically analyzed by one-way ANOVA and post-hoc Student-Newman-Keuls test. **P<0.01, ***P<0.001 as compared with control group. **P<0.01, ***P<0.001 as compared with MPTP treatment. STM: Short-term memory, LTM: Long-term memory.

壁報論文摘要及內容(附件三)

The protective and therapeutic effects of cortex Magnoliae on neuronal damage and behavioral deficits induced by neurotoxin

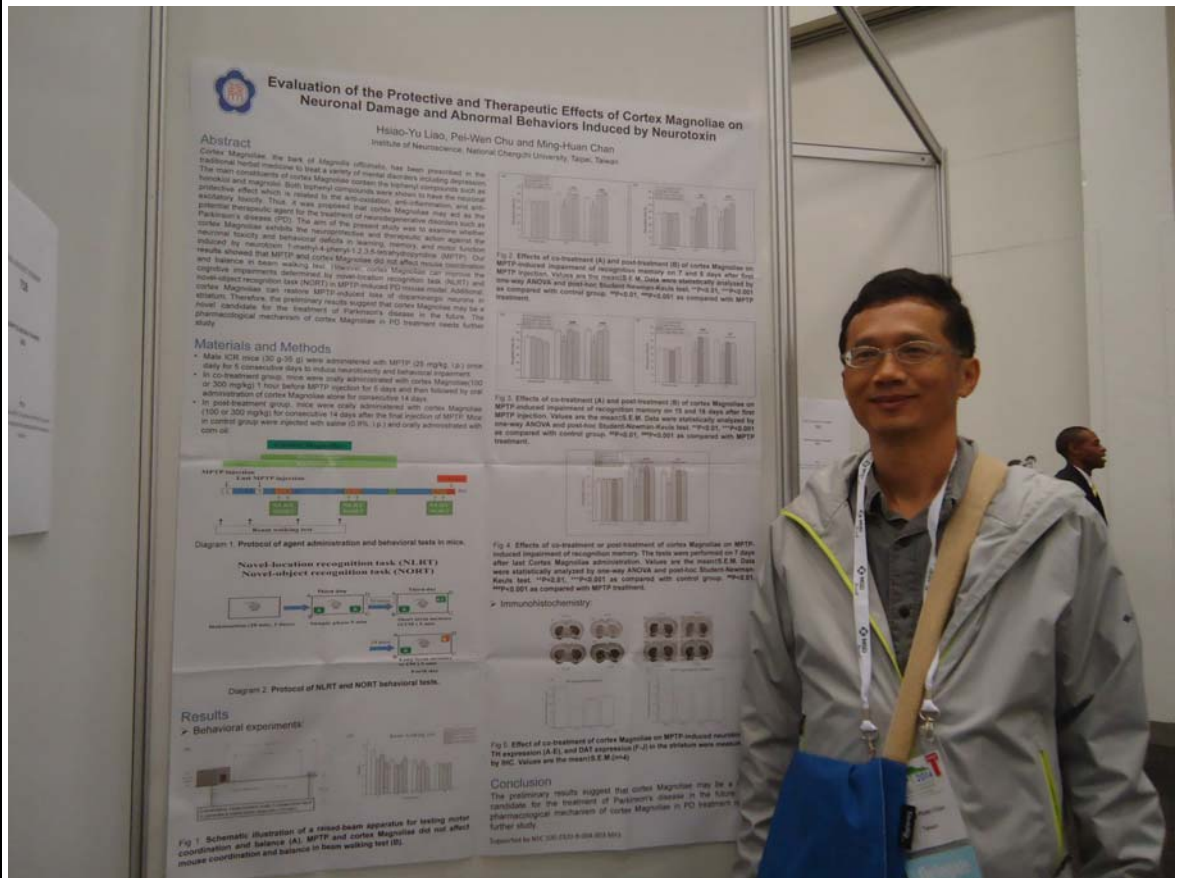
Hsiao-Yu Liao, Pei-Wen Chu and Ming-Huan Chan*

Institute of Neuroscience, National Chengchi University, Taipei, Taiwan

Cortex Magnoliae, the bark of *Magnolia officinalis*, has been prescribed in the traditional herbal medicine to treat a variety of mental disorders including depression. The main constituents of cortex Magnoliae contain the biphenyl compounds that were shown to have the anti-oxidation, anti-inflammation, and anti-excitatory toxicity leading to neuronal protection. Thus, it was proposed that cortex Magnoliae may act as the potential therapeutic agent for the treatment of neurodegenerative disorders such as Parkinson's disease (PD). The aim of the present study was to examine whether cortex Magnoliae exhibits the neuroprotective and therapeutic action against the neuronal toxicity and behavioral deficits in learning, memory, and motor function induced by neurotoxin. Male ICR mice (25-30 g) were administered with 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP, 25mg/kg, i.p.) once daily for 5 consecutive days to induce neurotoxicity and behavioral impairment. In co-treatment group, male mice were orally administrated with cortex Magnoliae (100 or 300 mg/kg) 1 hour before MPTP injection for 5 days and then followed by cortex Magnoliae alone for consecutive 14 days. Alternatively, mice in post-treatment group were orally administered with cortex Magnoliae for consecutive 14 days after the final injection of MPTP. Our results showed that MPTP and cortex Magnoliae did not affect mouse coordination and balance in beam walking test. However, cortex Magnoliae can improve the cognitive impairments determined by novel-location recognition task (NLRT) and novel-object recognition task (NORT) in MPTP-induced PD mouse model. Additionally, cortex Magnoliae can reserve MPTP-induced loss of dopaminergic neurons in striatum. Therefore, the preliminary results suggest that cortex Magnoliae may be a novel candidate for the treatment of Parkinson's disease in the future. The pharmacological mechanism of cortex Magnoliae in PD treatment needs further

study.

壁報會場(附件四)



(此研究成果尚未發表出版於國際期刊、請勿公開)

建議事項參採情形	出國人建議		單位主管覆核		
	建議採行	建議研議	同意立即採行	納入研議	不採行
1. 於本國舉辦生物醫學相關國際會議時，在會前或會後邀請神經藥理或神經科學研究領域之專家學者到本校神科所進行學術		V			

交流，精進研究量。					
2.鼓勵教師及學生參加國際會議並發表研究成果，增進國際觀及國際能見度。	V				
3. 推動本校神經科學在大學部的學程，以發展神經科學應用於人文社科的轉譯人才		V			

出國人簽名：詹銘煥

日期：7, 28, 2014

連絡人：

分機：67568

出國報告審核表

出國報告名稱：第 17 屆世界基礎及臨床藥理學會議		
出國人姓名	職稱	服務單位
詹銘煥	教授	神經科學所
出國類別	<input type="checkbox"/> 考察 <input type="checkbox"/> 進修 <input type="checkbox"/> 研究 <input type="checkbox"/> 實習 <input checked="" type="checkbox"/> 其他 國際會議 (例如國際會議、國際比賽、業務接洽等)	
出國期間：2014 年 7 月 12 日至 2014 年 7 月 18 日		報告繳交日期：2014 年 7 月 28 日
計畫主辦機關審核意見	<input type="checkbox"/> 1.依限繳交出國報告 <input type="checkbox"/> 2.格式完整(本文必須具備「目的」、「過程」、「心得及建議事項」) <input type="checkbox"/> 3.無抄襲相關出國報告 <input type="checkbox"/> 4.內容充實完備 <input type="checkbox"/> 5.建議具參考價值 <input type="checkbox"/> 6.送本機關參考或研辦 <input type="checkbox"/> 7.送上級機關參考 <input type="checkbox"/> 8.退回補正，原因： <input type="checkbox"/> 不符原核定出國計畫 <input type="checkbox"/> 以外文撰寫或僅以所蒐集外文資料為內容 <input type="checkbox"/> 內容空洞簡略或未涵蓋規定要項 <input type="checkbox"/> 抄襲相關出國報告之全部或部分內容 <input type="checkbox"/> 電子檔案未依格式辦理 <input type="checkbox"/> 未於資訊網登錄提要資料及傳送出國報告電子檔 <input type="checkbox"/> 9.本報告除上傳至出國報告資訊網外，將採行之公開發表： <input type="checkbox"/> 辦理本機關出國報告座談會(說明會)，與同仁進行知識分享。 <input type="checkbox"/> 於本機關業務會報提出報告 <input type="checkbox"/> 其他_____	
審核人	一級單位主管	機關首長或其授權人員

說明：

- 一、各機關可依需要自行增列審核項目內容，出國報告審核完畢本表請自行保存。
- 二、審核作業應儘速完成，以不影響出國人員上傳出國報告至「政府出版資料回應網公務出國報告專區」為原則。