Dr. Power Stem

Anti-Aging & Stem Cell Research

青春不老宝典

Rejuvenation Treasury

百威幹細胞博士生醫研發

Dr. Power Stem

Natural Precious Pure Healthy

Some of entrepreneurs from Swiss, USA and Taiwan consistently consider the excellent Biomedical-Stem Research techniques of Taiwan experts, and decided to integrate the whole resources of PhD, MD, and Professors. In July 2016, Dr. Power Stem was established and its RD team is composed of experts in anti-aging stem cells, genetic nutrition, neural health, flow liposome, protein and peptide, strategic marketing, product design, public relation, etc. This excellent RD team, headed by Dr. PS, leads this group to globally market made-in Taiwan Swiss brand. Through integration of bio-medical patent, manufacturing, packaging and marketing, brand establishment, quality control, Dr. Power Stem insists on the health values of high technology, high quality, green, non-toxic, and on bringing the best products to its customers. Dr. Power Stem insists forever on the direction of the Dream Kingdom, a place full of health, happiness, and welfare.

A SWISS BRAND
MADE IN TAIWAN
GLOBAL MARKETING

六大專利、六項技術

AMBASSADOR CLUB

AContact

Magazin des Ambassador Club **Schweiz**

Ausgabe 3 / September 2016

Kurz vor Redaktionsschluss kam noch unerwarteter Besuch nach Baden: Frau Professor Dr. Claire Lin aus Taiwan überbrachte die besten Grüsse vom RAC Taiwan und ihrem Mann, der dort Präsident ist.

Ihr Kurzaufenthalt wurde durch Mario Delvecchio und Ruedi Burger begleitet.
Auch so kann man Grenzen überwinden!

60. DELEGIERTEN-VERSAMMLUNG CHAC

Seiten 10/11

INTERNATIONALES BODENSEETREFFEN

Seite 24

Dr. Power Stem Research Center

/Ant-Aging Ambassador (AAA) International Association

Cooperative Research in National Ilan University

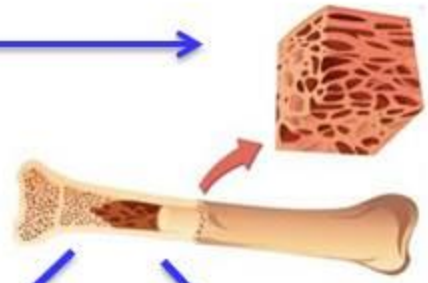
Bio-resource Research Building

Research Lab.

Biotech Food Factory

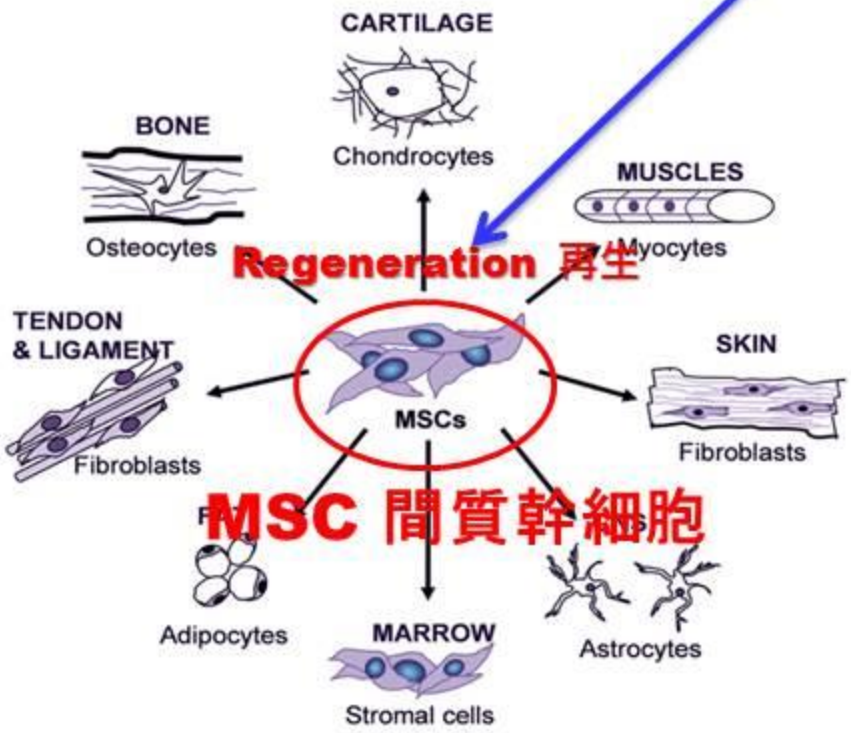
Biomedical Facilities

CD34 活性蛋白
(G-CSF + SCF Active protein)

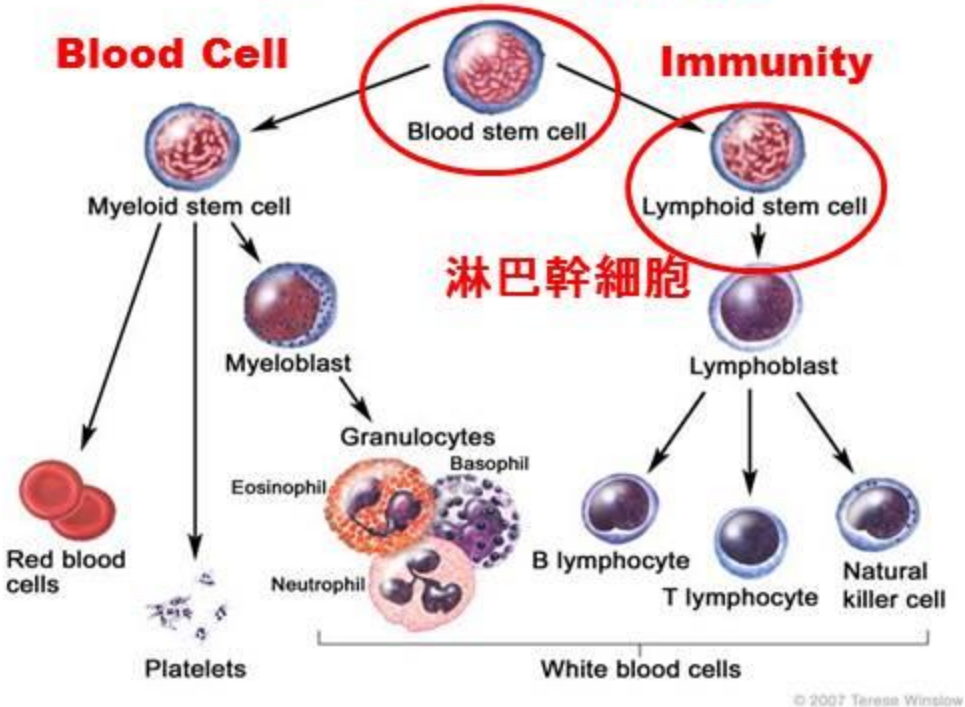


科學期刊已証實

1. 皮膚修復
2. 中風修復
3. 化療修復
4. 手術修復



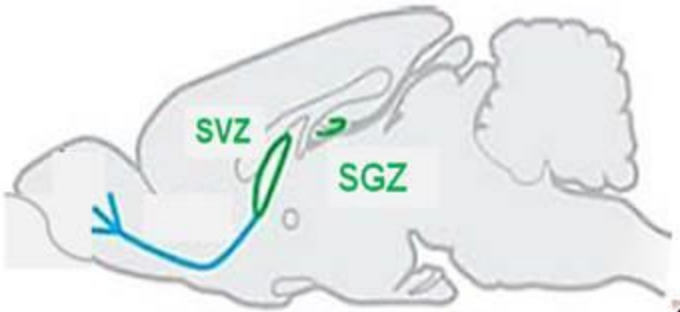
CD34 造血幹細胞

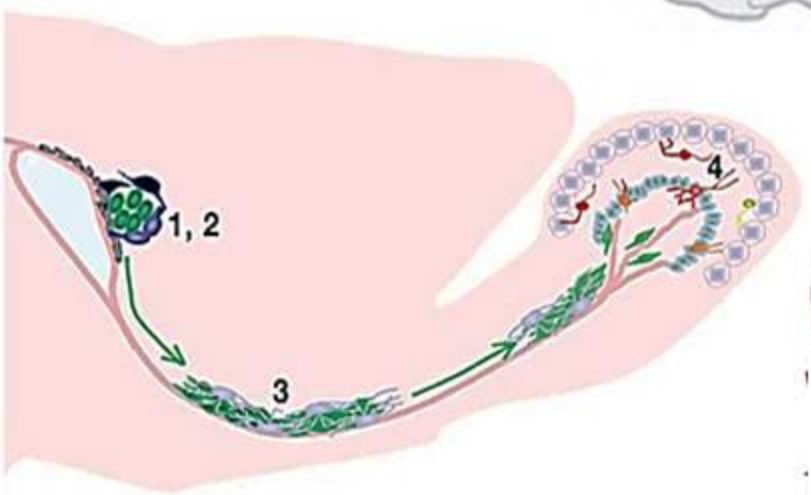


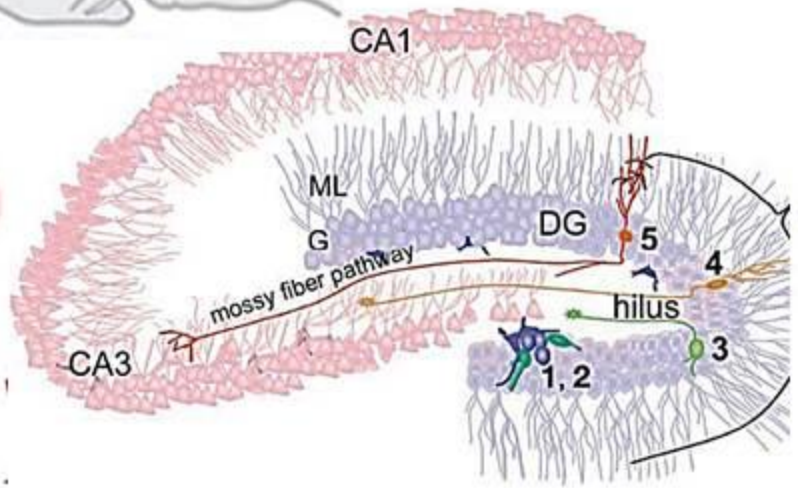
Neurogenesis 神經再生

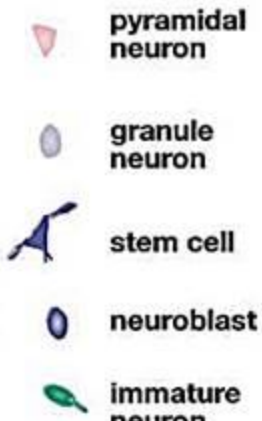
- **"No new neurons after birth" (Cajal, 1913)**
- **The developing tracking techniques**
  - **[H]-thymidine**
  - **BrdU**
- **Adult neural stem cell** (Ming and Song, 2005)

SVZ (subventricular zone, 下腦室)　　SGZ (subgranular zone, 海馬迴)





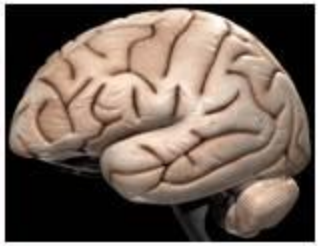




SCF (Stem Cell factor) and G-CSF (Granulocyte-Colony Stimulating Factor)

SCF & G-CSF
活性蛋白

1 **Pass through the blood-brain barrier**
通過血腦障蔽



(Zhao *et al.*, 2007)

2 **Anti-apoptosis**
抗凋亡

(Solaroglu *et al.*, 2006; Dhandapani *et al.*, 2005)

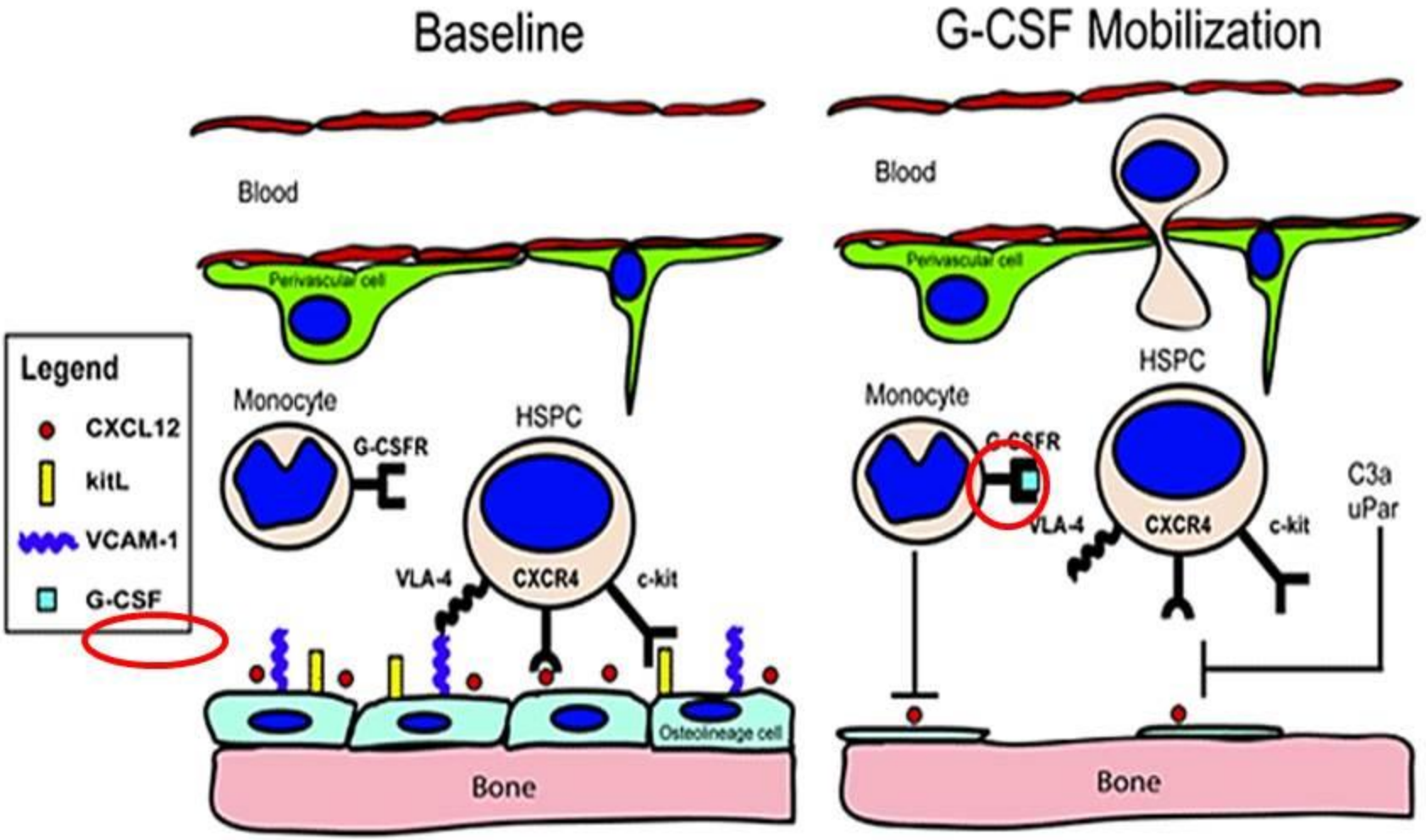
3 **Neurogenesis**
神經再生

(Jung *et al.*, 2006; Shyu et al., 2004; Jin *et al.*, 2002)

4 **Proliferation, Mobilization, and Differentiation of HSC and MSC**
造血幹細胞及間質幹細胞再生

(Li *et al.*, 2001; Hess *et al.*, 2002)

G-CSF is able to mobilize CD34 stem cells from bone marrow into blood. 趨動骨髓幹細胞到血液中



阿茲海默失智症病患血液中 G-CSF 偏低

J Alzheimers Dis. 2009;17(1):115-23. doi: 10.3233/JAD-2009-1017.

Decreased plasma levels of granulocyte-colony stimulating factor (G-CSF) in patients with early Alzheimer's disease.

Laske C[1], Stellos K, Stransky E, Leyhe T, Gawaz M.

Author information

Abstract

Alzheimer's disease (AD) is characterized by massive neuronal cell loss in the brain. Granulocyte-colony stimulating factor (G-CSF) is a hematopoietic growth factor that promotes neuroprotective effects and supports neurogenesis in the brain. In the present study, we found significantly lower G-CSF plasma levels in 50 early AD patients in comparison with 50 age-matched healthy controls. In AD patients, G-CSF levels showed a significant inverse correlation with amyloid-beta (Abeta1-42) levels in cerebrospinal fluid, but not with levels of tau protein in cerebrospinal fluid or Mini-Mental Status Examination scores. In addition, G-CSF plasma levels were significantly inversely correlated with age in AD patients and healthy controls. In conclusion, decreased G-CSF plasma levels in early AD patients may contribute to a deficient hematopoietic brain support with putative pathogenic relevance. Further studies are needed to examine whether a modulation of hematopoietic growth factors such as G-CSF could be a promising new therapeutic strategy for AD.

PMID: 19494436 DOI: 10.3233/JAD-2009-1017

[PubMed - indexed for MEDLINE]

Pilot study of granulocyte-colony stimulating factor for treatment of Alzheimer's disease.

Sanchez-Ramos J, Cimino C, Avila R, Rowe A, Chen R, Whelan G, Lin X, Cao C, Ashok R. J Alzheimers Dis. 2012;31(4):843-55.

以 G-CSF 進行阿茲海默失智症試驗成功

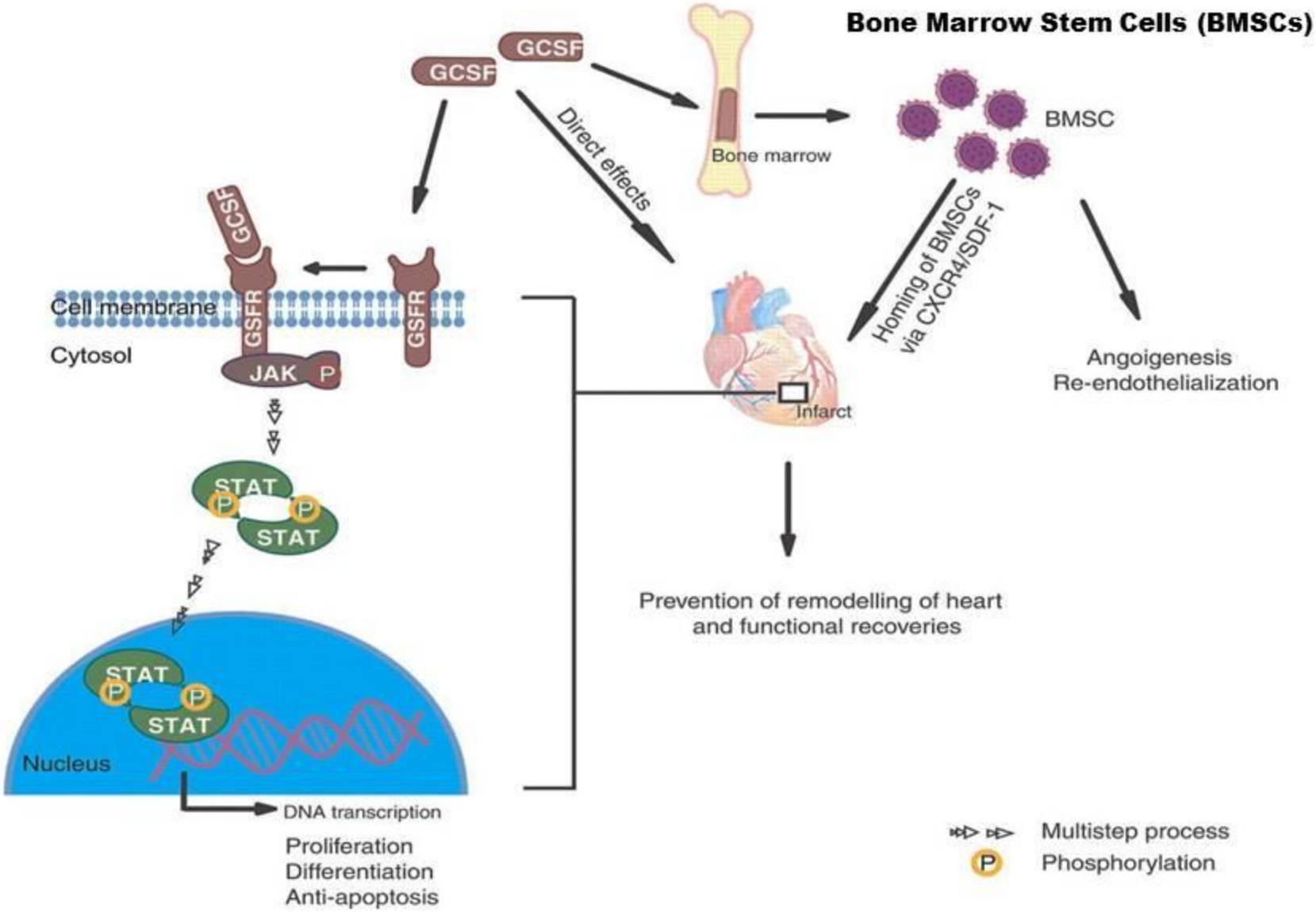
Granulocyte-colony stimulating factor (G-CSF) enhances recovery in mouse model of Parkinson's disease.

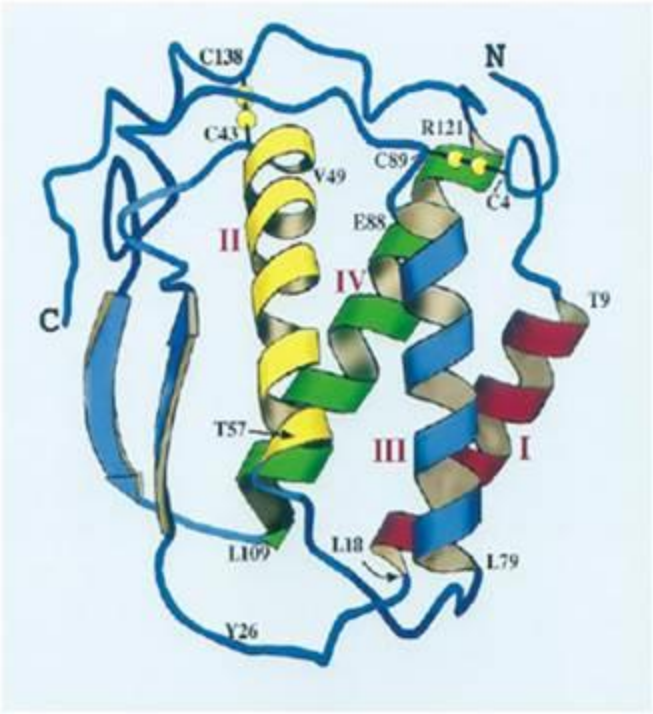
Song S, Sava V, Rowe A, Li K, Cao C, Mori T, Sanchez-Ramos J. Neurosci Lett. 2011 Jan 7;487(2):153-7.

以 G-CSF 進行巴金森氏症小鼠修復

G-CSF has been proven to prevent the infarct of heart and functional recoveries.

G-CSF 已証實可預防心肌梗塞與修復





Stem Cell Factor (SCF)

- ✓ **SCF is a peptide of 165 amino acids, which about 21 kDa.**
- ✓ **This cytokine plays an important role of proliferation, migration, and differentiation of stem cells.**

Hematopoiesis (造血作用)

Spermatogenesis (生精作用)

Mesenchyme stem cell regeneration (間葉幹細胞再生)

Neurogenesis (神經細胞再生)

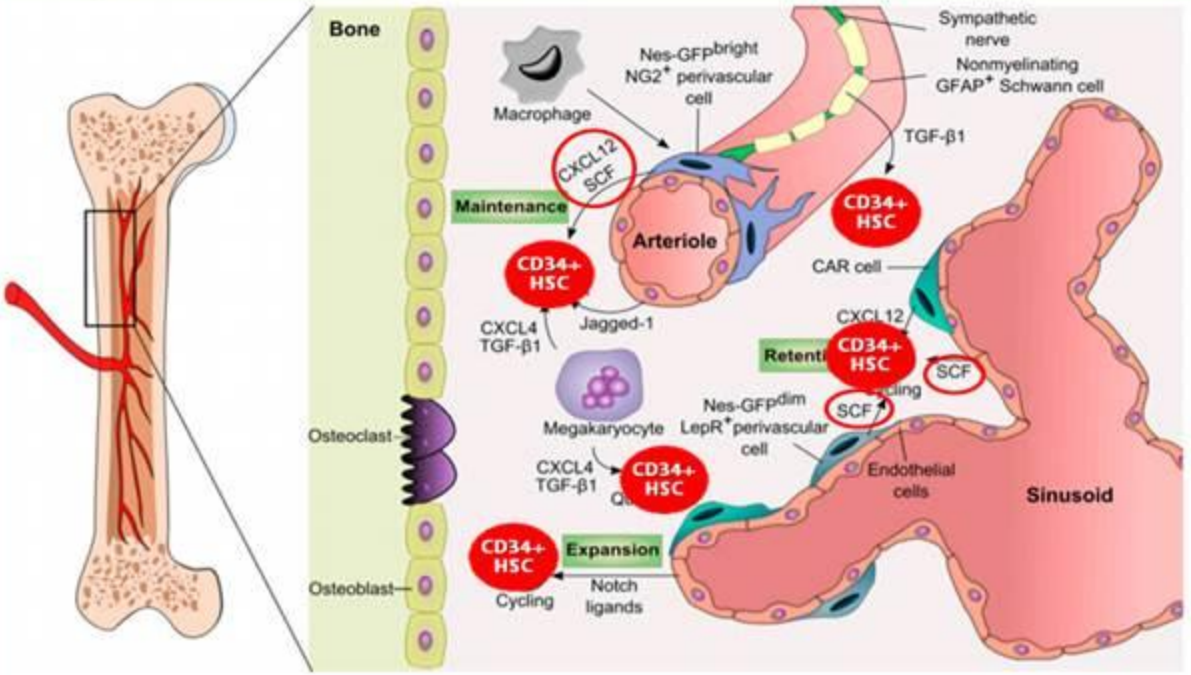
SCF is able to proliferate CD34 stem cells in niches of bone marrow. 促進骨髓幹細胞增生

Review Series

HEMATOPOIETIC STEM CELLS

Making sense of hematopoietic stem cell niches

Philip E. Boulais[1,2] and Paul S. Frenette[1-3]



G-CSF + SCF accelerates the stroke recovery.

二者加乘中風修復

SCIENCE VOLUNTEER | WARNING SIGNS | Search

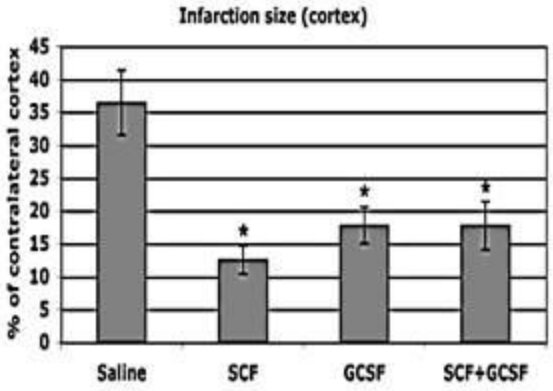
Stroke

ORIGINAL CONTRIBUTIONS

Brain Repair by Hematopoietic Growth Factors in a Rat Model of Stroke

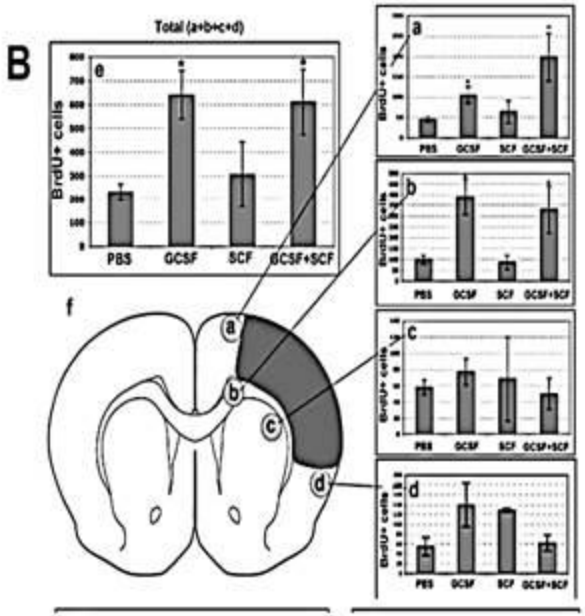
Li-Ru Zhao, Seema Singhal, Wei-Ming Duan, Jayesh Mehta and John A. Kessler

A



G-CSF + SCF 改善中風後的梗塞區塊

B



G-CSF + SCF 加速中風後的神經再生

After 6 months of stroke, SCF+G-CSF still shows the good recovery.

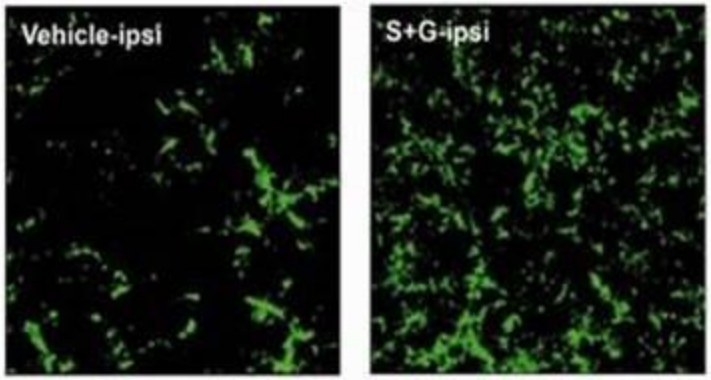
中風 Stroke 後 6 個月仍有治療希望：SCF + GCSF 效果顯著

ASN NEURO
ABOUT | EDITORIAL BOARD | INFO FOR AUTHORS | SUBMISSION
ASN Neuro. 2016 Jul-Aug; 8(4): 1759091416655010. PMCID: PMC4984318
Published online 2016 Aug 9. doi: 10.1177/1759091416655010

中風復健分為 3 個階段

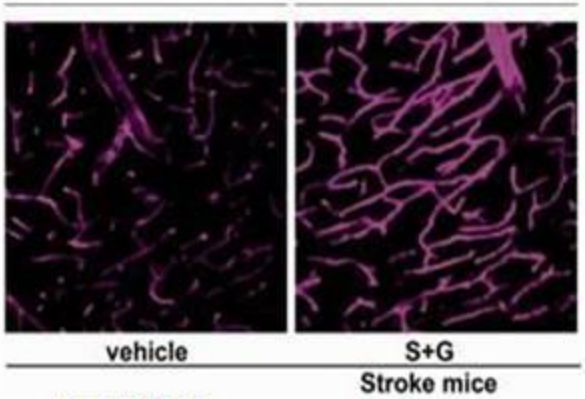
生命跡象穩定超過 24 小時	1 周內~3 個月	3 個月後
急性	亞急性	慢性

1. 如果錯過黃金治療期，中風 6 個月後才接受復健治療，則效果有限。
2. 已有科學報告指出：顱內出血中風的小鼠，無接受治療。直至 6 個月後，再處理 SCF+G-CSF，仍然具有神經再生及血管新生的功能，有助於中風後的持續復原。



中風對照組　中風 6 月後 S+G 處理組

中風區神經細胞再生 (PSD-95)



中風對照組　中風 6 月後 S+G 處理組

中風區血管生成

ASN Neuro. 2016 Jul-Aug
Repairing the Brain by SCF+G-CSF Treatment at 6 Months Postexperimental Stroke Mechanistic Determination of the Causal Link Between Neurovascular Regeneration and Motor Functional Recovery
Lili Cui,[1,2] Dandan Wang,[1] Sandra McGillis,[1] Michele Kyle,[1] and Li-Ru Zhao[1,2]

The neuron-regeneration and vascular-regeneration in the infarct area

SCF + G-CSF decreases the Aβ deposits in AD mice.

二者加乘失智症修復

Li et al. Alzheimer's Research & Therapy 2011, 3:8
http://alzres.com/content/3/2/8

RESEARCH Open Access

Stem cell factor and granulocyte colony-stimulating factor reduce β-amyloid deposits in the brains of APP/PS1 transgenic mice

Bin Li[1], Maria E Gonzalez-Toledo[1], Chun-Shu Piao[1], Allen Gu[2], Roger E Kelley[1,3] and Li-Ru Zhao[1,4*]

Abstract

Introduction: Alzheimer's disease (AD) is widely recognized as a serious public health problem and heavy financial burden. Currently, there is no treatment that can delay or stop the progressive brain damage in AD. Recently, we demonstrated that stem cell factor (SCF) in combination with granulocyte colony-stimulating factor (G-CSF) (SCF +G-CSF) has therapeutic effects on chronic stroke. The purpose of the present study is to determine whether SCF +G-CSF can reduce the burden of β-amyloid deposits in a mouse model of AD.

Methods: APP/PS1 transgenic mice were used as the model of AD. To track bone marrow-derived cells in the brain, the bone marrow of the APP/PS1 mice was replaced with the bone marrow from mice expressing green fluorescent protein (GFP). Six weeks after bone marrow transplantation, mice were randomly divided into a saline control group and a SCF+G-CSF-treated group. SCF in combination with G-CSF was administered subcutaneously for 12 days. Circulating bone marrow stem cells ($CD117^+$ cells) were quantified 1 day after the final injection. Nine months after treatment, at the age of 18 months, mice were sacrificed. Brain sections were processed for immunohistochemistry to identify β-amyloid deposits and GFP expressing bone marrow-derived microglia in the brain.

Results: Systemic administration of SCF+G-CSF to APP/PS1 transgenic mice leads to long-term reduction of β-amyloid deposition in the brain. In addition, we have also observed that the SCF+G-CSF treatment increases circulating bone marrow stem cells and augments bone marrow-derived microglial cells in the brains of APP/PS1 mice. Moreover, SCF+G-CSF treatment results in enhancement of the co-localization of bone marrow-derived microglia and β-amyloid deposits in the brain.

Conclusions: These data suggest that bone marrow-derived microglia play a role in SCF+G-CSF-induced long-term effects to reduce β-amyloid deposits. This study provides insights into the contribution of the hematopoietic growth factors, SCF and G-CSF, to limit β-amyloid accumulation in AD and may offer a new therapeutic approach for AD.

二者加乘肝臟修復

Molecular Biology Research Communications 2014;3(2):141-147

MBRC

Original Article

Open Access

Effects of associated SCF and G-CSF on liver injury two weeks after liver damage: A model induced by thioacetamide administration

Mohsen Esmaili[1], Durdi Qujeq[1, 2, *], Ali Asghar Yoonesi[1], Farideh Feizi[3], Mohammad Ranaee[4]

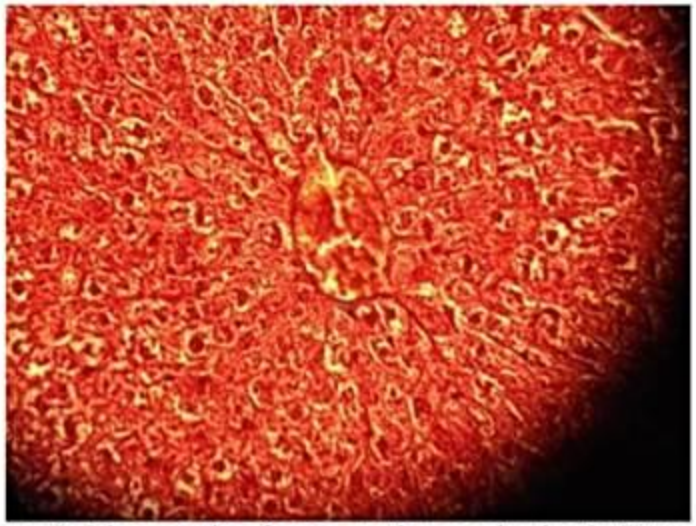


Figure 2: Liver section from a rat that received only TAA

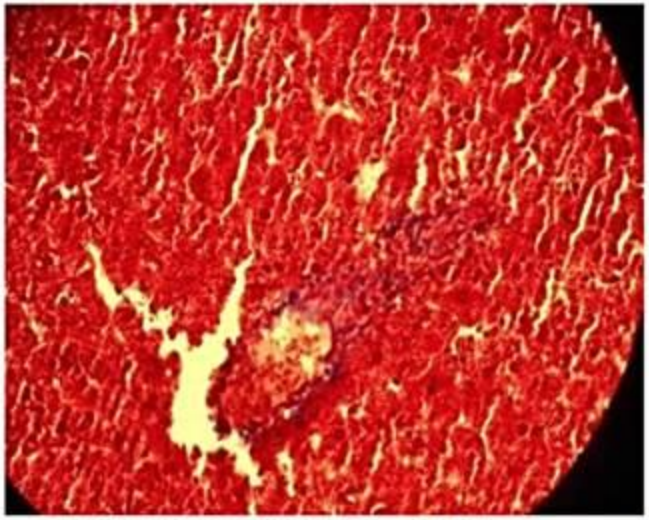
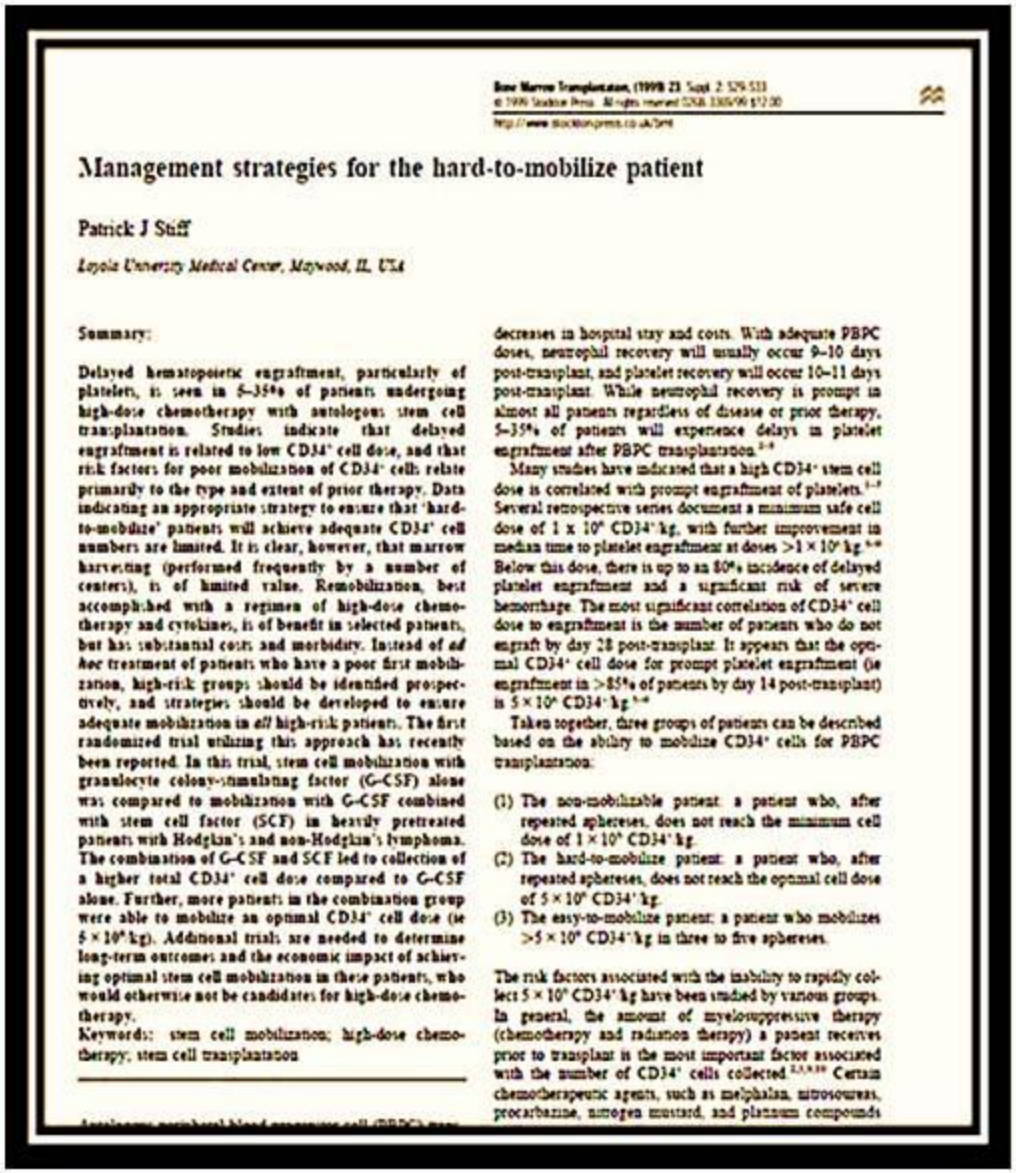


Figure 3: Liver section from a rat that received TAA plus G-CSF+SCF

二者加乘癌症化療時的修復



Bone Marrow Transplantation, (1999) 23, Suppl. 2, 529–533

http://www.stockton-press.co.uk/bmt

Management strategies for the hard-to-mobilize patient

Patrick J Stiff

Loyola University Medical Center, Maywood, IL, USA

Summary:

Delayed hematopoietic engraftment, particularly of platelets, is seen in 5–35% of patients undergoing high-dose chemotherapy with autologous stem cell transplantation. Studies indicate that delayed engraftment is related to low CD34⁺ cell dose, and that risk factors for poor mobilization of CD34⁺ cells relate primarily to the type and extent of prior therapy. Data indicating an appropriate strategy to ensure that 'hard-to-mobilize' patients will achieve adequate CD34⁺ cell numbers are limited. It is clear, however, that marrow harvesting (performed frequently by a number of centers), is of limited value. Remobilization, best accomplished with a regimen of high-dose chemotherapy and cytokines, is of benefit in selected patients, but has substantial costs and morbidity. Instead of *ad hoc* treatment of patients who have a poor first mobilization, high-risk groups should be identified prospectively, and strategies should be developed to ensure adequate mobilization in *all* high-risk patients. The first randomized trial utilizing this approach has recently been reported. In this trial, stem cell mobilization with granulocyte colony-stimulating factor (G-CSF) alone was compared to mobilization with G-CSF combined with stem cell factor (SCF) in heavily pretreated patients with Hodgkin's and non-Hodgkin's lymphoma. The combination of G-CSF and SCF led to collection of a higher total CD34⁺ cell dose compared to G-CSF alone. Further, more patients in the combination group were able to mobilize an optimal CD34⁺ cell dose (ie 5×10^6/kg). Additional trials are needed to determine long-term outcomes and the economic impact of achieving optimal stem cell mobilization in these patients, who would otherwise not be candidates for high-dose chemotherapy.

Keywords: stem cell mobilization; high-dose chemotherapy; stem cell transplantation

decreases in hospital stay and costs. With adequate PBPC doses, neutrophil recovery will usually occur 9–10 days post-transplant, and platelet recovery will occur 10–11 days post-transplant. While neutrophil recovery is prompt in almost all patients regardless of disease or prior therapy, 5–35% of patients will experience delays in platelet engraftment after PBPC transplantation.[1–4]

Many studies have indicated that a high CD34⁺ stem cell dose is correlated with prompt engraftment of platelets.[1–3] Several retrospective series document a minimum safe cell dose of 1×10^6 CD34⁺/kg, with further improvement in median time to platelet engraftment at doses $>1 \times 10^6$/kg.[4–6] Below this dose, there is up to an 80% incidence of delayed platelet engraftment and a significant risk of severe hemorrhage. The most significant correlation of CD34⁺ cell dose to engraftment is the number of patients who do not engraft by day 28 post-transplant. It appears that the optimal CD34⁺ cell dose for prompt platelet engraftment (ie engraftment in >85% of patients by day 14 post-transplant) is 5×10^6 CD34⁺/kg.[5,6]

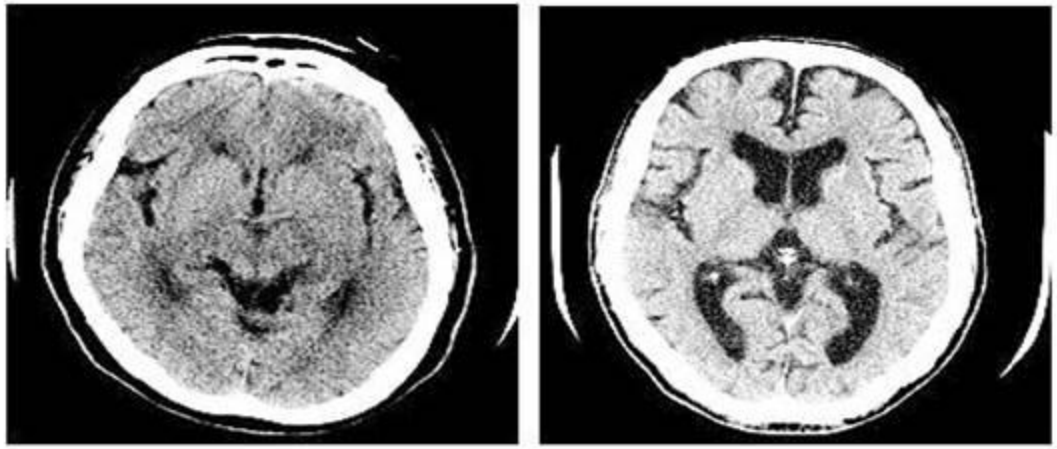
Taken together, three groups of patients can be described based on the ability to mobilize CD34⁺ cells for PBPC transplantation:

(1) The non-mobilizable patient: a patient who, after repeated aphereses, does not reach the minimum cell dose of 1×10^6 CD34⁺/kg.
(2) The hard-to-mobilize patient: a patient who, after repeated aphereses, does not reach the optimal cell dose of 5×10^6 CD34⁺/kg.
(3) The easy-to-mobilize patient: a patient who mobilizes $>5 \times 10^6$ CD34⁺/kg in three to five apheresis.

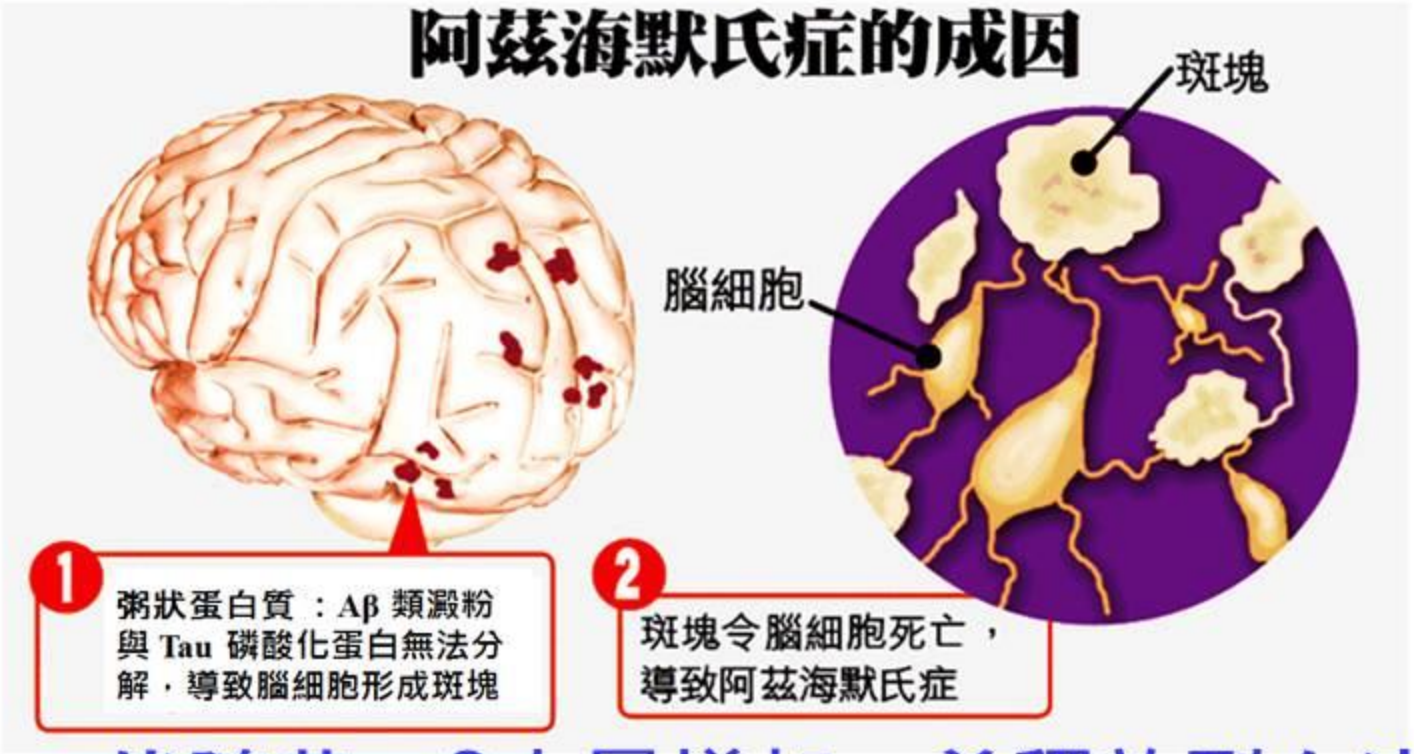
The risk factors associated with the inability to rapidly collect 5×10^6 CD34⁺/kg have been studied by various groups. In general, the amount of myelosuppressive therapy (chemotherapy and radiation therapy) a patient receives prior to transplant is the most important factor associated with the number of CD34⁺ cells collected.[3,7,8,10] Certain chemotherapeutic agents, such as melphalan, nitrosoureas, procarbazine, nitrogen mustard, and platinum compounds

Bone Marrow Transplantation
(1999) Suppl. 2. 529-533

CD34訊息:
兩種活性蛋白 G-CSF 及 SCF 共同使用，對於淋巴癌 (Lymphoma) 與 霍奇金病 (Hodgkin's Disease) 患者的自體復原力，確實比單獨使用 G-CSF 為更加顯著。



正常的腦部斷層　　萎縮腦（阿茲海默症）



ADD伴隨著 Aβ大量增加，並釋放到血液中

ADD血液檢測不同於ApoE4遺傳基因

- ApoE4：載脂蛋白基因 E 第四型基因（ApoE4）的變異型，這會提高阿茲海默症的發生率，但不一定會得病。只有小於 5% 的阿茲海默氏症患者是因基因遺傳而得到這個疾病。
- Neuro期刊: 12 週 (3個月)
- ApoE4 基因的老鼠，餵食 45% 的脂肪和 17%的蔗糖。很快出現妨礙認知和記憶功能的炎症標誌物血小板，大腦內發炎症。但餵食 10% 的脂肪和 7% 的蔗糖的健康飲食，大腦內則沒有出現這種情況。
- 2. ApoE4 增加環蛋白 A (cyclophillin A)，破壞血管，導致發炎物質以及毒素進入腦細胞。

阿茲海默失智症血液檢測: 準確度 94%

ADD伴隨著 Aβ大量增加，並釋放到血液中可做為血液指標

中華民國專利證書

發明第 I622771 號

發明名稱：診斷阿茲海默症方法

專利權人：百威研發股份有限公司

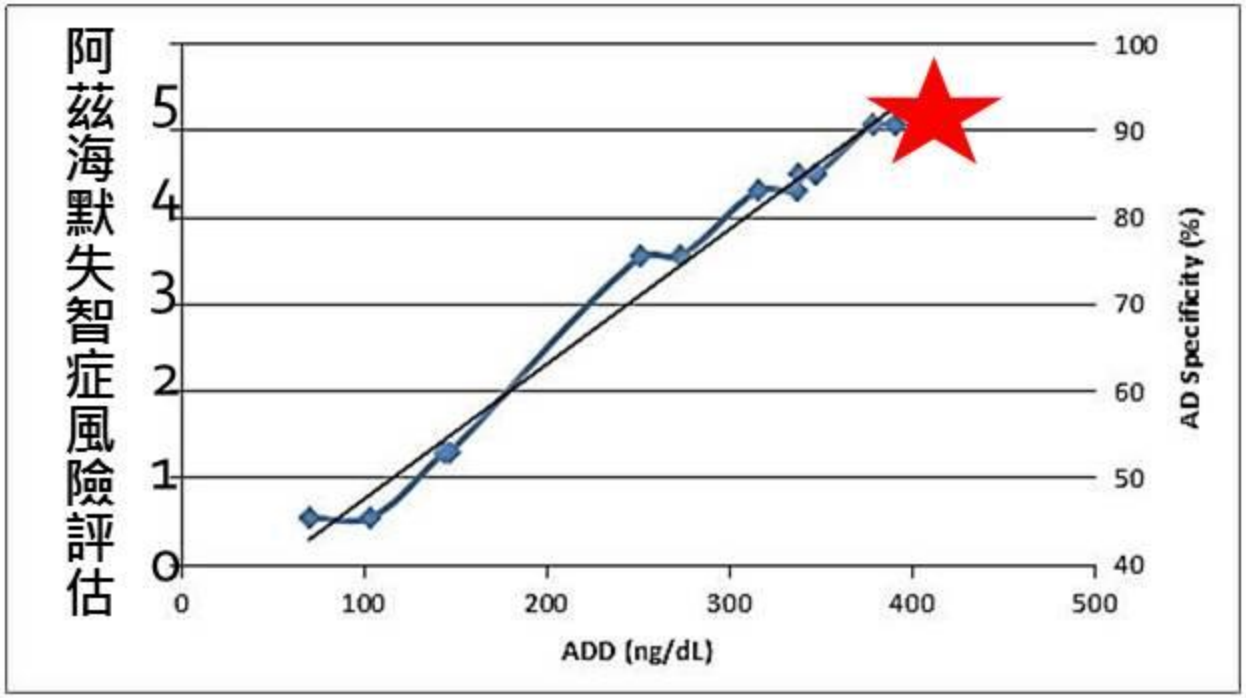
發明人：林信財、蔡澄偉

專利權期間：自2018年5月1日至2036年10月26日止

上開發明業經專利權人依專利法之規定取得專利權

經濟部智慧財產局 局長 洪淑敏

中華民國 107 年 5 月 1 日



1. 血液檢測: 45 歲以後每年定期檢測
2. ADD血液檢測，並非基因檢測 (ApoE4)
3. 提早 10-15 年的保健

多食用天然植化素 ~ 抗發炎

- ANTIOXIDANT ACTIVITY 抗氧化
- ANTI-CANCER 抗癌
- IMPROVING METABOLIC SYNDROME
  - ~ HIGH BLOOD PRESSURE 高血壓
  - ~ HIGH BLOOD LIPID 高血脂
  - ~ HIGH BLOOD SUGAR 高血糖
- PREVENTING FROM CARDIOVASCULAR DISEASES, STROKE, AND DIABETES 避免心血管疾病、中風及糖尿病
- BURNING BELLY FAT 燃燒腹部內臟脂肪

延緩老化與神經退化性疾病

1. 抗發炎:

- Phytochemicals 植化素
- 短、中鏈脂肪酸

2. 活性蛋白修復訊息:

- CD34 活性蛋白(G-CSF + SCF)