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樹下老人 Logo 很眼熟嗎？



The original Non Solus mark of Isaac Elsevir was used for the first time in Leyden in 1620.



ELSEVIER

- 1620年Elsevier的logo原始初稿首度誕生
- 榆樹 & 葡萄藤：象徵出版的豐碩智慧
- 老人：象徵研究的學者
- **Non Solus**：
 - 拉丁字 - 原意為Not alone（研究不孤獨）
 - 意涵：研究學者（老人）與 出版成果（榆樹）需相輔相成而共存
- ScienceDirect 為Elsevier的全文資料庫平台名稱，簡稱SDOL

ScienceDirect (SDOL) 簡介及其功能特點

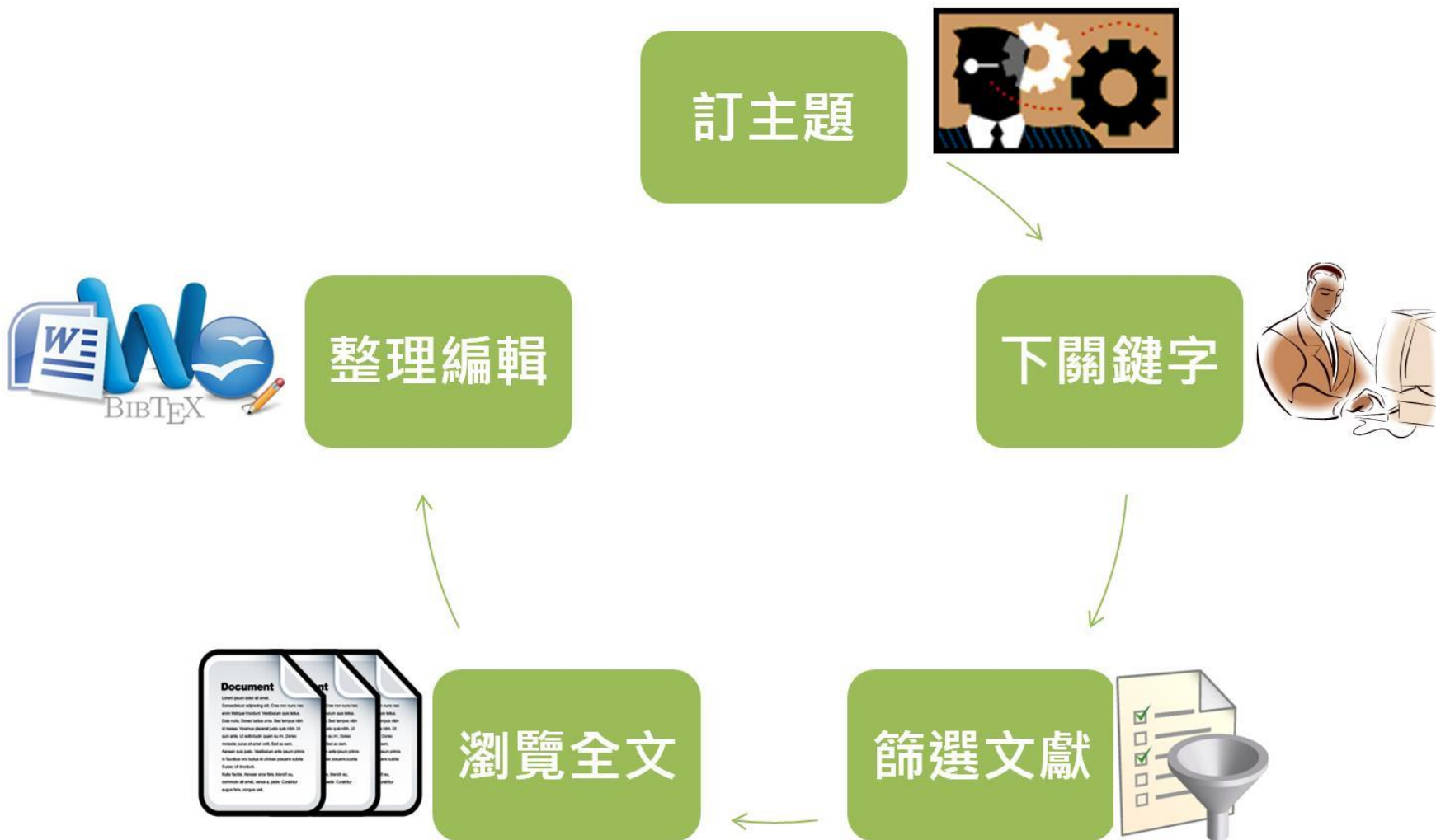
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General Overview



	屬性	學科領域	資料類型	特色	功能
Science Direct	全文資料庫 [世界最大]	綜合型	Elsevier旗下期刊、電子書、參考工具書	<ul style="list-style-type: none"> 一次下載25篇PDF, 並客製化檔名 圖片另存ppt 檔案, 並自動匯入來源出處 表格另存excel檔 	<ul style="list-style-type: none"> 匯出至書目管理軟體, 如 Mendeley ,EndNote 搶先看Articles in Press文章 未來相關文章主動找您(alert設定) 參考文獻提供全文連結與被引用次數 文章被引用次數

學術文獻回顧與分析程序



Association of high body lead store with severe intracranial carotid atherosclerosis

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ABSTRACT

Objective: Lead is involved in the pathogenesis of atherosclerosis and hypertensive disease and may be related to cerebrovascular disease. We studied the association of body lead level with stroke subtypes and severity of cerebral atherosclerosis in order to identify the significance of lead exposure to cerebrovascular disease.

Methods: From April, 2002 to March, 2005, we studied the lead level in all patients receiving digital subtraction angiography. Diameter stenosis at extracranial carotid, intracranial carotid and vertebro-basilar system was calculated according to the NASCET criteria. A blood sample and a mobilization test of 72-h urine sample were collected for lead measurement.

Results: In a total of 213 subjects, 19 were free of stroke (blood lead level = 4.62 ± 2.41 $\mu\text{g/dL}$, body lead store = 39.04 ± 20.91 μg) and 194 were stroke patients (4.89 ± 2.75 $\mu\text{g/dL}$, 45.13 ± 29.8 μg ; all stroke vs. non-stroke, $P > 0.05$). In the 153 subjects with atherosclerotic origin, body lead store but not blood lead level in the intracranial carotid system was significantly higher in $\geq 50\%$ group than $< 50\%$ group (blood lead: 5.61 ± 3.02 $\mu\text{g/dL}$ vs. 4.80 ± 2.50 $\mu\text{g/dL}$, Student's t -test, $P = 0.129$; body lead store: 51.7 ± 27.0 μg vs. 41.9 ± 23.5 μg , Student's t -test, $P = 0.038$, multivariate logistic regression, odds ratio = 1.02, 95% CI: 1.00–1.03, $P = 0.043$). However, there was no significant association between lead level and stenotic severity in extracranial and vertebrobasilar systems ($P > 0.05$).

Conclusion: Our study demonstrated that long-term lead exposure as measured by body lead store might carry a potential risk of intracranial carotid atherosclerosis.

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1. Introduction

Previous studies indicated that lead has specific toxicities in the proliferation, fibrinolysis, and extracellular matrix formation of vascular endothelial and smooth muscle cells, resulting in vascular disorders such as atherosclerosis in experimental animals (Kaji, 2004). Lead may induce aortic atherosclerosis in pigeons (Revis et al., 1981) and stimulate the proliferation of cultured rabbit aortic smooth muscle cells in varying degrees (Lu et al., 1990). Lead can also stimulate the proliferation of the vascular smooth muscle cells and fibroblasts (Fujiwara et al., 1995) and inhibit the repair process of damaged endothelial cell layer (Fujiwara et al., 1997) in in vitro studies. Animal study showed that lead may cause severe injury to endothelium of brain vasculature (Bradbury and Deane, 1988;

Linnamagi and Kaasik, 1995) and induces cerebral microvascular dysfunction with following changes in cerebral blood flow (Linnamagi and Kaasik, 1995). Hence, it is likely that lead is involved in the pathogenesis of cerebral atherosclerosis and may be related to cerebrovascular disease.

Cerebrovascular disease or stroke has been one of the first three leading causes of death in the past four decades in Taiwan (Jeng and Su, 2007) and is more common in Taiwanese than in Whites (Hu et al., 1992; Goldstein et al., 2006). The distribution of cerebral atherosclerosis in stroke patients is different between races, and atherosclerosis of the larger extracranial arteries is more prevalent in Whites, while occlusive disease of the intracranial arteries is more often seen in patients of Black or oriental origin (Feldmann et al., 1990; Leung et al., 1993; Liu et al., 1996; Jeng and Su, 2007). Regarding stroke subtype, small vessel occlusion and large artery

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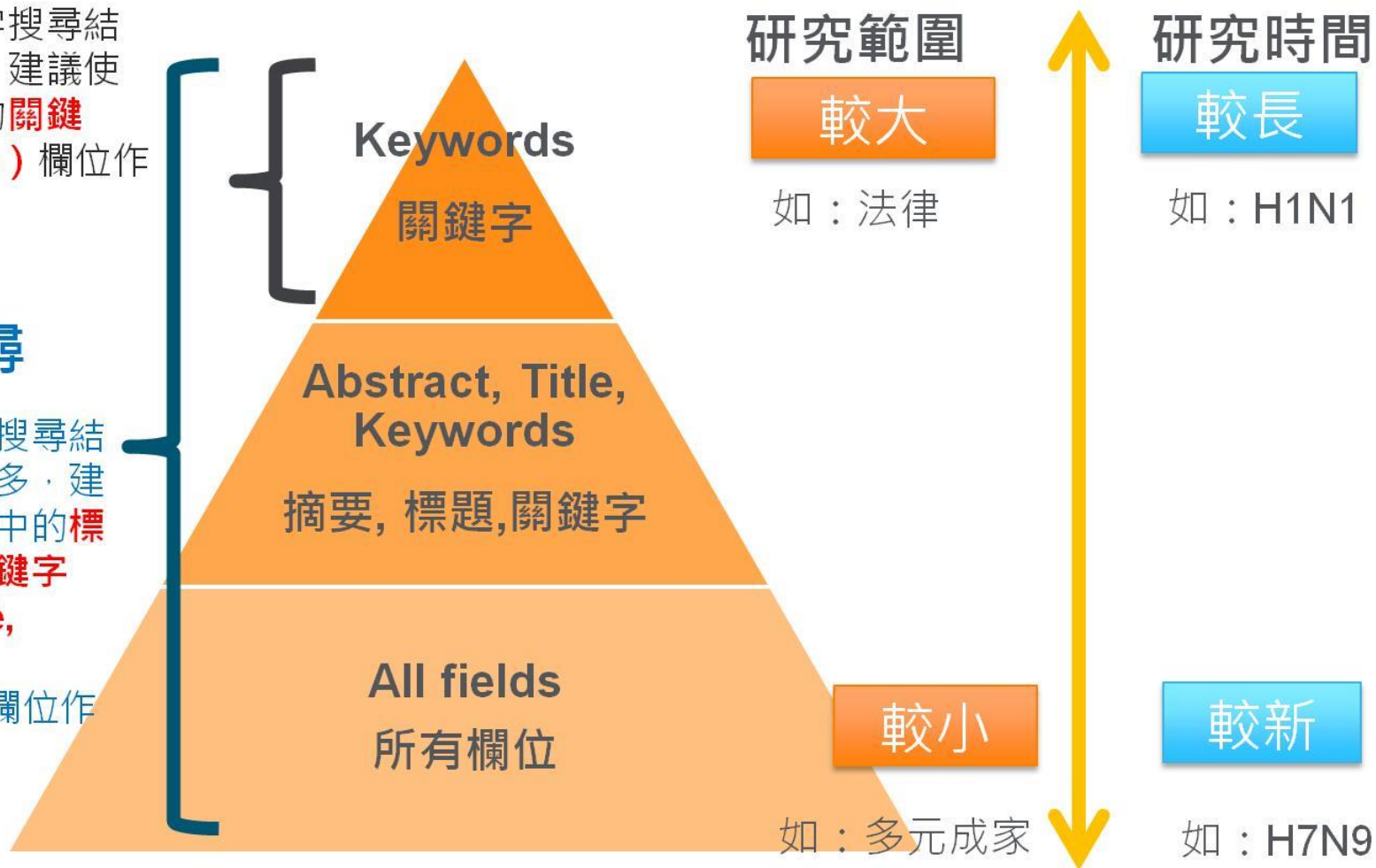
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 Falguni Pati, Jin-Hyung Shim, Jung-Seob Lee, Dong-Woo Cho

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The search results on the left include:

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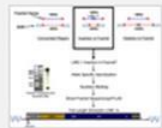
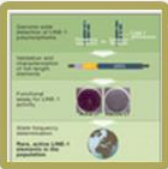
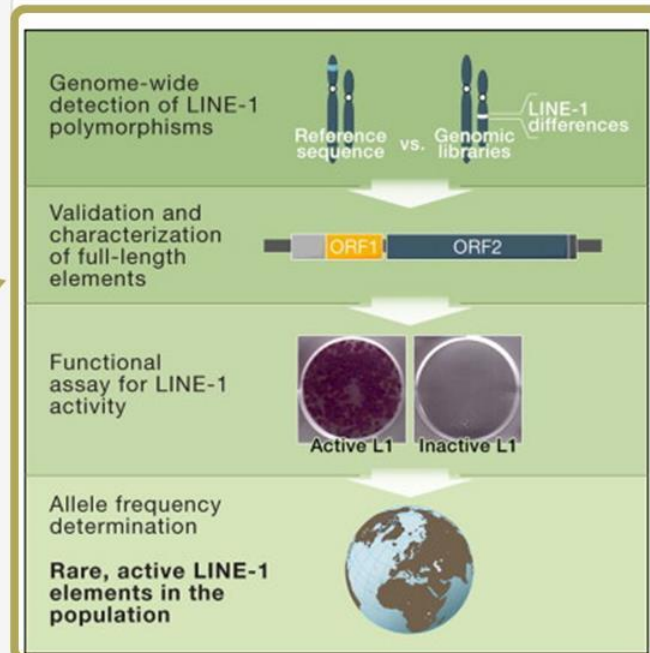


Table 1

Lineage	Active L1s	Inactive L1s
1	1	1
2	1	1
3	1	1
4	1	1
5	1	1
6	1	1
7	1	1
8	1	1
9	1	1
10	1	1

Graphical Abstract



Highlights

- The human population contains more active LINE-1s than pre

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Table 1

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2006). Column 9: the number of elements from column 7 that were active in retrotransposition assays. Column 10: elements from column 9 that retrotransposed at levels > 10% of L1.3, a known active element. Column 11: the number of the HGR hot elements that were present in each individual (Brouha et al., 2003).

a Daughters of Hap Map trios.

b Differs from Kidd et al. (2008).

c One element recurred in ABC11 & 12- #4-1 and #5-77. Neither allele is active, and the element is in dbRIP.

Table options ▲

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Fosmids fulfilling the above mapping criterion were subjected to a sequence-specific oligonucleotide hybridization using probes directed against diagnostic sequences in the L1Hs 5' UTR identified insertion fosmids that contained putative dimorphic L1Hs elements (Boissinot et al., 2000 and Tuzun et al., 2005). Sequence L19088 enabled the identification of L1Hs elements (Dombroski et al., 1993 and Sasagawa et al., 2003) and/or direct repeats (Badge et al., 2003) and/or direct repeats (length) L1Hs element in the fosmid library. The identified L1Hs elements were used to identify L1Hs elements in the fosmid library (command = start) (Kent, 2002). Flanking sequences also were used to determine whether any of the L1Hs elements were present in a database of known polymorphic retrotransposon insertions (dbRIP; <http://dbrip.brocku.ca/>) (Wang et al., 2006). Two additional L1Hs elements were identified through direct sequencing of the fosmids (#1-(2-1) and 3-(2-1)).

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