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Agenda 大綱

- ScienceDirect (SDOL) 簡介及特色
- 文獻回顧與分析程序
- 文章架構
- 首頁介紹
- 檢索功能介紹
- 設定新知通報



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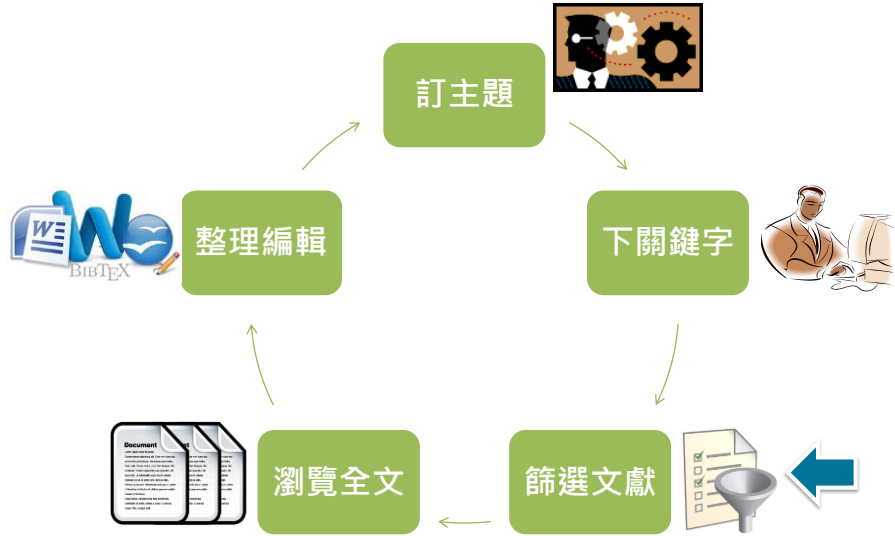


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學術文獻回顧與分析程序



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文章架構



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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 = $4.62 \pm 2.41 \mu\text{g/dL}$, body lead store = $39.04 \pm 20.91 \mu\text{g}$) and 194 were stroke patients ($4.89 \pm 2.75 \mu\text{g/dL}$, $45.13 \pm 29.8 \mu\text{g}$; 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 $>50\%$ group than $<50\%$ group (blood lead: $5.61 \pm 3.02 \mu\text{g/dL}$ vs. $4.80 \pm 2.50 \mu\text{g/dL}$, Student's t -test, $P = 0.129$; body lead store: $51.7 \pm 27.0 \mu\text{g}$ vs. $41.9 \pm 23.5 \mu\text{g}$, 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 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).

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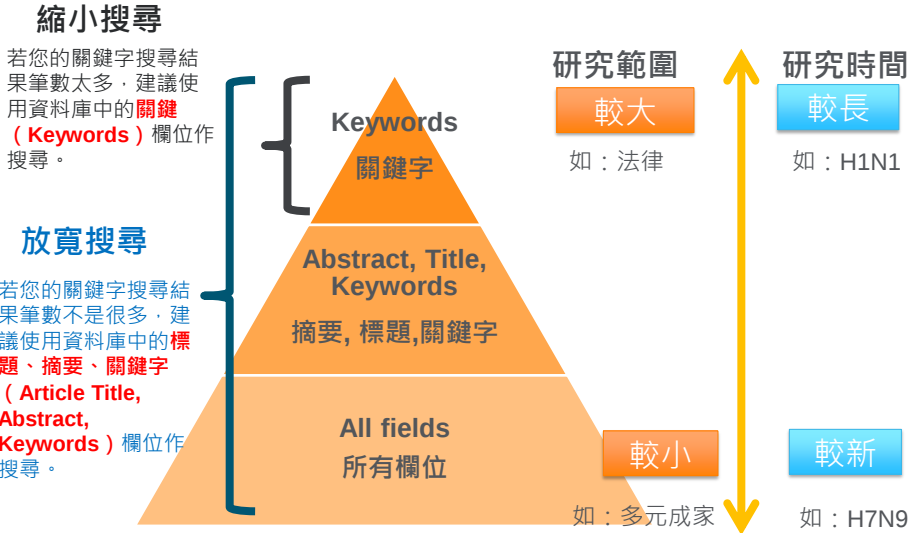
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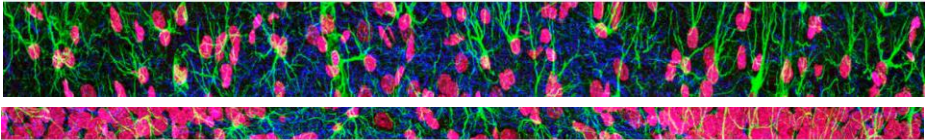
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Table 1

Table 2

Erythromelalgia (EM) is a heterogeneous condition characterized by attacks of red, hot and painful extremities (Fig. 1). Heating can induce or aggravate an attack, while patients find relief by cooling the affected site. The condition has been described for more than 100 years, but the pathophysiological mechanisms are still unclear (Mitchell, 1878). The condition may be primary or secondary. Secondary erythromelalgia can be caused by conditions such as myeloproliferative diseases, diabetes mellitus, infections, medication and mushroom poisoning (McCarthy et al., 2002).



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Fig. 1. The feet of an EM patient during an attack.

So far mainly circulatory changes in EM patients have been studied. The main final common pathway of the pathophysiological mechanism behind EM has been suggested to be maldistribution in micro-vascular skin perfusion with arterio-venous shunting causing a relative increase in thermoregulatory perfusion and lack of nutritive perfusion. The end result is skin

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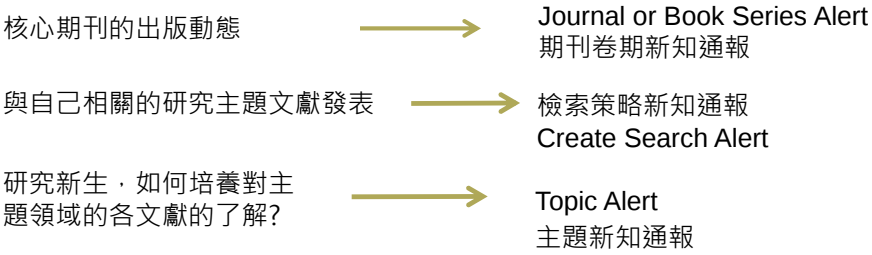
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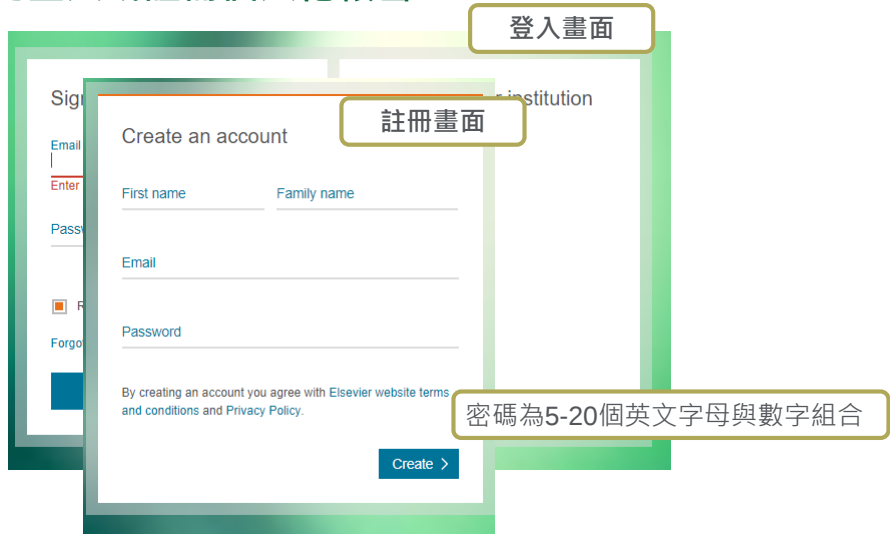
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