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整理編輯

Association of high body lead store with severe intracranial carotid atherosclerosis

Tsong-Hai Lee [a], Mei-Chun Tseng [b], Chi-Jen Chen [c], Ja-Liang Lin [d,*]

[a] Stroke Section, Department of Neurology and Stroke Center, Chang Gung Memorial Hospital, Linkou Medical Center and Chang Gung University College of Medicine, Taoyuan, Taiwan
[b] Department of Business Management, National Sun Yat-Sen University, Kaohsiung, Taiwan
[c] Department of Radiology, Taipei Medical University-Shuang Ho Hospital, Taipei, Taiwan
[d] Department of Nephrology, Division of Clinical Toxicology, Chang Gung Memorial Hospital, Linkou Medical Center and Chang Gung University College of Medicine, No. 5, Fu-hsing St., Kweishan, Taoyuan, 333 Taiwan

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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 vertebrobasilar 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 μg/dL, body lead store = 39.04 ± 20.91 μg) and 194 were stroke patients (4.89 ± 2.75 μg/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 ≥50% group than <50% group (blood lead: 5.61 ± 3.02 μg/dL vs. 4.80 ± 2.50 μg/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.

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).

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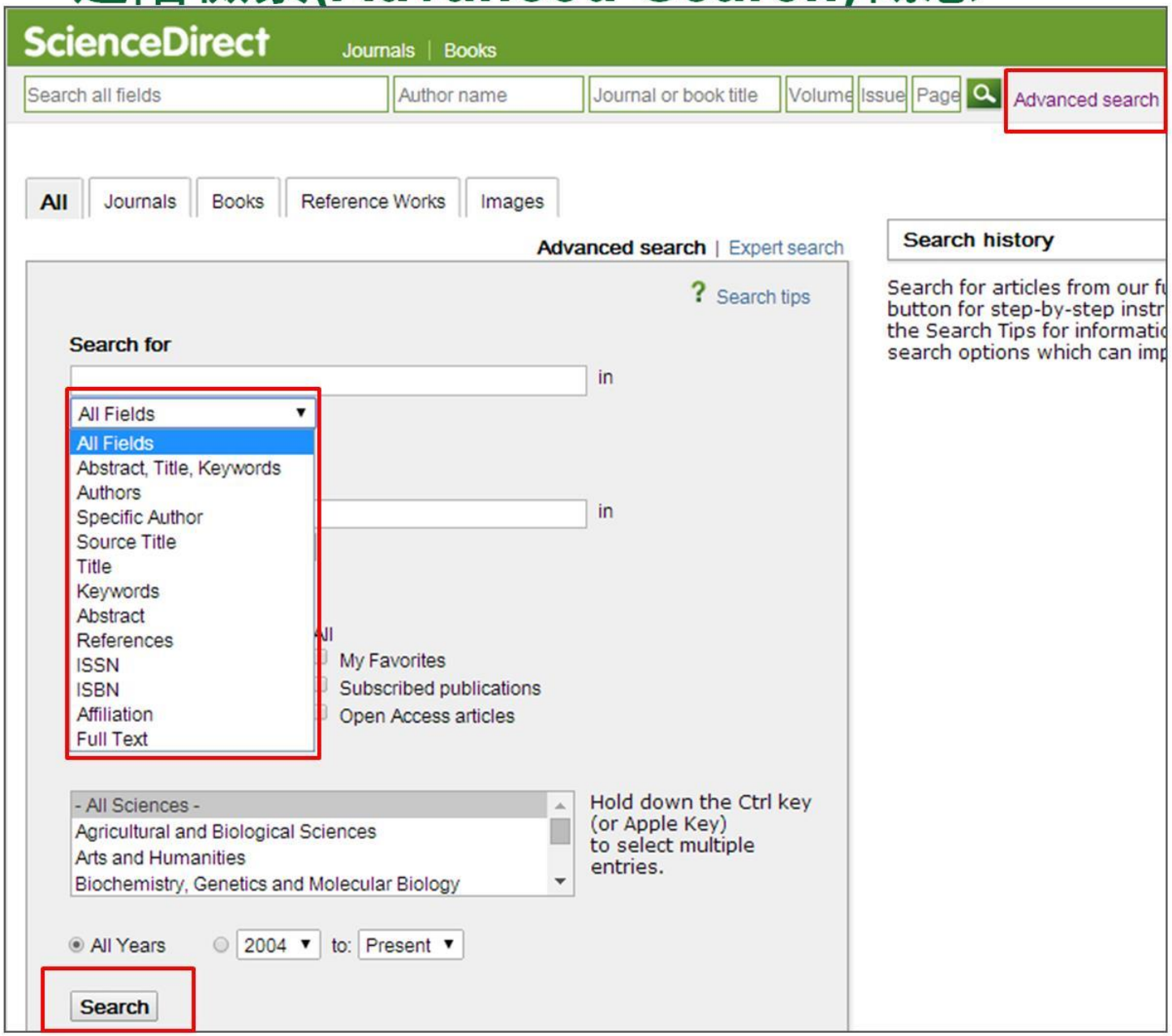

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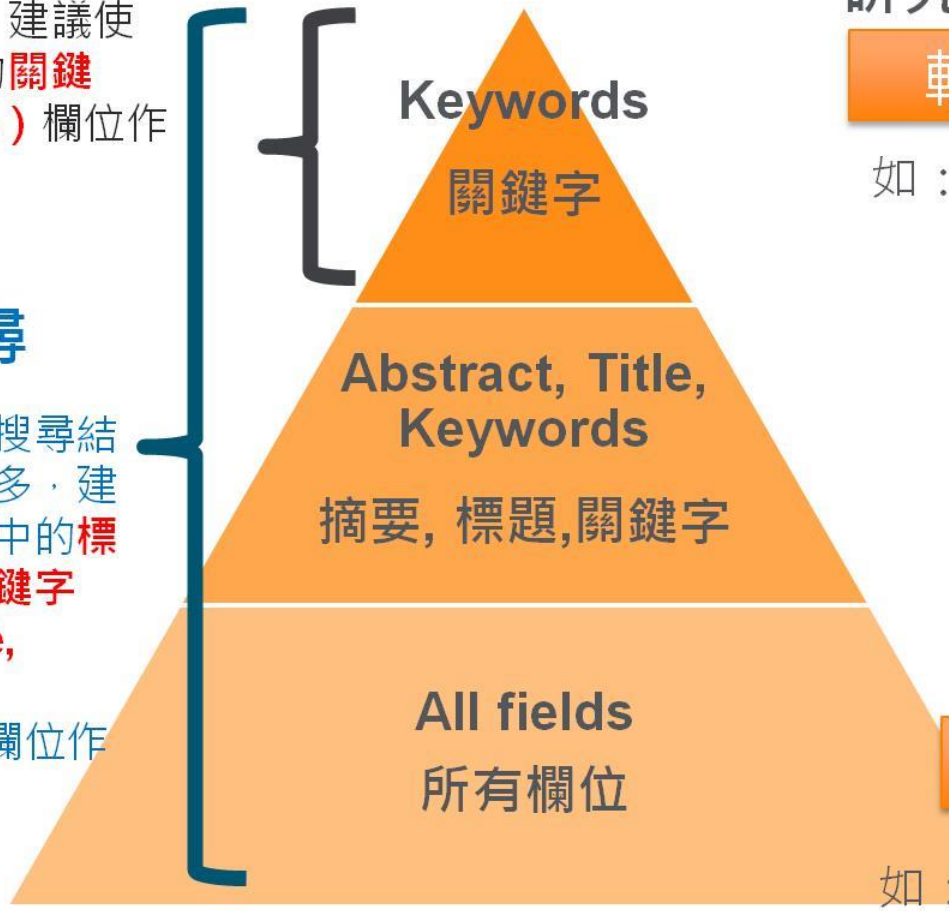

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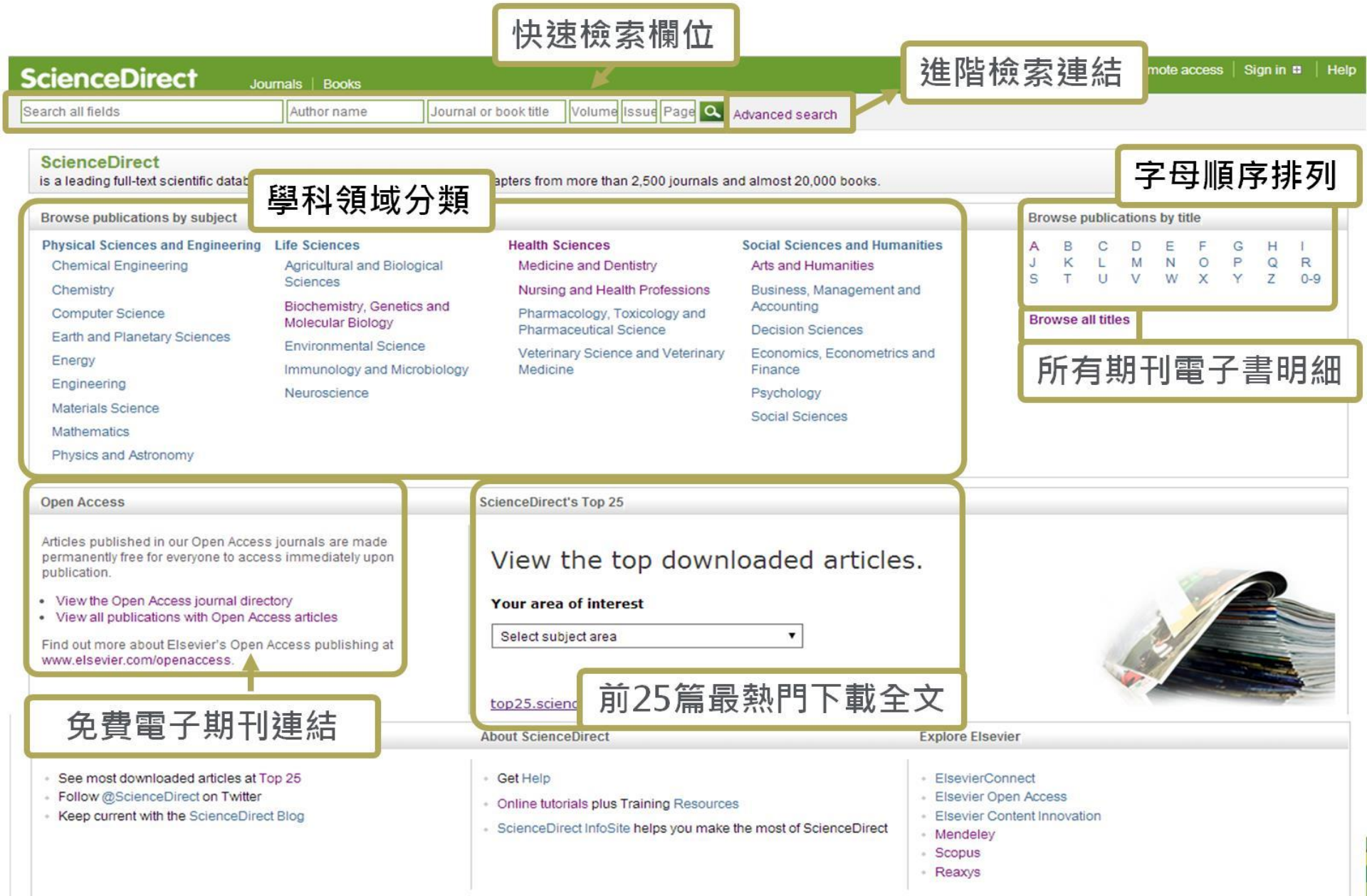
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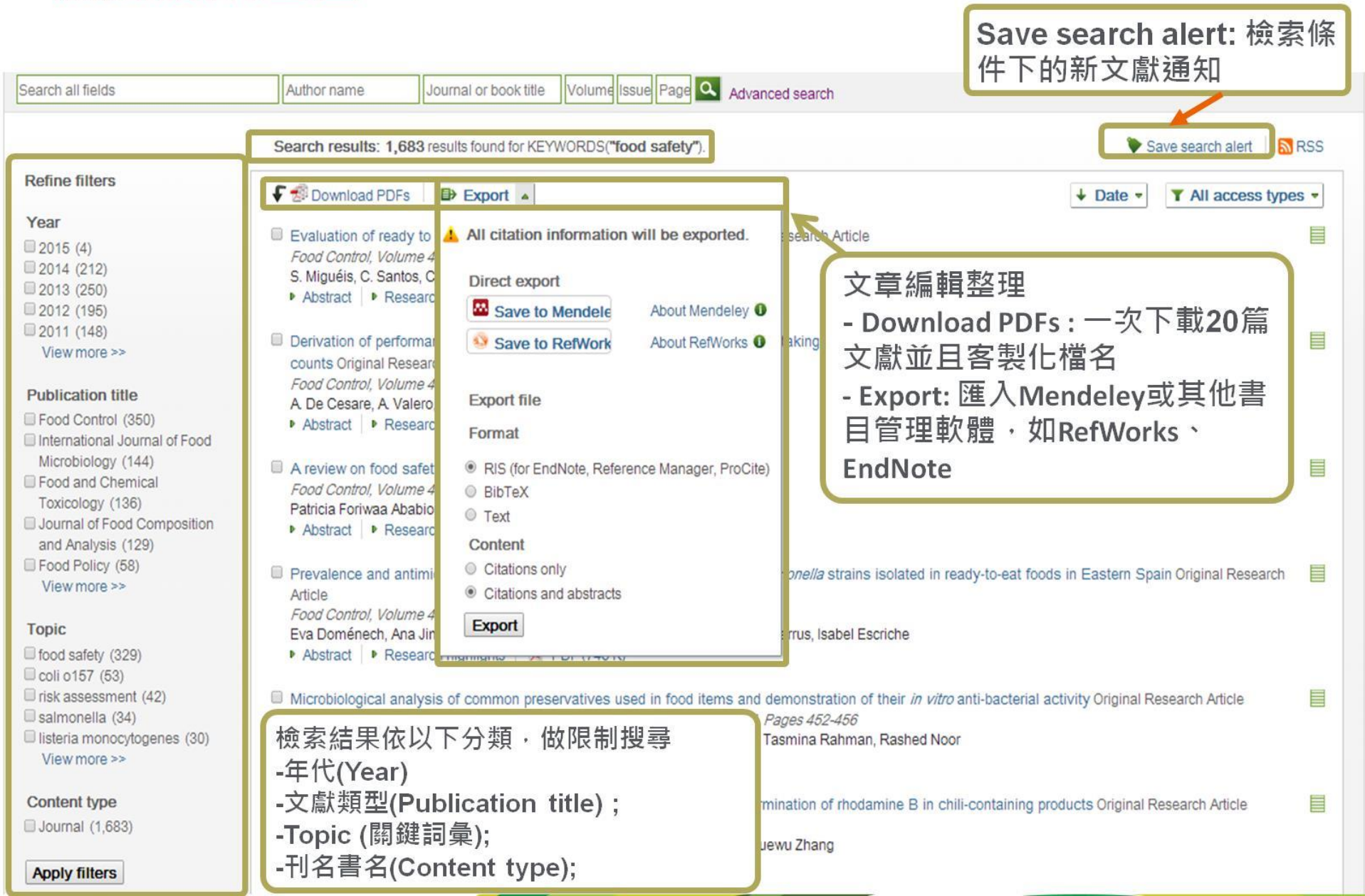
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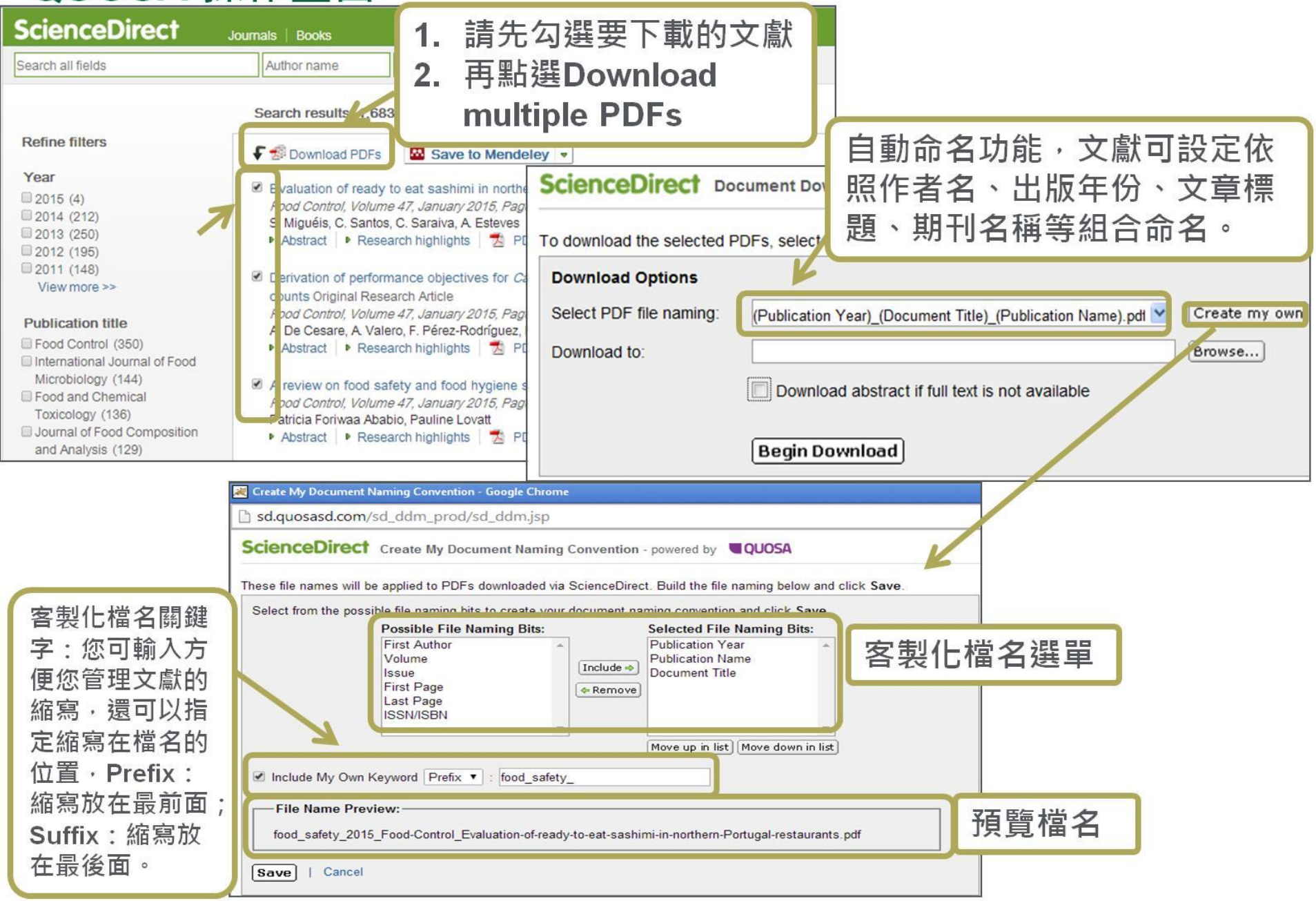
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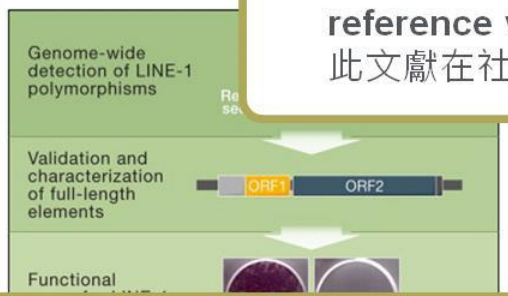

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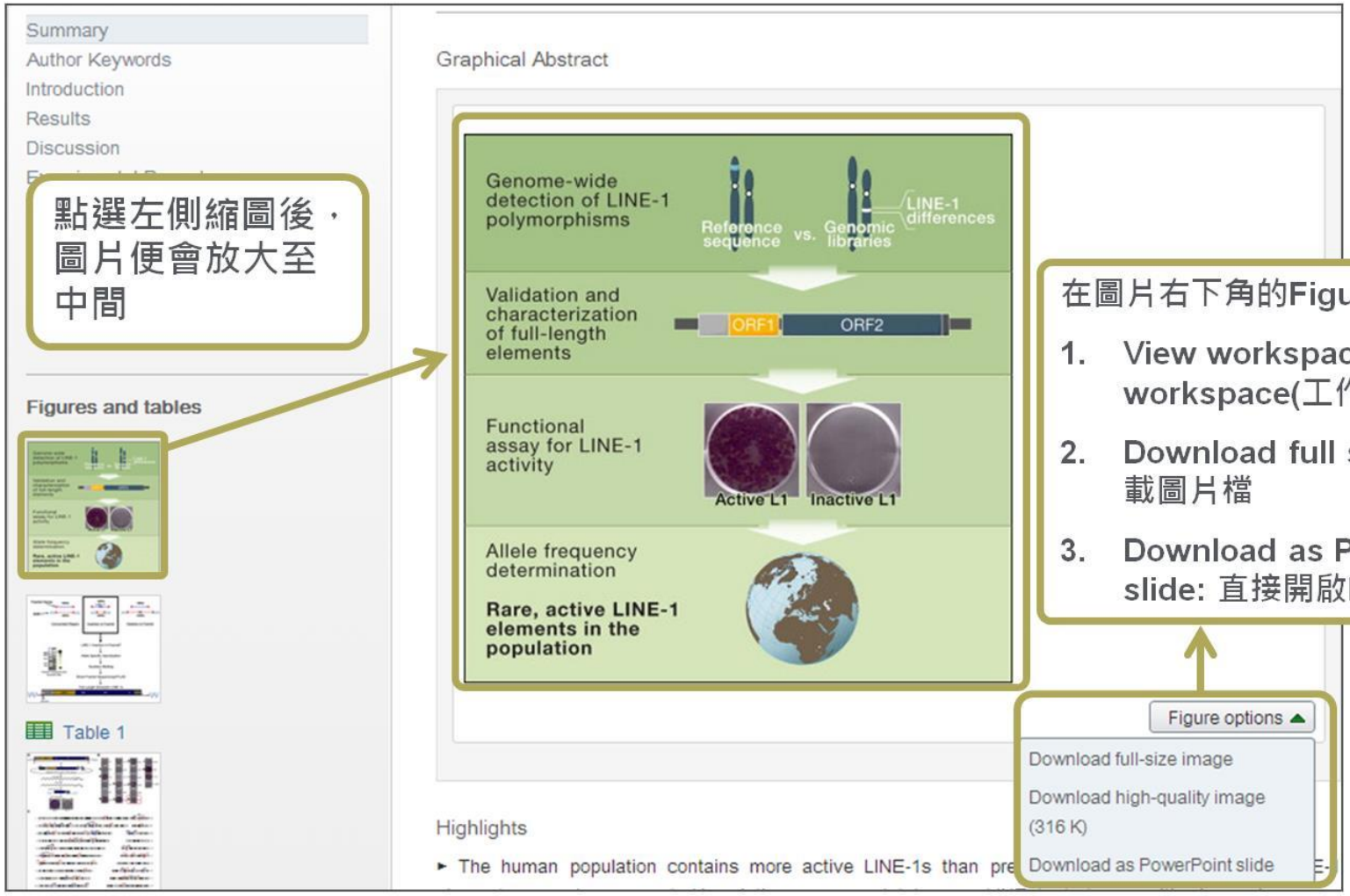

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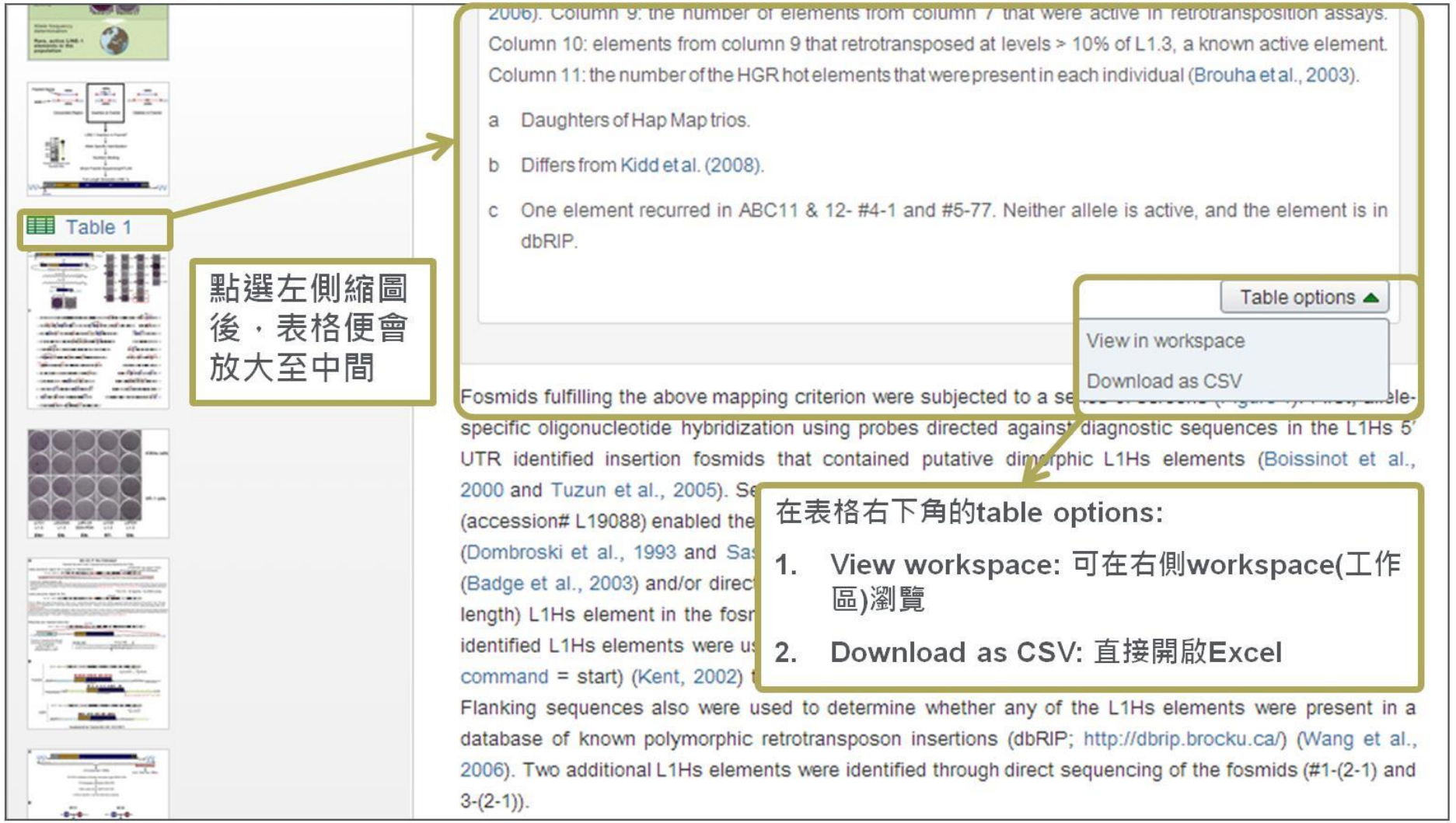

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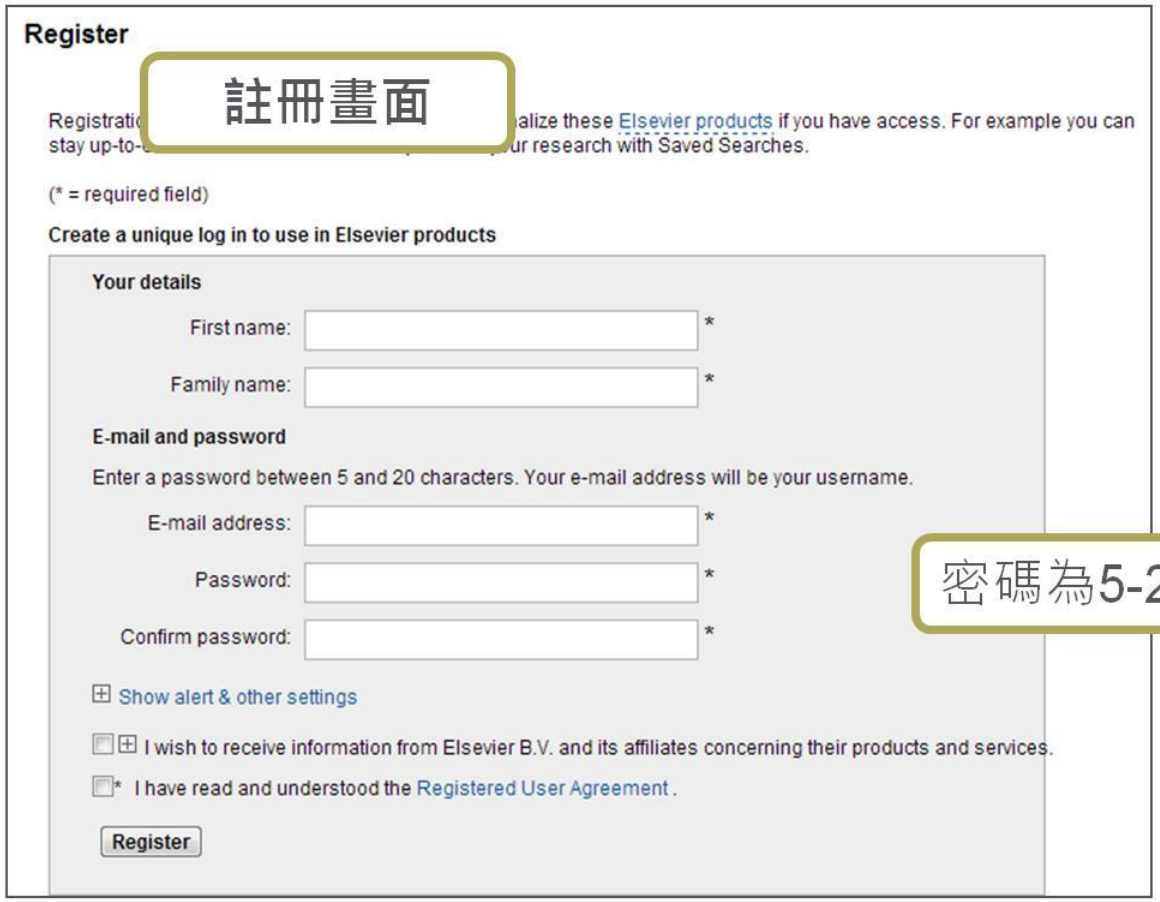

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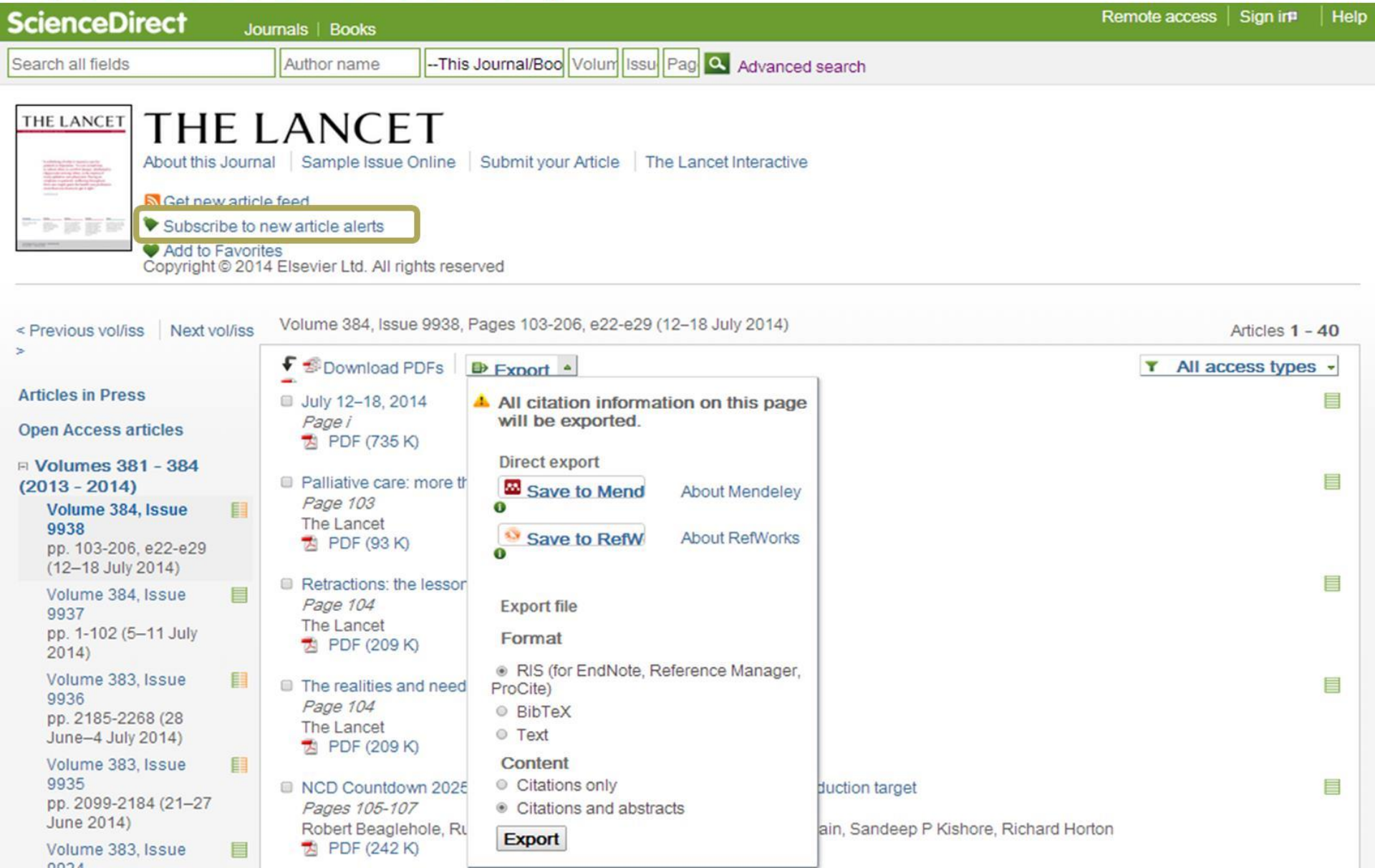

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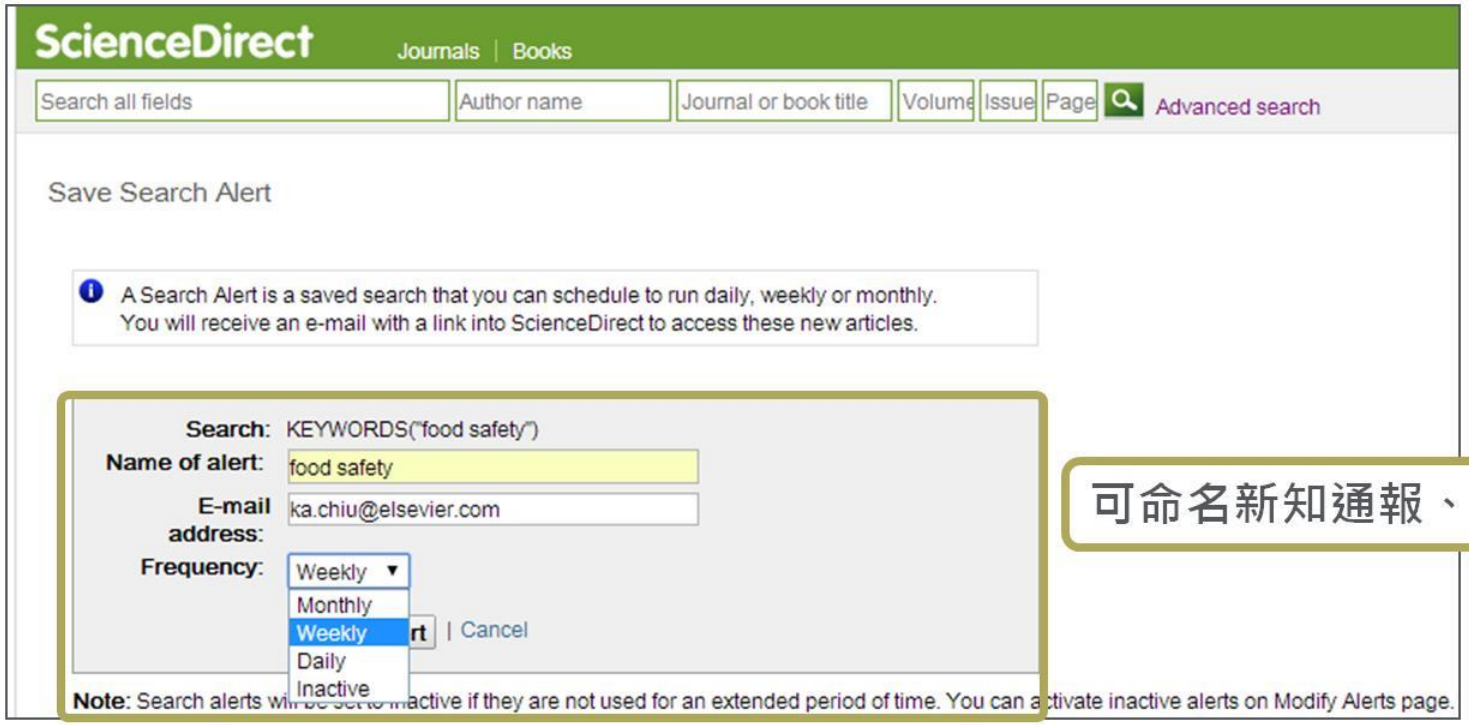

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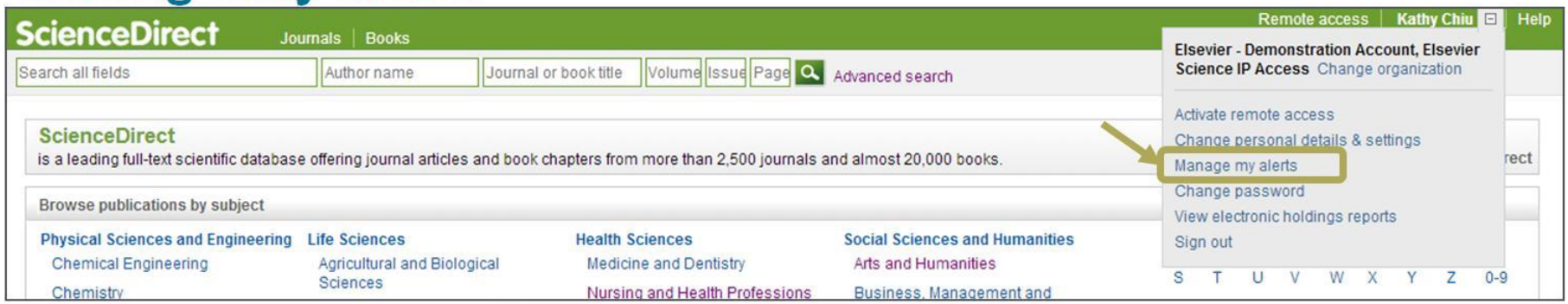

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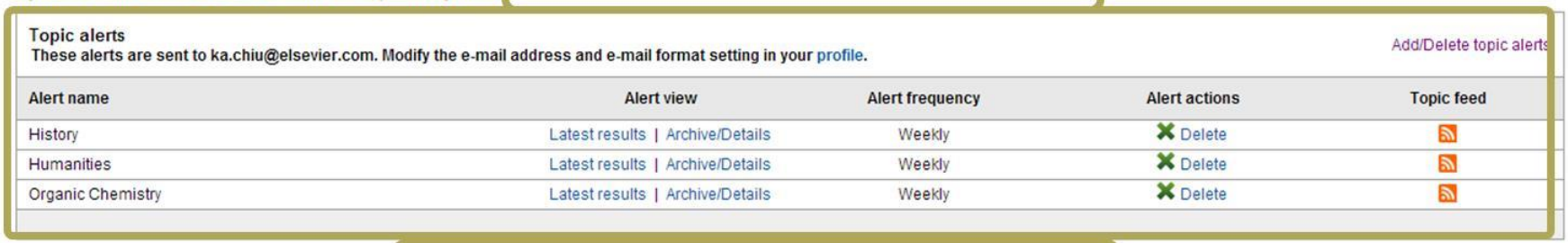

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