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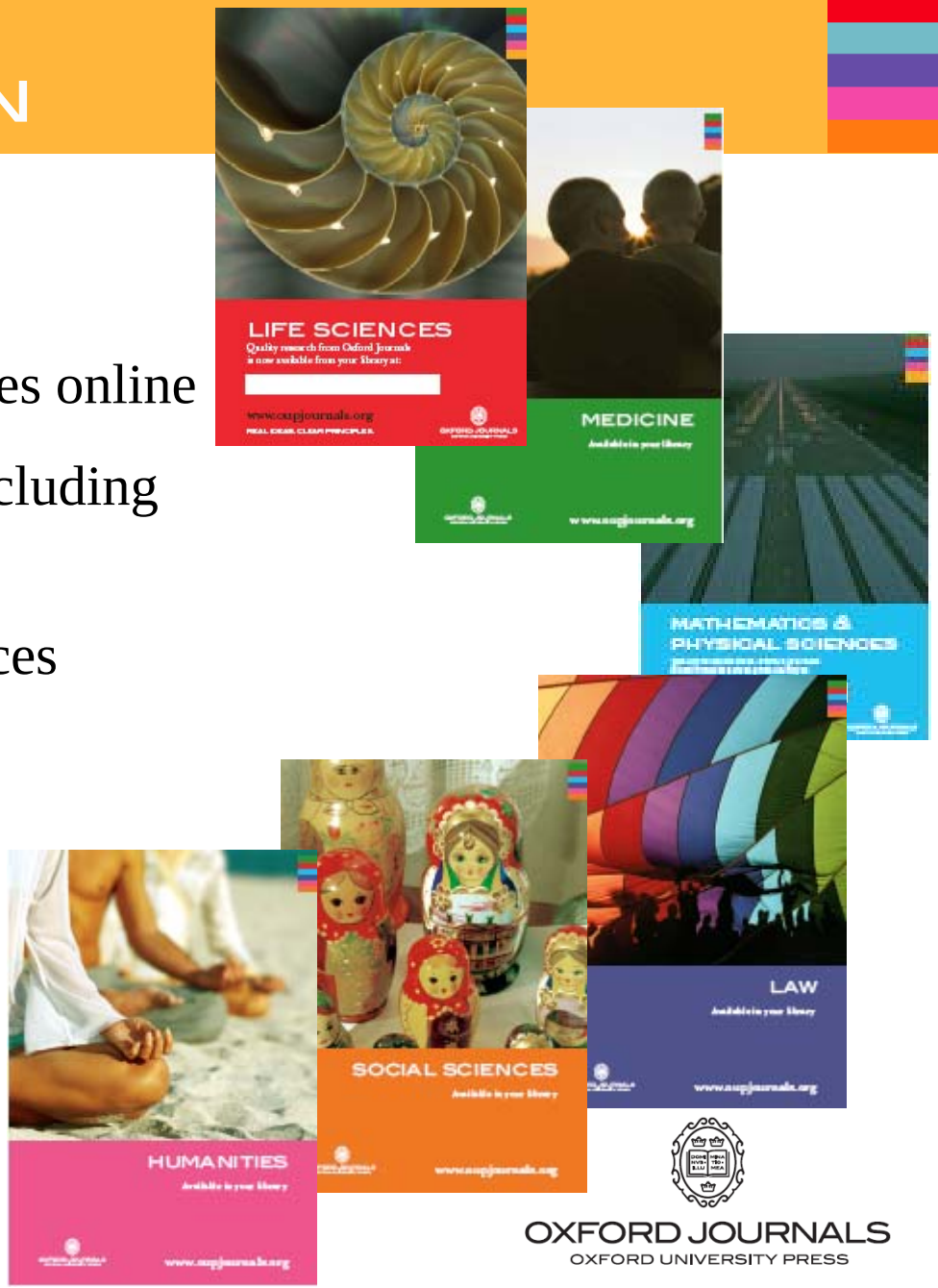

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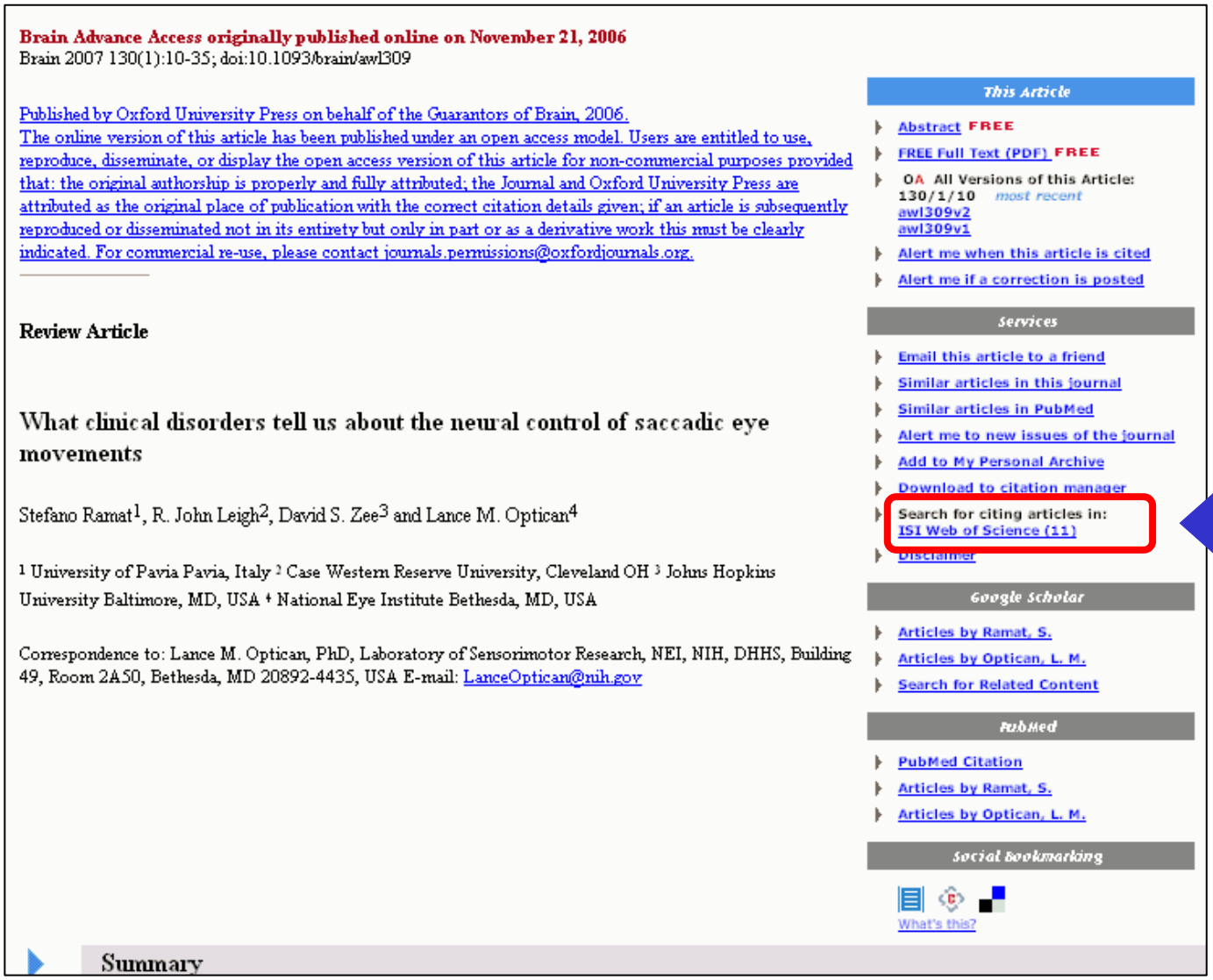

Brain Advance Access originally published online on November 21, 2006
Brain 2007 130(1):10-35; doi:10.1093/brain/awl309

Review Article

What clinical disorders tell us about the neural control of saccadic eye movements

Stefano Ramat[1], R. John Leigh[2], David S. Zee[3] and Lance M. Optican[4]

[1] University of Pavia Pavia, Italy [2] Case Western Reserve University, Cleveland OH [3] Johns Hopkins University Baltimore, MD, USA [4] National Eye Institute Bethesda, MD, USA

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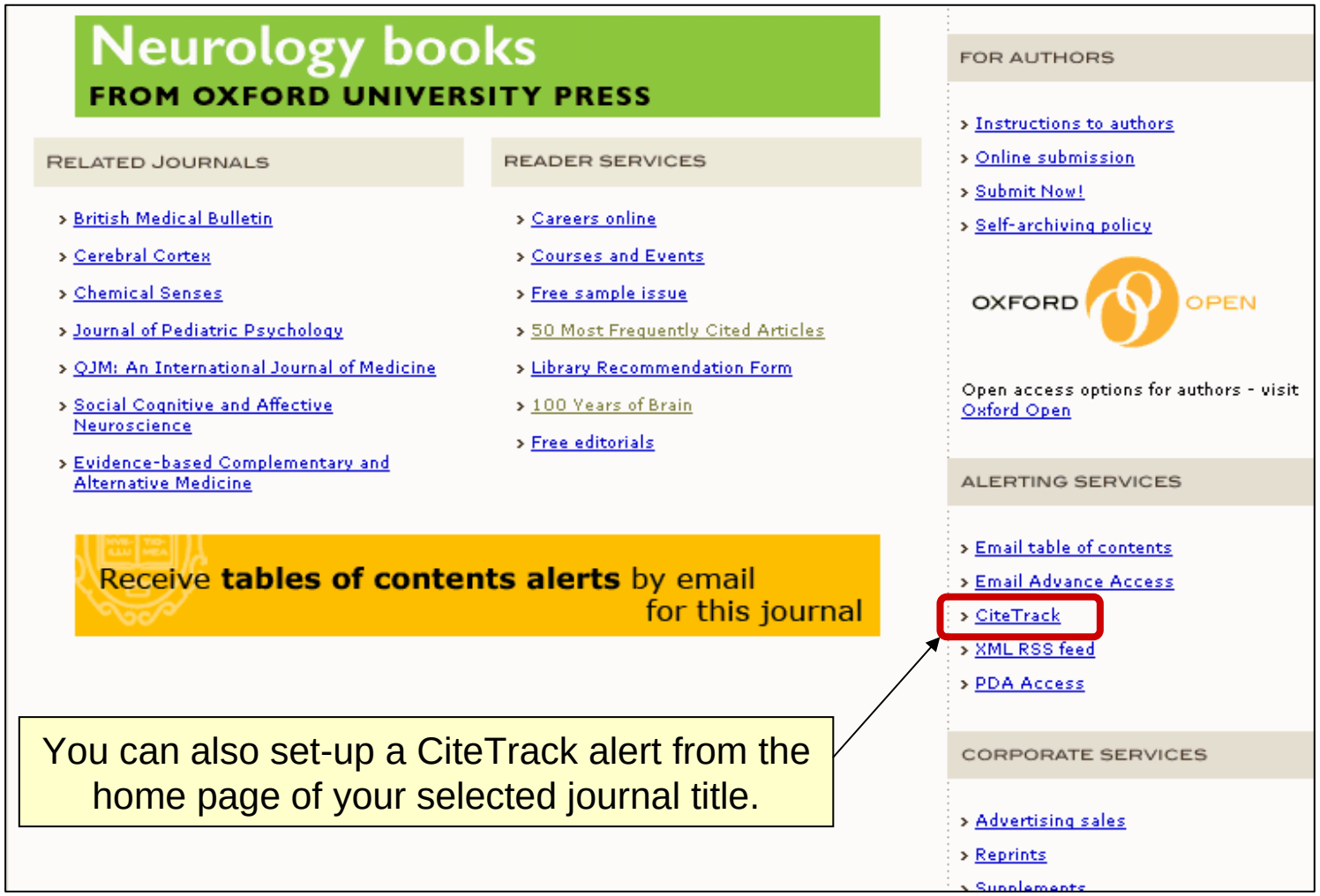

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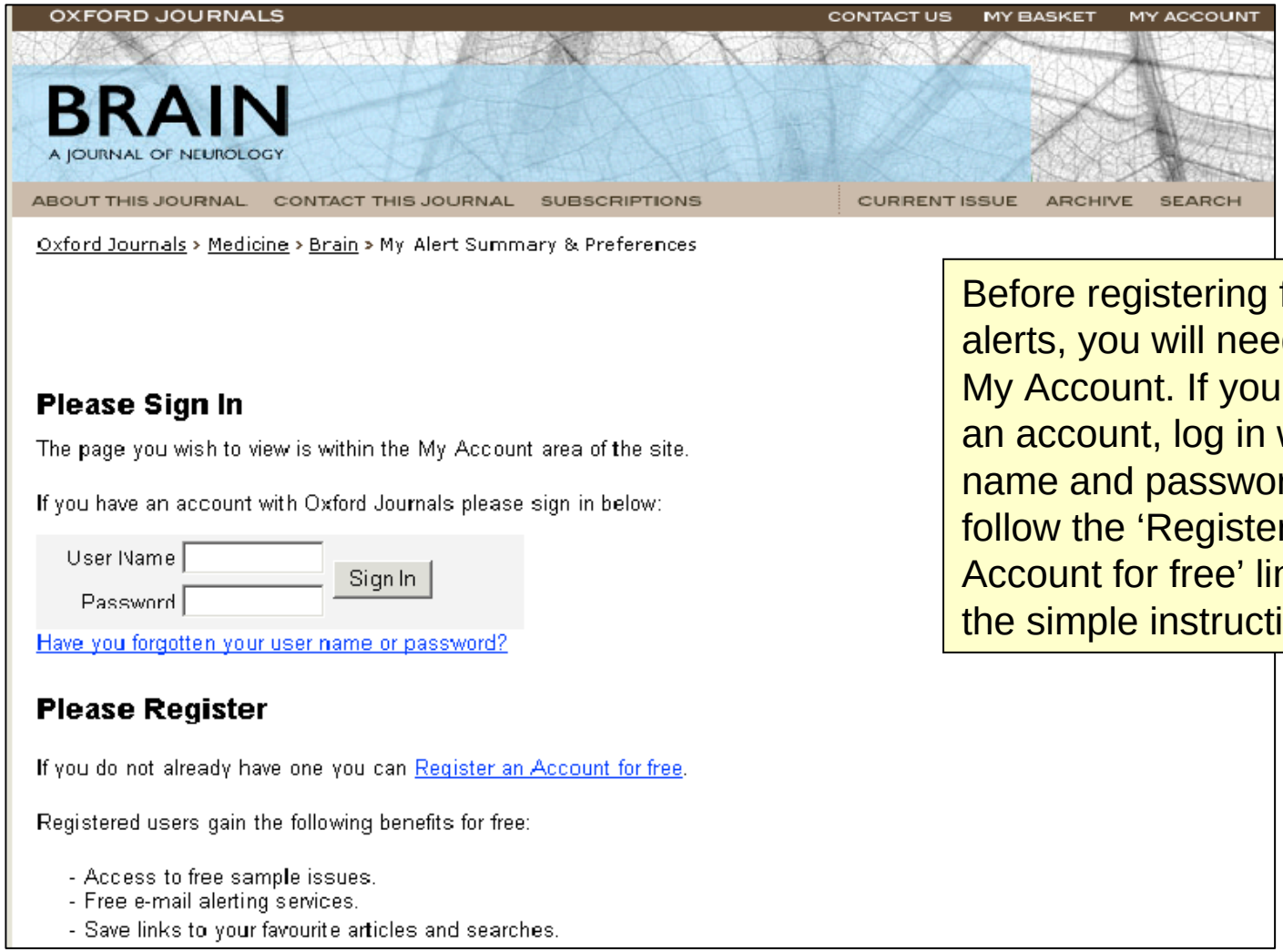

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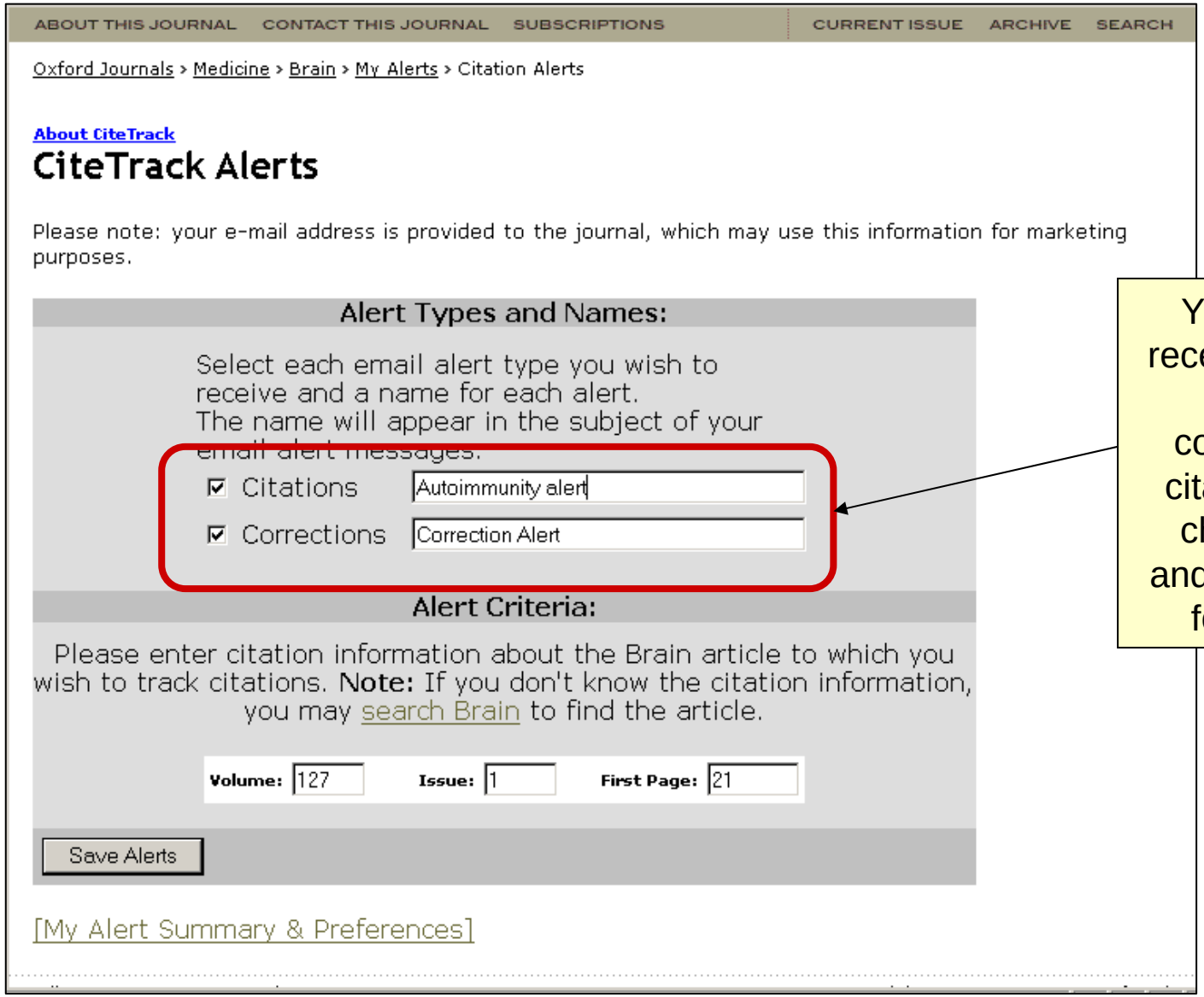

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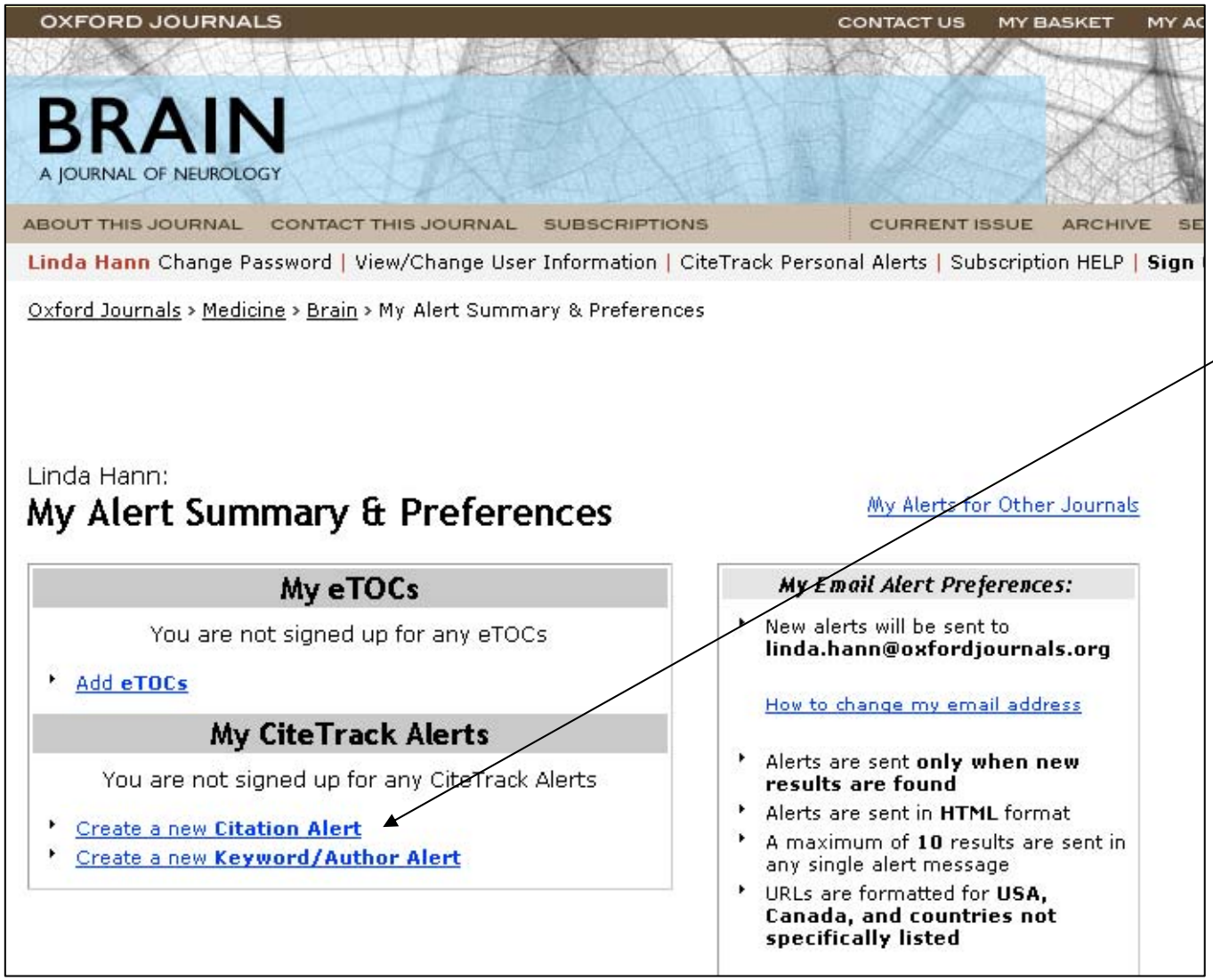

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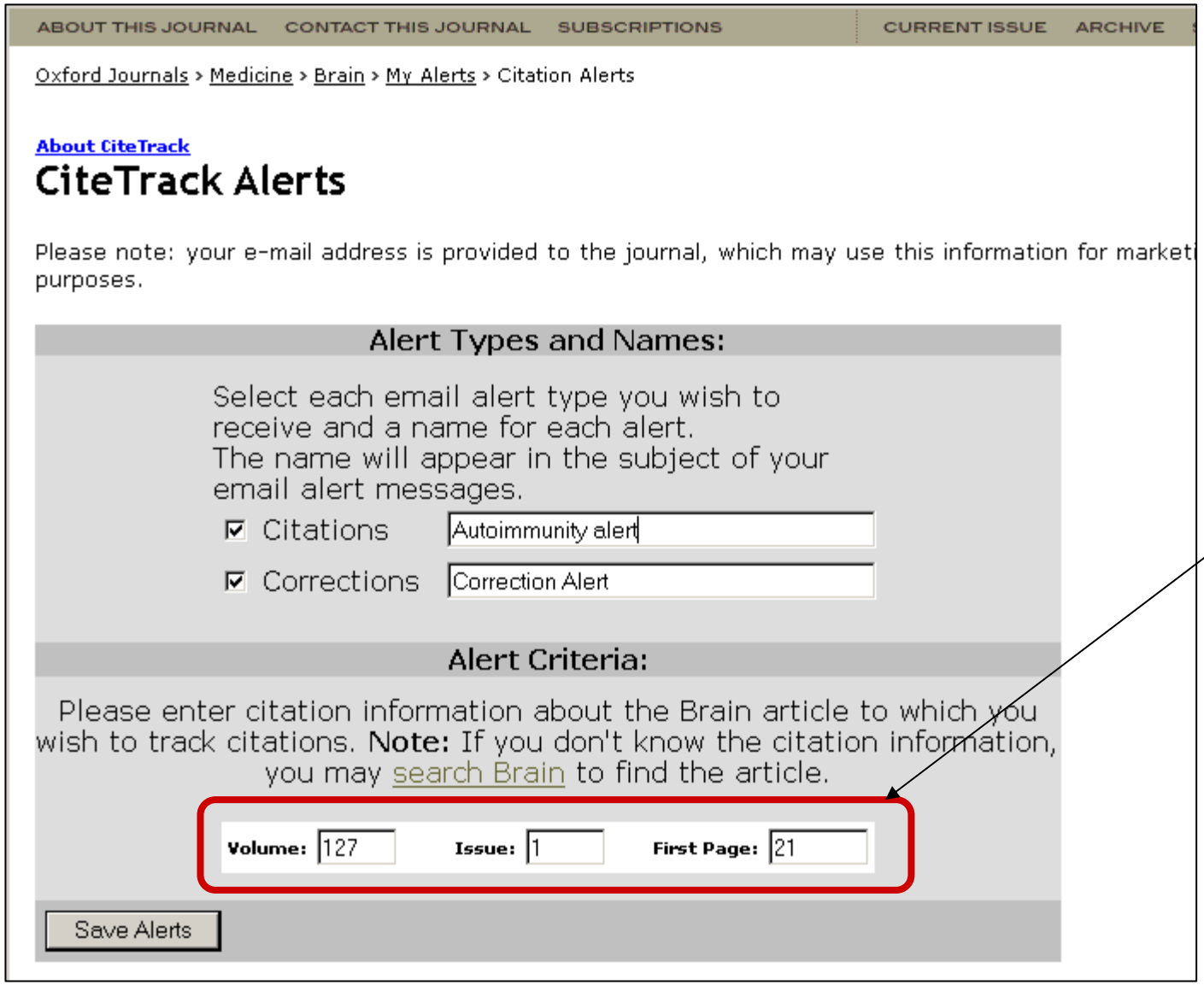

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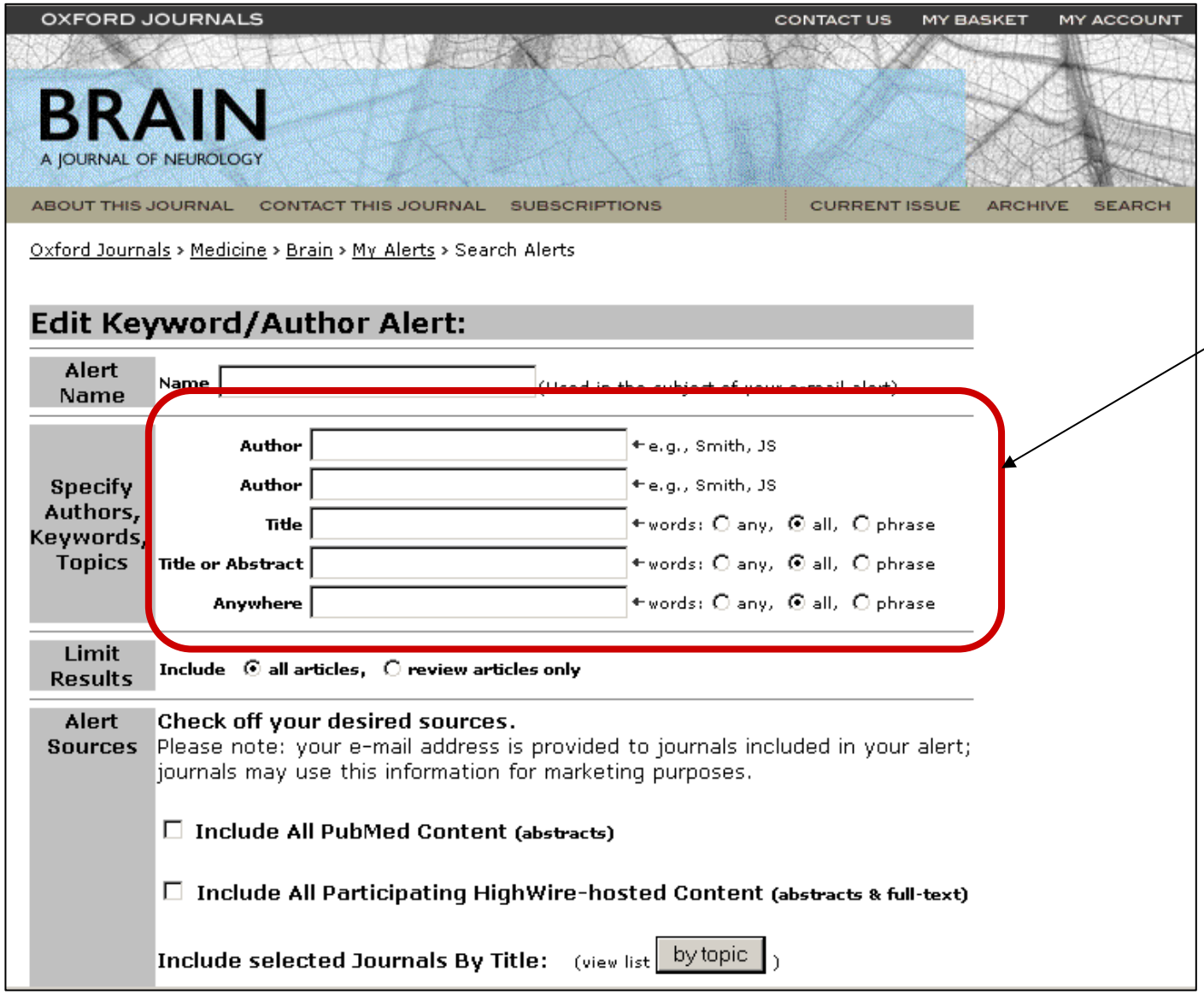

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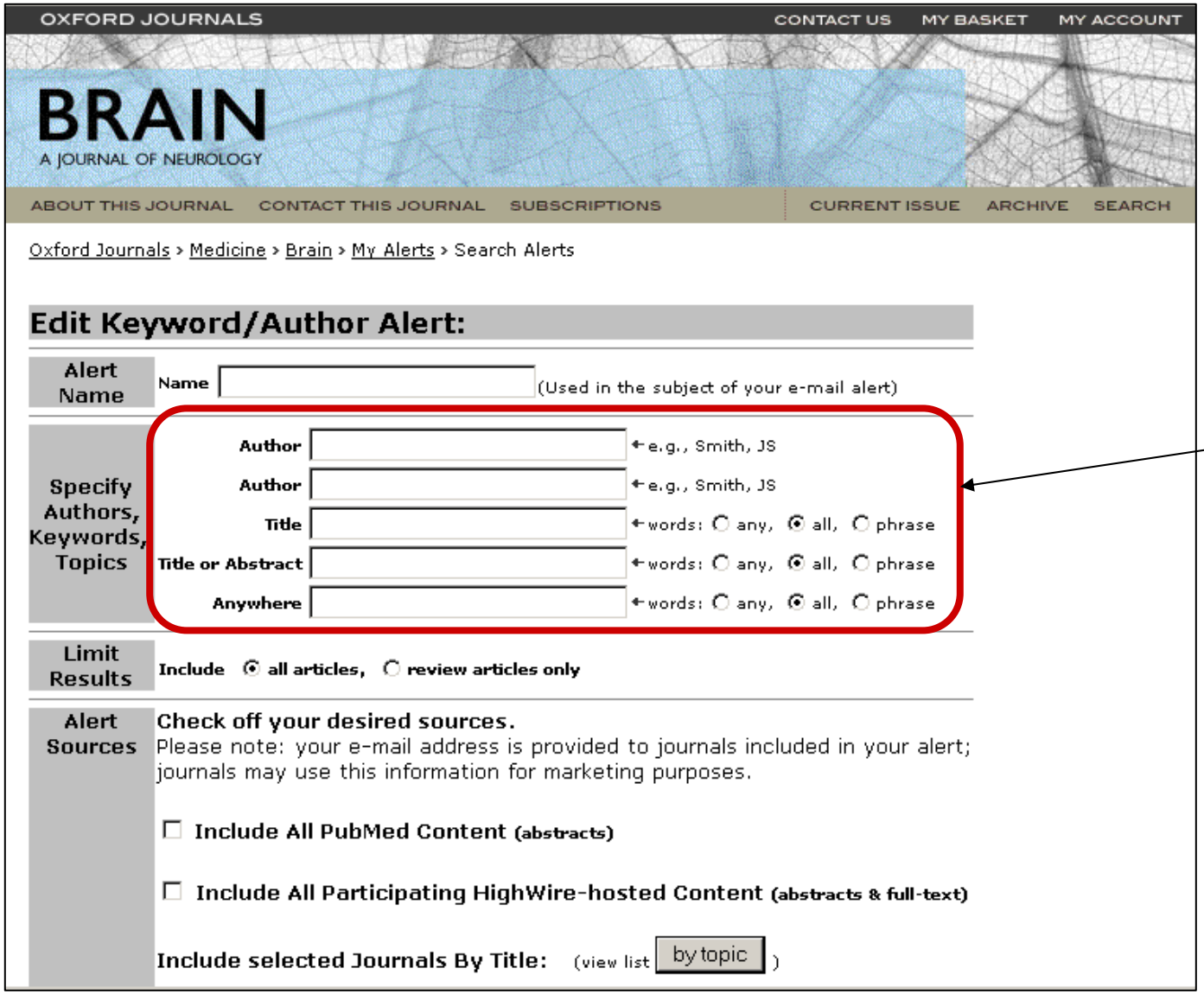

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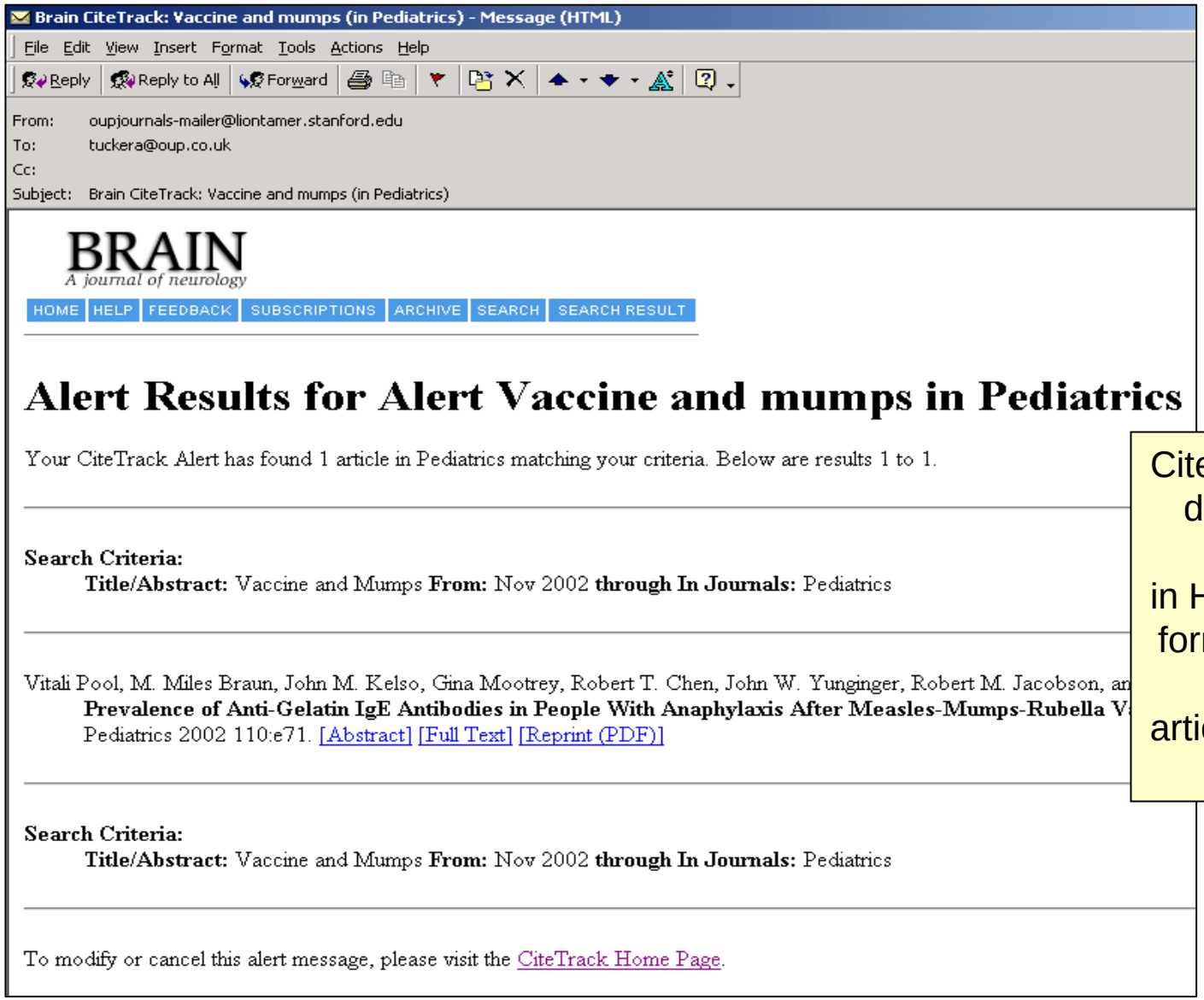

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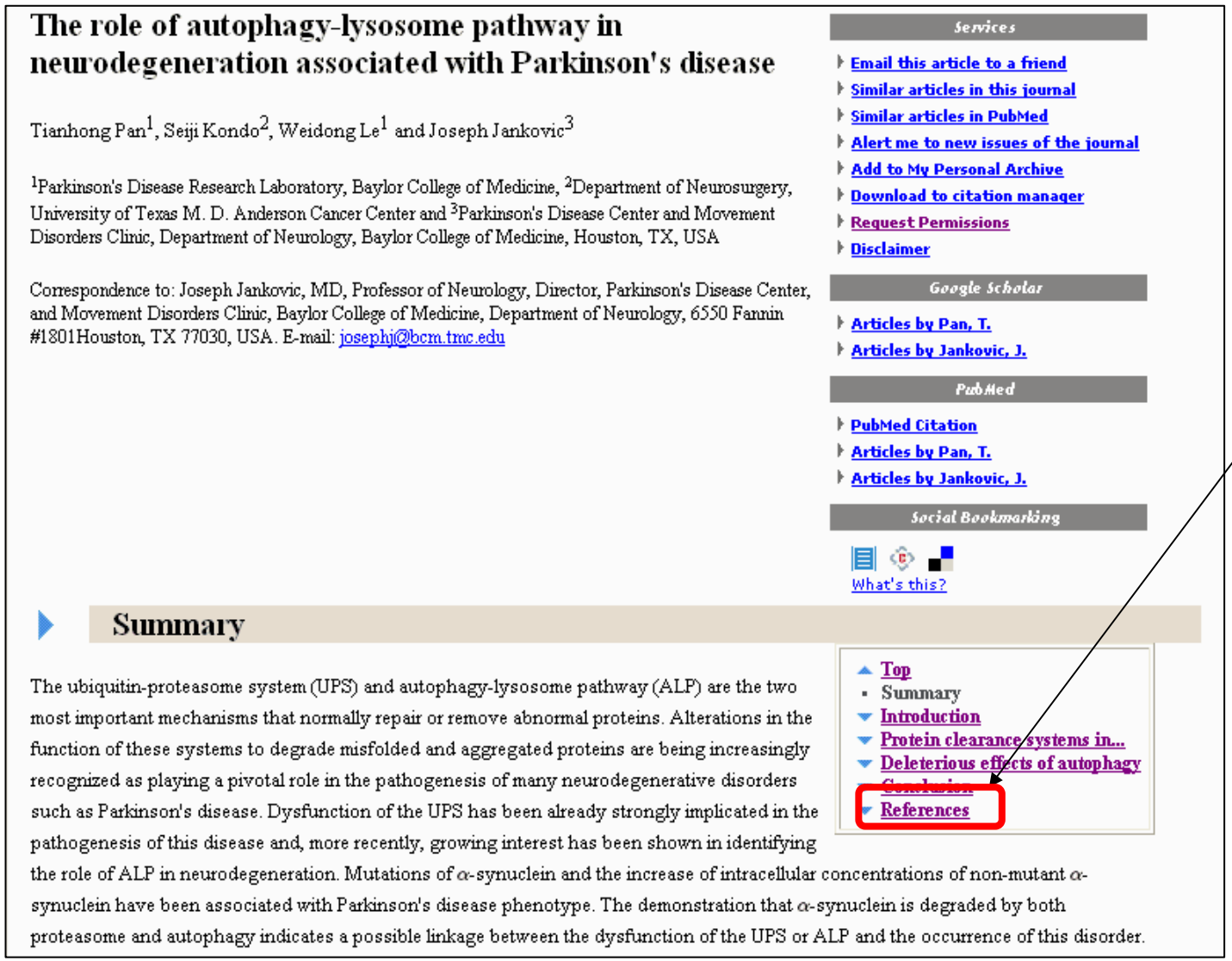

The role of autophagy-lysosome pathway in neurodegeneration associated with Parkinson's disease

Tianhong Pan[1], Seiji Kondo[2], Weidong Le[1] and Joseph Jankovic[3]

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Summary

The ubiquitin-proteasome system (UPS) and autophagy-lysosome pathway (ALP) are the two most important mechanisms that normally repair or remove abnormal proteins. Alterations in the function of these systems to degrade misfolded and aggregated proteins are being increasingly recognized as playing a pivotal role in the pathogenesis of many neurodegenerative disorders such as Parkinson's disease. Dysfunction of the UPS has been already strongly implicated in the pathogenesis of this disease and, more recently, growing interest has been shown in identifying the role of ALP in neurodegeneration. Mutations of α-synuclein and the increase of intracellular concentrations of non-mutant α-synuclein have been associated with Parkinson's disease phenotype. The demonstration that α-synuclein is degraded by both proteasome and autophagy indicates a possible linkage between the dysfunction of the UPS or ALP and the occurrence of this disorder.

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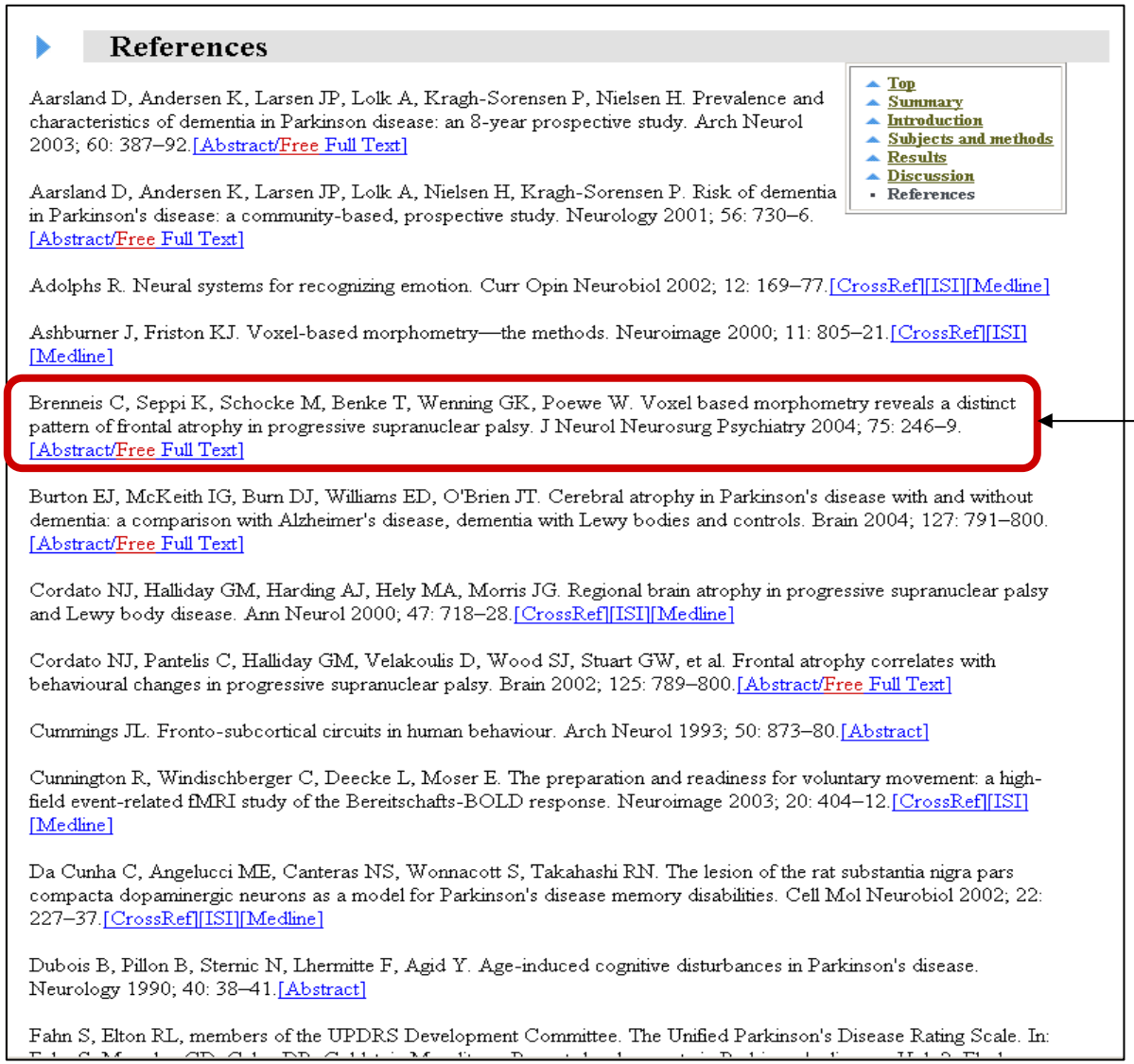

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