

one of a number of reference points for the motor control system that informs the predicted sensory feedback, or efference copy. Other factors, including joint position sense, the environment, previous experience and emotional factors, inevitably influence the efference copy. If any one of these becomes altered or distorted, the individual is rendered more vulnerable to an inaccurate or distorted sensory prediction. It is possible that some of these factors are differentially weighted and hence it is not surprising that the level of pain experienced has been shown to be significantly correlated with the level of cortical reorganization in some patients with phantom limb and in CRPS populations [6, 7]. In addition, functional MRI data show a dramatic increase in activity in the somatosensory cortex, as well as other cortical areas involved in cognitive and motor processing, when pin-prick hyperalgesia is induced in the CRPS-affected limb compared with the unaffected limb [8].

The authors have declared no conflicts of interest.

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1. Moseley GL, Gandevia SC. Sensory-motor incongruence and reports of 'pain'. *Rheumatology* 2005;44:1083–5.
2. McCabe CS, Haigh RC, Halligan PW, Blake DR. Simulating sensory-motor incongruence in healthy volunteers: implications for a cortical model of pain. *Rheumatology* 2005;44:509–16.
3. McCabe CS, Haigh RC, Ring EFR, Halligan PW, Wall PD, Blake DR. A controlled pilot study of the utility of mirror visual feedback in the treatment of complex regional pain syndrome (Type 1). *Rheumatology* 2003;42:97–101.
4. Ramachandran VS, Rogers-Ramachandran D. Synaesthesia in phantom limb induced by mirrors. *Proc R Soc B* 1996;263:377–86.
5. Sterr A, Muller MM, Elbert T, Rockstroh B, Pantev C, Taub E. Perceptual correlates of changes in cortical representation of fingers in blind multifinger Braille readers. *J Neurosci* 1998;18:4417–23.
6. Flor H, Elbert T, Knecht S *et al.* Phantom-limb pain as a perceptual correlate of cortical reorganisation following arm amputation. *Nature* 1995;375:482–4.
7. Maihöfner C, Handwerker HO, Neundörfer B, Birklein F. Cortical reorganization during recovery from complex regional pain syndrome. *Neurology* 2004;63:693–701.
8. Maihöfner C, Forster C, Birklein F, Neundörfer B, Handwerker HO. Brain processing during mechanical hyperalgesia in complex regional pain syndrome: a functional MRI study. *Pain* 2005; 114:93–103.

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Re: Sensory-motor incongruence and reports of 'pain', by G. L. Moseley and S. C. Gandevia. *Rheumatology* 2005;44:1083–1085: Reply

SIR, Thanks for the opportunity to comment on McCabe *et al.*'s response to our Editorial [1] concerning their earlier paper in

Rheumatology [2]. Our position was primarily one of caution that alternative explanations for both the results and the mechanisms underpinning them should not be excluded. Some of our concerns, for example selection bias, were allayed, but some remain. For example, while we appreciate that abnormal sensations stopped immediately when normal visual input was restored and that this suggests a direct relationship between the experimental condition and the abnormal sensations, the cogency of this finding would be strengthened if the onset of abnormal sensation occurred immediately too. Our contention that further work is required to determine how the experimental condition was related to the abnormal sensations still stands.

We agree, and it is well established, that many factors from across domains may impact on motor commands and their efference copies [3], and that changes in cortical reorganization (that is, the response profile of S1 neurons) cannot be solely responsible for pain. Furthermore, while S1 holds maps of the superficial and deep surfaces of the body, the understanding of the relationships between cutaneous afferent activity, proprioception and motor control is not well developed [4].

In summary, it is possible that mirror use in healthy volunteers is sufficiently incongruent to evoke abnormal sensations yet the same mirror use in patients is sufficiently congruent to alleviate these sensations. However, it seems critical to acknowledge that on the basis of current data this hypothesis can neither be accepted nor refuted.

The authors have declared no conflicts of interest.

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1. Moseley GL, Gandevia SC. Sensory motor incongruence and reports of 'pain'. *Rheumatology* 2005;44:1083–5.
2. McCabe CS, Haigh RC, Halligan PW, Blake DR. Simulating sensory-motor incongruence in healthy volunteers: implications for a cortical model of pain. *Rheumatology* 2005;44:509–16.
3. Von Holst H. Relations between the central nervous system and the peripheral organs. *Br J Anim Behav* 1950;2:89–94.
4. Collins DF, Refshauge KM, Todd G, Gandevia SC. Cutaneous receptors contribute to kinesthesia at the index finger, elbow, and knee. *J Neurophysiol* 2005;94:1699–706.

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β_2 -Glycoprotein I IgA antibodies and ischaemic stroke

SIR, Regarding the article by Kahles *et al.*, recently published in this journal [1], we would like to point out that our previous paper, published in 2003 [2] and not referred to by Kahles *et al.*, had already shown an association of IgA antibodies to β_2 -glycoprotein I (β_2 gpI) with ischaemic stroke. Like Kahles *et al.*, we conducted a case-control study with multivariate analysis. This also allowed us to evaluate risk association after adjustment for known risk factors for stroke. Our data showed that the presence of IgA anti- β_2 gpI antibodies was independently associated with the risk of acute cerebral ischaemia (adjusted odds

ratio 4.6; 90% confidence interval 1.5–14.3; $P=0.025$). β_2 gpI is present in atheroma plaques [3]. The occurrence of IgA anti- β_2 gpI antibodies in patients with ischaemic stroke brings about a possible link of autoimmunity with thrombophilia and/or atherosclerosis.

G.L.N. is an employee of INOVA Diagnostics Inc. He does not hold any stock or equity interest in the company.

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1. Kahles T, Humpich M, Steinmetz H, Sitzer M, Lindhoff-Last E. Phosphatidylserine IgG and beta2-glycoprotein I IgA antibodies may be a risk factor for ischaemic stroke. *Rheumatology* 2005;44:1161–5.
2. Staub HL, Norman GL, Crowther T *et al.* Antibodies to the atherosclerotic plaque components beta2-glycoprotein I and heat-shock proteins as risk factors for acute cerebral ischaemia. *Arq Neuropsiquiatr* 2003;61:757–63.
3. George J, Harats D, Gilburd B *et al.* Immunolocalization of beta2-gpI (apolipoprotein H) to human atherosclerotic plaques: potential implications for lesion progression. *Circulation* 1999;99:2227–30.

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β_2 -Glycoprotein I IgA antibodies and ischaemic stroke: reply

SIR, We appreciate the interest of Staub *et al.* in our recent article [1] and welcome their comment. We apologize for not referring to their paper published in *Arquivos de Neuropsiquiatria* in 2003 [2], in which they analysed the frequency of different phospholipid antibodies and antibodies to heat-shock proteins in patients with ischaemic stroke. They reported significantly higher positive test results for heat-shock protein 65 IgG and anti- β_2 -glycoprotein IgA in cases than in controls.

In accordance with our results, elevated titres of anti- β_2 -glycoprotein IgA appear to be associated with ischaemic stroke. In our study this was also the case after correction for multiple comparison. Additionally, we were able to show significantly higher titres of anti-phosphatidylserine IgG in patients with ischaemic stroke compared to healthy controls.

Moreover, we tested for an association of a broad panel of phospholipid antibodies within stroke subtypes with special regard to cryptogenic stroke. We found a trend for positivity for lupus anticoagulant and anti-phosphatidylinositol IgM in patients with cryptogenic stroke compared with those with a determined cause of stroke, which was not significant after modified Bonferroni correction for multiple comparison.

Establishing a causal link between cerebral ischaemia and elevated anti- β_2 -glycoprotein IgA titres on the one hand and atheroma plaques containing β_2 -glycoprotein on the other hand requires at least the separation of strokes into their aetiological subtypes. Such a link remains to be elucidated.

However, we fully agree with Dr Staub and colleagues that anti- β_2 -glycoprotein IgA is associated with ischaemic stroke. The results of our recent study may serve as the base for

upcoming prospective studies, which should focus on the relevant phospholipid antibodies found to be associated.

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1. Kahles T, Humpich M, Steinmetz H, Sitzer M, Lindhoff-Last E. Phosphatidylserine IgG and beta-2-glycoprotein I IgA antibodies may be a risk factor for ischaemic stroke. *Rheumatology* 2005;44:1161–5.
2. Staub HL, Norman GL, Crowther T *et al.* Antibodies to the atherosclerotic plaque components beta-2-glycoprotein I and heat-shock proteins as risk factors for acute cerebral ischemia. *Arq Neuropsiquiatr* 2003;61:757–63.

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Can research quality be estimated from journal titles?

SIR, In the article by Wooding *et al.* [1] the method used to estimate the impact of each individual paper published as the result of receiving an Arthritis Research Campaign grant is not clearly stated. However, if it was calculated indirectly from the impact factor of the journal of publication, the approach used to rank authors, I have serious concerns about the value of the information derived.

To investigate the reliability of this approach I have used a database of papers published from the (now defunct) MRC Clinical Research Centre (CRC) between 1972 and 1985, as listed in the CRC's bi-annual (later annual) reports. The data collected for each individual paper included the journal of publication, number of pages and total citations for that paper. Single-page papers were excluded as possibly being abstracts.

There were 20 journals that published more than 20 CRC papers each scoring more than 10 citations (mean 34) among several hundred journals used overall by CRC scientists, who were working in clinical and translational research relevant to many clinical disciplines. The distribution of the total citations received in the first 20 yr after publication by the 680 papers was found to be logarithmic, after subtracting nine citations from each (to correct for self-citation), the highest score being well over 1000. After log-transformation, analysis of variance was used to relate total score to journal, and journal was found to account for only 10% of the variance in the data. The residuals were exponentially distributed, so that after \log_{10} transformation the s.d. was 0.5. This means that the estimated mean and 95% confidence interval for predicting the citation count of an individual *Lancet* or *Nature* article (net of an estimated nine self-citations) was 42 (4.2, 420) and for the lowest scoring journal it was 10 (1, 100).

Estimating confidence intervals for the data published by Wooding *et al.* is impossible because they quote citations received annually. The CRC data showed clearly that clinical research had a much longer citation half-life than translational research for a similar number of total citations, or in other words had less early impact that was more sustained. This is appropriate for work