ratio 4.6; 90% confidence interval 1.5–14.3; \( P = 0.025 \). \( \beta_2 \)-gpI is present in atheroma plaques [3]. The occurrence of IgA anti-\( \beta_2 \)-gpI antibodies in patients with ischaemic stroke brings about a possible link of autoimmunity with thrombophilia and/or atherosclerosis.

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\( \beta_2 \)-Glycoprotein I IgA antibodies and ischaemic stroke: reply

Sir, We appreciate the interest of Staub \textit{et al}. in our recent article [1] and welcome their comment. We agree that it is important to provide information about the original study, but this should not be used to justify non-referring to their work. In Arquivos de Neuropsiquiatr 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-\( \beta_2 \)-glycoprotein IgA in cases than in controls.

In accordance with our results, elevated titres of anti-\( \beta_2 \)-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-phosphatidylserine 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-\( \beta_2 \)-glycoprotein IgA titres on the one hand and atheroma plaques containing \( \beta_2 \)-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-\( \beta_2 \)-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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Can research quality be estimated from journal titles?

Sir, In the article by Wooding \textit{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 paper is (mean \pm 1.65 \times s.d.).

Therefore, any single journal will have a citation count of an individual paper is described by 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 paper is (mean \pm 1.65 \times s.d.).

Estimating confidence intervals for the data published by Wooding \textit{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...
destined to be applied by careful clinicians, working in a framework in which patients are maximally protected. To use annual rates appropriately they have to be averaged over a substantial number of years.

These results show just how variable are the citations relating to different articles in the same journal. True, if the intention is to consider the mean impact of several articles the confidence intervals may be reduced approximately in proportion to the square root of the number of articles, and Wooding et al. were considering categories of grant each producing between 56 and 101 papers. One also has to consider uncertainties produced by the change in impact factor for journals over the passage of time, which might in part result from the submissions in question. Interestingly, for the period up to 1980, the effect of journal of publication was not a significant statistical determinant of citation count in published CRC work. One has to wonder if this situation will return because in future, as better information retrieval eases journal access, many well-known authors will feel less pressure to publish in demanding and time-consuming high-impact journals. Such high-profile scientists will be confident their work will be read, because so many searches are for the author who in the searcher’s mind is attached to an idea or a result.

Overall, there seems no alternative to the careful consideration of individual papers if it is desired to judge whether previous funding decisions gave good value. For clinical research it can take a decade for a paper’s importance to become clear. It is comparatively easy to manipulate averaged citation data from the first three post-publication years to generate support for basic vs clinical research, while industry-based authors are known to orchestrate impact by publishing repetitive supporting research that draws attention to previously published clinical data. When it comes to commissioning studies of publication impact, caveat emptor!

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