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Migrainous Stroke: Are Antiphospholipid Antibodies Pathogenetic, a Biological Epiphenomenon, or an Incidental Laboratory Aberration?

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Silvestrini et al. [1] suggest that circulating antiphospholipid antibodies (aPLs) may be an important marker for ischaemic stroke associated with migraine. The study of associations in the absence of a central hypothesis can fragment the research question [2], occasionally almost irretrievably. The excitement that an association generates generally sweeps aside gaps in logic heralding the evolution of yet another scientific myth. The principal psychosocial function of myths is to offer a vicarious resolution of the ignorance that lies between our insecurities and expectations [3]. Conflicting results inevitably follow [1]. Several autoantibodies function as biological, diagnostic or pathognomonic markers but only few of these have been conclusively linked *pathogenetically*. Although an association may be observed, determining causation involves a subjective inference, in the absence of the known cause. Even the truth or falsity of the statement ‘Smoking caused X’s lung cancer’ cannot be established [4]. The interpretation of no autoimmune phenomenon could be more incongruent than that of aPLs that are implicated in thrombotic disorders despite being often associated with prolonged activated partial thromboplastin clotting time (APTT) – *the precise effect of heparin activity itself* While the syndrome of aPLs itself is associated with thrombocytopenia [1, 5], the latter does not preclude the development of migraine [6] or its complications. The institution of platelet antiaggregating drugs is central to the suggestion that early recognition of aPLs has important practical management implications [1]. Recurrent thrombotic events have occurred in subjects with aPLs associated cerebral ischaemia while receiving warfarin sodium and/ or antiplatelet agents [5]. Realistically, given the concurrent haemio-biological profile, such a therapy in the aPLs syndrome with or without migraine is illogical. It is hardly surprising that the results have been highly inconsistent [5]. The overall significance of the presence of aPLs in the pathogenesis of migraine is attenuated by (1) the detection of such antibodies in only 3 out of 197 consecutive patients [1]; (2) the absence of difference in the *extent* and *location* of cerebral ischaemic lesions in migraine subjects with or without aPLs [1]; (3) the wide spectrum of diverse neurological diseases – several of which are unrelated to stroke – reported in association [5], and, (4) a *negative* association between migraine and aPLs in systemic lupus erythematosus (rather than merely no association) [7], suggesting that migraine was more common in the antibody-negative group – a *biologically* intriguing but statistically insignificant finding. These investigators emphasize the frequency, severity, duration and refractory nature of the underlying migraine disorder in the subjects with strokes associated with aPLs [1]; the key feature advanced for the putative role of aPLs is the absence of alternative explanations for the strokes. The weakest circumstantial evidence is that which is supported by default. Dehydration, platelet activation, cerebrovascular constriction by non-selective ̢-adrenergic antagonists, ergotism, ictal and amitriptyline-augmented orthostatic hypotension can trig-

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