

Eighth International Congress of Parkinson's Disease and Movement Disorders

June 13-17, 2004; Rome, Italy

After the disappointment of Beijing last year, when the Parkinson's disease congress was postponed because of the SARS epidemic, delegates descended upon Rome from all corners of the world, bursting to show their abstracts. Situated in the Palazzo Dei Congressi, a Mussolini-inspired building set apart, it seems, from every hotel in Italy's capital city by at least a long walk or metro ride, the organisation was enthusiastic, if not efficient.

Were their any over-arching themes to emerge from this meeting? There was certainly an emphasis on basic science and its contribution to our understanding of movement disorders. There is an increasing importance attached to the non-motor complications of Parkinson's disease, notably dementia. Therapeutically, the meeting seemed to consolidate knowledge, rather than trail-blaze. With over 1300 posters and eight parallel sessions, together with four plenary sessions, it is impossible to be comprehensive, but some highlights are selected below.

In the first plenary session, Bill Langston (CA, USA) discussed the role of environmental factors in the aetiology of Parkinson's disease (PD), covering smoking and pesticide exposure, in particular. A large NIH-funded agricultural health study should hopefully help to resolve uncertainty over whether PD is associated with an increased risk of pesticide exposure, by comparing rates in applicators with spousal controls. John Hardy (MD, USA) followed with a discussion of genetic causes of PD, in his inimitable thought-provoking style. He covered, amongst other things, the recent discovery of an α -synuclein gene triplication and its link with a PD/dementia with Lewy bodies clinical phenotype. The next speaker (Serge Przedborski, NY, USA) discussed the role of mitochondria, oxidative stress, and inflammation in the pathogenesis of PD. This was a great overview of a complex topic. Potential mechanisms for modulating the glial inflammatory response were discussed, including vaccination strategies, although his conclusion was that the optimal protective strategy of neurones in PD is likely to be a cocktail of agents. Finally, in this session Kevin McNaught (NY, USA) described a new animal model, whereby systemic exposure to proteasomal inhibitors (PSI or epoxomicin) can produce behavioural and pathological features resembling PD, including reduced 11C-CFT binding on PET scanning, loss of nigral neurones and Lewy body-like inclusions. Do all pathogenic roads now point to the ubiquitin-proteasomal system? There is certainly a lot of environmental and genetic data converging on dysfunction of this system in PD.

The afternoon plenary session focused on the basal ganglia pathophysiological model, its contributions and limitations. This was not a session for those predisposed to post-prandial drowsiness. The shortcomings of animal models are recognised (Yanagisawa, Japan), while a number of misconceptions of the current motor loop model were highlighted (Bergman, Israel), including the co-localisation of dopamine receptors, branching of striatal axons, basal ganglia-brainstem, thalamo-striatal and direct cortico-subthalamic nucleus projections. An "action selection model" was proposed, in which dopamine acts to narrow the focus of a chosen action and dyskinesias are associated with an increased "aperture". It is not the level of basal ganglia output but the pattern that is important with a marked excess of ϵ -synchrony seen in the local field potential in PD, probably linked to akinesia (Brown, UK).

David Williams (London, UK) presented a cogent argument for two distinct clinical phenotypes observed in

pathologically proven progressive supranuclear palsy, with immunoblot differences in protein banding pattern to support this notion. Essentially, "Richardson's syndrome" was suggested to represent text-book PSP, with falls, supranuclear gaze palsy and an aggressive disease course, refractory to L-dopa. "PSP-P" was proposed to represent a more benign variant, with great likelihood of L-dopa response, longer disease duration, tremor and asymmetric onset (and thus more likely to be mis-diagnosed). He won a junior award for this work.

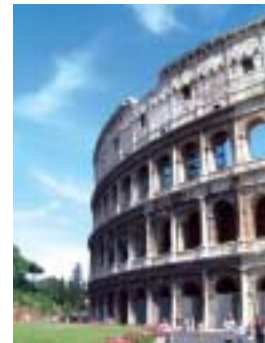
A number of posters and speakers (notably Rascol, France and Brotchie, Canada) touched upon potential new agents for dyskinesias that included levetiracetam, adenosine A2A antagonists and sarizotan (a 5HT1A agonist). An excellent parallel session featured updates on dystonia, Huntington's disease, Friedreich's ataxia, psychogenic movement disorders and essential tremor. Unfortunately, from a personal perspective, this clashed with the session on cognitive and behavioural dysfunction in movement disorders. As well as a succinct review of dementia associated with PD, this session also featured lectures on other interesting and frequently less well-covered aspects, notably apathy and motivation, reward and executive function.

Experimental interventional therapeutics for movement disorders (focusing upon PD) were addressed in a plenary session, including gene therapy, stem cells, trophic factors, and transplantation strategies. This was high-powered and exciting stuff. Latest results from the GDNF trial were presented and the use of lentiviral vectors for GDNF delivery proposed (using doxycycline to switch production on and off). In the meantime, the results of a double-blind placebo-controlled multicentre GDNF infusion trial are awaited. Potential pathogenic mechanisms for "off" (runaway) dyskinesias and strategies to optimise success for cell replacement therapy were also considered, amongst other topical themes.

From a surgical perspective, there were numerous posters and a dedicated parallel session. Is the zona incerta (ZI) a better target than the subthalamic nucleus (STN) for deep brain stimulation (DBS) for PD? From a preliminary study of 29 patients conducted in Bristol, UK (Plaha), stimulation of the ZI produced better motor outcomes. Bilateral DBS of the STN can improve motor function and reduce medication requirements for up to four years post-surgery (Liang, Pennsylvania, USA). The outcome of a double-blind multicentre study of bilateral DBS-STN (SPARK study, France) was reported, in which 97 patients in four centres underwent surgery. Mean L-dopa equivalent dose was reduced by 59%. Off medication, motor scores improved by 57% and activities of daily living by 48%. The pre-operative on-medication motor rating score was predictive of the 12-month post-operative motor outcome. In a cohort of 38 patients with advanced PD, DBS of the STN led to benefits in quality of life in both short and long (mean follow up 30.2 months) term follow up (Siderowf, Philadelphia, USA).

And finally, if you suffer from "distressful belching" (actually in the context of neuroacanthocytosis; Cuny, Bordeaux, France), DBS of the internal pallidum might be the treatment of choice, proving the range of conditions that DBS can tackle knows no bounds!

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The Colosseum, Rome
(Picture courtesy of Dr Naomi M Warren)

For a report on coma and impaired awareness, see the website at www.acnr.co.uk/conferences.htm