MSA Trust Research Strategy

Multiple System Atrophy Trust
Founded by Sarah Matheson

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What is MSA

Multiple system atrophy (MSA) is a sporadic, progressive, adult-onset neurodegenerative disease characterized clinically by autonomic dysfunction, parkinsonism, and cerebellar ataxia in any combination. Symptoms of MSA vary in distribution, onset and severity from person to person. Because of this, MSA was originally thought to be 3 different diseases: Shy-Drager syndrome, striatonigral degeneration, and sporadic olivopontocerebellar atrophy. These terms are no longer in use, and these diseases are now considered forms of MSA.

Epidemiology and aetiology

MSA occurs more commonly in men than women (approximately 1.3:1). Onset is typically in the 6th decade (average 57 years).

A study of incidence MSA indicated there are 3 new cases per 100,000 person-years in those between ages 50 and 99 There were no cases with onset younger than age 50 in this study. However, it can start as young as age 31, although there are no proven cases that started before age 30. No ñbeyond ageò for onset has been defined. Prevalence studies suggest there are 2 to 5 cases per 100,000 people. This compares with about 180 per 100,000 for Parkinsonôs Disease (which is therefore 36 times commoner). At any one time there are at least 3,000 people living with MSA in the UK.

There are no known genetic causes of MSA, and no familial pattern suggesting a strong genetic contribution to the disease. Inherited cerebellar degenerations are sometimes mistaken for MSA, but genetic investigations can often differentiate the two. However intricate modern genetic studies (so called ñgenome wide association studies) have indicated that there is a genetically determined predisposition. Environmental factors have also been implicated in MSA. Specifically, risk might be increased by an occupational history of farming, and decreased by smoking.

Pathology

Neurodegeneration in MSA-P is most prominent in the striatonigral system, while in MSA-C it is more marked in the olivopontocerebellar system.

Autonomic system involvement is accompanied by cell loss in the dorsal motor nucleus of the vagus nerve, the locus coeruleus, and the ventrolateral medulla, as well as parasympathetic preganglionic nuclei in the spinal cord. Other cell populations may also be affected.

The pathologic hallmark of MSA is the presence of cytoplasmic inclusion bodies in glial cells of the basal ganglia, supplementary and primary motor cortices, reticular formation, and pontocerebellar system. These inclusions contain ubiquitin, tau, and alpha-synuclein. These glial cytoplasmic inclusions (GCIs) are definitive for the diagnosis of MSA. The discovery of alpha-synuclein in MSA inclusions linked the disease with Parkinsonôs disease and Lewy body dementia, together known as

ñsynucleinopathies.òThe pathogenic significance of the inclusions, and how an excess of protein is involved in the disease process, is unknown.

Symptoms

MSA can cause a wide range of symptoms as shown in Table 1

Table 1

Stiffness or rigidity
Freezing or slowed movements
Postural instability; loss of balance; incoordination
Tremor
Orthostatic hypotension
Male erectile dysfunction
Urinary incontinence or incomplete bladder emptying
Constipation
Speech and swallowing difficulties
Sleep disorder

There are 2 forms of motor presentation in MSA:

MSA as parkinsonism - MSA-P

Parkinsonism is seen in the majority of patients at onset. Akinesia, rigidity, and tremor are often asymmetrical, and postural tremor more common than rest tremor. Flexion of the head and neck is common.

MSA as cerebellar ataxia - MSA-C

Cerebellar symptoms are seen in about 20% of patients at onset, presenting as limb ataxia, gait ataxia, scanning dysarthria, and oculomotor dysfunction. At first presentation MSA-C is difficult to distinguish from late-onset pure cerebellar ataxia until the disease progresses to include other MSA signs and symptoms.

In addition, *autonomic disturbance* is seen in both MSA-P and MSA-C. It is present at diagnosis in about 40% of patients, and develops shortly after diagnosis in virtually all other patients. In males, erectile dysfunction is the most frequent initial symptom, while in females, urinary incontinence is seen most often. Orthostatic hypotension is present in about two-thirds of patients in the later stages of the disease although sometimes it occurs early. Faecal incontinence affects about one-third of patients. Heat intolerance may be a problem in warmer climates due to impaired sweating.

Dysphagia is common early in the disease. Sleep disorders, particularly during the rapid eye movement (REM) phase and sleep behaviour disorders, occur in about two-thirds of patients. Confusion and hallucinations are uncommon, and much less frequent than in Parkinsonôs disease (PD).

MSA progresses over the course of several years to cause more widespread and severe symptoms. Orthostatic hypotension can cause fainting and falls. Loss of coordination, slowed movements, and rigidity can interfere with activities of daily living. Some patients with MSA develop mild cognitive impairment.

Diagnosis

The diagnosis of MSA is currently a clinical one. Consensus criteria originally formulated in 1998 were refined and updated by a group of experts in the field, and published in late 2008 (Gilman et al., 2008). The diagnosis can be described as ñpossibleò, ñdefiniteòor ñprobableòaccording to the stage of progression.

Treatment

There are as yet no treatments that halt or slow the neurodegenerative process, although trials are currently in progress to investigate putative disease-modifying drugs. There are also no symptomatic treatments for the cerebellar symptoms. Treatments are available for parkinsonism using the same medications as are given in Parkinsonôs Disease and also for autonomic symptoms, although these tend to become less effective as the disease progresses.

Prognosis

The interval from symptom onset to combined motor and autonomic dysfunction is predictive of functional deterioration and survival in MSA. In a Japanese study of 230 (mostly MSA-C) patients, the median time from symptom onset to multi-system involvement was 2 years. Median survival time was 9 (range 2-17) years. Time to assisted walking, wheelchair requirement, bed confinement, and death were proportional to time between symptom onset and multisystem involvement. Patients in whom multisystem involvement occurred within one year of symptom onset proceeded to each of these milestones 2 to 3 times as fast as those for whom multisystem involvement occurred after 3 years or more.

In the London brain bank series of 100 (mostly MSA-P) cases average survival was 7 (range 2-16) years.

References

Gilman S et al. The North American Multiple System Atrophy Study Group. J Neural Transmission. 2005;112:1687-1694.

S Gilman, GK Wenning, PA Low, DJ Brooks, CJ Mathias, JQ Trojanowski, NW Wood, C Colosimo, A Durr, CJ Fowler, H Kaufmann, T Klockgether, A Lees, W Poewe, N Quinn, T Revesz, D Robertson, P Sandroni, K Seppi, M Vidailhet. Second consensus statement on the diagnosis of multiple system atrophy. Neurology. 2008;71:670-676

Wenning GK, Colosimo C. Geser F, Poewe W. Multiple system atrophy. Lancet Neurology 2004;3:93-103.

MSA Trust Research Strategy

MSA Trust and Research

When Sarah Matheson was diagnosed with MSA in 1993, she was dismayed at the lack of readily available information on MSA for both those diagnosed with the condition, and the health and social care professionals. Sarah felt this lack of information led to isolation for people living with MSA due to the general ignorance about the condition. She conceived the idea of a Support Group that would both support those with the condition and those involved in their care. The Sarah Matheson Trust became a registered charity in 1997. She died on the 17th September 1999, six years after being diagnosed with MSA. The work of the Trust (renamed the Multiple System Atrophy Trust in June 2010) continues in her memory and is growing, thanks to the support of its membership and the dedication of its staff, Trustees and Patrons. The Multiple System Atrophy Trust is the only UK support charity for people with MSA. It is funded entirely by donations.

In 2005 Prof Clare Fowler was asked by the then Sarah Matheson Trust to set up a Research Advisory Panel (RAP) to advise on and develop the Trustôs support for research and oversee the allocation of its grant money. This is an important activity for any charity and there are recommendations for the governance of the process laid out by the Association of Medical Research Charities (AMRC). A panel of experts was duly invited to join the RAP, Prof John Newsom-Davis (Oxford), Prof Martin Rossor (London), and Prof David Burn (Newcastle). The panel was unanimous in agreeing that the call for research should be targeted at ñproposals for research aimed at discovering the cause of MSAò, rather than spending the money on a number of smaller palliative care projects.

The Trustôs Treasurer advised that he thought there would be £60K a year available for the foreseeable future. Prof Newsom-Davis was also able to secure a further £60k for 3 years to the Sarah Matheson Trust, to fund a Research Fellow from the Welton Foundation Trustees. The Sarah Matheson trust nominated Ms Darcy Hare as the Trustee appointed to liaise with the panel and assistance from the Sara Koe PSP Research Centre was gratefully received, allowing us to modify their grant

application forms. Subsequently we received assistance from Parkinsonôs UK with the grant assessment process.

Following the tragic death of Prof Newsom-Davis in 2007, Prof Nicholas Wood (London) was invited to join the RAP and then in 2008, Prof Gregor Wenning from Innsbruck, who had been instrumental in setting up European MSA- SG (see page 13).

In June 2006 the first call advertising for applications failed to attract any responses. The call was for a research fellow to write their own application to work in a suitable host laboratory but at that time the impact of ñModernising Medicine Careersòhad had a very negative impact on UK junior doctors wishing to do research. The terms of the grant funding were therefore changed to an appeal for proposals for small ñProject Grantsò. The call was widely advertised in 2007 and three applications received which were independently reviewed by experts from institutions different from the applicants. Prof Tamas Revesz and Dr Janice Holton (London) were awarded a one year grant - the Margaret Watson Memorial Grant - in Jan 2008. Further money was made available by the Trust in 2008 and, following due process and a proper review, two 3 year grants were awarded in June 2008 to Dr Henry Houlden and Dr Janice Holton (London), respectively. Regrettably no money was available in 2009 but in 2010, a widely advertised grant of £30K for one year, resulted in an award to Dr Shahin Zibaee a post-doctoral scientist from Cambridge.

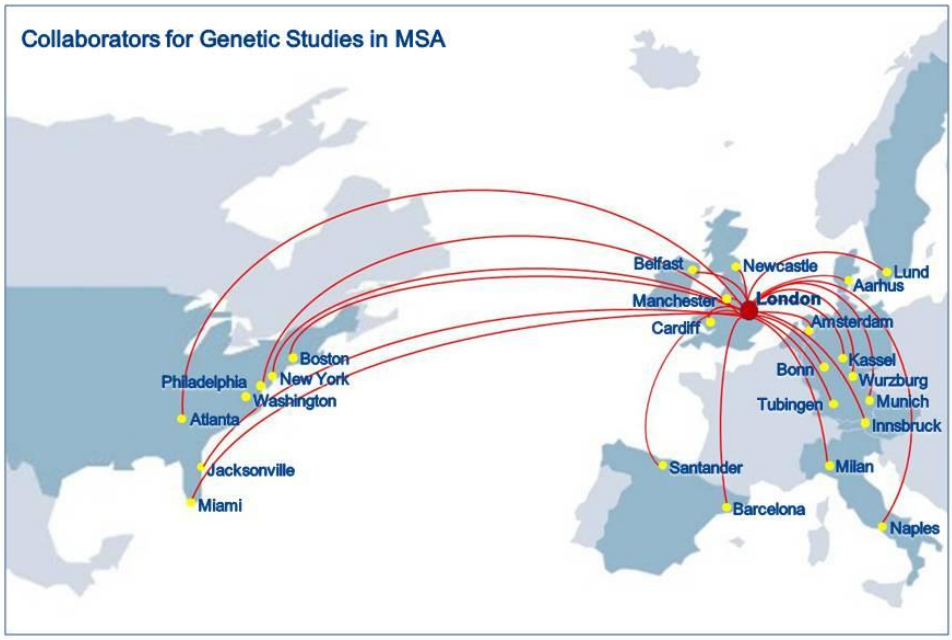
Previous grants awarded
St Mary's Development Trust
Dr Nin Bajaj
Dr Timothy Young
Dr Anette Schrag

Reports from currently funded research

Genetic Analysis of MSA – Professor Houlden

Why people develop MSA is still largely unknown but needs to be elucidated in order to better understand the disease and to develop effective treatment. In similar disorders such as Parkinsonôs and Alzheimerôs disease laboratory research has been established for many years, hence we know a great deal about the causes and treatment trials are already underway. The important research findings have come from finding disease-causing genes for these disorders.

MSA research has so far lagged behind in this field, but thanks to a Multiple System Atrophy Trust grant awarded to Professor Houlden we are now intensifying research into genetics of MSA at the Institute of Neurology, London. We are currently performing a genome wide association study screening 1000 MSA patients for genetic risk factors. Many collaborators all over Europe and the US have collected DNA from MSA patients and are contributing these to our study (see map). By this study we want to identify genetic risk factors that contribute to the development MSA. Identifying such genetic causes and understanding how they contribute to disease development is very important for future research into treatment possibilities of MSA.



Neuropathological changes – Dr Janice L. Holton JL, and Prof Tamas Revesz

Neuropathological research has played an important role in advancing our understanding of a number of neurodegenerative diseases such as Alzheimer ôs Disease, Parkinsonôs Disease and Progressive Supranuclear Palsy. Further neuropathological studies in MSA are expected to contribute to our understanding of the disease process. In MSA a protein called Ŭ-synuclein accumulates within nerve cells and is even more prominent in the glial cells which support them, the oligodendrocytes. This abnormal build-up of Ŭ-synuclein inside oligodendrocytes is not only important for the microscopic diagnosis of MSA, but is also thought to be a fundamental process in developing the disease. Thanks to funding from the Multiple System Atrophy Trust we are investigating important questions such as how and why Ŭ-synuclein accumulates in oligodendrocytes in MSA and how this may cause damage to nerve cells. As part of this research we are investigating whether the precursors of oligodendrocytes are also damaged by the disease process. Ultimately, answers to these fundamental questions will improve our understanding of MSA and pave the way for much needed treatments.

The importance of the Queen Square Brain Bank

The Queen Square Brain Bank (QSBB) gives access to a unique resource of human brain tissue, provided by those who have died of MSA and who expressed in life the wish to contribute to research. This collection of brains from patients with MSA is the largest in the world. The active programme of movement disorder research at the QSBB provides considerable added value for MSA research, as improved understanding of other diseases can give information and insights into MSA. This is particularly relevant in studies of Parkinsonôs Disease which, in common with MSA, is a member of the group of diseases known as Ŭ-synucleinopathies, and also Progressive Supranuclear Palsy which shares with MSA the major feature of prominent abnormalities affecting glial cells in addition to nerve cells.

Publications arising from MSA Trust funded research.

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2. LRRK2 and parkin immunoreactivity in multiple system atrophy inclusions. Huang Y, Song YJ, Murphy K, Holton JL, Lashley T, Revesz T, Gai WP, Halliday GM. (2008) Acta Neuropathol. 2008 Dec;116(6):639-46

3. Clinical outcomes of progressive supranuclear palsy and multiple system atrophy. O'Sullivan SS, Massey LA, Williams DR, Silveira-Moriyama L, Kempster PA, Holton JL, Revesz T, Lees AJ. (2008) Brain. 2008 131:1362-1372

4. Glucocerebrosidase mutations in 108 neuropathologically confirmed cases of multiple system atrophy. Segarane B, Li A, Paudel R, Scholz S, Neumann J, Lees A, Revesz T, Hardy J, Mathias CJ, Wood NW, Holton J, Houlden H. (2009) Neurology 72(13):1185-6

5. SNCA variants are associated with increased risk for multiple system atrophy. Scholz SW, Houlden H, Schulte C, Sharma M, Li A, Berg D, Melchers A, Paudel R, Gibbs JR, Simon-Sanchez J, Paisan-Ruiz C, Bras J, Ding J, Chen H, Traynor BJ, Arepalli S, Zonozi RR, Revesz T, Holton J, Wood N, Lees A, Oertel W, Wüllner U, Goldwurm S, Pellecchia MT, Illig T, Riess O, Fernandez HH, Rodriguez RL, Okun MS, Poewe W, Wenning GK, Hardy JA, Singleton AB, Gasser T. (2009) Ann Neurol. 65(5):610-4

6. Concomitant progressive supranuclear palsy and multiple system atrophy: More than a simple twist of fate? Silveira-Moriyama L, González AM, O'Sullivan S, Williams DR, Massey L, Parkkinen L, Ahmed Z, de Silva R, Chacón JR, Revesz T, Lees AJ, Holton JL. (2009) Neurosci Lett. 467:208-11

7. Degeneration in different parkinsonian syndromes relates to astrocyte type and astrocyte protein expression. Song YJ, Halliday GM, Holton JL, Lashley T, O'Sullivan SS, McCann H, Lees AJ, Ozawa T, Williams DR, Lockhart PJ, Revesz TR. (2009) J Neuropathol Exp Neurol. 68:1073-83

8. Mutational analysis of parkin and PINK1 in multiple system atrophy. Brooks JA, Houlden H, Melchers A, Islam AJ, Ding J, Li A, Paudel R, Revesz T, Holton JL, Wood N, Lees A, Singleton AB, Scholz SW. (2009) Neurobiol Aging. Dec 22 Epub

9. Ahmed Z, Tabrizi SJ, Li A, Houlden H, Sailer A, Lees AJ, Revesz T, Holton JL. (2010) A Huntington's disease phenocopy characterised by pallido-nigro-luysian degeneration with brain-iron accumulation and p62-positive glial inclusions. Neuropathol Appl Neurobiol. May 20. [Epub ahead of print]

10. Zibaee S, Fraser G, Jakes R, et al: Human {beta}-Synuclein rendered fibrillogenic by designed mutations. J Biol Chem 285:38555, 2010

Presentations:

XVIIth International Congress of Neuropathology, Salzburg, September 11th ï 15th 2010

1. Oligodendrocyte precursor cells do not contain pathological inclusions in multiple system atrophy or progressive supranuclear palsy. Ahmed Z, Asi YT, Lees AJ, Revesz T and Holton JL.
2. Disequilibrium in MSA phenotype distribution between populations: genetics or environment? Ozawa T, Holton JL, Paviour D, Lees AJ, Tada M, Kakita A, Onodera O, Wakabayashi K, Takahashi H, Nishizawa M, Revesz T.

International MSA Research

The graph below (Figure 1) shows the 1060 publications, found by searching titles in the database PubMed with the term ñmultiple system atrophyò, plotted by year of publication.

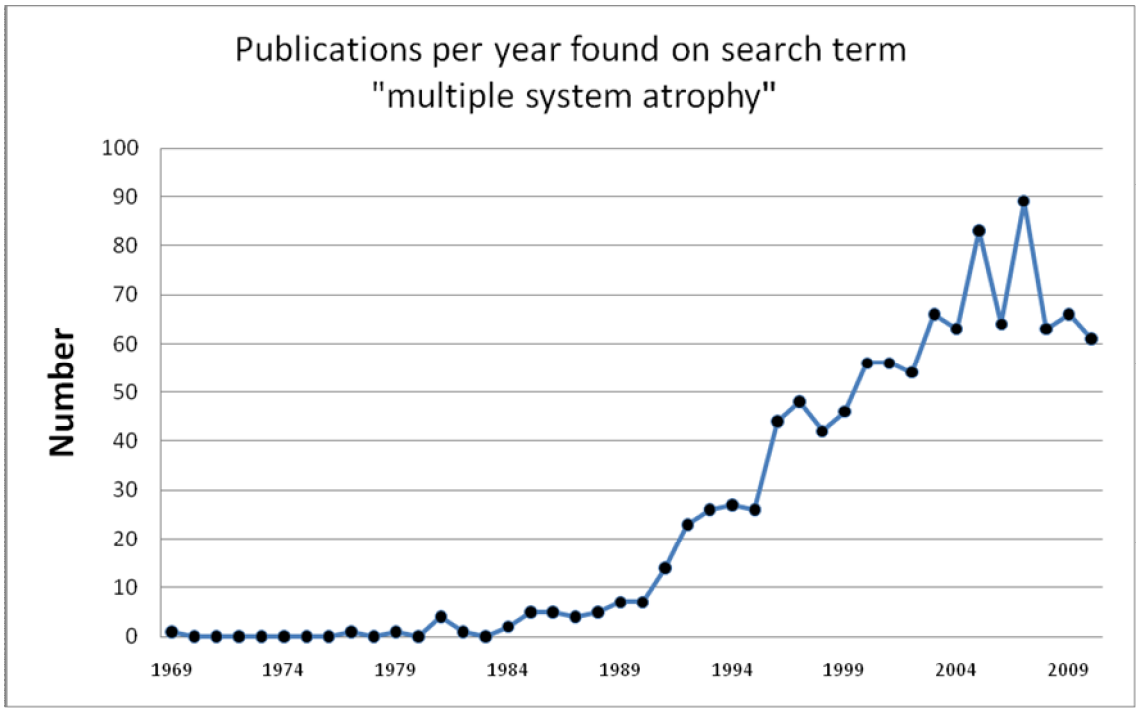

Figure 1 Publications by year found in PubMed with the search phrase "multiple system atrophy"

Although Bannister and colleagues starting publishing on aspects of orthostatic hypotension in this disease in 1967, the first publication which included MSA in the title was by Graham and Oppenheimer in 1969 ñOrthostatic hypotension and nicotine sensitivity in a case of multiple system atrophyò. The next was in 1977, but in 1981 there were 4. From the early 1980s onwards there was a steady increase and in the last 10 years (2000-August 2010) there have been 616 publications.

Figure 2 shows the country of origin, when given, of the addresses of authors of 992 of the above papers. Note that before the early 1990s the address was not always captured by PubMed.

This shows that research into MSA is now of international concern, reflected by the location of interested groups. UK researchers have excellent links with groups in US and there are also connections between researchers on a personal level between the UK and researchers in Japan. Connections and collaborations within Europe, incorporated in the ñEuropean MSA Study Groupò (see below) are strong, and regular annual meetings are attended by several UK researchers.

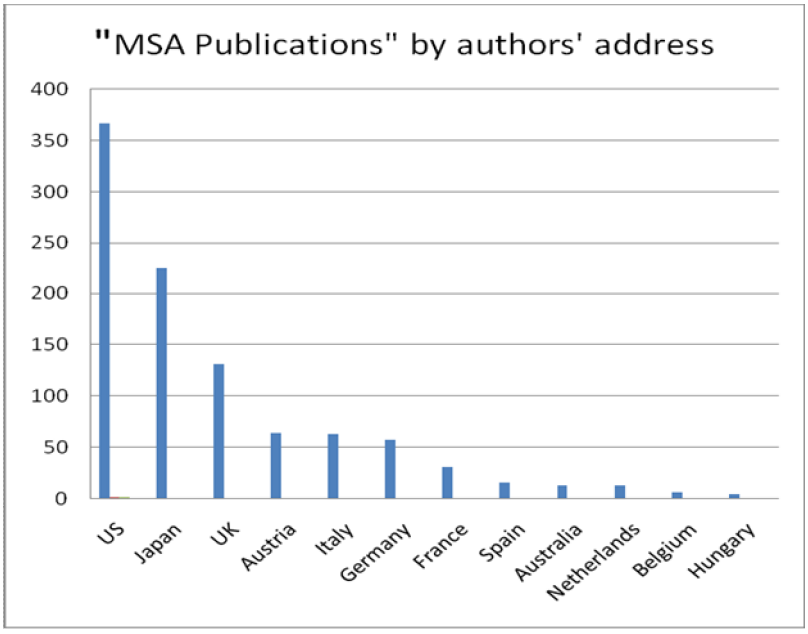

Figure 2 Coutry of origin, when give, for address of authors of papers in PubMed found by the search phrase "multiple system atrophy"

International collaboration has been important in the currently funded Genome Wide Association Study (see page 8) and the attendence of UK researchers at American and European meetings has been important in establishing working relationships.

The European Multiple System Atrophy-Study Group (EMSA-SG) is an academic network comprising 23 centres across Europe and Israel that was set up in January 1999. This international forum of recognized experts was established under the guidance of the University Hospital of Innsbruck as the coordinating centre, and was initially supported by a European Union grant. The primary goals of the network were (1) to create a central Registry for European Multiple System Atrophy (MSA)

patients, (2) to create a decentralized DNA Bank, (3) to develop and validate the novel Unified MSA Rating Scale (UMSARS), (4) to conduct a Natural History Study, and (5) the planning or implementation of interventional therapeutic trials. Many of these initial goals have now been sucessfully achieved and EMSA-SG continues to be a very valuable group with which our current grant holders work closely.
There has not, however, until recently been an identifiable UK nucleus of MSA researchers. Fortunately the awards of the MSA Trust grants to researchers at the Institute of Neurology at Queen Square, London has now reached a critical mass of researchers who, building on their previous work on the clinical aspects, neuropathology and genetics of MSA and enabled by complementary skills and experience, have formed an MSA Study Group. They enjoy frequent opportunities to exchange ideas in regular meetings and on a day to day basis. The group is further strengthened because it is embedded in the wider research environment of the Institute of Neurology which has a track record of excellence in multidisciplinary research into movement disorders involving neuropathologists, geneticists, molecular biologists, neurologists, neuroradiologists and others.

A better UK-wide research organization could certainly enhance co-operation within the country and lead to improved pooling of resources and exchange of ideas. A ñUK MSA Research Dayòwould go some way to promoting this. and one is now planned for November 2011.

Future Research Strategy

The current stated research strategy for the MSA Trust is to find the Cause of MSA and a Cure. Continued research into the mechanisms underlying this fatal neurodegenerative disease must be a prime aim of the Trust, because without this knowledge the development of prevention and treatment strategies for MSA cannot be realised. An additional aim is education ï of patients, their carers, relatives and the general public about the condition.

In a recent survey (Sept 2010) of 358 patients with MSA and their carers funding research was considered one of the highest priorities (Figure 3). Those affected by the disease wish for a cure for themselves but must be aware that this may not be found in their life time. They want to know that the medical world is actively engaged in trying to understand why people develop MSA and what can be done about it ï and that is through research.

Funding research has a powerful appeal to those who have first hand knowledge of MSA and also provides a persuasive platform from which to launch an educational campaign.

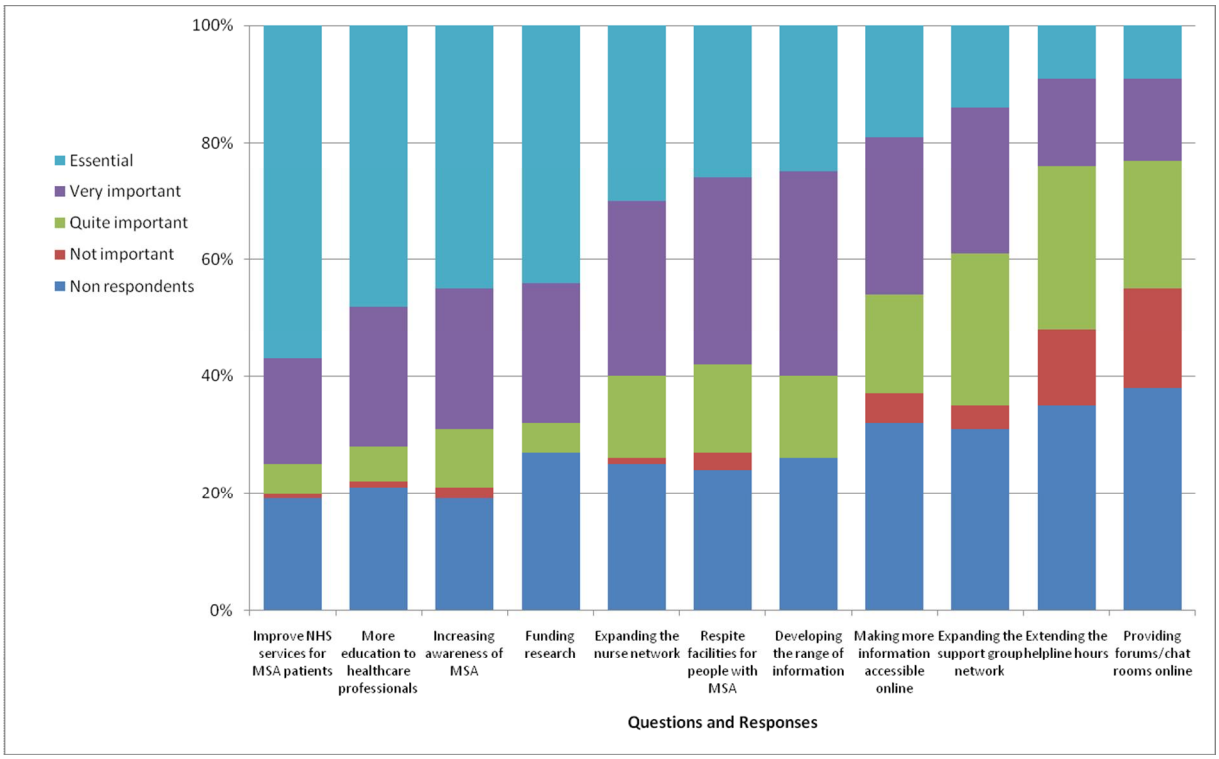

Figure 3 Respondents (358) believe the top priority for the Trust is to encourage government to improve NHS services, followed by increasing awareness about MSA amongst healthcare professionals and the general population, then funding MSA research (63%). (From Survey Analysis, September 2010 by Jackie Davis)

In an ideal world the MSA Trustôs Research Strategy would include investigations into Care and Treatment but facing the problems of very limited resources, prioritization has been necessary. Recognizing the importance of research into those needs the Trust will direct and actively encourage applicants who wish to research into symptomatic management of MSA to alternative sources of funding whilst supporting their applications with evidence of ñpatient opinionòor putting them in touch with patients should that be required.

Cause and Cure

The finding of the cause of MSA, be it either at an environmental or genetic level ï or both - would be a crucial advance of such magnitude as to be secondary in importance only to finding a cure, but a cure is only likely through understanding the cause.

It is quite possible that the cause of MSA may be discovered incidentally by workers in another but related field, and likewise some effective treatment that halts or modifies disease progression found. Certainly progress in understanding the molecular basis of MSA has advanced considerably through research into Parkinsonôs and Alzheimerôs disease and this is likely to continue, as has been highlighted in the section on ñInternational MSA Researchò. Meanwhile however funds spent on fundamental research into MSA in the UK are paying significant dividends ï see ñCurrently Funded Researchò.

Early recognition of MSA

This is clearly important both for individual patients and against the day when some intervention is found which halts or slows the progression of disease. It is important, too, in understanding the epidemiology of the condition and hence possibly finding a cause for the disease. It is however an area of clinical activity which has already been much studied (Gilman, et al 2008 and EMA-SG publications) with less than the hoped for result. A novel approach identifying a ñbiomarkerò would seem a promising alternative approach and a proposal for such a project would be encouraged by the MSA Trust.

Why “treatments” should not be funded

The discovery of treatments which modify the progression of the disease will be extremely expensive and must be the province of the pharmaceutical industry and encouragingly some studies are now in progress. Drug trials nowadays require complex and stringent regulatory approvals and a significant work force to organize and execute.

Although the Multiple System Atrophy Trust cannot fund drug trials, it will set up a system whereby patients wishing to take part in treatment trials can find out from the MSA Trust website what studies are in progress or expected to be recruiting in the near future. The intention is that the information provided there will give some background about the scientific aim of the clinical trial and other relevant details. It will also give contact details of the neurologist leading the trial so that patients can

either ask to be referred to that centre or find out if there is a neurologist closer to where they live who is a trial investigator.

In due course the MSA Trust intends to establish a database of the names of patients willing to take part in drug trials and who have consented to have their details forwarded to a neurologist recruiting for a treatment trial. The MSA Trust will also engage with the NIHR topic-specific Dementia and Neurodegenerative Diseases Research Network (DeNDRoN-UK) to discover when trials will become available.

Through the website the MSA Trust aspires to empower people affected by the condition and their carers, providing downloadable information, updating them with regard to ongoing and nascent studies and promoting the general ethos of trial participation.

Symptomatic management

This is currently the activity which the MSA Trust does best. Two highly experienced nurses are funded by the Trust to provide information and support for patients. Furthermore in the UK the NHS is committed to providing care for patients at all stages of their illness. What is constantly required on their behalf, and is made clear from the recent survey, is better education of healthcare workers in many different spheres ï both primary and secondary care, including better knowledge amongst neurologists, ENT and urological surgeons and palliative care physicians, so that there can be the best utilization of existing resources. Those with progressive and fatal neurological diseases do not at the moment receive the same palliative support as do patients with cancer.

Applications to NHS funding sources such as the National Institute for Health Research: Research for Patient Benefit Programme (RfPB) will be actively encouraged by the MSA Trust. For those seeking to symptomatically improve care of patients with MSA, this can provide a suitable alternative appropriate source of

funding. (RfPB has been providing up to £25 million each year. It supports projects of to 36 months in duration with a total maximum cost of £250,000.)

Also see comments above with regard to NIHR DeNDRoN regarding engagement of research active centres and the opportunity to improve recruitment to clinical trials.

Education

Better knowledge of the disease MSA will clearly be a priority of the Trustsô Information and Support Officer. With a comparable incidence to Motor Neurone Disease it is surprising that there is so little public awareness of MSA. The explanation for this is probably complex and must include the diagnostic difficulty of cases, some confusion in the minds of the medical profession about the precise nature of MSA, and the fact that medical research has only focused intensively on the condition in the last 10 years (Figure 1). Education of the public will be crucial and will engender a better understanding of the importance of scientific research to patients and carers of those with MSA.

Funding of applications

In the current financial climate it will be necessary to focus on our identified priorities ï which will both enhance the aim of finding a ñCause and Cureòand add value to what has already been achieved. However all applications will be properly reviewed in line with AMRC criteria, and funding awarded on scientific merit.

1st UK MSA Senior Research Fellow

The focus of the 2012 MSA Trust anniversary appeal has been identified as obtaining funding for the post of a 3-5 year senior research fellow dedicated to MSA research.

MSA Trust Research Strategy

Summary of Recommendations

1. Plan 1st UK MSA ResearchersôMeeting ï 4th November 2011.

2. Raise money to fund the 1st UK MSA Senior Research Fellow to lead future research towards finding a ñCause and Cureò

3. Publicise the importance of MSA scientific research to patients, carers and the general public.

4. Apply for Membership of AMRC

5. Aid and encourage research applications to National Institute for Health Research: Research for Patient Benefit Programme (RfPB) by those seeking to symptomatically improve care of patients with MSA.

6. Provide information via the website about current and future clinical treatment trials. Contact names of centres participating in trials will also be provided so that patients can consider volunteering as subjects.

7. Aim to establish a UK Registry of people diagnosed with the disease who have given consent to be approached by investigating neurologists to take part in treatment trials.

8. Promote efforts of the MSA Trust to join other progressive neurological disease alliances to improve accessibility to NHS facilities for symptomatic and palliative care.

9. Develop closer links with NIHR DeNDRoN.

10. Encourage patients to consider the benefit to science of arranging for their brain, at death, to be donated to Queen Square Brain Bank.

Acknowledgements

The contributions of The MSA Study Group at the Institute of Neurology, Dr Janice Holton and of Prof Niall Quinn in particular are gratefully acknowledged.

Professors Rossor, Burn and Wenning kindly commented and all their suggested changes have been incorporated in this version.

Eileen Lady Strathnaver and Nickie Roberts suggested alterations to better align the proposals for research with the overall aims of the MSA Trust.

This document is the result of revisions and clarifications requested at the Trusteesô meeting, held on 14th December 2010 and subsequent suggestions made by Sir Roger Bannister, Patron of the Trust.