1. The Academy of Medical Sciences welcomes the opportunity to respond to the Nuffield Council of Bioethics consultation 'Dementia – Ethical Aspects'. The Academy promotes advances in medical science and campaigns to ensure that these are translated as quickly as possible into benefits for society.

2. The Academy has previously addressed issues relating to neurological conditions in the following publications:
   - 'Restoring neurological function' published in 2004.[1]
   - The Academy of Medical Sciences' response to the 2004 House of Lords Science and Technology Committee inquiry into the scientific aspects of ageing.[2]
   - Academy of Medical Sciences, Medical Research Council, Royal College of Physicians and Wellcome Trust briefing ahead of the Second Reading of the Mental Capacity Bill in the House of Lords in 2005.[3]
   - 'Use of non-human primates in research', published in 2006.[4]
   - 'Brain science, addiction and drugs', published in 2008.[5]

   Current Academy activity also includes a focus on the strategic direction of future research into ageing and age-related disease.[6] We have chosen to respond primarily with reference to particular research aspects of dementia and would be happy to expand on any of the points made in this response.

Summary

3. Worldwide, dementia is responsible for 11.2% of all years lived with disability among people aged over 60 years. In the UK alone, there are now almost 700,000 people living with dementia, equivalent to 1.1% of the UK population, at a cost of £17 billion per year, and prevalence is projected to rise by 38% over the next 15 years. Yet just 1.4% of research papers published since 2002 focus on dementia, compared to 23.5% for cancer and 17.6% for cardiovascular disease.[7] Significant increases in research investment will thus be required as the impact of dementia grows. Continued support for epidemiological, basic, clinical, and health services research is needed, combined with the development of initiatives to encourage collaboration between researchers and to attract new researchers to the field to ensure that there is adequate capacity for dementia research in the future.

4. To date, medical research has yielded significant advances in our understanding of the pathogenesis of dementia and ongoing genetic and biochemical studies hold much promise for refining current models and highlighting novel drug targets. Specific areas in which significant developments are likely to arise include:

[1] http://www.acmedsci.ac.uk/p48prid15.html
[2] http://www.acmedsci.ac.uk/p100puid32.html
[3] http://www.acmedsci.ac.uk/p100puid71.html
[4] http://www.acmedsci.ac.uk/p99puid83.html
[5] http://www.acmedsci.ac.uk/p99puid126.html
[6] http://www.acmedsci.ac.uk/p47.html
[7] Knapp M & Prince M *et al.* (2007). *Dementia UK. The full report.* http://www.alzheimers.org.uk/downloads/Dementia_UK_Full_Report.pdf

- Gene association and large cohort studies.
- Molecular refinement of the amyloid cascade hypothesis of pathogenesis, which implicates the amyloid-beta (Aβ) protein in neuronal destruction.
- Advanced understanding of the molecular pathways of neurodegeneration.
- Characterization of biomarkers for dementia.
- Development of quick, accurate and non-invasive diagnostic tests.

5. We emphasise the need to continue research involving individuals that lack capacity in order to bring potential benefits to people with mental incapacity in the future. We support the Mental Capacity Act 2005 in assuring that strict safeguards are in place to protect vulnerable individuals who participate in research.

Key developments in dementia research

Genetic and molecular studies

6. To date, genetic and biochemical studies have been critical in enabling advances in our understanding of the molecular basis of dementia. Over the past 25 years, it has become clear that the proteins forming abnormal deposits in the brains of people with dementia (namely Aβ, tau and synuclein) are central to the disease process. Genetic studies have been crucial for these findings, together with the development of the widely accepted amyloid cascade hypothesis, which implicates Aβ as a critical early step in the process leading to neuronal dysfunction and eventual destruction.[8] In the future, it is likely that continuing molecular and genetic analysis will bring significant advances in our understanding of the neurodegenerative pathways leading to pathogenesis and will subsequently lead to the generation of novel treatments.

7. In particular, we believe that significant developments will arise from whole genome studies performed on large populations, through the identification of susceptibility genes for various dementias. To date, studies have demonstrated the role of autosomal genes such as MAPT and progranulin in fronto-temporal dementia,[9] the Amyloid Precursor Protein gene (APP), the presenilins (PS-1 and PS-2),[10] and the ε4 allele of Apolipoprotein E (ApoE)[11] in the pathogenesis of Alzheimer's disease (AD), and recent data also suggest that variants of the *CALHM1* gene may influence the risk for late-onset AD.[12] Yet, the precise mechanism by which ApoE4 functions remains to be clarified[13] and recent genetic studies have highlighted the probability that a unique constellation of multiple genetic risk factors, and complex

[8] Reviewed in Goedert M & Spillantini MG (2006). *A century of Alzheimer's disease*. Science **314(5800)**, 777-81, Lambert JC & Amouyel JP (2007). *Genetic heterogeneity of Alzheimer's disease: complexity and advances*. Psychoneuroendocrinology **32**, S62-S70.
[9] Reviewed in Boeve BF & Hutton M (2008). *Refining frontotemporal dementia with Parkinsonism linked to chromosome 17: introducing FTDP-17 (MAPT) and FTDP-17 (PGRN).* Archives of Neurology **65(4)**, 460-464.
[10] Hardy J (2006). *Alzheimer's disease: the amyloid cascade hypothesis: an update and reappraisal.* Journal of Alzheimer's Disease **9(3 Suppl)**, 151-3, Lambert JC & Amouyel P (2007). *Genetic heterogeneity of Alzheimer's disease: complexity and advances.* Psychoneuroendocrinology **Suppl 1**, S62-70.
[11] Roses AD (1996). *Apolipoprotein E alleles as risk factors in Alzheimer's disease*. Annual Reviews of Medicine **47**, 387–400, Russo C *et al.* (1998). *Opposite roles of apolipoprotein E in normal brains and in Alzheimer's disease.* PNAS **95**, 15598-15602.
[12] Dreses-Werringloer U *et al.* (2008). *A Polymorphism in CALHM1 Influences Ca^{2+} Homeostasis, Aβ Levels, and Alzheimer's Disease Risk.* Cell **133**, 1149-1161.
[13] Strittmatter WJ *et al.* (1996). *Apolipoprotein E: high-avidity binding to beta-amyloid and increased frequency of type 4 allele in late-onset familial Alzheimer disease*. PNAS **90(5),** 1977-81.

interactions between genes and environmental triggers, give rise to late-onset AD.

8. Recent molecular studies have demonstrated that many neurological diseases previously thought to be unrelated may share common mechanisms. For instance, it is thought that α-synuclein in Parkinson's disease might have similar neurotoxic effects to the amyloid oligomers in AD.[14] Whilst AD is the most prevalent cause of dementia, identification of overlapping mechanisms between different forms of dementia would bring welcome advances.

9. In the future, it will be important for molecular studies to achieve a more detailed understanding of the current model of dementia pathogenesis. Recent progress supports the amyloid cascade hypothesis but further refinement of the model is required.[15] For instance, the degree of amyloid deposits in the brain does not always correlate with the extent of cognitive impairment. Additionally, whilst AD-causing mutations increase Aβ deposition, *in vitro* experiments show that the degree to which a given mutation affects Aβ production does not always correlate with the age at which it first produces symptoms. Despite identification that amyloid, tau, presenilins and ApoE4 all have important roles, the precise mechanisms of neurodegeneration also remain to be elucidated.

Epidemiological cohort studies

10. To complement genetic studies, long-term cohort studies, to include a range of ethnic groups, will generate further advances in preventing dementia through an enhanced understanding of the influence of risk factors earlier in life, such as hypertension, diet and lifestyle. A complex array of genetic and environmental factors are involved in the development of dementia and whilst understanding these interactions represents a significant research challenge, such studies will yield important data to facilitate the development of prevention strategies and novel therapeutics.

11. Epidemiological research should also be viewed in the global context. The incidence of dementia is rising fastest in low- and middle-income countries, yet less than 10% of all population-based research into dementia has been directed towards the two-thirds or more of people with dementia who live in these countries. Research studies establishing incidence, together with care facility capacity and health system challenges such as the long-term study by the 10/66 dementia research group,[16] co-ordinated through the Institute of Psychiatry at King's College London, should be expanded.

In vivo studies

12. The generation of more complex and appropriate animal models for dementia will be key to confirming *in vitro* findings and testing novel hypotheses. Over recent years *in vivo* studies have enabled hypotheses of pathogenic mechanisms to be experimentally demonstrated. For instance, transgenic mouse models have indicated the pathogenic importance of an inflammatory immune response and the relationship between the load of Aβ

---

[14] Obi K *et al.* (2008). *Relationship of phosphorylated alpha-synuclein and tau accumulation to Abeta deposition in the cerebral cortex of dementia with Lewy bodies*. Experimental Neurology **210**, 409-420.

[15] Hardy J & Selkoe DJ (2002). *The amyloid hypothesis of Alzheimer's disease: progress and problems on the road to therapeutics*. Science **297**, 353-356, Lambert JC & Amouyel P (2007). *Genetic heterogeneity of Alzheimer's disease: complexity and advances*. Psychoneuroendocrinology **32(S1)**, S62-S70.

[16] 10/66 Dementia Research Group. http://www.alz.co.uk/1066/

and the degree of tau pathology, which is most closely associated with neuronal loss.[17] In coming years, animal models will make it possible to:

- Unravel the molecular detail of pathways linking protein aggregation to neuronal dysfunction and death.
- Understand mechanisms by which Aβ and tau cause cell death.
- Study genetic and pharmacological modifiers of the disease process.
- Assess the role of specific risk factors.

Diagnosis and monitoring

13. There is a strong case for investing in research in the field of diagnostics to improve the accuracy with which dementia is diagnosed in the early, rather than later, stages of the disease (see paragraphs 22-25 for further discussion of the benefits and risks of early diagnosis). Accurate diagnosis facilitates epidemiological research and enables early therapeutic intervention, monitoring of clinical progression and assessment of the efficacy of treatment. At present it is not possible to distinguish between clinical symptoms in the early stages of dementia and those of Mild Cognitive Impairment (MCI) or mild cognitive manifestations of other neuropsychiatric conditions such as depression; MCI is prodromal for dementia caused by AD in just 50% of cases. Distinguishing between dementia and distinct causes of cognitive decline, such as Creutzfeldt-Jakob disease, HIV-associated dementia, neurosyphilis and cerebrovascular disease, is a further consideration.[18]

14. The identification of biomarkers that enable early diagnosis is a pivotal feature of diagnostic research. Monitoring of disease progression and response to therapy through links between biomarkers and clinical outcomes will be important for long-term epidemiological studies. Further precise biomarkers specific to the various forms of dementia are required.

15. Promising research is identifying genetic, biochemical and neuroimaging biomarkers, particularly in the context of AD,[19] where proteomic approaches have identified possible markers in the cerebrospinal fluid (CSF) and blood.[20] Structural neuroimaging is being used in clinical trials, functional neuroimaging using positron emission tomography (PET) is able to detect abnormal protein deposits in the brain,[21] and analysis of the distribution of ^{18}F—fluorodoxyglucose (FDG) throughout the brains of patients and healthy subjects can demonstrate whether cognitive decline has occurred. Promising

---

[17] Rapoport M *et al.* (2002). *Tau is essential to beta-amyloid-induced neurotoxicity*. PNAS **99(9)**, 6364-9, Gotz *et al.* (2001). *Formation of neurofibrillary tangles in P301L tau transgenic mice induced by Abeta 42 fibrils*. Science **293(5534)**, 1491-5, Lewis J *et al.* (2001). *Enhanced neurofibrillary degeneration in transgenic mice expressing mutant tau and APP*. Science **293(5534)**, 1487-91.

[18] Reviewed in Silverman DHS *et al.* (2008). *Positron emission tomography scans obtained for the evaluation of cognitive dysfunction*. Seminars in Nuclear Medicine **38(4)**, 251-261.

[19] Hye *et al.* (2006). *Proteome-based plasma biomarkers for Alzheimer's disease*. Brain **129(Pt 11)**, 3042-50, Jellinger *et al.* (2008). *Biomarkers for early diagnosis of Alzheimer disease: 'ALZheimer ASsociated gene' - a new blood biomarker?* Journal of Cellular and Molecular Medicine (Epub ahead of print), Zhang R *et al.* (2004). *Mining biomarkers in human sera using proteomic tools*. Proteomics **4(1)**, 244-56. Dubois *et al.* (2007*). Research criteria for the diagnosis of Alzheimer's disease: revising the NINCDS-ADRDA criteria*. Lancet Neurology **6**, 734-36.

[20] Hye *et al.* (2006). *Proteome-based plasma biomarkers for Alzheimer's disease*. Brain **129(Pt 11)**, 3042-50, Davidson P & Sjogren M (2006). *Proteome studies of CSF in AD patients*. Mechanisms of Ageing and Development **127(2)**, 133-7, Puchades M *et al.* (2003). *Proteomic studies of potential cerebrospinal fluid protein markers for Alzheimer's disease*. Brain research. Molecular Brain Research **118(1-2)**, 140-6, Abdi F *et al.* (2006). *Detection of biomarkers with a multiplex quantitative proteomic platform in cerebrospinal fluid of patients with neurodegenerative disorders*. Journal of Alzheimer's Disease **9(3)**, 293-348.

[21] Silverman DHS *et al.* (2001). *Positron emission tomography in evaluation of dementia: regional brain metabolism and long-term outcome*. JAMA **286(17)**, 2120-2127, Silverman DHS *et al.* (2008). *Positron emission tomography scans obtained for the evaluation of cognitive dysfunction*. Seminars in Nuclear Medicine **38**, 251-61.

data are emerging from PET studies regarding conversion of MCI to AD. Further illustration of how PET-based measurements change in healthy ageing individuals and how to distinguish between cognitive impairment in patients with depression or thyroid disease will also be important.[22]

Clinical research

16. Developments on the horizon in clinical research may arise from current efforts to find effective anti-inflammatory agents, reduce the production of Aβ, reduce the phosphorylation of the abnormal tau protein, and to target the tau and α-synuclein proteins, which are most closely associated with neuronal loss.[23] Broad clinical research aims include large clinical studies to: assess the effectiveness of diagnostic tests; trial drugs currently prescribed for other diseases; and study methods of primary care. In this regard, initiatives that encourage collaboration between basic researchers, clinical scientists, health professionals and pathologists will provide important novel perspectives for dementia research.

17. Further studies are focused on improving the clinical effectiveness of cognition-enhancing drugs, including cholinesterase inhibitors such as donepezil and rivastigmine, which are currently licensed for the treatment of dementia. Reviews of clinical trials of these drugs have shown significant, but somewhat limited, efficacy in ameliorating cognitive symptoms of AD and preliminary findings for treatment of Lewy Body dementia are promising. However, the increasing availability and possible 'off-label' use of such cognition enhancers by healthy people has ethical implications. This field is explored further in the recent Academy of Medical Sciences report 'Brain science, addiction and drugs'.[24]

18. Ongoing investment in clinical trials capacity and existing networks will be required to facilitate large, multi-site population studies. The '*Dementias and Neurodegenerative Diseases Research Network'* (DeNDRoN)[25] provides one such vehicle for this. DeNDRoN aims to develop NHS infrastructure for clinical research by investing in regional research networks and multi-centre trials through clinical studies groups (CSGs). Through UKCRC and UKCRN, DeNDRoN also works with the healthcare industry to facilitate commercial trials.

Research funding

19. The Academy emphasises that funding should always be allocated on the basis of academic excellence. As the incidence and socio-economic burden of dementia grows in coming years, investment may need to be raised to facilitate further research. At present, the Department of Health allocates 3% of its research and development budget to dementia research[26] and dementia-related studies account for far fewer publications than those related to cancer or cardiovascular disease. It will be necessary to ensure that all priority areas are adequately supported and that the broad spectrum of research necessary for scientific advancement is maintained.

---

[22] Reviewed in Silverman DHS *et al.* (2008). *Positron Emission Tomography Scans Obtained for the Evaluation of Cognitive Dysfunction*. Seminars in Nuclear Medicine **38**, 251-61.
[23] Sorrentino G and Bonavita V (2007). *Neurodegeneration and Alzheimer's disease: the lesson from tauopathies*. Neurological Sciences **28**, 63-71, Saha AR *et al.* (2000). *Induction of neuronal death by alpha-synuclein*. European Journal of Neuroscience **12(8)**, 3073-7.
[24] Academy of Medical Sciences (2008). *Brain Science, Addiction and Drugs*. http://www.acmedsci.ac.uk/p47prid47.html
[25] Dementias and Neurodegenerative Diseases Research Network. http://www.dendron.org.uk/
[26] Department of Health (2008). *Transforming the quality of dementia care. Consultation on a National Dementia Strategy*. http://www.dh.gov.uk/en/Consultations/Liveconsultations/DH_085570

20. Research allocations should support a range of basic, epidemiological, clinical and health system research studies. In this way, basic medical research aimed at the generation of candidates and leads for clinical research takes place alongside translational research, health services research, cohort and community studies and clinical trials to assess the impact of modifying risk factors and use of novel therapeutics. A balanced programme of research will have the greatest chance of impacting on the health, well being, and quality of life of patients and carers, whilst reducing the growing economic and social costs.

21. The importance of allocating funding to attracting new researchers to the field must be emphasised, since there is a shortage of international leaders in dementia research within the UK, despite the UK's strength in the broader neuroscience field. A fellowship programme aimed at attracting the best neuroscientists and researchers into dementia research could redress this imbalance. Initiatives could also be developed in priority areas of research, with an emphasis on new approaches, new collaborations and new disciplines.

22. Additional efforts to encourage closer interactions between basic scientists, neuropathologists and clinicians would be welcomed. In particular, greater collaboration between researchers in the field would promote the sharing of data gathered through large patient cohort studies. Such collaboration could generate datasets potentially covering thousands of patients that facilitate robust epidemiological analysis and deliver crucial results.

Benefits and risks of early diagnosis

23. It is broadly accepted that early diagnosis is beneficial for the patient and their families, allowing them to plan for the future when the patient is able to give reasoned input. Early diagnosis can explain symptoms that were causing distress to an individual, enable practical help to be mobilised and facilitate the use of disease-modifying interventions as early as possible (or at a time when interventions might be most effective). Many patients will be able to prepare advance directives and others will be able to decide whether to participate in a clinical trial. The inclusion of early diagnosis and intervention as one of three focus areas in the government's forthcoming National Dementia Strategy is most welcome.[27]

24. Clinical guidelines issued by the National Institute for Health and Clinical Excellence (NICE) do not support the use of pharmaceutical treatments in the early stages of dementia caused by AD.[28] Donezepil, Rivastigmine and Galantamine are recommended only for patients where Mini Mental State Examination (MMSE) scores lie between 10-20 points.[29] Thus, the *health* benefits of early diagnosis may be somewhat limited until national clinical guidelines support the use of therapeutics in the early stages of dementia. The accuracy of early diagnosis may be affected by the continuum of cognitive change between MCI and dementia, and the timing of switch between conditions is not always uniform between patients.[30] Thus advances

[27] Department of Health (2008). *Transforming the quality of dementia care. Consultation on a national dementia strategy*. http://www.dh.gov.uk/en/Consultations/Liveconsultations/DH_085570
[28] NICE (2007). *Technology Appraisal. Donepezil, galantamine*. http://www.nice.org.uk/guidance/TA111#documents
[29] *Ibid.*
[30] Whitehouse P, Frisoni GB & Post S (2004). *Breaking the diagnosis of dementia.* Lancet Neurology **3(2)**, 124-128.

in diagnostic methodology are needed to enable early diagnosis and treatment to bring added clinical benefits to patients.

25. Recent studies have highlighted that just 30-50% of people with dementia received a formal diagnosis,[31] and that around two thirds of General Practitioners (GPs) in the UK felt that they did not have enough training to diagnose dementia.[32] Similar findings have been reported in Europe, the US and Australasia. In many cases, concerns that people with dementia might experience increased anxiety and/or depression, hypervigilence, restricted abilities, isolation and the effects of stigma, prevent GPs from disclosing a diagnosis. Yet recent data indicate that the incidence of depression does not increase following a diagnosis of dementia and in some cases receiving a diagnosis may actually provide some relief.[33]

26. There is a strong case for initiating training programmes for primary care practitioners to improve rates of diagnosis and enable GPs to tailor the diagnosis to the individual (taking into account the awareness level and experiences of the individual with dementia). In combination with early diagnosis, it is important that adequate support services are readily available for patients (and carers/families if necessary) following a diagnosis and that we have an improved understanding of how best such services can provide appropriate support for patients, their carers and their families.

Research involving individuals that lack capacity

27. The Mental Capacity Act 2005 provides an important statutory framework to clarify and enshrine in law requirements relating to the participation in research of adults lacking the capacity to consent.

28. Studies involving individuals with dementia who lack capacity are essential to advance our understanding of the causes and molecular mechanisms of dementia, and the effectiveness of potential treatments. Strict safeguards are thus required to protect vulnerable individuals. Whilst it has been argued that studies should only proceed where there is a clear benefit to the study participants, such research provides opportunities to transform the lives of thousands of dementia patients in the future through improved treatments, care and quality of life.

29. Research studies including individuals with incapacity have been crucial for advancing therapy for conditions such as phenylketonuria (PKU), head injury and stroke.[34] For instance, the clinical outcome of PKU has been significantly altered following studies of adults and children with severe learning disability, whilst the 'CRASH' trial, led to improvements in treatment and likely outcome of patients suffering from head injuries.[35]

30. When involving people with dementia in research studies, the wishes expressed when a person has capacity should remain the guiding ones when capacity has been lost. Patients expect certainty when they make advance

---

[31] King's Fund (2008). *Paying the price. The cost of mental health care in England to 2026*. http://www.kingsfund.org.uk/publications/kings_fund_publications/paying_the_price.html
[32] National Audit Office (2007). *Improving services and support for people with dementia*. http://www.kingsfund.org.uk/publications/kings_fund_publications/paying_the_price.html
[33] Carpenter BD *et al.* (2008). *Reaction to a dementia diagnosis in individuals with Alzheimer's disease and mild cognitive impairment*. Journal of the American Geriatrics Society **56(3)**, 405-12.
[34] Academy of Medical Sciences, Medical Research Council, Royal College of Physicians, Wellcome Trust (2005). *Briefing on the research provisions of the mental capacity bill, pending second reading in the House of Lords*. http://www.acmedsci.ac.uk/p100puid71.html
[35] *Ibid.*

decisions about the disposition of their property after death, thus it is reasonable to expect that where a patient has given careful thought to their wishes when they have capacity, advance decisions about their healthcare should be respected.

31. Similarly, in cases where individuals have given authority to a welfare attorney, their decision should prevail over that of the healthcare provider, since the individual has entrusted them to act on their behalf. Once Advance Directives have been made, they should also be respected, since the importance of such Directives is dependent upon the fact that they reflect views of an individual with capacity, even if that person is no longer able to express them.

32. Ideally, research studies would be performed on individuals in the early stages of dementia who still have capacity and have given advance consent to future participation. Where subsequent research is planned when capacity has been lost, the consent of the partner, nearest relative, or appropriate representative should be obtained. For those who lack capacity and have not given advanced consent, the research should be discussed with the participant and a close family member or partner in order to obtain the assent of the former and consent of the latter. If there are no such appropriate representatives, research should not usually proceed. It may be necessary for consideration to be given to who might be most appropriate to give consent by proxy, whether next-of-kin, partner or carer, since circumstances may vary between cases.

33. Notwithstanding the importance of protecting the best interests of individuals with dementia, there may be a need to consider safeguards for researchers in this context. Although researchers take informed consent from patients, and from next-of-kin (or an alternative appropriate person), it is not always clear how this absolves researchers from legal liability.

Ethical review

34. The Academy cautions against the complexity of ethical review, which may, in principle, discourage researchers from undertaking studies in the field. In light of the extent of paperwork required and the need to monitor progress with Primary Care Trust research and development offices, we consider that a more manageable system could be encouraged.

35. We also highlight that the ways in which ethical review committees interpret the Mental Capacity Act (2005) may vary and there may be a need for some monitoring to ensure that there are no inconsistencies between regions.

36. One further ethical consideration arises from our increased knowledge regarding risk genes for dementia, combined with growing access to full genetic profiles and proposed susceptibilities. Improved regulations of genetic tests may be required. The recent move by the Department of Public Health in California requiring start-up companies that provide genetic tests to demonstrate that they meet quality and reliability standards provides a good example of this.[36] This issue is considered in the Academy's recent submission to the House of Lords Science and Technology Committee Inquiry into Genomic Medicine.[37]

[36] New Scientist (2008). *Gene-Scan Crackdown*. New Scientist **2661**, 7
[37] Academy of Medical Sciences (2008). *Response to the House of Lords Science & Technology Committee inquiry into genomic medicine*. http://www.acmedsci.ac.uk/p100.html

This response was prepared by Dr GJ MacArthur and was reviewed by the Academy's Officers. We are grateful to the following Fellows for their contributions: Professor John Aggleton FMedSci, Professor Brian Anderton FMedSci, Professor Simon Lovestone FMedSci, Professor Ian McKeith FMedSci, Professor Sheila McLean FMedSci, Professor A. David Smith FMedSci and Professor Robert Stout FMedSci.

The Academy of Medical Sciences
The Academy of Medical Sciences promotes advances in medical science and campaigns to ensure these are converted into healthcare benefits for society. Our Fellows are the UK's leading medical scientists from hospitals and general practice, academia, industry and the public service.

The Academy seeks to play a pivotal role in determining the future of medical science in the UK, and the benefits that society will enjoy in years to come. We champion the UK's strengths in medical science, promote careers and capacity building, encourage the implementation of new ideas and solutions – often through novel partnerships – and help to remove barriers to progress.

Academy of Medical Sciences
10 Carlton House Terrace
London, SW1Y 5AH
Tel: +44(0)20 7969 5288
Fax: +44(0)20 7969 5298

E-mail: info@acmedsci.ac.uk
Web: www.acmedsci.ac.uk
Registered Charity No. 1070618
Registered Company No. 35202