Dear Friends and Colleagues,

we are delighted to welcome you to the 2nd Annual meeting of The Lupus Academy, which we hope will be one of the most stimulating and rewarding meetings you will attend this year.

This meeting, which has full Continuing Medical Education (CME) accreditation, aims to provide cutting edge insights into advances in global research and clinical practice in Lupus and allied diseases. Delegate feedback from our inaugural meeting in Barcelona (2012) has guided us in our selection of topics and speakers to ensure that a top-class educational programme is provided.

The scientific component of this programme, developed by our Steering Committee of six international experts in Lupus, is designed to create a highly interactive forum through which we can develop a logical approach to the management of Lupus across the globe. It will give you the opportunity to meet world-leading clinicians and scientists and, through the sharing of clinical experience, develop your knowledge in this high-profile therapeutic area.

We sincerely hope that the meeting will provide you with new ideas and enhanced enthusiasm for collaborative research and discussion with your colleagues who care for patients with lupus.

We look forward to meeting and talking with you here in Buenos Aires.

With kind regards,

The Lupus Academy Steering Committee

Professor Munther A. Khamashta  
Meeting Course Director, Co-Chairman

Professor David A. Isenberg  
Programme Director, Co-Chairman

Professor Ricard Cervera; Professor Roger A. Levy; Professor Sandra V. Navarra; Professor Ronald F. van Vollenhoven

The ‘European CME Forum’ is accredited by the European Accreditation Council for Continuing Medical Education (EACCME) to provide the following CME activity for medical specialists: ‘2nd Annual Meeting of the Lupus Academy’. It is designated for a maximum of 8 hours of European external CME credits. Each medical specialist should claim only those hours of credit that he/she actually spent in the educational activity. Go to www.lupus-academy.org for more information.

Mission Statement

The Lupus Academy is a long-term initiative committed to improving patient outcomes in SLE and allied diseases. By providing a highly interactive educational forum, the Lupus Academy is dedicated to sharing best clinical practice through the dissemination and discussion of cutting edge scientific and clinical research.
### Programme

#### Wednesday 17 April

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<thead>
<tr>
<th>Time</th>
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<tbody>
<tr>
<td>18:15</td>
<td>Opening Address</td>
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<tr>
<td></td>
<td>Munther A. Khamashita (UK), David A. Isenberg (UK) &amp; Bernardo A. Pons-Estel (Argentina)</td>
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#### Keynote Lectures

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<tr>
<th>Time</th>
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<tbody>
<tr>
<td>18:30</td>
<td>The genetics of lupus: where are we now?</td>
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<tr>
<td></td>
<td>Marta E. Alarcón-Riquelme (Spain/USA)</td>
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<tr>
<td>19:15</td>
<td>Decreasing morbidity and mortality and improving outcomes in SLE</td>
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<td>Murray B. Urowitz (Canada)</td>
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<tr>
<td>20:00</td>
<td>Welcome Dinner</td>
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#### Thursday 18 April

**Breakfast**

**Plenary I: Lupus Manifestations**

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<th>Time</th>
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<tr>
<td>08:30</td>
<td>Getting to the heart of the matter: improving cardiovascular outcomes</td>
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<tr>
<td></td>
<td>Ian N. Bruce (UK)</td>
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<tr>
<td>09:10</td>
<td>Clinical manifestations and evaluation of cutaneous lupus</td>
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<td>Victoria P. Werth (USA)</td>
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**Hot Topic Lecture**

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<th>Time</th>
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<tr>
<td>09:50</td>
<td>The burden of fatigue in lupus: from patient perspectives to effective management</td>
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<td>Meenakshi Jolly (USA)</td>
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**Case Study Workshops (AM)**

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<tr>
<td>11:00</td>
<td>Biologics for SLE: the future is here. When and how to use biologic therapies in clinical practice</td>
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<tr>
<td></td>
<td>Ronald F. van Vollenhoven (Sweden) &amp; Marta Mosca (Italy)</td>
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<tr>
<td>11:00</td>
<td>Management and prevention of infections in SLE</td>
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<tr>
<td></td>
<td>Ricard Cervera (Spain) &amp; Sandra V. Navarra (Philippines)</td>
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<tr>
<td>11:00</td>
<td>Lupus, pregnancy and APS: current issues in management</td>
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<td></td>
<td>Munther A. Khamashita (UK) &amp; Roger A. Levy (Brazil)</td>
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<tr>
<td>11:00</td>
<td>Lupus nephritis: update on modern management</td>
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<td></td>
<td>Liz Lightstone (UK) &amp; Paula Alba (Argentina)</td>
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**Lunch**

**State-of-the-art Lecture**

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<tr>
<td>15:10</td>
<td>T-cell signalling in patients with SLE: what every rheumatologist needs to know!</td>
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<td>Liz Jury (UK)</td>
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**Plenary II: New horizons in the basic science and clinical practice of SLE**

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<tr>
<td>15:50</td>
<td>Immunopathogenic mechanisms driving SLE</td>
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<td></td>
<td>Thomas Dörner (Germany)</td>
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<tr>
<td>16:30</td>
<td>Biologics in lupus: clinical practice to clinical trials… and now...</td>
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<tr>
<td></td>
<td>David A. Isenberg (UK) &amp; Joan T. Merrill (USA)</td>
</tr>
<tr>
<td>17:10</td>
<td>Summary</td>
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<td>Eloisa Bonfá (Brazil)</td>
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**Close**

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<td></td>
<td>Munther A. Khamashita (UK) &amp; David A. Isenberg (UK)</td>
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Biographies

Professor Graciela S. Alarcón, MD, MPH
The University of Alabama at Birmingham, Alabama, USA

Graciela S. Alarcón is the Jane Knight Lowe Chair of Medicine in Rheumatology, Emeritus at the University of Alabama at Birmingham, Birmingham, Alabama. Professor Alarcón graduated as an MD from Universidad Peruana Cayetano Heredia in Lima, Peru in 1967, and as Master of Public Health from Johns Hopkins University in 1972. She trained in Rheumatology under Drs. Lawrence Schumman and Alex Townes, in Baltimore, Maryland. Upon her return to Peru she founded the first clinical and teaching Rheumatology Unit at her alma mater. In 1980 she returned to the US and has been in the Faculty at the University of Alabama at Birmingham (UAB) since, now as an Emeritus Professor.

For the past 20 years Professor Alarcón has been involved in the study of lupus in minority populations, establishing the LUMINA cohort. The work from this study has contributed to the understanding of the mechanisms through which anti-nucleolar autoantibodies contribute to lupus disease activity. In 2011, Professor Alarcón received the Evelyn Hess Award from the Lupus Foundation of America (LFA) in recognition of her studies in lupus and is currently a member of its medical scientific advisory board.

Professor Marta E. Alarcón-Riquelme, MD, PhD
Universidad de Granada, Spain and Oklahoma Medical Research Foundation, USA

Marta Alarcón-Riquelme is Head of the Human DNA Variability Area at the Center for Genomics and Oncological Research (GENYO), Granada, Spain and an Associate Member of the Oklahoma Medical Research Foundation (OMRF), USA. Professor Alarcón-Riquelme studied Medicine in Mexico, before moving to Sweden in 1987, where she was awarded her PhD in Immunology from Stockholm University. After her PhD she began forming research collaborations, which allowed her to build banks of samples for genetics studies, families and case-control sets. These efforts, and financing from the European 4th Framework Programme, allowed her to publish the identification of the first gene resulting from genetic linkage analyses of systemic lupus erythematosus (SLE) families, PDCD1, and the very first example of how to analyse a genetic polymorphism with impact on transcription factor binding and gene expression. In several subsequent studies, Professor Alarcón-Riquelme identified BANK1 and developed understanding of the mechanisms through which genetic polymorphisms modulate gene function for AIFS, CD226 and fine mappings of various genes published in top journals. Following these research activities, Professor Alarcón-Riquelme was invited to become a member of the International Lupus Genetics Consortium.

Professor Paula Alba, MD, PhD
State University of Córdoba, Argentina

Paula Alba is a Consultant Rheumatologist, Assistant Professor and Clinical Researcher in Internal Medicine and Rheumatology at the Córdoba Hospital, State University of Córdoba. She graduated from Medical School at the State University Córdoba in 1992 and finished her Internal Medicine and Rheumatology specialties at the Córdoba Hospital. She subsequently completed a fellowship programme in the Lupus Research Unit at St Thomas’ Hospital London in 2001, and she received her PhD in Medicine and Surgery from the Faculty of Medicine at the State University of Córdoba in 2008.

Disclosures

Professor Graciela S. Alarcón: None.
Professor Marta E. Alarcón-Riquelme: None.
Professor Paula Alba: None.
**Biographies**

**Professor Mary-Carmen Amigo**, MD, FACP

Mary-Carmen Amigo is Professor of Rheumatology and Head of the Rheumatology Division at ABC Medical Centre, Mexico City. She qualified in medicine (Summa Cum Laude) from Universidad Nacional Autónoma de México (UNAM) Medical School in 1974, after which she undertook her residency at Hospital Español, Mexico City before starting her fellowship in rheumatology at the Instituto Nacional de Cardiología Ignacio Chávez in 1980. Professor Amigo served as Associate Professor of Rheumatology at UNAM (1986-2006) and was a member of the national system of researchers in Mexico between 1994 and 2006. She became a Master of the Mexican College of Rheumatology in 2010 and has held academic affiliations with St. Thomas’ Hospital London in 1997, 2000 and 2012. Professor Amigo is a member of numerous professional societies and has held several posts on the Mexican Board of Rheumatology and at the Mexican College of Rheumatology. She is on the Editorial Boards for the journals Lupus, Reumatología Clínica and Current Opinion in Rheumatology. Professor Amigo’s main clinical research interests focus on antiphospholipid syndrome and systemic lupus erythematosus, in particular cardiovascular, pulmonary, renal and pregnancy complications. To date, she has published 27 book chapters and 54 scientific papers.

**Professor Zahir Amoura**, MD

Groupe Hospitalier Pitié-Salpêtrière, Paris, France

Zahir Amoura is Professor of Internal Medicine at the French National Reference Centre for Systemic Lupus Erythematosus at Pitié-Salpêtrière Hospital, a role that he has held since 2003. In 2005, Professor Amoura became Head of the Department of Internal Medicine in the same institution. He completed his Paris Hospitals Medical Internship in 1981, obtained a Masters degree in Immunopharmacology in 1989, and was subsequently awarded his MD (Silver Medal) in 1993 and his qualifying certification in Internal Medicine in 1994. Professor Amoura joined the Department of Internal Medicine at Pitié-Salpêtrière Hospital in 1995 as a Senior Lecturer and Senior Practitioner. In the last 10 years, Professor Amoura has published over 294 peer-reviewed papers, of which 70 focused on the immunopathological features of lupus.

**Professor Eloisa Bonfá**, MD, PhD

University of São Paulo Medical School, Brazil

Professor Eloisa Bonfá is the Physician-in-Chief of the Rheumatology Division of the University of São Paulo, the largest tertiary referral centre for autoimmune rheumatic disorders of Latin America. She was also elected Clinical Director of the same Hospital for 4 years. Graduating from the University of São Paulo Medical School, Professor Bonfá undertook specialist training in rheumatology in the same University, followed by a 4-year Rheumatology Research Fellowship at the Hospital for Special Surgery, New York, under the supervision of Professor Keith Elkon. Professor Bonfá’s main clinical and research interests are systemic lupus erythematosus and autoimmunity with contributions in the field of autoantibodies, the association of anti-ribosomal P antibodies with psychiatric manifestation of lupus and the first in vitro demonstration of the antihypermagogenic potential of purified Ro/SSA antibodies. More recently, she has described the association of anti-ribosomal P antibodies and lupus membranous nephritis and published several papers on autoimmune disorders and influenza vaccine. She has published more than 150 original papers and several book chapters.

**Professor Ian N. Bruce**, MD, FRCP

University of Manchester, UK

Ian Bruce is Professor of Rheumatology at the Arthritis Research UK Epidemiology Unit, School of Translational Medicine, University of Manchester. He is co-Chair of the Inflammatory Musculoskeletal Conditions Division, and lead for the Cardiovascular Research Group. He qualified in medicine from Queen’s University Belfast in 1988 and gained his MRCP in 1991. He trained in medicine and rheumatology in Northern Ireland and completed his MD thesis on the pathogenesis of systemic vasculitis in 1995. He was the Geoff Carr Lupus Fellow at the University of Toronto, before moving to Manchester in 1998 as an NHS consultant, transferring to the University in 2003. Professor Bruce is on the Editorial Board of the Journal Rheumatology. He is a member of the Systemic Lupus International Collaborating Clinics (SLICC) and the British Isles Lupus Assessment Group, and participates in a number of national and international multicentre studies that are seeking to refine our understanding of systemic lupus erythematosus (SLE). He leads the Cardiovascular Group within the Arthritis Research UK Epidemiology Unit and is joint Principal Investigator on the Norfolk Arthritis Registry (NORAP), Cardiovascular Substudy. He is involved in the Welcome Trust Case Control Consortium and the British Society for Rheumatology Biologics Registry Control Consortium. Professor Bruce’s major research focus is on the association between inflammatory rheumatic diseases and premature atherosclerosis/ coronary heart disease. In particular, his focus is on SLE and rheumatoid arthritis. He has published 100 papers in his field.
Biographies

Professor Luis J. Catoggio, MD, PhD
Hospital Italiano de Buenos Aires, Argentina

Luis J. Catoggio is an Associate Professor of Medicine at the Instituto Escuela de Medicina, Hospital Italiano de Buenos Aires and Associate Director of the Rheumatology Training Program of the School of Medicine at the University of Buenos Aires. He is also a Research Coordinator of the Department of Education and Research of the Hospital Italiano, and has been a member of the Comité de Ética de Protocolos de Investigación at Hospital Italiano since 1984. He was Chief of the Rheumatology Section at Hospital Italiano between 1983 and 2010 and remains an active member of the section.

After completing his Residency in Internal Medicine and being Chief Resident, Professor Catoggio trained in Rheumatology as Clinical Research Fellow and Honorary Registrar at the Royal National Hospital for Rheumatic Diseases in Bath, UK between 1980 and 1982. Upon his return to Buenos Aires in 1983, he became Chief of Rheumatology at the Hospital Italiano, a department that currently has 10 members of staff, 10 fellows in training and currently sees over 1500 outpatients each month.

Disclosures
None.

Professor Ricard Cervera, MD, PhD, FRCP
Hospital Clinic, Barcelona, Catalonia, Spain

Ricard Cervera is Head of the Department of Autoimmune Diseases (which he co-founded in 1995), at Hospital Clinic, Catalonia, Barcelona. He is also Director of the Research Group on Systemic Autoimmune Diseases at the Institut d’Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS). He qualified in Medicine in 1983 from the University of Barcelona and received his PhD in 1988 for his thesis on anticardiolipin antibodies. He spent two years at the Lupus Research Unit at The Rayne Institute, St Thomas’ Hospital, London.

Professor Cervera is on the Editorial Boards of 20 medical journals. He is coordinator of the European Forum on Antiphospholipid Antibodies and of the European Working Party on Systemic Lupus Erythematosus, Chairman of the Medical Advisory Board of the Catalan Association of Lupus Patients, and Medical Advisor to Lupus Europe. He chaired the 6th and 8th International Congresses on Autoimmunity, the 1st and 2nd Latin-American Congresses on Autoimmunity, the 5th Meeting of the European Forum on Antiphospholipid Antibodies, and the 8th European Lupus Congress.

Professor Cervera’s research interests include clinical and epidemiological aspects of systemic autoimmune diseases, particularly systemic lupus erythematosus and the antiphospholipid syndrome, with special focus on its ‘catastrophic’ variant. He has presented over 300 invited lectures and published more than 800 scientific papers (h factor, 52), including original articles at the New England Journal of Medicine, The Lancet, Annals of Rheumatic Diseases, Arthritis and Rheumatism, American Journal of Medicine, and Medicine (Baltimore). He is co-editor of 20 books, including ‘The antiphospholipid syndrome’, ‘Vascular Manifestations of Systemic Autoimmune Diseases’, and ‘Diagnostic Criteria in Autoimmune Diseases’.

Disclosures
Consultant/Advisor: GlaxoSmithKline, Immunomedics, UCB
Professor Cervera is a member of the Lupus Academy Steering Committee and has been involved in the planning and development of the meeting programme and materials.

Professor Andrea Doria, MD
University of Padova, Italy

Andrea Doria is Associate Professor of Rheumatology, Director of the Academic Postgraduate School of Rheumatology and Head of the Unit of Connective Tissue Disease and Rare Rheumatic Diseases, University of Padova.

Professor Doria received his medical degree and qualification in rheumatology from the University of Padova. He was Council member of the Italian College of Rheumatology (CRO) (1999–2005) and a Council member of the Italian Society of Rheumatology (SIR) (2007–2013). He is also a member of the American College of Rheumatology (ACR).

Professor Doria has organised over 10 international congresses on autoimmunity and was involved as “expert” in the European League Against Rheumatism (EULAR) Standing Committee for the development of clinical and therapeutic recommendations: (1) EULAR recommendations for the management of systemic lupus erythematosus (SLE)—Assessment of the SLE patient (2008–2009); (2) EULAR recommendations for the management of SLE Part II—Neuropsychiatric disease (2008–2009); (3) Joint EULAR and European Renal Association-European Dialysis and Transplant Association (EULAR/ERA-EDTA) recommendations for the management of adult and paediatric lupus nephritis (2012).

Professor Doria is on the Editorial Boards of several rheumatology and immunology journals, including Lupus, Autoimmunity, Clinical and Experimental Rheumatology, Autoimmunity Reviews, Journal of Autoimmunity, Experimental Biology and Medicine, Rheumatology Reports, Journal Autoimmunity, Highlights and Reumatismo (the official journal of the SIR). He has authored over 200 EU publications on SLE and other connective tissue diseases. These include clinical studies describing new manifestations or subgroups of autoimmune disorders, prognostic risk factors, diagnostic tests and therapeutic interventions as well as immunochemical studies that evaluate autoantibodies, epitopes and complementary autoantibodies, epitopes and complementary.

Professor Doria has a long-standing experience in clinical management of connective tissue disease patients. The Unit in which he works is a 3rd referral rheumatology centre, within Italy, for the diagnosis and management of patients affected with systemic connective diseases. In addition, he has expertise in the management and follow-up of pregnant patients with systemic rheumatic diseases. Professor Doria has also trained over 30 students in rheumatology.

Disclosures
Consultant/Advisor: GlaxoSmithKline
Speakers’ Bureau: GlaxoSmithKline

Professor Luis J. Catoggio, MD, PhD
Hospital Italiano de Buenos Aires, Argentina

Professor Andrea Doria, MD
University of Padova, Italy

Disclosures
None.

Professor Cervera is a member of the Lupus Academy Steering Committee and has been involved in the planning and development of the meeting programme and materials.
Biographies

Professor Thomas Dörner, MD
Charite University Hospitals, Berlin, Germany

Thomas Dörner is a board certified Rheumatologist and Professor of Rheumatology and Hemostaseology at Charite University Hospitals, Berlin and group leader at the German Research Centre of Rheumatology, Berlin. He qualified in medicine in 1990 at Charite University Hospitals, Berlin and received his board certification in internal medicine in 1996 before undertaking a post-doctoral fellowship at the University of Texas, Southwestern Medical Centre at Dallas, where he researched delineating molecular aspects of the B-cell receptor gene usage.

Professor Dörner has received a number of international and national awards, including the Senior Scholar Award of the American Society of Rheumatology, the H Schultze Award of the German League against Rheumatism and the Schoen Award of the German Society of Rheumatology.

Professor Dörner has served as a member of Editorial Boards of leading journals in rheumatology, including Arthritis and Rheumatism, Arthritis Research and Therapy, Annals of the Rheumatic Diseases, Global Arthritis Research Network (GARN), Current Reviews in Rheumatology, Brazilian Journal of Rheumatology, European Journal of Immunology and Rheumatology Reviews.

Professor Dörner has led various clinical trials of rheumatic diseases, including systemic lupus erythematosus, Sjögren’s syndrome, rheumatoid arthritis and seronegative spondylarthropathies. His research interests focus on the characterisation of disturbances of humoral autoimmunity and abnormalities of B cell differentiation in autoimmune diseases (lupus, neonatal lupus syndromes, Sjögren’s syndrome, immune thrombocytopeniai) and exploring innovative therapeutic approaches with particular focus on B-cell directed therapy as well as improving diagnostic tools in autoimmune diseases.

Professor David A. Isenberg, MD, FRCP, FAMS
University College London, UK

David Isenberg is the Arthritis Research UK Diamond Jubilee Professor of Rheumatology at University College London (UCL). He graduated from St Bartholomew’s Hospital, London in 1973, and trained in general medicine, rheumatology, neurology, psychiatry, and gastroenterology, becoming a Research Fellow at UCL. The Middlesex Hospital in 1979. He was awarded his MD in 1984, based on his studies of myositis. During a year of research at Tufts University, Boston, he became interested in autoantibody structure, function and origin. He was appointed Consultant Rheumatologist in 1984, Professor in 1991, and became the arC Diamond Jubilee Chair of Rheumatology at UCL in 1996. He has Fellowships of both the Royal College of Physicians and the Academy of Medical Sciences.

Professor Isenberg is on the Editorial Boards of five journals, including the Journal of Rheumatology. He is Chair of the British Isles Assessment Group and was Chair of the Systemic Lupus International Collaborating Clinics group (SLICC). During the past 20 years, Professor Isenberg has undertaken many roles at Arthritis Research UK, for whom he currently chairs the autoimmune rheumatic disease clinical trials sub-committee. He is Past President of the British Society for Rheumatology (2004–2006) and has chaired the Society’s Biologics Register Committee (2006–2011). Professor Isenberg is the 2010 recipient of the Evelyn Hess Prize from the Lupus Foundation of America for his contribution to lupus research and treatment.

Professor Isenberg’s principal clinical interests are the development of disease activity and damage assessment tools in patients with lupus. His specialist interest is autoimmune rheumatic diseases, notably systemic lupus erythematosus, Sjögren’s syndrome, myositis, and antiphospholipid antibody syndrome.

Professor Meenakshi Jolly, MD
Rush University Medical Centre, Chicago, USA

Meenakshi Jolly is the Director of the Rush Lupus Clinic and Attending Physician at Rush University Medical Centre, Chicago. She is also an Associate Professor in the Department of Internal Medicine and Associate Director of the Rheumatology Fellowship Program at Rush Medical College. Professor Jolly received her medical degree from Maulana Azad Medical College, Delhi, India. She completed her residency in internal medicine at the Cleveland Clinic Foundation, her fellowship in rheumatology at the University of Chicago and has also received a master’s degree in health studies from the University of Chicago. She served as Section Chief of Rheumatology at Christ Medical Centre, University of Illinois prior to joining the Rush Medical Centre.

Professor Jolly serves on the medical Advisory Board and Board of Directors of the Lupus Foundation of America, Illinois Chapter, and on the Physician Advisory Council of the Lupus Research Institute. She is a member of the American College of Rheumatology (ACR), the Arthritis Foundation, the International Society of Quality of Life, Society of Behavioral Medicine and the Central Society of Clinical Research.

She is engaged in investigator-initiated, collaborative lupus research and clinical trials. Her research interests include psychosocial aspects (body image and sexual health) of lupus and patient-reported health outcomes (including quality of life measures). Her work has been published in journals such as Annals of Rheumatic Disease, Arthritis and Rheumatism, Journal of Rheumatology and Lupus.

Dr Elizabeth Jury
University College London, UK

Elizabeth Jury is an Arthritis Research UK Career Progression Fellow at the Centre for Rheumatology Research, University College London. Dr Jury started her career at St Bartholomew’s Hospital, London developing the immunopathology service specialising in the diagnosis of autoimmunity, immunodeficiency and immunophenotyping. She moved to a more research-focused career joining Professor David Isenberg’s team in 2000 and now leads her own research group within the Centre for Rheumatology Research at University College London. Dr Jury’s work has developed new areas of research into signalling abnormalities in T- and B-cells from patients with systemic lupus erythematosus (SLE) making a significant contribution towards understanding the nature of these abnormalities and how they relate to disease pathogenesis. The main focus of her research is to understand the role of plasma membrane, cellular and serum lipids on immune cell activation. Dr Jury was one of the first to identify defects in plasma membrane lipids in T-cells from patients with SLE.

She revealed that these defects contributed to T-cell hyperactivity and has shown that by manipulating cholesterol biosynthesis using atorvastatin some of the signalling and functional defects seen in T-cells from patients can be reversed.

More recently Dr Jury has used a new approach to visualise the organisation (order) of plasma membrane lipids and found that T-cells have specific patterns of membrane lipid-order that correlate with their function. Importantly, she has shown that manipulation of membrane order can control T-cell function, adding weight to her hypothesis that therapeutic targeting of membrane lipids can correct aberrant immune cell activation. Dr Jury has also shown that invariant Natural Killer T-cells (INKT), a unique subset of T-cells that recognise lipid antigen, are deficient in SLE patients. Interestingly INKT cell defects were reversed in patients responding to rituximab therapy.
Biographies

Professor Munther A. Khamashta, MD, PhD, FRCP
St Thomas’ Hospital, London, UK

Munther Khamashta is Professor/Consultant Physician and Director of The Graham Hughes Lupus Research Laboratory at St Thomas’ Hospital, London, and runs a large lupus pregnancy clinic. He studied medicine in Barcelona and internal medicine in Madrid, Spain, where he developed an interest in connective tissue diseases and received his PhD. He was awarded the MRCP in 1999 and FRCP in 2002. He joined the Lupus Unit in London over 25 years ago and has been instrumental in developing it into an internationally recognized tertiary centre receiving referrals from all over the UK.

Professor Khamashta has served on the Editorial Boards of many journals, including Clinical and Experimental Rheumatology, Lupus, and Current Rheumatology Reviews. He is a member of several professional societies, including the International Society of Internal Medicine, the American College of Rheumatology (ACR), and the Spanish Society of Rheumatology. He is a member of the Steering Committee of the International Board on the Study of Antiphospholipid Antibodies and of the Steering Committee of the International Advisory Board for Systemic Lupus Erythematosus. He has received several international awards for his work in lupus, including The Eularian League Against Rheumatism (EULAR) and International League Against Rheumatism (ILAR) prizes.

Professor Khamashta has a strong research interest in lupus and connective tissue diseases, with a special interest in pregnancy and antiphospholipid syndrome. He has published extensively in lupus, Hughes’ syndrome and related areas, with more than 600 original papers.

Professor Roger A. Levy, MD, PhD
The State University of Rio de Janeiro, Brazil

Roger Levy is Adjunct Professor of Rheumatology at The State University of Rio de Janeiro, Brazil. Graduating from medical school at the University of Rio de Janeiro in 1986, he subsequently completed a fellowship programme at the Hospital for Special Surgery, Cornell Medical College, New York in 1989 and received his PhD in Biological Sciences from the Biophysics Institute – Immunology, at the Federal University of Rio de Janeiro. Professor Levy holds positions on a number of Editorial Boards including the Journal of Clinical Rheumatology, Lupus, Seminars of Arthritis and Rheumatism, Rheumatology, Autoimmunity reviews, and The Brazilian Journal of Rheumatology (of which he is a former Editor). He was the Scientific Director of the XV Brazilian Congress of Rheumatology and chaired the 2nd Latin American Congress of Autoimmunity (Rio de Janeiro, 2006). Professor Levy was President of the Rio de Janeiro Rheumatology Society (2007–2008). He is currently coordinating the vasculopathies committee of the Brazilian Society of Rheumatology and will chair the XV International Antiphospholipid Congress (AFLA) and the Latin American Congress of Autoimmunity (LACA) in Rio de Janeiro in September 2013.

Professor Levy’s research is based around the clinical and immunologic aspects of systemic lupus erythematosus, antiphospholipid syndrome, Sjögren’s syndrome, and pregnancy in rheumatic patients. He has published over 80 articles in medical journals, over 100 abstracts, and over 20 book chapters, and has lectured in many countries.

Professor Loreto Massardo, MD
Pontificia Universidad Católica de Chile, Santiago, Chile

Loreto Massardo is a Full Professor and Attending Rheumatologist at the Pontificia Universidad Católica de Chile in Santiago. She first graduated from Pontificia Universidad Católica de Chile with a medical degree in 1979 and with a post-graduate degree in Clinical Immunology and Rheumatology in 1982. Following graduation, Professor Massardo worked as a General Practitioner at Hospital Regional de Talca, Chile, and as a Rheumatologist at the Hospital Dr Sotero, Santiago, Chile. She returned to Pontificia Universidad Católica de Chile in 1985, and accepted her current post in 2010. During her career, Professor Massardo has undergone training in rheumatology at the University of Bristol, UK and has also held the position of Visiting Clinician at the Mayo Clinic, Minnesota, USA.

Professor Massardo has held positions with several societies, including Secretary of the Sociedad Chilena de Reumatologia (1991–1993), Director of the Sociedad Chilena de Reumatologia (1997–1998) and Chairperson of the Rheumatoid Arthritis Study Group of PANLAR (2002–2006). At the 2009 American College of Rheumatology (ACR) meeting in Philadelphia, she moderated the session on Challenges of Rheumatoid Arthritis in Latin America.

Professor Massardo’s research interests include systemic lupus erythematosus and rheumatoid arthritis, particularly in Latin American populations, and Neuropsychiatric lupus. She has presented findings from her research at major international congresses and has more than 45 articles published in peer-review journals, including Arthritis and Rheumatism, Rheumatology and Lupus.

Disclosures
Consultant/Advisor: GlaxoSmithKine, Jansen

Dr Liz Lightstone, PhD, FRCP
Imperial College London, UK

Liz Lightstone is a Reader in Renal Medicine in the Division of Immunology and Inflammation, Department of Medicine, Imperial College London, and an Honorary Consultant Renal Physician in the Imperial College Healthcare NHS Trust Renal and Transplant Centre (ICH-NT/RCT). After an undergraduate degree at Cambridge, she graduated in medicine from the University of London in 1983, and trained in nephrology at the Royal Postgraduate Medical School. She won a Medical Research Council Training Fellowship in 1986, and undertook a PhD in immunology at University College London. This was followed by a Medical Research Council Clinician Scientist Fellowship at the Royal Postgraduate Medical School. She was appointed Senior Lecturer and Honorary Consultant Physician in 1995.

Dr Lightstone has major roles in undergraduate and postgraduate medicine at Imperial College, in particular in her role as Director of the Academic Foundation programme within North West Thames Foundation School. She is a member of the LUPUS UK Peer Review Panel. She was an elected member of the UK Renal Association Executive and remains active in the Renal Association Programme Planning group and Equal Opportunities Committee.

Disclosures
Grant/Research: Roche
Consultant/Advisor: Aspiva Pharmaceuticals, Biogen Idec, GlaxoSmithKine, Roche, Medimmune

Dr Lightstone’s research is now focused on lupus nephritis and renal disease in ethnic communities. Together with colleagues in the ICH-NT/R, she has pioneered the use of steroid-minimising regimens in lupus nephritis. She is chief investigator on the planned international multicentre randomised RITUXILUP trial which has recently been awarded funding by Arthritis Research UK. The trial will address the question as to whether the regimen of two doses of methyl prednisolone plus two doses of rituximab plus mycophenolate mofetil (MMF) but no oral steroids is as good as the standard combination of MMF and steroids in treating lupus nephritis. She is also working on identifying urine biomarkers that better predict the outcome of lupus nephritis. She is joint Principal Investigator on a study, funded by Kidney Research UK, to identify the incidence and progression of chronic kidney disease in the Indian Asian community in West London. Her main clinical interests are in lupus nephritis and the management of women with kidney disease in pregnancy.
Professor Joan T. Merrill, MD
University of Oklahoma, USA

Joan T. Merrill is the Head of the Clinical Pharmacology Research Program at the Oklahoma Medical Research Foundation (OMRF) and OMRF Professor of Medicine and Adjunct Professor of the School of Pharmacy at the University of Oklahoma. She graduated from Medical School at Cornell University, New York in 1985 and is the recipient of the Edmund Dubois Award/lecture from the American College of Rheumatology (ACR), 2000 the Ira Goldstein Award and Lectureship at the New York University Advanced Course in Rheumatology (2006) and the Pemberton Lecture of the Philadelphia Rheumatism Society in 2010.

Professor Merrill is Medical Director of the Lupus Foundation of America (2004–present) and has served on the Medical and Scientific Committee of the National Arthritis Foundation (2000–2003), Research Committee and Clinical Research Subcommittee of the ACR (2002–2004), as Chair of the Antiphospholipid Subcommittee of the ACR (1999–2002), Planning Committee member of the ACR Meeting (1999–2002), ACR Sponsored Programs Committee (1999–2002) and as Vice-Chair of the Medical and Scientific Committee, New York, Chapter Arthritis Foundation (2000–2001).

Professor Merrill has been active in the design and implementation of many interventional clinical trials for lupus with a focus on defining clinical and biologic subgroups to optimize treatment selection and dosing.

Professor Marta Mosca, MD
University of Pisa, Italy

Marta Mosca is Associate Professor in Rheumatology at the Department of Internal Medicine of the University of Pisa, Italy. Professor Mosca graduated from Medical School at the University of Pisa in 1992.

Her research interests are represented by systemic lupus erythematosus (SLE) and undifferentiated connective tissue diseases, with particular interest to clinimetrics. Recently she has worked on the development of “EULAR Recommendations for monitoring SLE patients in clinical practice and observational studies”, of quality measures in SLE and of a core set of measures to be used in clinical practice to standardise patients’ care.

Professor Bernardo A. Pons-Estel, MD
Cardiovascular Institute of Rosario, Rosario, Argentina

Bernardo Pons-Estel is Head of Rheumatology at the Cardiovascular Institute of Rosario, Rosario, Argentina. Professor Pons-Estel obtained his medical degree from the National University of Rosario and received a Rheumatology Scholarship from the Rosario University Medical Centre before completing a Fellowship within the Division of Rheumatology at the Bellevue Hospital, New York University Medical Centre, New York, USA in 1982. During 1983–1984, Professor Pons-Estel completed his MD at the Division of Rheumatology and Immunology, University of Missouri Cancer Research Centre, Columbia, Missouri and Irvington House Institute, University Hospital, New York University Medical Center, New York.

Professor Pons-Estel is the Principal Co-ordinator for the Latin American Group for the Study of Systemic Lupus Erythematosus (GLADEL) and the Genomic Study of Latin-American Patients with Systemic Lupus Erythematosus. He is also President of the Argentine Rheumatology Society for 2011–2013 and is an International Member of the American College of Rheumatology (ACR).

Professor Pons-Estel has authored and co-authored more than 65 papers in international peer-reviewed journals, including Nature Genetics, American Journal of Human Genetics, Lupus, Journal of Clinical Rheumatology, Arthritis and Rheumatism and Genes and Immunity.
Biographies

Professor Murray Urowitz, MD
University of Toronto, Canada

Murray Urowitz received his MD from the University of Toronto in 1963. He completed his postgraduate training in rheumatology at the Johns Hopkins University, Baltimore and at the University of Toronto in 1968. He was a Staff Rheumatologist at the Wellesley Hospital in Toronto (1974–1987) and Physician in Chief (1987–1996). He has been a Senior Staff Rheumatologist at the Toronto Western Hospital and Senior Scientist at the Toronto Western Research Institute since 1995. Professor Urowitz is currently Professor of Medicine at the University of Toronto and Director of the Centre for Prognosis Studies in the Rheumatic Diseases and the University of Toronto and the Lupus Clinic at the Toronto Western Hospital.

Professor Urowitz established the University of Toronto Lupus Clinic and Lupus Databank Research Program in 1970. This extensive longitudinal database is one of the largest such databanks in the world, with over 1600 patients, and has allowed for numerous findings, which have changed the way lupus is diagnosed and managed. His teaching excellence is exemplified by his having won the outstanding clinical teacher award in the medical school for a remarkable eight times and he was the Associate Dean of Postgraduate Medical Education with the University of Toronto during 1995–2005. This life-long commitment to medical education has resulted in his being the recipient of the Royal College of Physicians and Surgeons of Canada 2004 Duncan Graham Award.

Professor Urowitz was a founding member of the Ontario Lupus Association (now Lupus Ontario). He was president of the Lupus Council of the American Rheumatology Association. He was a founding member of the Systemic Lupus International Collaborating Clinics (SLICC) group and currently directs the SLICC Registry for Atherosclerosis. In 1995 he was the recipient of the Distinguished Rheumatologist Award of the Canadian Rheumatology Association and in 2009 he was recipient of the Evelyn V. Hess Award for outstanding contributions to lupus research, awarded by the Lupus Foundation of America. In 2013 he received the Queen’s Jubilee Medal for contributions to Canada.

Professor Urowitz has been an invited speaker around the world, has published over 300 peer-reviewed papers and 40 book chapters, and has supervised the training of over 100 fellows in rheumatology, mainly in systemic lupus erythematosus.

Disclosures
None.

Professor Ronald F. van Vollenhoven, MD, PhD
The Karolinska Institute and Karolinska University Hospital, Sweden

Ronald van Vollenhoven is Professor and Chief of the Unit for Clinical Therapy Research, Inflammatory Diseases (ClinTRD) at the Karolinska Institute and Chief of the Clinical Trials Unit in Rheumatology at Karolinska University Hospital, Sweden. He received his MD and PhD from the University of Leiden, The Netherlands. After graduating in 1984, he pursued immunology research at Cornell Medical College, New York, followed by specialty training in internal medicine at the State University of New York. He did a fellowship in rheumatology at Stanford University, California, and received American Board of Internal Medicine certification in both internal medicine and rheumatology. He was Assistant Professor of Medicine, then Medical Services Chief and Fellowship Director, in the Division of Immunology and Rheumatology at Stanford University. In 1998 he moved to Sweden and became Senior Physician and Chief of the Clinical Trials Unit in the Department of Rheumatology at the Karolinska University Hospital; he was appointed to his current position in 2010.

Professor van Vollenhoven is Editor-in-Chief of European Musculoskeletal Review, a member of several Editorial Boards, including Annals of the Rheumatic Diseases, Chair of the Swedish health economics working group (HERA) and co-founder of the International Registry for Biologics In Systemic lupus erythematosus (IRBIS) and of the NORD-STAR collaboration for Nordic trials in the rheumatic diseases.

Professor van Vollenhoven’s research interests focus on the development and systematic evaluation of biological and immunomodulatory treatments for rheumatic diseases, including clinical efficacy, pharmacology, outcomes, and pharmacoconomics. He has been Principal Investigator in many clinical trials of novel therapies in rheumatic diseases. He has published over 150 original papers, book chapters and reviews, and is editor of the textbook ‘Targeted Treatment of the Rheumatic Diseases’ and associate editor of ‘Dubois’ Lupus Erythematosus’.

Disclosures
Grant/Research: Abbott, Bristol Myers, Squibb, GlaxoSmithKline, Merck Sharp & Dorhne, Pfizer, Roche, UCB.
Consultant/Advisor: Abbott, Bristol Myers, Squibb, GlaxoSmithKline, Merck Sharp & Dorhne, Pfizer, Roche, UCB.

Professor van Vollenhoven is a member of the Lupus Academy Steering Committee and has been involved in the planning and development of the meeting programme and materials.
Biographies

Professor Victoria P. Werth, MD
Philadelphia Veterans Affairs Medical Center, USA

Victoria P. Werth is Chief of the Division of Dermatology at the Philadelphia Veterans Affairs Medical Centre (PVAMC), a position she has held since January 1989. She is also Professor of Dermatology and Medicine at the University of Pennsylvania. After graduation from Johns Hopkins School of Medicine in 1980, she completed an internal medicine residency in 1983 at Northwestern Memorial Hospital and a dermatology residency in 1986 at New York University. She then completed an immunodermatology research fellowship, sponsored by the National Institutes of Health (NIH) and the Dermatology Foundation, at New York University just prior to moving to her current position at PVAMC.

Professor Werth has a laboratory investigative effort devoted to the effects of ultraviolet light on skin and skin cells and has received four consecutive Merit Review grants supporting this research programme. She is also participating in a VA cooperative trial related to prevention of skin cancers that involves a number of VA dermatology divisions across the USA. In addition, she has received numerous NIH and foundation grants that support translational research in autoimmune skin diseases, with a focus on cutaneous lupus. Her research has been funded by the Lupus Research Institute for investigative studies of a polymorphism associated with sub-acute cutaneous lupus, the Alliance for Lupus Research for translational studies related to an investigator-initiated study in cutaneous lupus, and the Lupus Foundation of America for research related to further characterisation of glycosaminoglycans in the skin in cutaneous lupus. Professor Werth led a research effort to develop and validate the cutaneous lupus erythematosus disease area and severity index (CLASS), which is now being used in many international trials. She has an ongoing effort to characterise response to therapy in a cohort of over 250 patients, and has studied the impact of cutaneous lupus and photosensitivity on quality of life in this cohort. She has published over 140 papers in these areas in peer-reviewed journals.

Dr. Werth co-founded the Medical Dermatology Society, an organisation that emphasises the importance of education, patient care, and research related to complex medical dermatologic disease and is a recipient of the lifetime achievement award from that society. She is a founding member of the North American Rheumatologic Dermatology Society, is current president of the group, and has been on the medical advisory board for the Lupus Foundation of America (LFA) for many years. She has worked with the LFA on many of their initiatives related to lupus, with a focus on cutaneous disease. Her clinical expertise in these areas has been recognised with her listing in several local and national listings, including Best Doctors in America, “Guide to America’s Top Dermatologists”, and Best Doctors in Philadelphia Magazine. She received the Lady Colyton Prize for Autoimmune Research at the University of Pennsylvania in recognition of her research studies.

Disclosures
Grant/Research: Amgen, Celgene, NIH, Rigel, VA
Consultant/Advisor: Biogen Idec, Celgene, Cephalon, Genentech, Lupus Foundation, Madimux, Novartis, Pfizer, Sanofi-Aventis, Stiefel, UBC
Stock Shareholder: UV Therapeutics

Notes
**Keynote Presentation**

**Professor Marta E. Alarcón-Riquelme**
MD, PhD  
Universidad de Granada, Spain and Oklahoma Medical Research Foundation, USA

**The genetics of lupus:**  
**where are we now?**

In the last 5 years enormous advances have been gained in the identification of genes for systemic lupus erythematosus (SLE). Over 50 new genes have been identified, but their overall contribution to SLE is relatively small. Studies are also underway to describe the genetic contribution in populations other than the European, and several studies have been published and completed in the Asian populations of China and Korea. Furthermore, individuals of Latin American origin, a complex admixture of European and Amerindian (mestizos), are now under surveillance, as well as patients of African ancestry from the United States and Latin America. These studies will allow us to determine the pathways of disease development for specific populations.

SLE is a very heterogeneous disease, however, and more insight is still needed. To complement genetic analyses, studies in epigenetics, gene expression, cellular populations and tissue imaging are all required to define the groups of individuals where a given molecular pathway is affected. New and more precise classification criteria of the SLE based not only on clinical and serological parameters are required. These should also incorporate molecular information that will allow us to test and validate the best treatments for each differentiated group of patients. In this way we shall be able to develop true diagnostic tools that may help guide physicians to keep the most cost-effective approach towards their patient.

**References**


**Learning Objectives**

At the end of the presentation, participants will be able to:

- Understand how genetic association studies are accomplished.
- Have learnt the new technological and analytical advances in SLE.
- Have learnt the new methodologies to complement genetic work with epigenetics and gene expression in SLE.
- Have learnt the genetic basis of SLE in several populations.
Decreasing morbidity and mortality and improving outcomes in SLE

Systemic lupus erythematosus (SLE) is a protean autoimmune disease affecting multiple organ systems. As such, treatment and management of SLE is often challenging. Despite this, over the past few decades advances in diagnosis, management and introduction of newer therapies have led to improved life expectancies for patients with SLE. However, with this have come new challenges.

In patients with SLE, standardised mortality ratios have improved significantly, decreasing from 13.8 for patients seen in the 1970s to 3.8 for those seen in the early 2000s. Nevertheless, prolonged remission remains elusive, while damage accrual increases with disease duration. Of particular note is the increasing incidence of coronary artery disease, osteoporosis and osteonecrosis, and neurocognitive abnormalities. There is accumulating evidence that a considerable proportion of this damage can be attributed to corticosteroid use. Strategies to manage these complications and to minimise the exposure to corticosteroids are crucial to improve survival and quality of life for patients with SLE.

Learning Objectives
At the end of the presentation, participants will be able to:
- Appreciate the improving survival and decreasing mortality rates in SLE patients seen over the past four decades.
- Recognise the decrease in disease activity in patients at presentation and within cohorts over the past four decades.
- Recognise the impact of both damage accruing in SLE patients due to the disease and, importantly, therapies on patients’ quality of life.
- Diagnose the specific damage morbidities such as accelerated atherosclerosis, bone disease and neurocognitive abnormalities.
- Develop treatment strategies to prevent new morbidities.

References
The increased cardiovascular burden in patients with SLE is now well recognised and represents a significant cause of mortality and morbidity in these conditions. Cardiovascular risk includes an increased risk of coronary heart disease (CHD) as well as stroke syndromes. Recent studies continue to highlight the excess burden of cardiovascular disease in SLE and this continues to be a problem 30 years after its first description. A number of studies have also highlighted the importance of studying sub-clinical markers of cardiovascular risk as these might help us to better understand the pathogenesis of accelerated atherosclerosis in lupus.

We have recently focussed attention on the development of metabolic syndrome in the context of SLE as this is an interesting model of future cardiovascular risk. The presence of metabolic syndrome in the general population is associated with an excess risk of future CHD and we have noted that metabolic syndrome is more common in patients with SLE. Recent work from a large international cohort has also demonstrated that corticosteroid therapy contributes to the development of metabolic syndrome. Interestingly, however, we have also noted a significant contribution of active inflammatory disease and certain ethnic backgrounds are particularly prone to metabolic syndrome, most notably among patients from a Korean and Hispanic background. All of this suggests that stratiﬁcation of patients according to cardiovascular risk may be possible from early on in the disease course, and future work will focus on how changing our therapeutic regimes may impact on cardiovascular risk in lupus.

The role of chronic inﬂammation has also recently been highlighted, and recent data do suggest that suppression of inﬂammation has a modifying effect on surrogate cardiovascular markers. Overall, therefore, the management of cardiovascular risk should be multifactorial and multidisciplinary. Close attention to traditional risk factors should be a cornerstone of management. We should also aim to suppress disease as completely as possible whilst minimising the use/dose of corticosteroid therapy and ensuring patients without contraindications are treated with antimalarial agents. Clinical trials will be required to prove the efficacy of key therapies but such trials will require a concerted international effort.

Learning Objectives

At the end of the presentation, participants will be able to:

- Be aware that cardiovascular disease risk is increased in patients with SLE.
- Understand that from early in the disease, metabolic syndrome is more common in SLE.
- Know that corticosteroid therapy is likely to have a detrimental effect on the cardiovascular system.
- Appreciate that antimalarial drugs have potential beneﬁcial effects on the cardiovascular system.
- Be mindful that a targeted approach to cardiovascular modiﬁcation is recommended.
Patients can have cutaneous lupus erythematosus (CLE) with or without systemic disease. Patients with CLE can meet the diagnosis of systemic lupus erythematosus (SLE) solely because of their skin criteria. Identifying and diagnosing the type of skin lesions in lupus patients is important for identifying triggering medications, as well as prognosis and treatment.1-3

Skin lesions are divided into those that are specific and those that are non-specific for CLE. Specific cutaneous lesions include acute, subacute and chronic forms.4, 5 Acute cutaneous lupus erythematosus (ACLE) is often transient and typified by malar erythema. Subacute cutaneous lupus erythematosus (SCLE) is a photosensitive eruption characteristically more long-lasting than ACLE, but without the potential for scarring. Discoid LE (DLE) is characterised by lesions that can lead to permanent disfiguring scars and is frequently long-lived and intensely inflammatory. Subacute cutaneous lupus erythematosus (SCLE) is a photosensitive eruption characteristically more long-lasting than ACLE, but without the potential for scarring. Discoid LE (DLE) is characterised by lesions that can lead to permanent disfiguring scars and is frequently long-lived and intensely inflammatory. In addition to DLE, chronic CLE includes the less common LE tumidus, LE panniculitis, LE/lichen planus overlap, and chilblain lupus.

Non-specific cutaneous lesions can be seen at increased frequency in patients with lupus, particularly in those who have systemic disease. Among others, these lesions include Raynaud’s phenomenon, livedo reticularis, palmar erythema, and periungual telangiectasia. Purpura, urticarial papules or ulcerations due to vasculitis may occur, as well as cutaneous infarction resembling Degos’ disease or atrophie blanche. Patients with cutaneous lupus who have any of these findings should be evaluated for systemic disease. Proper identification of lupus specific and non-specific skin lesions is critical to appropriate management of patients.

References

Abstracts

Clinical manifestations and evaluation of cutaneous lupus

Learning Objectives
At the end of the presentation, participants will be able to:
- Recognise lupus specific cutaneous lupus.
- Recognise lupus nonspecific skin lesions.
- Understand the relative risks for SLE with specific cutaneous lesions of lupus.
- Understand diagnostic approaches for cutaneous lupus.
- Understand potential triggers of cutaneous lupus.

Learning objectives
At the end of the presentation, participants will be able to:

- Recognise lupus specific cutaneous lupus.
- Recognise lupus nonspecific skin lesions.
- Understand the relative risks for SLE with specific cutaneous lesions of lupus.
- Understand diagnostic approaches for cutaneous lupus.
- Understand potential triggers of cutaneous lupus.
Notes

The burden of fatigue in lupus: from patient perspectives to effective management

Fatigue is experienced by almost all patients with lupus at some point in time. It has been identified as a significant concern by patients and thus is included in various patient-reported outcome measures used in lupus for research or clinical care. Fatigue may not only impact the patient’s own health-related quality of life, but its effects may transcend to their families and friends, making it difficult for them to fulfill personal or professional aspirations and responsibilities.

It is very difficult to discern where the patient’s fatigue originates. It may be due to their active disease that is not well controlled, or from pain, fibromyalgia, poor sleep, depression or stress. Clinicians and researchers alike have attempted to understand the issue of fatigue in patients with lupus, with some describing the fatigue to be “of its own kind” in lupus. Screening and measurement of fatigue is important during the provision of lupus patient care. In some cases the treatment of fatigue may be as simple as discontinuing an offending medication. Several strategies to address fatigue have been tried; these include, but are not limited to, medications, acupuncture, cognitive behavioral therapy and even Wii-Fit™.

References

Abstracts

Hot Topic Lecture

Meenakshi Jolly, MD Rush University Medical Center, Chicago, USA

Learning Objectives

At the end of the presentation, participants will be able to:

- Understand the prevalence of fatigue and its correlates in lupus.
- Know how to measure fatigue in patients with lupus using common patient-reported outcome measures.
- Understand how to approach fatigue in patients with lupus.
- Know the various interventions that can be offered to patients with lupus with fatigue.

Notes
### Case Study Workshops

#### Thursday 18 April

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<td>Biologics for SLE: the future is here. When and how to use biologic therapies in clinical practice</td>
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<td><strong>Moderator/Facilitator:</strong> Graciela S. Alarcón (USA)</td>
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<td>Management and prevention of infections in SLE</td>
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<td><strong>Moderator/Facilitator:</strong> Mary-Carmen Amigo (Mexico)</td>
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<td>Lupus, pregnancy and APS: current issues in management</td>
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<td><strong>Moderator/Facilitator:</strong> Luis J. Catoggio (Argentina)</td>
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<td>Lupus nephritis: update on modern management</td>
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Please Note

These workshops will be held in breakout rooms. Please follow the appropriate signs/symbols corresponding to the workshops you are registered to attend. Please note workshops are repeated at 11:00 and 13:30 hours.
Case Study Workshop

Professor Ronald F. van Vollenhoven, MD, PhD

Case 1
A 32-year-old female was diagnosed with systemic lupus erythematosus (SLE) 8 years ago after presenting with widespread inflammatory skin rash and vasculitis changes on the fingers and toes, arthritis, alopecia, normocytic anaemia, elevated acute phase reactants, positive ANA, anti-ds-DNA, SSA, SSB, and very low complement. The patient never had renal involvement and has been treated with glucocorticoids in the close range 10–30 mg/d, antimalarials and azathioprine. Despite these medications, moderate lupus activity persists. Her clinical course was complicated by multiple infections, some very severe (i.e. bacterial meningitis and pneumococcal sepsis). Evaluation for underlying immune deficiency was negative.

What is the best course of treatment for this patient with typical smoldering non-renal lupus? Consider the possibility of biologics treatment.

Case 2
A 35-year-old female with was diagnosed with SLE 10 years ago on the basis of malar rash, arthritis, an episode of pleurisy, alopecia, anaemia, ANA, SSA, and SSB. She was treated with low-dose glucocorticoids and antimalarials. About 1 year ago she presented with progressively worsening general symptoms. Evaluation revealed mild proteinuria (350 mg/day) and electroneuropathy, and positive anti-DNA antibodies and glomerular filtration rate (GFR), by isohemese clearance, is 77 ml/min. A renal biopsy showed extensive membrano-proliferative glomerulonephritis with high activity and moderate chronicity. Treatment with cyclophosphamide in monthly IV boluses was initiated. After six infusions, clinical improvement was marginal and urinary abnormalities persist and GFR decreased to 64 mg/min. Repeat renal biopsy showed no improvement of the renal pathology.

What treatment options, including biologics, should be considered for this patient?

Case 3
A 17-year-old patient was diagnosed with SLE 4 months ago after presenting with malar rash, joint involvement, leucopenia, positive ANA, positive anti-ds-DNA and anticardiolipin antibodies (low titre). She started treatment with medium-dose corticosteroid and antimalarials with good response, but still she complains of some joint pain (with mild synovitis on ultrasound) and fatigue.

Case 4
A 29-year-old patient presented with diffuse subacute cutaneous lupus, arthritis in her wrist, and haemolytic anaemia. She wanted to become pregnant and, therefore, could not be treated with teratogenic drugs. She was treated with high-dose corticosteroids and azathioprine with initial remission of her manifestations followed by flaring with steroid tapering below 12 mg/day. After 8 months she was given methotrexate and a complete response of cutaneous manifestations was obtained. Arthritis in her wrists has persisted and she continues using steroids >16 mg/day. She is cushingoid. Patient has reported losing her job, increased in weight, and is unhappy with delayed pregnancy.

Case 5
A Caucasian female was diagnosed with SLE in 1992 based on malar rash, arthritis, pericarditis and severe thrombocytopenia (10,000 p/l). She was treated with corticosteroids, antimalarials and azathioprine until 1998. She remained in remission until April 2003, when she presented with fever, pleuritis, acute diffuse rash, anaemia and hypocomplementaemia; corticosteroids, antimalarials drugs and azathioprine are subsequently reintroduced. In May 2004, she exhibited a persistently active non-erosive arthritis; azathioprine is changed to methotrexate. In March 2006, she presented with arthralgias, hypocomplementaemia, proteinuria (approximately 2 g/24 hrs) with urinary casts; a renal biopsy revealed diffuse proliferative glomerulonephritis so treatment in line with the Eurolupus protocol was initiated. In October 2008, she had a renal flare and treatment with NH protocol was initiated followed by mycophenolate mofetil. Between 2009 and 2012 she had serum creatinine 1.8 mg/dL, proteinuria 1 mg/24 hrs, relapsing remitting arthritis and relapsing remitting cutaneous manifestations, so treatment was given with adjusted doses of corticosteroids, antimalarials and methotrexate. Best control of disease activity was achieved so no further changes in treatment were made. At 39-years-old (and with a disease duration of 20 years) the patient exhibits kidney, articular deformities (Jaccoud’s arthropathy) and osteoporosis (no fractures).

What treatment options, including biologics, should be considered for this patient?

Further Reading

Learning Objectives

At the end of the workshops, participants will be able to:
- Name the biologic therapies available for the treatment of SLE.
- Evaluate targets for treatment of SLE patients.
- Critically evaluate data needed to define when these therapies should be considered in routine clinical practice.
Management and prevention of infections in SLE

Professor Ricard Cervera, MD, PhD, FRCP

Case 1
A 22-year-old Caucasian woman with history of systemic lupus erythematosus (SLE) was admitted at the Hospital Clínic of Barcelona with fever, acute tonsillitis, nausea and diarrhoea. Diagnosis of SLE had been made 8 months before based on arthritis, photosensitivity, serositis, autoimmune haemolytic anaemia, positive antinuclear and anti-dsDNA antibodies, hypocomplementaemia, and presence of anticardiolipin IgG (57 GPL) and anti-β2-glycoprotein I IgG (68 IU/ml) antibodies (confirmed in a second determination). Four months before the current admission, diffuse segmental proliferative lupus nephritis was diagnosed by renal biopsy. The patient reported 6 days of fever (39ºC), tonsillitis and general malaise. Despite amoxicillin treatment, she persisted with fever, adding nausea and vomiting. Her medications on admission included mycophenolic acid 720 mg bd, prednisone 20 mg/day, hydroxychloroquine 200 mg/day and calcium.

The patient was in a regular state. Abdominal examination revealed mild tenderness in the epigastrium without hepatosplenomegaly. Her pharynx was oedematous and presented ulcerative lesions. Laboratory tests showed: WBC count 2.8x10⁹/L (89% neutrophils), platelets 82x10⁹/L, creatinine 3.6 mg/dL, AST 1,166 IU/L, ALT 1,776 IU/L, LDH 5,892 U/L, GGT 206 IU/L, Ap 214 IU/L, bilirubin 0.5 mg/dL, ESR 35 mm/hour and CRP 11.3 mg/dL.

By the third week of confinement, fever was persistent and she had new onset lung crackles. Haemoglobin was 77 g/L, WBC 12.5 x 10⁹/L, platelets 60 x 10⁹/L; Staphylococcus aureus grew from the catheter access site. Repeat chest radiograph showed new-onset lung infiltrates suggestive of hospital-acquired pneumonia; reticulonodular infiltrates were stationary. She was also started on valacyclovir for painful oral herpetic lesions. Over the next few days, she grew lethargic; there were no focal deficits or meningeal signs; fundoscopy showed indistinct optic disc margins. Cranial MRI (without contrast) disclosed early hydrocephalus. She was intubated and put on a mechanical ventilator. Antibiotics were shifted for broader spectrum coverage, valacyclovir was discontinued, anti-TB medications continued and antifungal (caspofungin) started. She received regular haemodialysis.

Her sensorium gradually improved over the remainder of her hospital confinement, she was weaned off mechanical ventilation, and she became afebrile. Antimicrobial regimens were completed, and she was discharged on anti-TB medications, tapering prednisone regimen and continued haemodialysis as an outpatient.

Professor Sandra V. Navarra, MD, FACP, FPAA

Case 2
A 39-year-old female with lupus nephritis is being maintained on prednisone 20 mg/day, mycophenolate mofetil 2 g/day, and hydroxychloroquine; she is also taking tinzaparin for recent deep venous thrombosis associated with antiphospholipid syndrome. Two weeks ago, she started to have intermittent low-grade fever attributed to an infected leg ulcer for which she received a course of oral antibiotics.

Despite improvement in the leg ulcer, she continued to have daily spikes in temperature with occasional chills. The present admission was prompted by episodes of diarrhoea and vomiting of one day duration accompanied by oliguria. Haemoglobin was 86 g/L, leucocyte 1.1 x 10⁹/L (85% neutrophils, 60% lymphocytes, 5% monocytes), platelet 40 x 10⁹/L, serum creatinine 5.6 mg/dL, and complement (C3) 0.92 g/L (LLN 0.90). She received intravenous fluids, prednisone was increased to 60 mg/day, and she was started on broad spectrum antibiotics and haemodialysis. Mycophenolate mofetil and tinzaparin were discontinued.

Over the next few days, she continued to be febrile with persistent pancytopenia; blood and urine cultures did not reveal any microorganism. A chest radiograph showed diffusely scattered reticulo-nodular infiltrates suggestive of military tuberculosis (TB). Anti-TB regimen consisting of isoniazid, rifampicin, ethambutol and pyrazinamide was started.
**Notes**

**Professor Munther A. Khamashita, MD, PhD, FRCP**

Case 1
A 35-year-old female sustained a stroke 2 years ago and is on warfarin. Her laboratory profile shows IgG ACA: 85 GPL (high) and IgG Anti-β2 GPI: 75 (high). She has been married for 10 years and she wishes to conceive. She seeks advice for the management of her anticoagulation therapy should she become pregnant.

Case 2
A 29-year-old female married for 7 years has had no successful pregnancies. Her laboratory profile revealed IgG ACA: 90 GPL (high), IgM ACA: 75 MPL (high), and positive lupus anticoagulant (LAC). This patient is planning for in vitro fertilisation (IVF) and has read extensively about antiphospholipid syndrome (APS); she is therefore seeking advice on the best approach to prevent complications secondary to APS should her pregnancy be successful. She is also questioning whether or not she should receive anticoagulation during her IVF cycle.

Case 3
A 24-year-old female has systemic lupus erythematosus (SLE) with positive LAC, IgG ACA: 45 GPL (moderate) and IgM ACA: 45 MPL (moderate). Her medical history reveals one pregnancy loss secondary to intrauterine foetal demise during the second trimester. Her lupus is well controlled and she wishes to become pregnant. She tested positive for anti-Ro antibodies and is seeking advice on the best medical approach for management during her pregnancy.

**Professor Roger A. Levy, MD, PhD**

Case 4
A 38-year-old patient of mixed ethnicity has cutaneous-articular SLE and is 6 weeks pregnant, with a history of four second-trimester foetal losses – she had used low-dose aspirin during her fourth pregnancy. Her LAC is positive and ACA positive at high titre (60 GPL., 44 MPL).

Case 5
A 31-year-old patient with isolated APS is 20 weeks pregnant and taking prednisone 10 mg/day, but tapering dose because her platelet count was 86,000/µL on 20 December 2011 compared with 36,000/µL on 19 January 2011. Her previous pregnancy, 2 years ago, was complicated with massive proteinuria and thrombocytopenia (lowest reading: 5,000 platelets), for which she was treated with pulse methylprednisolone and intravenous immunoglobulin.

Case 6
A 25-year-old patient with SLE, and two previous uneventful pregnancies, had negative tests for antiphospholipid antibodies and anti-dsDNA, and no protein in her urine. She is not planning to conceive in the next 2–3 years and asks for advice about contraception.

**Learning Objectives**

At the end of the workshop, participants will be able to:
- Offer the most appropriate contraceptive scheme for individual SLE patients.
- Understand how to manage a pregnant woman with positive anti-Ro/SS-A antibodies.
- Diagnose and manage antiphospholipid syndrome (APS) during pregnancy.
- Differentiate between pre-eclampsia, lupus nephritis and APS microangiopathy, and treat accordingly.
Case Study Workshop

Professor Paula Alba, MD, PhD and Dr Liz Lightstone MD PhD

Case 1
A 24-year-old mestizo (mixed race) girl, in her first pregnancy, presented with severe preeclampsia at 30 weeks. Her blood pressure on presentation was 200/130 mmHg and she had a serum creatinine of 122 mmol/l, nephrotic syndrome and microscopic haematuria. She delivered a live baby boy of 1.2 kg. One month later she was persistently hypertensive and treatment with enalapril 20 mg/d and amlodipine 10 mg/d was required. She still has proteinuria 3 grs/d, microscopic haematuria, anaemia, thrombocytopenia, and joint pain. Her serum complement levels were normal and double stranded DNA (ds-DNA) antibodies and lupus anticoagulant were positive.

Questions
Would you do a renal biopsy?
What would it show?
How would you treat her?
What would you advise for her next pregnancy?

Case 2
In January 2009, a 21-year-old mestizo male presented with fever, arthritis, malar rash, positive antinuclear antibody, high anti-ds-DNA levels and low serum complement levels. The diagnosis of systemic lupus erythematosus was made and treatment with prednisolone 10mg/d, hydroxychloroquine 400mg/d and azathioprine was started. Three months later, he developed proteinuria (3300 mg/24 h), microscopic haematuria with normal serum creatinine. The renal biopsy showed Class III lupus nephritis and treatment with cyclophosphamide according Euro-lupus protocol was started and renal response was achieved at the end of the induction therapy. Maintenance treatment with azathioprine was introduced. Three years later, he presented with fever, hypertension, proteinuria (3500 mg/d), microscopic haematuria, increase of anti-ds-DNA levels and decrease of complement levels and serum creatinine of 135 mmol/l.

Questions
Would you biopsy him again?
What would it show?
How would you treat him?

Learning Objectives
At the end of the workshop, participants will be able to:
- Understand the indications of renal biopsy in lupus nephritis.
- Understand the definition and management of renal flare.
- Describe the recommended induction treatment in class III/IV lupus nephritis.
- Describe the recommended maintenance treatment in class III/IV lupus nephritis.
- Describe the approach and advice of lupus nephritis in pregnancy.
Notes

T-cell signalling in patients with SLE:
what every rheumatologist needs to know!

Systemic lupus erythematosus (SLE) is seen classically as a disease of auto-reactive B cells producing auto-antibodies that cause pathology in organs and tissues. However, much evidence shows that the immune system cannot be considered in isolation and while B cells are the most obvious candidates underlying disease pathogenesis, many other immune cells contribute to the overall pathology seen in patients. T cells have long been known to be abnormal in patients with SLE. They have a lower activation threshold, undergo increased apoptosis, produce high levels of cytokines including IL10 and IL6 and provide excessive help to B cells thereby contributing to the production of auto-antibodies.1

T cells from patients with SLE have many defects in the way these messages are delivered and translated. Some of these defects are associated with genetic abnormalities in patients whereas some are acquired due to the pro-inflammatory environment associated with lupus disease.2

Recently, great advances have been made in understanding exactly how T cell signalling defects affect SLE pathogenesis. Increased signalling via the T cell antigen receptor has been attributed to an altered profile of plasma membrane lipids that changes the position of key signalling molecules affecting the balance of ‘on’ or ‘off’ signals; an altered expression of important signalling molecules changes the intensity of the signals transmitted and an enhanced rate of calcium-associated signalling predisposes T cells to undergo accelerated cell death.3, 4

Understanding these processes has enabled the prospect of developing novel and targeted intervention strategies.

References


Immunopathogenic mechanisms driving SLE

Given certain genetic risk factors, systemic lupus erythematosus (SLE) develops following environmental triggering and subsequent interplay of innate and adaptive immunity critically linked by antigen-presenting cells (APCs), resulting in the breakdown of tolerance. This loss of immune homeostasis includes the generation of various autoantibodies (including those against dsDNA), formation of immune complexes and cytotoxic T cells. Although the precise cellular and humoral mechanisms, and what determines which organs are involved in this heterogeneous disease, remain poorly understood, the initiating tissue likely provides a decisive immune microenvironment. Here tissue-resident dendritic cells that have different capacities to direct the immune response (balance of humoral and cellular components), together with invading plasmacytoid dendritic cells, define organ manifestations.

A common denominator of the autoimmune response is the utilisation of type I interferon, but other cytokine networks can be involved. Different immune pathways are likely driven by distinct dendritic cells in the target tissue that apparently provide a critical link to both the organs involved and activating innate and adaptive immunity. Promising new therapies may simultaneously and specifically target several of the critical pathways in autoimmunity. Autoantibodies (i.e. anti-dsDNA) are induced based on a genetic MHC class II predisposition and are able to fuel immunopathogenesis by formation of immune complexes, but also by activating pDCs. Thus, autoantibodies appear to be involved in a positive forward loop and, therefore, critically linking adaptive and innate immunity in SLE.

Learning Objectives

At the end of the presentation, participants will be able to:

- Be familiar with the currently accepted interaction of innate and adaptive immunity driving SLE pathogenesis.
- Understand the genetic background of SLE linked to immune pathways.
- Learn the heterogeneous immune consequences of increased type I interferons and other cytokine networks considered to be involved in SLE.
- Learn that immune memory provided by lymphocytes is important for the maintenance of autoimmunity.
- Be familiar with pathways of how autoreactive B cells are likely selected to differentiate in plasma cells producing autoantibodies.

References


Professor Thomas Dörner, MD
Charite University Hospitals Berlin, Germany
Biologics in lupus: clinical practice to clinical trials... and now...

With the successful introduction of biologic therapies for patients with rheumatoid arthritis more than a decade ago, it seemed likely that a wide array of precisely targeted treatments would soon be available to patients with systemic lupus erythematosus (SLE). An improved understanding of the immune response, together with better knowledge of the aetiopathogenesis of lupus, identified cells and molecules to block that were likely to be critically involved in the development and/or perpetuation of the disease.

Initial open-label studies with rituximab (blocking the CD20 molecule on B cells) appeared promising, especially when they were widely reproduced. However, double-blind, placebo-controlled trials of this drug (EXPLORE1 and LUNAR1 for lupus nephritis) failed to meet their primary or secondary endpoints.1,2 In fact, lupus treatment development was rapidly becoming a graveyard for biologics, with virtually every treatment encountering extremely disappointing Phase II outcomes. Development has been delayed for rituximab and abatacept (CTLA4i, which interferes with communications between antigen-presenting cells and T cells) despite widespread off-label use of both of these treatments, which are marketed for rheumatoid arthritis and have been used in some lupus clinics. Development has been halted completely for several agents, including DHEA (a β-cell modulator), LJP394 (designed to block anti-dsDNA antibodies) and two agents targeting CD40 Ligand (DEC 131 and Biogen Bg9688) and the future appears dim for a number of other treatments that have since failed in Phase II studies. However, the developers of one such treatment that did not meet Phase II endpoints, belimumab, which blocks the activity of BLyS (a B-cell activating factor) did use an exploratory analysis of that failed trial to design and completed a large Phase III programme3,4 and became the first new internationally regulatory-approved treatment for lupus in many decades.

Similar agents which target BLyS and other B-cell activating factors (e.g. atacicept) and epratuzumab, which blocks the CD22 molecule on B cells, have similarly entered Phase II trials, neither of which had substantial sized, or definitive results in Phase II. During this time, the technology for clinical trials has progressed, and an appreciation of the likely reasons why earlier trials did not work has emerged. Detailed post-hoc analyses of earlier trials provided evidence that efficacy results may be masked both by too little disease and too much background treatment in trials, and a re-evaluation of specific clinical endpoints used in renal and non-renal trials provides some confidence of obtaining increasingly interpretable data from these extremely expensive endeavours. At the same time, biologic subsets of patients, and the differential impact of certain background medications on these heterogeneous patients, are beginning to be better resolved, providing a framework for more rational clinical trial designs in the near future.

References