Foreword

On behalf of the Conference Programme Committee and the Organising Committee, I would like to extend a warm welcome to everyone attending the Osteoporosis and Bone Conference 2012 in Manchester, UK. The meeting this year is being hosted for the first time by the National Osteoporosis Society, the Bone Research Society and the Paget’s Association. With the combined expertise of the three host organisations, the Osteoporosis and Bone Conference offers a varied scientific programme that will be of interest to clinicians, health professionals and basic scientists working in the field of bone disease. Our keynote speakers are of international standing and come from a range of disciplines that reflect the diverse interests of the host organisations. The Conference is being held at the Manchester Central Convention Complex, an award-winning, flexible and state-of-the-art conference venue located in the heart of Manchester’s city centre.

Scientific Programme

This year’s Osteoporosis and Bone Conference comprises an exciting and varied programme – including both clinical and basic science aspects of bone disease – that we hope you will find thought-provoking and inspiring. There will be a comprehensive range of oral and poster presentations selected from submitted abstracts. As in previous meetings, our popular Educational Update sessions will provide updates on recent developments in osteoporosis and bone disease. A number of interactive workshops will address topical and clinically relevant issues including FRAX, clinical use of bone markers, and (for the first time) pre-clinical and basic science topics. A symposium on Paget’s disease will include an update on the major clinical and scientific developments in the field. We are delighted also to have a number of leading pharmaceutical and healthcare companies exhibiting at the conference. A key component of the meeting will be the extensive opportunities for informal discussion and networking. Finally, for those with an interest in a research career, a New Investigator session will feature talks on career development and funding by some of the major providers in our field.

Abstracts

The response to our call for abstracts this year has been excellent. Each abstract has been marked anonymously by reviewers and given an overall mean score. The final abstracts will be available to view as poster presentations or in oral presentation sessions throughout the conference.

Awards

This year there will be a ‘Young Scientist’ prize that recognises the work of an investigator pursuing research into osteoporosis and/or fragility fractures. In addition, Young Investigator awards and Allied Health Professional awards will also be presented to those abstract authors at the meeting who have acquired the highest mean review scores. There will also be Premier Poster prizes for highly commended poster presentations chosen at the meeting. Premier Poster nominations are marked in this handbook and highlighted on poster boards. Finally, we would like to thank you all for attending and contributing to the success of this conference, and hope you have an enjoyable and informative experience.

Professor Terence W O'Neill
Conference Chair

On behalf of the Osteoporosis and Bone Conference 2012 Programme and Organising Committees.
Programme Committee
Professor Terence O’Neill (Manchester) – Conference Programme Committee Chair and Organising Committee Chair

Organising Committee Members
Prof. Richard Eastell (Sheffield)
Prof. Roger Francis (Newcastle)
Prof. David Reid (Aberdeen)
Anne Sutcliffe (Manchester)
Prof. Jonathan Tobias (Bristol)

Programme Committee Members
Prof. Judith Adams (Manchester)
Prof. Tim Arnett (London)
Prof. Juliet Compston (Cambridge)
Prof. Cyrus Cooper (Southampton)
Dr Graham Davenport (Keele)
Dr Alison Gartland (Sheffield)
Dr Nick Harvey (Southampton)
Dr Helen Macdonald (Aberdeen)
Dr Nicola Peel (Sheffield)
Debbie Stone (Aberystwyth)
Dr Mike Stone (Cardiff)
Dr Kate Ward (Cambridge)
Mark Wilkinson (Sheffield)

Additional Abstract Reviewers
Prof Nigel Arden (Oxford)
Dr Pam Brown (Swansea)
Dr Alun Cooper (Crawley)
Dr Richard Keen (Stanmore)
Dr Rachel Lewis (Bristol)
Shirley Love (Cambridge)
Prof David Marsh (Stanmore)
Prof Chris Moran (Nottingham)
Dr Opinder Sahota (Nottingham)
Dr Peter Selby (Manchester)
Angie Snow (Derby)
Dr Susan Steel (Hull)

Conference Organisers
The event is organised by the National Osteoporosis Society, a registered charity no. 1102712 in England and Wales, no. SC039755 in Scotland and registered as a company limited by guarantee in England and Wales no. 4995013.

Events Department
National Osteoporosis Society
Camerton, Bath, BA2 0PJ, UK

Barry Jordan
Tel: 01761 473123
Fax: 01761 479271
Email: conferences@nos.org.uk

The event is hosted by three organisations: the National Osteoporosis Society, the Bone Research Society and the Paget’s Association.

Conference Website
www.nos.org.uk/conference

Registration Desk Opening
The registration desk for the conference will be located inside the entrance in the lower foyer and will be open as follows:
- Sunday 1st July: 13.30–19.30
- Monday 2nd July: 08.15–19.15
- Tuesday 3rd July: 08.00–19.00
- Wednesday 4th July: 07.30–13.00

Oral Presenters
All speakers are asked to report to the Speaker Preparation Room to upload their finalised presentation slides with the room technician, where possible, the day before the presentation. The Speaker Preparation Room is networked with the meeting room computers. We ask that you finalise your presentation before loading it onto the PC in the preparation room as later changes are difficult to accommodate.

The Speaker Preparation Room will be open as follows:
- Sunday 1st July: 13.30–18.30
- Monday 2nd July: 07.30–17.30
- Tuesday 3rd July: 07.30–17.30
- Wednesday 4th July: 07.30–12.00

Speakers are then asked to check in at the meeting room 30 minutes prior to the start of the session to become familiar with the equipment and to meet with the session chair(s). In the auditorium, please sit in the reserved seats for speakers.

Please note
Photography (and audio-recording) is not permitted during sessions or of abstract posters.

Anyone found taking photographs in these circumstances will be asked to leave the premises. Thank you for your co-operation.
**Evaluation Form**

Your comments and views on the event are highly valued, and we would encourage you to complete the evaluation form that will be sent to you via email post-conference.

**CPD and Certificates of Attendance**

Continuing professional development accreditation from the Royal College of Physicians of London has been granted for the Osteoporosis and Bone Conference 2012. 22 credits will be provided for attendance at the full event.

To gain CPD accreditation, please ensure that, in addition to registering for the conference when you arrive, you sign in at the registration desk on each day that you attend. A CPD sign-in list will be available on the registration desk.

To assist health professionals with their CPD needs, official certificates of attendance listing the learning objectives and topics covered in this conference will be available.

To apply for your accreditation and/or certificate of attendance, please visit the registration desk.

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**Conference Dinner**

Tuesday 3rd July, 20.00–midnight  
The Midland Hotel, Manchester  
The dinner is included in the registration fee for all registered full conference delegates, and tickets are placed in badge holders. The event will include a welcome pre-dinner drink, dinner and entertainment. A cash bar will be available with dinner.

**Internet Café**

Don’t forget to visit the internet café in the exhibition area of the Exchange Hall.
The Bone Research Society (BRS) is delighted to be a partner in this year’s Bone Conference, along with the National Osteoporosis Society and Paget’s Association.

Here’s a taste of what we do …

**MEMBERSHIP**
Join the Society and receive:
- Reduced registration for BRS annual meeting
- Eligibility to apply for New Investigator Awards
- Eligibility for travel grants to attend BRS and other meetings
- Access to the Members’ Area of the BRS website containing a number of educational resources, including slide handouts

COST: £50 per year (£25 for students)

**ANNUAL MEETING**
Bone Research Society/
British Orthopaedic Research Society
Joint Meeting
4-5 September 2013
OXFORD, UK
Local Organisers: Cyrus Cooper/Claire Edwards/
James Edwards/Richie Gill/Kassim Javaid

**TRAINING COURSES**

**BRS Training Course: Osteoporosis and Other Metabolic Bone Diseases**
20-22 March 2013
ST CATHERINE’S COLLEGE, OXFORD, UK
Organisers: Tash Masud (Nottingham)/Jon Tobias (Bristol)

The Society also organises training courses for basic scientists – keep an eye on our website for more information.

**RESOURCES**
The BRS website includes information about
- Jobs
- Fellowships
- Studentships
- Educational resources
- Members’ Area: more resources, including slide handouts

Visit the website to find out more!

www.brsoc.org.uk

**COMMITTEE 2011/12**
President: Jon Tobias (Bristol), President Elect: Tim Arnett (London), Secretary: Eugene McCloskey (Sheffield), Treasurer: Nigel Loveridge (Cambridge), Gavin Clunie (Ipswich), Kate Ward (Cambridge), Isabel Orriss (London), Vicky MacRae (Edinburgh), Allie Gartland (Sheffield), Celia Gregson (Bristol), Sanjeev Patel (London), Deborah Mason (Cardiff)
Whilst the acceptance of an exhibitor or sponsor does not imply endorsement of a company or its products by the National Osteoporosis Society, we are very grateful for the generous support all the companies have given to the Osteoporosis and Bone Conference 2012.

Their support helps to keep registration fees as low as possible and thereby maximises attendance. The companies host a number of delegate places and contribute to the objectives of the conference by providing educational satellite sessions and by covering the expenses of key international and eminent national speakers.

In return, please take the time to look around the exhibition and find out more about the companies, their products and their services, and please do support the satellite sessions.
Osteoporotic vertebral fractures are common and serious, yet under-diagnosed and under-reported. SpineAnalyzer™ is a software workflow tool that helps you to address these problems. It is quick and easy to use, works with x-rays and DXA-based VFA images.

SpineAnalyzer enables you to:
- Assess and report on all vertebrae (T4-L4) in less than 3 minutes
- Produce a clear, standardized report indicating fracture / no fracture
- Review trending of vertebral fracture over time
- Provide a key risk factor for FRAX® assessment
- Communicate fracture status with a patient

"I believe that, in the hands of physicians, the SpineAnalyzer Program represents a major step forward in facilitating and standardizing their assessment of vertebral fractures across a broad spectrum of osteoporosis clinical and research practice."

DR. HARRY K. GENANT
Professor Emeritus of Radiology, Orthopedic Surgery, Medicine, and Epidemiology at the University of California San Francisco

"A vertebral fracture, found on a radiograph, is the most important risk factor for future fracture. We have used SpineAnalyzer to assess hundreds of spine films and I am impressed that it would change practice by making accurate assessment of vertebral deformities an easy, routine part of radiologic practice; this would substantially improve patient care."

DR. STEVEN R. CUMMINGS
Professor of Medicine and Epidemiology, at the University of California in San Francisco

www.optasiamedical.com

Optasia Medical
Innovation in X-Ray Imaging Software

ASK FOR A SOFTWARE DEMONSTRATION ON BOOTH 24
Alexion
Exhibition Stand: 26
352 Knotter Drive
Cheshire
CT 06410
USA
Telephone: +1 203-272-2596
Website: www.alexionpharma.com

Alexion Pharmaceuticals, Inc. is a biopharmaceutical company focused on serving patients with severe and ultra-rare disorders through the innovation, development and commercialization of life-transforming therapeutic products. Alexion developed and markets Soliris® (eculizumab) as a treatment for patients with paroxysmal nocturnal hemoglobinuria (PNH) and atypical hemolytic uremic syndrome (aHUS), two debilitating, ultra-rare and life-threatening disorders caused by chronic uncontrolled complement activation. Alexion is evaluating other potential indications for Soliris and is developing four other highly innovative biotechnology product candidates, including asfotase alfa, an investigational targeted enzyme replacement therapy for patients with hypophosphatasia (HPP), an ultra-rare, genetic, life-threatening metabolic disease.

Amgen
Exhibition Stand: 1
Amgen UK
1 Uxbridge Business Park
Uxbridge
UB8 1DH
Telephone: 01895 525581
Website: www.amgen.co.uk

Amgen discovers, develops, manufactures, and delivers innovative human therapeutics. A biotechnology pioneer since 1980, Amgen was one of the first companies to realize the new science’s promise by bringing safe, effective medicines from lab to manufacturing plant to patient. Amgen therapeutics have changed the practice of medicine, helping millions of people around the world in the fight against cancer, kidney disease, rheumatoid arthritis, bone disease, and other serious illnesses. With a deep and broad pipeline of potential new medicines, Amgen remains committed to advancing science to dramatically improve people’s lives. For more information, visit www.amgen.com

Arthritis Research UK
Arthritis Research UK
Copeman House
St Mary’s Gate
Chesterfield
Derbyshire
S41 7TD
Tel: +44 (0) 300 790 0400
Website: www.arthritisresearchuk.org

Arthritis Research UK is the charity leading the fight against arthritis. We’re working to take the pain away for sufferers of all forms of arthritis and helping people to remain active.

We do this by funding high class research, providing information and campaigning. Everything we do is underpinned by research. Our core remits are funding world-class research and providing a comprehensive range of information for patients, the public and health professionals and we are committed to raising the profile of arthritis and making a real difference to people’s lives.
Eli Lilly

KEY SUPPORTER

Exhibition Stand: 29

Eli Lilly and Company Limited
Lilly House
Priestley Road
Basingstoke
Hampshire
RG24 9NL

Telephone: 01256 315000
Website: www.lilly.co.uk

Eli Lilly and Company Limited is one of the world’s largest research-based bio-pharmaceutical companies, dedicated to creating and delivering innovative pharmaceutical healthcare solutions that enable people to live longer, healthier and more active lives. Our research and development efforts constantly strive to address the world’s growing unmet medical needs in several different clinical areas.

Eli Lilly has a significant presence in the UK at several locations:

Erl Wood, Surrey: Home to our European HQ and largest R&D facility outside the US.
Speke, Merseyside: A bulk biotech manufacturing plant which makes both human and animal health products.
Basingstoke, Hampshire: The Headquarters for the UK pharmaceutical business.

For information about our products or services, please come and talk to us at the Lilly stand, or log on to the Eli Lilly website at www.lilly.com We look forward to meeting you. Jeff Howe, Sales and Marketing Manager, Osteoporosis email:howe_jeffrey_r@lilly.com

Haymarket Medical

Exhibition Stand: 15

Haymarket Medical
174 Hammersmith Road
London
W6 7JP

MIMS, or the Monthly Index of Medical Specialities, has been providing healthcare professionals with concise and reliable information on prescription medicines since its launch in 1959. Published monthly, MIMS is one of the most up-to-date prescribing guides in the UK. MIMS is independently compiled by our editorial team, who incorporate around 200 changes every month. In addition to summarising information on more than 2000 prescription products, MIMS includes a number of helpful quick-reference prescribing resources, including drug comparison tables and guideline summaries. Complementing the print edition, MIMS is also available as a fully searchable, interactive website: www.mims.co.uk/ and as an app for iPhone, iPad and Android.
IDS (Immunodiagnostic Systems)  
Exhibition Stand: 19

Immunodiagnostic Systems (IDS) Plc  
10 Didcot Way  
Boldon Business Park  
Boldon  
Tyne & Wear  
NE35 9PD

IDS excels at providing scientifically advanced solutions that allow laboratories to efficiently perform and confidently report results for important specialty markers in the areas of bone, calcium, growth, cartilage and Hypertension.

The IOF and IFCC experts recommend that s-PINP and s-CTX are used as reference analytes for bone turnover markers in observational and intervention studies. Together with the co-specific 25 Vitamin D, the s-PINP and s-CTX can be consolidated onto the automated IDS-iSYS system for a complete bone turnover and nutritional status.

Please come and visit stand 19 to:
- Discuss our Vitamin D tests
- How our bone and growth assays can support your research
- Specialty ELISA kits from our partners

GE Health Care

Email: Paul.Stevens@med.ge.com  
Website: www.gehealthcare.com

GE is dedicated to helping you transform healthcare delivery by driving critical breakthrough in biology & technology. Our expertise in medical imaging & information technologies, medical diagnostics, patient monitor systems, drug discovery, and biopharmaceutical manufacturing technologies is enabling healthcare professionals around the world discover new ways to predict, diagnose and treat disease earlier.

Med Imaging
Exhibition Stand: 14

Telephone: 0870 241 1397

MedImaging is a leading independent solutions provider for specialist diagnostic imaging equipment established in the UK in 1995. Over the past 16 years a solid reputation has been established with customers for the provision of innovative service and product solutions. Innovation is the result of teamwork and success. This has been achieved by the company working closely with customers to identify their requirements and provide tailored solutions efficiently and within budget. It is also recognised that cost efficiency and savings are frequently driving elements for many projects. MedImaging's aim is to achieve high standards whilst providing significant cost savings.
medi UK Ltd.
Exhibition Stand: 20

medi UK Ltd.
Plough Lane
Hereford
HR4 0EL

Telephone: 01432 373500
Fax: 01432 373510
Email: enquiries@mediuk.co.uk
Website: www.mediuk.co.uk

Offering bracing and bespoke spinal bracing medi UK Ltd prides itself on the highest possible standards. We are a family owned company that is based in Hereford. We offer excellence in delivery, with a free next working day delivery service. Customer service is a keystone for the company. We offer advice from highly experienced and courteous staff.

National Osteoporosis Society

ORGANISER AND EXHIBITOR
Exhibition Stands: 9, 22 & 30

National Osteoporosis Society
Manor Farm
Camerton
Bath
BA2 0PJ

Telephone: 0845 130 3076
Website: www.nos.org.uk

The National Osteoporosis Society is the only UK-wide charity offering support to people with osteoporosis, their families and carers through a range of information booklets, a national telephone helpline and a network of regional support groups. The National Osteoporosis Society works to raise awareness of osteoporosis and bone health, fundraises for research, works with healthcare professionals across a wide range of disciplines to facilitate greater understanding of the needs of people with osteoporosis and encourages the government and health services to resource and deliver appropriate services to prevent and treat fractures.

Ocean Media Magazines Ltd

Exhibition Stand: 21

Ocean Media Group
One Canada Square
Canary Wharf
London
E14 5AP

Telephone: 020 7772 8300
Website: www.oceanmedia.co.uk

GM is the leading monthly peer-reviewed journal for primary and secondary care physicians with an interest in the 50+ patient and the conditions that impact upon them. It successfully reaches 23,000 named healthcare professionals with each issue across both primary and secondary care. With the majority of issues requested it is important to request your own copy free of charge at the GM booth. In addition six therapy issues under the banner GM2 - are also published every year, each dedicated to an individual therapy area, including Cardiology / Diabetes / Mental Health / Neurology, Oncology and conditions of Gender. Visit the GM website: www.gerimed.co.uk/ for more information.
Osteocare is the UK’s most popular calcium formula, providing nutritional protection for healthy, strong bones. From Vitabiotics, the leading supplement company, it has been developed by doctors and pharmacists on the very latest international findings. Its natural source calcium is the most concentrated form of calcium available. Magnesium is also essential for bones but many supplements only provide a token amount; Osteocare provides 300mg magnesium, the full recommended amount. As well as the exact RDA of calcium and magnesium, Osteocare’s unique formula also provides essential cofactors like vitamin D, zinc, manganese and selenium. Latest additions to the Osteocare range include Osteocare Chewable and Osteocare Plus with soy isoflavones and Omega-3.

Optasia Medical makes software that facilitates the reading of X-rays for the management of musculoskeletal diseases. Our products are used by identify and monitor patients who will benefit from treatment. With Optasia software, x-rays can be evaluated more rapidly, accurately and reproducibly. Our flagship product, SpineAnalyzer™ facilitates the assessment and documentation of osteoporotic vertebral fracture. Prevalent vertebral fracture is a key component in the calculation of future fracture risk but is largely under-diagnosed and under-reported. SpineAnalyzer provides a rapid workflow for the assessment of deformity that may indicate vertebral fracture, including a convenient report for the referring physician.

The Paget’s Association is the only UK organisation that focuses solely on Paget’s disease. It acts as a resource for those with Paget’s disease of bone, as well as offering high-quality information and support to health professionals and the general public when necessary.
ProStrakan
Exhibition Stand: 6, 23

ProStrakan
ProStrakan Group plc
Galabank Business Park
Galashiels
TD1 1QH

ProStrakan Group is a rapidly growing specialty pharmaceutical company engaged in the development and commercialisation of prescription medicines for the treatment of unmet therapeutic needs in major markets.

A member of the Kyowa Hakko Kirin group of companies, ProStrakan is headquartered in Galashiels in Scotland. The company’s development capabilities are centred on Galashiels and Bridgewater, New Jersey, US. Sales and marketing of ProStrakan’s portfolio of products are handled by commercial subsidiaries in the UK, US, France, Germany, Spain, Italy and other EU countries.

Bone Research Society
Website: www.brsoc.org.uk

The Bone Research Society (BRS) was founded in 1950 to promote research into bone and other mineralised tissues. It was the forerunner of the European Calcified Tissue Society (ECTS), with which it is affiliated, and the American Society of Bone and Mineral Research (ASBMR). The BRS supports research into the full range of bone diseases, including osteoporosis. This comprises both basic research in the laboratory, and clinical research in patients and the wider population.

Rosemont Pharmaceuticals
Exhibition Stand: 12

Rosemont Pharmaceuticals Ltd
Rosemont House
Yorkdale Industrial Park
Braithwaite Street
Leeds LS11 9XE

Rosemont Pharmaceuticals are the leading specialists for licensed and “specials” oral liquid medicines for people who have difficulty swallowing solid medication. With over 40 years experience of R&D, manufacturing, and marketing of liquid alternatives; and an extensive range of over 140 different formulations*, all our products are made to a formulation with active ingredients and excipients. Unlike other special manufacturers we never crush, dissolve or suspend solid formulations to make them.

Rosemont is dedicated to the promotion of best practice in medication administration; and provides training and educational materials to healthcare professionals. Rosemont is ‘The Source of Liquid Solutions’.
Rothband
Exhibition Stand: 13

W S Rothband & Co. Ltd
4-6 Knowsley Road
Haslingden
Rossendale
Lancashire
BB4 4RX

Telephone: 01706 830086
Website: www.rothband.com

Rothband are pleased to attend the NOS Conference as the new UK distributor for the DXL Calscan. Rothband are an established Health imaging supplier with staff who are experienced in the Imaging Market and Bone densitometry in particular. The DXL Calscan is becoming established as the equipment of choice for peripheral Bone densitometry. Please come and visit our stand where we would be pleased to discuss the DXL Calscan and the exciting new prospective fracture study which validates the DXL Calscan as a valuable tool in Osteoporosis Diagnosis. For further information, please visit our website.

Servier Laboratories Ltd

Exhibition Stand: 4

Servier Laboratories Ltd
Rowley
Wexham Springs
Framewood Road
Wexham
Slough
SL3 6PJ

Telephone: 01753 662744
Website: www.servier.co.uk

Servier Laboratories is the UK subsidiary of The Servier Research Group, the leading independent French research based pharmaceutical company. The key franchises of the Servier Research Group are rheumatology, cardiovascular disease, diabetes, Central Nervous System and oncology. Servier regularly invests more than 25% of its annual turnover into research, discovering and delivering therapeutic innovations to patients through their healthcare professionals. For more information about Protelos, go to www.servier.co.uk/products/protelos/

Vertec
Exhibition Stand: 27

Vertec Scientific Limited
Unit 44, Easter Park
Benyon Road
Silchester
Reading
Berkshire
RG7 2PQ

Telephone: 01189 702100
Website: www.vertec.co.uk

Based in Berkshire, Vertec has grown in 30 years to become one of the biggest distributors of world class medical devices in the UK. Representing a wide range of technologies from DXA and digital x-ray to radiotherapy consumables and tumour location systems, the company has earned the highest reputation for customer focus and support. In DXA exams for osteoporosis, Vertec is the leading supplier of diagnostic systems in the UK, beginning with the Hologic QDR pencil beam system and culminating in the Discovery series which offers peak precision APEX software, FRAX 10 year integrated reporting and advanced whole body composition. For further information visit our website.
Are your clients getting enough calcium & vitamin D?

Vitamin D is known as the sunshine vitamin and contributes to normal absorption and utilisation of calcium. It may also help contribute to overall wellbeing. Osteocare’s expert formula contains **D3, the preferred form of vitamin D, plus the full RDA of calcium**, along with **magnesium**, **zinc** and other essential co-factors, so you can give your clients the support they need.
Abstracts IS1–IS7, O1–O52 and P1–P134 can be found in the special supplement of Osteoporosis International. Materials relating to EU1–EU6 and Workshops can be found in this handbook along with biographies of plenary speakers IS1–IS7 and Educational Update speakers EU1–EU6.

### Sunday 1st July 2012

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<th>Time</th>
<th>Event</th>
<th>Location</th>
<th>Chair(s)</th>
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<tbody>
<tr>
<td>13.30</td>
<td>Registration Opens</td>
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<tr>
<td>15.30 – 17.30</td>
<td>Parallel Session: Bone: The Basic Facts</td>
<td>Exchange Auditorium</td>
<td>Debbie Stone, Dr Nicola Peel &amp; Anne Sutcliffe</td>
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<td>New Investigator Session</td>
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<td></td>
<td>Location: Exchange 11</td>
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<tr>
<td></td>
<td>Session Chairs: Dr Nick Harvey, Dr Celia Gregson, Dr Alison Gartland</td>
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<tr>
<td>15.30 – 16.00</td>
<td>What Makes Healthy Bone Unhealthy?: Professor Roger Francis</td>
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<tr>
<td>14.00 – 17.30</td>
<td>How to Achieve a Successful Research Profile Led by Dr Kay Guccione</td>
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<td></td>
<td>Location: Exchange 11</td>
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<tr>
<td></td>
<td>Session Chairs: Dr Kay Guccione, Researcher Development Advisor, University of Sheffield</td>
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<td></td>
<td>This session will include guidance on how to network effectively with colleagues at all levels, and how to increase your profile as a researcher, and will include some practical (friendly) exercises!</td>
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<td>15.45 – 16.00</td>
<td>How Does a Clinician Manage Osteoporosis?: Dr Nicola Peel</td>
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<td>16.00 – 16.30</td>
<td>Tea Break</td>
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<td>16.30 – 17.00</td>
<td>How Does a Clinician Manage Paget’s Disease?: Anne Sutcliffe</td>
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<td>16.00 – 17.30</td>
<td>‘CV clinic’ – Bring Your Own CV for Feedback and Advice!</td>
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<td>This session focuses on how you present yourself on paper. How to tailor your CV for the post you want. Confidential, constructive feedback will be available from our panel of established researchers: clinical, laboratory-based and translational</td>
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<td>17.00 – 17.30</td>
<td>What Does an Allied Health Professional Contribute to the Management of Bone Disease?: Debbie Stone</td>
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<td>17.30 – 18.00</td>
<td>An Opportunity to Put Your Newly Acquired Networking Skills to the Test!</td>
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<td>(Sojourn in a local hostelry)</td>
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<tr>
<td>17.30 – 18.00</td>
<td>Pre-conference Session Refreshments. Location: Exchange Foyer</td>
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Sunday 1st July 2012

18.00-19.30 Conference Session Supported by the National Osteoporosis Society

Launch of the UK Allied Health Professional Network

Location: Exchange Auditorium

Session Chair: Nina Copping

18.00 Welcome and Introduction
Caroline Johnson, Country Development Manager for England, National Osteoporosis Society

18.10 Professional Peer Support Networks – Personal Reflections
Shirley Love, Service Manager & Nurse Specialist in Osteoporosis, Metabolic Bone Unit & Bone Density Unit, Addenbrooke's Hospital, Cambridge and Angie Snow, Acting Assistant Subject Head Radiography & Programme Leader MSc Advanced Practise, School of Health, University of Derby

18.20 Sharing Good Practice
Colin Beevor, Matron, Rheumatology Department, Queen Alexandra Hospital, Cosham

18.30 A Bit of Give and Take!
Debbie Stone, Specialist Osteoporosis Nurse, Bronglais Hospital, Aberystwyth

18.40 The National Osteoporosis Society Allied Health Professional Network
Hilary Arden, Training & Professional Development Manager, National Osteoporosis Society

19.00 Regional Workshops

19.20 Summary and Close
Caroline Johnson, Country Development Manager for England, National Osteoporosis Society

End of Day 1

Monday 2nd July 2012

8.15 Exhibition Opens

9.00 – 10.15 Educational Update

Location: Exchange Auditorium

Session Chairs: Professor David Reid and Professor Judy Adams

9.00 EU1 Pathophysiology of Osteoporosis: Professor Stuart Ralston

9.25 EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey

9.50 EU3 Diagnosis and Fracture Risk Assessment: Professor Eugene McCloskey

10.15 – 10.45 Refreshments in the Exchange Hall

10.45 – 11.00 Official Opening: Professor Nancy Rothwell, University of Manchester

Location: Exchange Auditorium

Introduction by: Professor Terry O’Neill and Professor Jon Tobias

11.00 – 12.30 Educational Update

Location: Exchange Auditorium

Session Chairs: Dr Mike Stone and Debbie Stone

Bone Research Society Symposium on Extracellular Signalling

Location: Exchange 9

Session Chairs: Dr Alison Gartland and Dr Deborah Mason
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<tr>
<th>Time</th>
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<tr>
<td>11.00</td>
<td>EU4 Non-Pharmacological Interventions: Tash Masud</td>
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<td>11.30</td>
<td>EU5 Pharmacological Therapy for Osteoporosis: David Hosking</td>
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<td>12.00</td>
<td>EU6 Prevention and Management of Falls: Dawn Skelton</td>
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<td>12.30</td>
<td>Poster Viewing Session A and Lunch</td>
<td>Exchange Hall</td>
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<td>14.00</td>
<td>Plenary Invited Speaker</td>
<td>Exchange Auditorium</td>
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<tr>
<td>14.30</td>
<td>Please make your way to workshop sessions</td>
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<tr>
<td>14.40</td>
<td>Parallel Workshops</td>
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<tr>
<td>15.00</td>
<td>Refreshments in the Exchange Hall</td>
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<tr>
<td>16.00</td>
<td>IS2 Paget’s Disease Symposium</td>
<td>Exchange Auditorium</td>
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<tr>
<td>17.15</td>
<td>Pre-satellite refreshments sponsored by Amgen</td>
<td>Exchange Foyer</td>
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<tr>
<td>17.45</td>
<td>Satellite Session sponsored by Amgen</td>
<td>Exchange Auditorium</td>
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<td>19.15</td>
<td>End of Day 2</td>
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## Tuesday 3rd July 2012

### 8.30–9.00
**Parallel Session: Update from the National Osteoporosis Society:** Claire Severgnini and Professor Terry O’Neill  
**Location:** Exchange Auditorium  
**Session Chair:** Professor Roger Francis

**Parallel Session: Meet the Professor:** Professor Alan Boyde  
**Location:** Exchange 11  
**New Bone for Old Cartilage: The Bizarre Tale of the Elephant’s 6th Toe**

### 9.00–10.20
**Parallel Session: NOS Abstracts:** Presentations from Submitted Abstracts  
**Location:** Exchange Auditorium  
**Session Chair:** Dr Nicola Peel and Mark Wilkinson

**Parallel Session: BRS Abstracts:** Presentations from Submitted Abstracts  
**Location:** Exchange 9  
**Session Chair:** Dr Sophie Jamal and Dr Vicky Macrae

### 9.00
**O1 HABITUAL LEVELS OF HIGH, BUT NOT MODERATE OR LOW, IMPACT ACTIVITY ARE POSITIVELY RELATED TO HIP BONE MINERAL DENSITY AND STRENGTH: RESULTS FROM A POPULATION BASED STUDY OF ADOLESCENTS**  
Kevin Deere (1) Adrian Sayers (1) Joern Rittweger (2) Jon Tobias (1); University of Bristol, Bristol, UK (1) Institute of Aerospace Medicine, Cologne, Germany (2)

**O9 THE MEPE-ASARM AXIS REGULATES CHONDROCYTE MATRIX MINERALISATION**  
Katherine Staines (1) presenting; Neil Mackenzie (1) Matt Prideaux (4) Claire Clarkin (2) Lesya Zelenchuk (3) Lynda Bonevald (4) Peter Rowe (3) Vicky MacRae (1) Colin Farquharson (1); University of Southampton, The Roslin Institute and R(D)SVS, University of Edinburgh, Edinburgh, UK (1) University of Southhampton, Southhampton, UK (2) KUMC, Kansas City, USA (3) University of Missouri-Kansas City, Kansas City, USA (4)

### 9.10
**O2 BRIEF HIGH IMPACT EXERCISE IMPROVED FEMORAL NECK BONE MINERAL DENSITY AND HIP STRUCTURAL PARAMETERS IN OLDER MEN: A RANDOMISED UNILATERAL INTERVENTION**  
Sarah J Allison (1) Jonathan P Folland (1) Winston J Rennie (2) Gregory D Summers (3) Katherine Brooke-Wavell (1); Loughborough University, Leicestershire, UK (1) University Hospitals of Leicester, Leicester, UK (2) Royal Derby Hospital, Derby, UK (3)

**O10 INTRA-ARTICULAR AMPA/KAINATE GLUTAMATE RECEPTOR ANTAGONISTS ALLEVIATE INFLAMMATION, PAIN AND PATHOLOGY IN RAT ANTIGEN INDUCED ARTHRITIS**  
Cleo Bonnet presenting; Anwen Williams, Ann Harvey, Sophie Gilbert, Abdul Moideen, Mari Nowell, Deborah Mason; Cardiff University, Cardiff, UK

### 9.20
**O3 THE RISK OF FRACTURE IN INCIDENT MULTIPLE SCLEROSIS PATIENTS: THE DANISH NATIONAL HEALTH REGISTERS**  
Marloes Bazelier, Joan Bentzen, Peter Vestergaard, Egon Stenager, Frank de Vries; Utrecht University, Utrecht, The Netherlands (1) University of Southern Denmark, Copenhagen, Denmark (2) Aarhus University Hospital, Aarhus, Denmark (3) University of Southampton, Southhampton, UK (4)

**O11 NON-ENZYMATIC OMEGA-3-FATTY ACID BREAKDOWN PRODUCTS ARE DIFFERENTIALLY EXPRESSED IN RHEUMATOID ARTHRITIS AND SUPPRESS NFkB ACTIVITY TO INHIBIT OSTEOCLAST FORMATION**  
Saint Lwin (1) Joshua Brooks (1,2) Lynett Danks (1) Karen Lyndberg (1) Ginger Milne (1,2) Jason Morrow (1) James Edwards (1) presenting; University of Oxford, Oxford, UK (1) VUMC, Nashville, USA (2)

### 9.30
**O4 CAN FUNCTIONAL MUSCLE TESTING IMPROVE FRACTURE RISK ASSESSMENT IN AN AGEING FEMALE POPULATION?**  
Nicola Crabtree, Natalie Bebbington, Katie Stant, Helen Duffy, Jim Parle, Neil Gittoes; Queen Elizabeth Hospital, Birmingham, UK (1) Primary Care Clinical Sciences, University of Birmingham, Birmingham, UK (2)

**O12 DEATH RECEPTOR 3 AND TL1A: A REGULATORY PATHWAY OF BONE TURNOVER AND TARGET FOR OSTEOPOROSIS THERAPY?**  
Fraser Collins (1) presenting; Michael Stone (2) Edward Wang (1) Anwen Williams (1); Institute of Infection and Immunity, School of Medicine, Cardiff University, Cardiff, UK (1) Bone Research Unit, Cardiff University Academic Centre, University Hospital Llandough, Penarth, UK (2)

### 9.40
**O5 ACCRUAL OF LEAN MASS IN EARLY CHILDHOOD IS ASSOCIATED WITH HIP STRENGTH AND GEOMETRY AT SIX YEARS OLD: THE SOUTHAMPTON WOMEN’S SURVEY**  
Elizabeth Curtis, Zoe Cole, Sarah Crozier, Georgia Ntani, Sian Robinson, Keith Godfrey, Avan Sayer, Hazel Inskip, Cyrus Cooper, Nicholas Harvey; MRC Lifecourse Epidemiology Unit, University of Southampton, Southampton, UK (1) Southampton Nutrition Biomedical Research Unit, Southampton, UK (2)

**O13 VITAMIN D, BUT NOT PTH, MODULATES VEGF AND IL-6 SECRETION BY OSTEOCYTES IN VITRO**  
Madeleine Adams (1) presenting; Meriel Jenney (2) John Gregory (1) Bronwen Evans (1); Institute of Molecular and Experimental Medicine, Cardiff University, Cardiff, UK (1) Paediatric Oncology Department, Children’s Hospital for Wales, Cardiff, UK (2)
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<tr>
<th>Time</th>
<th>Session 1</th>
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<tr>
<td>9.50</td>
<td>06 THE EFFECT OF OBESITY ON BONE MINERAL DENSITY MEASURED AT FOUR FRACTURE SITES</td>
<td>014 HOST-DERIVED MMP-7 DECREASES MYELOMA PROGRESSION IN VIVO: AN UNEXPECTED ROLE FOR MMP-7 IN MYELOMA BONE DISEASE</td>
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<td>Amy Evans, Jennifer Walsh, Richard Eastell; University of Sheffield, Sheffield, UK (1) Sheffield Teaching Hospitals Foundation NHS Trust, Sheffield, UK (2)</td>
<td>Seint Lwin (1,2) Conor Lynch (2) Jessica Fowler (2) James Edwards (1,2) Claire Edwards (1,2) presenting; University of Oxford, Oxford, UK (1) Vanderbilt University, Nashville, TN, USA (2)</td>
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<td>10.00</td>
<td>07 WHAT ARE THE ASSOCIATIONS BETWEEN WAIST HIP RATIO, BODY MASS INDEX AND HIP FRACTURES? COHORT OF NORWAY</td>
<td>015 BMP2-ENOS AXIS REGULATES BONE VOLUME AND STATIN-INDUCED BONE FORMATION</td>
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<td>Anne Johanne Seggaard (1) Tone K Omland (1,2) Haakon E Meyer (1,3) Division of Epidemiology, Norwegian Institute of Public Health, Oslo, Norway (1) Department of Public Health and Primary Health Care, University of Bergen, Bergen, Norway (2) Institute of General Practice and Community Medicine, University of Oslo, Oslo, Norway (3) NOREPOS Core Research Group, Oslo, Norway (4)</td>
<td>Megan Moore-Weivoda (1,2) Seint Lwin (1) Ross Garrett (1,2) Jonathon Lowrey (1,2) Gregory Mundy (1,2) Gloria Gutierrez (1,2) James Edwards (1) presenting; University of Oxford, Oxford, UK (1) VUMC, Nashville, USA (2)</td>
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<td>10.10</td>
<td>08 RISK OF FRACTURE IN PATIENTS WITH BARIATRIC SURGERY AND MATCHED CONTROLS: A POPULATION-BASED COHORT STUDY IN THE UNITED KINGDOM</td>
<td>016 THE ROLE OF DUAL SPECIFICITY PHOSPHATASE-1 IN LIMITING INFLAMMATORY OSTEOLYSIS AND ITS ROLE IN COLLAGEN-INDUCED ARTHRITIS</td>
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<td>Arief Lalmohamed (1) presenting; Cyrus Cooper (2) Marloes Bazelier (1) Alun Cooper (2) Corinne Klopf (1) Frank de Vries (1) Nick Harvey (2); Utrecht University, Utrecht, The Netherlands (1) Southampton General Hospital, Southampton, UK (2)</td>
<td>Youridies Vattakuzhi, Andy Clark, Nikki Horwood; Kennedy Institute of Rheumatology, London, UK</td>
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<tr>
<td>11.00–11.30</td>
<td>Refreshments</td>
<td>Invited Speaker</td>
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<td>Location: Exchange Auditorium</td>
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<td>Session Chairs: Dr Nick Harvey and Dr Kate Ward</td>
<td>Session Chairs: Dr Celia Gregson and Dr Isabel Orriss</td>
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<td>Location: Exchange Auditorium</td>
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<td>11.30</td>
<td>017 POWER COMPARISON OF DIFFERENT SCREENING TOOLS (FRAX WITHOUT BMD, OST, ORAI, ORISIS, SCORE AND AGE ALONE) TO IDENTIFY WOMEN WITH INCREASED RISK OF FRACTURE. ARE COMPLEX TOOLS BETTER</td>
<td>023 RARE MUTATIONS ASSOCIATED WITH OSTEOCLAST-POOR OSTEOPETROSIS PROVIDE MOLECULAR INSIGHTS INTO RECEPTOR ACTIVATOR OF NFKB SIGNALLING</td>
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<td>Katrine Hass Rubin (1,2), Bo Abrahamsen (1,3), Teresa Friis-Holmberg (4), Jacob VB Hjelmborg (5), Mickael Bech (6), Anne Pernille Hermann (2), and Kim Brixen (1,2); Institute of Clinical Research, University of Southern Denmark (1) Department of Medical Endocrinology, Odense University Hospital, Odense, Denmark (2) Department of Medicine F, Copenhagen University Hospital Gentofte, Copenhagen, Denmark (3) National Institute of Public Health, University of Southern Denmark, Copenhagen, Denmark (4) Department of Biostatistics, Institute of Public Health, University of Southern Denmark, Odense, Denmark (5) Institute of Public Health, Health Economics, University of Southern Denmark, Odense, Denmark (6)</td>
<td>Subhajit Das, Ilnaz Sepahi, David Mellis, Angela Duthie, Miep Helfrich, Julie Crockett; University of Aberdeen, Aberdeen, UK</td>
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Tuesday 3rd July 2012

11.40  **O18 ANXIETY, FRACTURE RISK AND ADHERENCE TO TREATMENT: THE MRC SCOOP TRIAL**
Nicholas Harvey (1) Janet Cushnaghan (1) Wendy Lawrence (1) Georgia Ntani (1) Karen Collins (1) Liz Lenaghan (1) Lee Shepstone (2) Eugene McCluskey (3) John Kanis (4) Cyrus Cooper (1); MRC Lifecourse Epidemiology Unit, Southampton, UK (1) University of East Anglia, Norwich, UK (2) University of Sheffield, Sheffield, UK (3) WHO Collaborating Centre for Metabolic Bone Diseases, Sheffield, UK (4)

11.50  **O19 VERTEBRAL FRACTURE ASSESSMENT (VFA) IS MORE USEFUL IN POPULATION-BASED SETTINGS THAN AS PART OF FRACTURE LIAISON SERVICES (FLS)**
Emma Clark (1,2) presenting; Virginia Gould (1) Louise Carter (2) Leigh Morrison (1) Jon Tobias (1); University of Bristol, Bristol, UK (1) North Bristol NHS Trust, Bristol, UK (2)

12.00  **O20 VERTEBRAL FRACTURE ASSESSMENT BY THE GE LUNAR IDXATM VERSUS RADIOGRAPHIC ASSESSMENT IN CHILDREN**
Nicola Crabtree, Nick Shaw, Wolfgang Hogler, Natalie Bebbington, Dee Chapman, Steve Chapman, Birmingham Children's Hospital, Birmingham, UK

12.10  **O21 PLACENTAL SIZE AT 19 WEEKS PREDICTS NEONATAL BONE MASS: RESULTS FROM THE SOUTHAMPTON WOMEN’S SURVEY**
Christopher Holroyd (1) presenting; Sarah Crozier (1) Pamela Mahon (1) Nicola Winder (1) Keith Godfrey (1,2) Hazel Inskip (1) Nicholas Harvey (1) Cyrus Cooper (1,3); MRC Lifecourse Epidemiology Unit, Southampton, Hampshire, UK (1) Southampton NIHR Biomedical Research Unit in Nutrition, Diet and Lifestyle, Southampton, UK (2) NIHR Biomedical Research Unit in Musculoskeletal Sciences, Oxford, UK (3)

12.05  **O25 A GENOME-WIDE ASSOCIATION META-ANALYSIS AND A MOUSE GENE DELETION MODEL IDENTIFY WNT16 AS A POTENTIAL REGULATOR OF CORTICAL BONE THICKNESS**
Jonathan Tobias (1) presenting Class Ohlsson (2) Lavinia Paternoster (1) Terho Lehtimaki (3) Mika Kahonen (3) Olli Raitakari (4) Marika Laaksonen (5) Jorma Vilkki (4) Dan Mellstrom (2) Sofia Moverare-Skitric (2) Magnus Karlsson (6) Osten Ljunggren (1) John P Kemp (1) Maria Nethander (2) Liesbeth Vandenput (2) Robert Brommage (6) Jeff Liu (8) David M Evans (1) Mathias Lorentzon (7); University of Bristol, Bristol, UK (1) University of Gothenburg, Gothenburg, Sweden (2) University of Tampere, Tampere, Finland (3) University of Turku, Turku, Finland (4) University of Helsinki, Helsinki, Finland (5) Lund University, Malmo, Sweden (6) Uppsala University, Uppsala, Sweden (7) Lexicon Pharmaceuticals, Texas, USA (8)

12.10  **O28 THE ANTI-DIABETIC DRUG METFORMIN HAS NO DELETERIOUS EFFECT ON BONE MASS IN VIVO BUT DOES NOT INDUCE OSTEOGENESIS NOR FRACTURE HEALING**
Jeshmi Jeyabalan (1) presenting; Benoit Viollet (2) Peter Smitham (3) Yasin Undre (1) Stephanie Ann Ellis (1) Allen Goodship (3) Chantal Chen (1); Royal Veterinary College, London, UK (1) INSEIR M567, Paris, France (2) Institute of Orthopaedics & Musculoskeletal Science, Stanmore, UK (3)

12.15  **O29 HIGH CIRCULATING SEROTONIN IN CARCINOID SYNDROME IS NOT ASSOCIATED WITH ALTERATIONS IN BONE TURNOVER OR BONE DENSITY**
Jennifer Walsh (1) presenting; John Newell-Price (2) Miguel Debono (2) Richard Eastell (2); Sheffield Teaching Hospitals NHS Foundation Trust, Sheffield, UK (1) University of Sheffield, Sheffield, UK (2)
12.20  O22 LUMBAR SPINE AND HIP BONE MINERAL DENSITY ARE IMPORTANT RISK FACTORS FOR STRESS FRACTURE IN ROYAL MARINE RECRUITS
Trish Davey (1,2) presenting; Susan A. Lanham-New (2) Adrian. J. Allsopp (1) Pat Taylor (4)
Cyrus Cooper (3) Joanne L. Fallowfield (1); Institute of Naval Medicine, Gosport, UK (1)
University of Surrey, Guildford, UK (2) University of Southampton, Southampton, UK (3)
Southampton General Hospital, Southampton, UK (4)

12.20  O30 STRONTIUM DIRECTLY INHIBITS MINERAL DEPOSITION IN BONE-FORMING PRIMARY OSTEOBLAST CULTURES
Daniel Wornham, Mark Hajjawi, Timothy Arnett, University College London, London, UK

12.25  O31 ACCELERATION OF FRACTURE REPAIR WITH MUSCLE
James Chan, Lorraine Harry, Graeme Glass, Nikki Horwood, Jagdeep Nanchahal; The Kennedy Institute of Rheumatology, London, UK

12.30–14.00  Poster Viewing Session B and Lunch
Location: Exchange Hall
See separate poster programme within this section for details

14.00–15.00  Parallel Workshops
See additional workshop materials within this handbook for details of each session. Please refer to the separate workshop handout in your delegate pack for workshop rooms and refer to your conference badge for details of your pre-booked workshops.

T1 Bone Markers – Laboratory Issues and Clinical Value
T2 Vertebral Fracture Assessment
T3 Imaging the Musculoskeletal System: Beyond DXA
T4 Bone Biology – The Essentials
T5 Bone-Unfriendly Drugs
T6 Treatment Conundrums
T7 How Do We Really Manage Paget’s Disease?
T8 Novel In Vitro Cell Culture and Signalling Approaches

15.00–15.30  Refreshments

15.30–16.00  Plenary Invited Presentation
Location: Exchange Auditorium
Session Chairs: Professor Tim Arnett and Professor Richard Eastell

IS5 Sarcopenia: Causes and Consequences: Professor Avan Aihie Sayer

16.00–16.30  Joint Presentations from Submitted Abstracts
Location: Exchange Auditorium
Session Chairs: Professor Tim Arnett and Professor Richard Eastell

16.00  O32 EFFECTS OF AGE ON GENETIC INFLUENCE ON BONE DENSITY AND BONE LOSS OVER 17 YEARS IN WOMEN: A LONGITUDINAL TWIN STUDY
Alireza Moayyeri, Deborah Hart, Chris Hammond, Tim Spector; Department of Twin Research and Genetic Epidemiology, King’s College London, London, UK

16.10  O34 MUSCLE SIZE, STRENGTH AND PHYSICAL PERFORMANCE AS PREDICTORS OF BONE STRUCTURE IN THE HERTFORDSHIRE COHORT STUDY
Mark Edwards, Karen Jameson, Celia Gregson, Nicholas Harvey, Avan Aihie Sayer, Elaine Dennison, Cyrus Cooper; MRC Lifecourse Epidemiology Unit, Southampton, Hampshire, UK
**O36 META-ANALYSIS OF GENOME-WIDE SCANS FOR TOTAL BODY BMD REVEALS AN INTERACTION WITH WEIGHT BEARING AT THE WNT16 LOCUS**

John P Kemp (1,2) Carolina Medina-Gomez (3,4,5,6) Karol Estrada (3,5,6) Joel Eriksson (7) Jeff Liu (8) Sjur Reppe (9), David M Evans (1,2) Denise Heppe (4,5,10) Liesbeth Vandenput (7) Lizbeth Herrera (3) Susan M Ring (2) Claudia Kruthof (4,5) Nicholas J Timpson (1,2) M Carola Zillikens (3,6) Ole K Olstad (9) Hou-Feng Zheng (11,12) Brent Richards (11,12,13) Beate St Pourcain (1) Albert Hofman (4,5,6) Vincent W Jaddoe (4,5,10) George Davey Smith (1,2) Mattias Lorentzon (7) Kaare M Gautvik (9,14) André G. Uitterlinden (3,4,5,6) Robert Brommage (8) Claes Ohlsson (7) Jonathan H Tobias (15) Fernando Rivadeneira (3,4,5,6); MRC CAiTE Centre, School of Social and Community Medicine, University of Bristol, Bristol, UK (1) ALS PAC, School of Social & Community Medicine, University of Bristol, Bristol, UK (2) Department of Internal Medicine, Erasmus University Medical Center, Rotterdam, The Netherlands (3) Generation R Study Group, Erasmus University Medical Center, Rotterdam, The Netherlands (4) Department of Epidemiology, Erasmus University Medical Center, Rotterdam, The Netherlands (5) Netherlands Genomics Initiative (NGI)-sponsored Netherlands Consortium for Healthy Aging (NCHA), The Netherlands (6) Center for Bone and Arthritis Research, Institute of Medicine, Sahlgrenska Academy, University of Gothenburg, Sweden (7) Lexicon Pharmaceuticals, The Woodlands, Texas, USA (8) Department of Medical Biochemistry, Oslo University Hospital, Oslo, Norway (9) Department of Pediatrics, Erasmus University Medical Center, Rotterdam, The Netherlands (10) Department of Medicine, Human Genetics, McGill University, Montreal, Quebec, Canada (11) Department of Epidemiology and Biostatistics, Lady Davis Institute for Medical Research, Jewish General Hospital, Montreal, Quebec, Canada (12) Twin Research and Genetic Epidemiology, King’s College London, London, UK (13) Department of Medical Biochemistry, Oslo Deacon Hospital, Oslo, Norway (14) School of Clinical Sciences, University of Bristol, Bristol, UK (15)

**O33 THE RESPONSE OF CORTICAL BONE TO HIGH IMPACT ACTIVITY IS ATTENUATED IN GIRLS: FINDINGS FROM A CROSS-SECTIONAL PQCT STUDY IN ADOLESCENTS**

Kevin Deere (1) presenting; Adrian Sayers (1) Joern Rittweger (2) Jon Tobias (1); University of Bristol, Bristol, UK (1) Institute of Aerospace Medicine, Cologne, Germany (2)

**O35 HIGH BONE MASS IS ASSOCIATED WITH A GREATER PREVALENCE OF HIP BUT NOT KNEE REPLACEMENT**

Sarah A Hardcastle (1) presenting; Celia L Gregson (1,2) Kevin Deere (1) George Davey-Smith (3) Jon H Tobias (1); MRC CAiTE Centre, University Musculoskeletal Research Unit, University of Bristol, Bristol, UK (1) MRC Lifecourse Epidemiology Unit, University of Southampton, Southampton, Southhampton, UK (2) MRC CAiTE Centre, University of Bristol, Bristol, UK (3)

**O37 CIRCULATING SCLEROSTIN AND DICKKOPF-1 IN PRE-DIALYSIS CHRONIC KIDNEY DISEASE: RELATIONSHIP WITH BONE DENSITY AND ARTERIAL STIFFNESS**

Rakshita Roplekar, Subashini Thambiah, Padmini Manghat, Ignac Fogelman, William Fraser, David Goldsmith, Geeta Hampson; Renal Department of Clinical Chemistry, Guy’s and St Thomas’ Hospital, London, UK (1) Osteoporosis Clinic, Guy’s Hospital, London, UK (2) Renal Unit, Guy’s Hospital, London, UK (3) Nuclear Medicine, Guy’s Hospital, Kings College London, London, UK (4) Norwich Medical School, University of East Anglia, Norwich, UK (5) Department of Pathology, University Putra, Selangor, Malaysia (6)

Pre-conference session refreshments

Location: Exchange Foyer

Conference Session: Putting Patients First

Location: Exchange Auditorium

Session Chair: Professor Cyrus Cooper

**Osteoporosis - The Patient’s Perspective:** Professor Patrick Amman

**Osteoporosis and Fractures - The Gender Divide:** Professor Roger Francis

Commissioning Services for Osteoporosis: Julian Given

End of Day 3

Confence Dinner

At the Midland Hotel. Details can be found on the ticket in your badge holder.
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<th>Session Chairs</th>
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<tr>
<td>8.00–9.00</td>
<td><strong>Conference Session Sponsored by Arthritis Research UK</strong>&lt;br&gt;<strong>Clinical Trial Opportunities in Metabolic Bone Diseases</strong>&lt;br&gt;<strong>Location:</strong> Exchange Auditorium</td>
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<td><strong>Programme</strong></td>
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<td>08.00</td>
<td><strong>Introduction and Welcome</strong>&lt;br&gt;David M Reid, Chair Arthritis Research UK Clinical Studies Group for Metabolic Bone Diseases</td>
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<td>08.05</td>
<td><strong>Trial Design – Effects of Bisphosphonates on Colles Fracture Healing and Outcome</strong>&lt;br&gt;Jon Tobias, Professor of Rheumatology, University of Bristol</td>
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<td>08.15</td>
<td><strong>Trial Design – Vitamin D in Older People (VDOP): A Randomised Controlled Trial of Vitamin D Supplementation</strong>&lt;br&gt;Terry Aspray, consultant physician &amp; hon. Senior Lecturer, Freeman Hospital, Newcastle</td>
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<td>08.25</td>
<td><strong>Trial Planning – How Can We Examine the Value of Monitoring Therapy in Osteoporosis</strong>&lt;br&gt;Richard Eastell, Professor of Bone Metabolism, University of Sheffield</td>
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<tr>
<td>08.35</td>
<td><strong>Trial Planning – How Can We Design a Trial to Examine the Value of Fracture Liaison Services</strong>&lt;br&gt;Kassim Javaid, Senior Lecturer in Rheumatology, University of Oxford</td>
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<td>08.45</td>
<td><strong>Trial Funding – How to Have a Bone Disease Trial Funded by Arthritis Research UK</strong>&lt;br&gt;David M Reid, Chair Arthritis Research UK Clinical Studies Group for Metabolic Bone Diseases</td>
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<td>08.55</td>
<td><strong>Open Questions</strong></td>
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<td>9.00–9.30</td>
<td><strong>Plenary Invited Speaker</strong>&lt;br&gt;<strong>Location:</strong> Exchange Auditorium</td>
<td>Dr Helen Macdonald and Professor Roger Francis</td>
<td><strong>IS6 Calcium and Vitamin D – Kill or Cure:</strong> Professor Bo Abrahamsen</td>
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<td>9.30–11.00</td>
<td><strong>Parallel Session: NOS Abstract:</strong>&lt;br&gt;<strong>Presentations from Submitted Abstracts</strong>&lt;br&gt;<strong>Location:</strong> Exchange Auditorium</td>
<td>Dr Helen Macdonald and Professor Roger Francis</td>
<td><strong>Parallel Session: BRS Abstract:</strong>&lt;br&gt;<strong>Presentations from Submitted Abstracts</strong>&lt;br&gt;<strong>Location:</strong> Exchange 11&lt;br&gt;<strong>Session Chairs:</strong> Dr Sanjeev Patel and Nigel Loveridge</td>
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<td>9.30</td>
<td><strong>O38 MATERNAL VITAMIN D LEVELS IN PREGNANCY AND OFFSPRING BONE MASS AT AGE 9: FINDINGS FROM A UK PROSPECTIVE BIRTH COHORT STUDY</strong>&lt;br&gt;Andrew Wills (1) presenting; Adrian Sayers (1) William Fraser (2); Jon Tobias (1); Debbie Lawlor (1); University of Bristol, Bristol, UK (1); University of East Anglia, Norwich, UK (2)</td>
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<td><strong>O47 INVESTIGATING NOVEL REGULATORS AND INHIBITORS OF AORTIC VALVE CALCIFICATION</strong>&lt;br&gt;Neil Mackenzie (1) presenting; Dongxing Zhu (1); Daniel Lerman (1,2); Mark Dweck (2); David Newby (2); Vicky Macrae (1); Roslin Institute, Edinburgh, UK (1); Centre for Cardiovascular Science, Edinburgh, UK (2)</td>
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<td>9.40</td>
<td><strong>O39 VITAMIN D LEVELS IN HIP FRACTURES: RATIONALE AND GUIDELINES FOR RAPID SUBSTITUTION THERAPY</strong>&lt;br&gt;Andy de Jong, Matthew Porteous; West Suffolk Hospital, Bury St. Edmunds, UK</td>
<td></td>
<td><strong>O48 RAB27 KNOCKOUT MICE HAVE LOW BONE DENSITY DUE TO DEFECTIVE OSTEObLAST FUNCTION</strong>&lt;br&gt;Fraser Coxon (1) presenting; Angela Douglass (1); Alun Hughes (1); Miguel Seabra (2); Tanya Tilmachova (2); University of Aberdeen, Aberdeen, UK (1); Imperial College, London, UK (2)</td>
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<td>09.50</td>
<td><strong>O40</strong> ACTIVE VITAMIN D (1,25-DIHYDROXYVITAMIN D) AND BONE HEALTH IN MIDDLE AGED AND OLDER MEN</td>
<td>Stephen Pye (1) presenting; Dirk Vanderschueren (2) Terence O'Neill (1) David Lee (1) Ivo Jans (2) Jaak Billen (2) Evelien Gielen (2) Frank Claessens (2) Judith Adams (3) Kate Ward (4) Gyorgy Bartfai (5) Felipe Casanueva (6) Joseph Finn (1) Gianni Forti (7) Aleksander Giwercman (8) Ilpo Huhtaniemi (9) Krzysztof Kula (10) Margus Punab (11) Frederick Wu (1) Steven Boonen (2); University of Manchester, Manchester, UK (1) Katholieke Universiteit Leuven, Leuven, Belgium (2) Radiology and Manchester Academic Health Science Centre in the Royal Infirmary, Manchester, UK (3) MRC Human Nutrition Research, Cambridge, UK (4) Albert Szent-Gyorgy Medical University, Szeged, Hungary (5) Santiago de Compostela University, Santiago de Compostela, Spain (6) University of Florence, Florence, Italy (7) University of Lund, Malmo, Sweden (8) Imperial College London, London, UK (9) Medical University of Lodz, Lodz, Poland (10) United Laboratories of Tartu University Clinics, Tartu, Estonia (11)</td>
<td>Stephen Pye (1) presenting; Dirk Vanderschueren (2) Terence O'Neill (1) David Lee (1) Ivo Jans (2) Jaak Billen (2) Evelien Gielen (2) Frank Claessens (2) Judith Adams (3) Kate Ward (4) Gyorgy Bartfai (5) Felipe Casanueva (6) Joseph Finn (1) Gianni Forti (7) Aleksander Giwercman (8) Ilpo Huhtaniemi (9) Krzysztof Kula (10) Margus Punab (11) Frederick Wu (1) Steven Boonen (2); University of Manchester, Manchester, UK (1) Katholieke Universiteit Leuven, Leuven, Belgium (2) Radiology and Manchester Academic Health Science Centre in the Royal Infirmary, Manchester, UK (3) MRC Human Nutrition Research, Cambridge, UK (4) Albert Szent-Gyorgy Medical University, Szeged, Hungary (5) Santiago de Compostela University, Santiago de Compostela, Spain (6) University of Florence, Florence, Italy (7) University of Lund, Malmo, Sweden (8) Imperial College London, London, UK (9) Medical University of Lodz, Lodz, Poland (10) United Laboratories of Tartu University Clinics, Tartu, Estonia (11)</td>
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<td>10.00</td>
<td><strong>O41</strong> ESTABLISHING REFERENCE INTERVALS FOR BONE TURNOVER MARKERS IN HEALTHY POST MENOPAUSAL WOMEN</td>
<td>Fatma Gossiel (1) presenting; Judith Finigan (1) Richard Jacques (1) David Reid (2) Dieter Felsenberg (3) Christian Roux (4) Claus Glueer (5) Richard Eastell (1); University of Sheffield, Sheffield, UK (1) University of Aberdeen, Aberdeen, UK (2) Charite Universitätsmedizin, Berlin, Germany (3) Rene Descartes Universite, Paris, France (4) Universitaetsklinikum Schleswig-Holstein, Kiel, Germany (5)</td>
<td>Fatma Gossiel (1) presenting; Judith Finigan (1) Richard Jacques (1) David Reid (2) Dieter Felsenberg (3) Christian Roux (4) Claus Glueer (5) Richard Eastell (1); University of Sheffield, Sheffield, UK (1) University of Aberdeen, Aberdeen, UK (2) Charite Universitätsmedizin, Berlin, Germany (3) Rene Descartes Universite, Paris, France (4) Universitaetsklinikum Schleswig-Holstein, Kiel, Germany (5)</td>
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<td>10.10</td>
<td><strong>O42</strong> ETHNICITY AND BONE PHENOTYPE IN UK MEN</td>
<td>Kate Ward (1,2) presenting; Judith Adams (2,3) Stephen Pye (2) Joseph Finn (2) Frederick Wu (2) Terence O’Neill (2); MRC Human Nutrition Research, Cambridge, UK (1) University of Manchester, Manchester, UK (2) The Royal Infirmary, Manchester, UK (3)</td>
<td>Kate Ward (1,2) presenting; Judith Adams (2,3) Stephen Pye (2) Joseph Finn (2) Frederick Wu (2) Terence O’Neill (2); MRC Human Nutrition Research, Cambridge, UK (1) University of Manchester, Manchester, UK (2) The Royal Infirmary, Manchester, UK (3)</td>
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<td>10.20</td>
<td><strong>O43</strong> DIFFERENTIAL EFFECTS OF TERIPARATIDE ON TRABECULAR AND CORTICAL BONE MEASURED BY 18 F-FLUORIDE POSITRON EMISSION TOMOGRAPHY</td>
<td>Michelle Frost (1) presenting; Musib Siddique (1) Glen Blake (1) Amelia Moore (1) Paul Schleyer (1) Paul Marsden (1) Richard Eastell (2) Ignac Fogelman (1); King’s College London, London, UK (1) University of Sheffield, Sheffield, UK (2)</td>
<td>Michelle Frost (1) presenting; Musib Siddique (1) Glen Blake (1) Amelia Moore (1) Paul Schleyer (1) Paul Marsden (1) Richard Eastell (2) Ignac Fogelman (1); King’s College London, London, UK (1) University of Sheffield, Sheffield, UK (2)</td>
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<td><strong>O49</strong> TNF-α: A THERAPEUTIC TARGET IN BONE REPAIR</td>
<td>James Chan presenting; Graeme Glass, Nikki Horwood, Jagdeep Nanchahal; Kennedy Institute of Rheumatology, London, UK</td>
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<td><strong>O50</strong> IGF-2 PROMOTES BMP-9-INDUCED VASCULAR CALCIFICATION</td>
<td>Dongxing Zhu (1) presenting; Neil Mackenzie (1) Jose Luis Millan (2) Colin Farquharson (1) Vicky Macrae (1); Roslin Institute, Edinburgh, UK (1) Sanford-Burnham Research Institute, California, USA (2)</td>
<td>Dongxing Zhu (1) presenting; Neil Mackenzie (1) Jose Luis Millan (2) Colin Farquharson (1) Vicky Macrae (1); Roslin Institute, Edinburgh, UK (1) Sanford-Burnham Research Institute, California, USA (2)</td>
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<td><strong>O51</strong> EXPRESSION OF 11ß-HYDROXYSTEROID DEHYDROGENASE ENZYMES IN HUMAN OSTEOSARCOMA: POTENTIAL ROLE IN PATHOGENESIS AND AS TARGETS FOR TREATMENTS</td>
<td>Pushpa Patel (1) presenting; Rowan Hardy (1) Sumathi Vaiyapuri (2) Paul Stewart (1) Elizabeth Rabbitt (1) Neil Gittoes (1) Mark Cooper (1); University of Birmingham, Birmingham, UK (1) Royal Orthopaedic Hospital, Birmingham, UK (2)</td>
<td>Pushpa Patel (1) presenting; Rowan Hardy (1) Sumathi Vaiyapuri (2) Paul Stewart (1) Elizabeth Rabbitt (1) Neil Gittoes (1) Mark Cooper (1); University of Birmingham, Birmingham, UK (1) Royal Orthopaedic Hospital, Birmingham, UK (2)</td>
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<td><strong>O52</strong> AGE-RELATED STRUCTURAL CHANGES IN THE INTERTROCHANTER AND TROCHANTER OF THE HIP</td>
<td>Benjamin Gabcott (1) presenting; Ken Poole (2) Richard Eastell (1) Lang Yang (1); Academic Unit of Bone Metabolism, Sheffield, UK (1) Dept. of Medicine and Dept of Radiology, Cambridge, UK (2)</td>
<td>Benjamin Gabcott (1) presenting; Ken Poole (2) Richard Eastell (1) Lang Yang (1); Academic Unit of Bone Metabolism, Sheffield, UK (1) Dept. of Medicine and Dept of Radiology, Cambridge, UK (2)</td>
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</table>
### 044 Hip Fracture Prediction Using Active Shape (ASM) and Appearance (AAM) Modelling

S Goodyear (1) R Barr (1) R Aspden (1) D Reid (1) I Reid (2) J Gregory (1); University of Aberdeen, Aberdeen, UK (1) University of Auckland, Auckland, New Zealand (2)

### 045 Epidemiology of Atypical Femoral Fractures in the Bristol Population

Cecilia Mercieca (1,2) presenting; Virginia Gould (1) Jon Tobias (1,2) Ashley Blom (1,2) Emma Clark (1,2); Academic Rheumatology; Musculoskeletal Research Unit, University of Bristol, Bristol, UK (1) North Bristol NHS Trust, Bristol, UK (2)

### 046 Outcome Following Hip Fracture: Long Term Mortality and Post-Discharge Residence

Antony Johansen, Maizura Mansor, Sue Beck, Heather Mahoney, Suzanne Thomas; University Hospital of Wales, Cardiff, UK

### 10.30 Annual General Meeting of the Bone Research Society

**Location:** Exchange 11

**Reports from:**
- The Secretary (Professor Eugene McCloskey)
- The Treasurer (Nigel Loveridge)
- The President (Professor Jon Tobias)

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### 11.00–11.30 Refreshments

### 11.30–12.00 Linda Edwards Memorial Lecture

**Location:** Exchange Auditorium

**Session Chair:** Professor Richard Eastell

**IS7 Legends of Osteoporosis:** Bone: A Target for Systemic Diseases Professor Juliet Compston

### 12.00–13.00 Award Presentations: National Osteoporosis Society & Bone Research Society

**Location:** Exchange Auditorium

**Session Chairs:** Professor Terry O’Neill and Professor Jon Tobias

12.00 **National Osteoporosis Society Young Scientist Award Lecture**

12.15 **Award Presentations**

**National Osteoporosis Society**
- Young Investigator Award
- Allied Health Professional Award
- Premiere Poster Award
- Hilary Noakes Award

**Bone Research Society**
- New Investigator Award
- BRS prizes for best poster abstract
- BRS prizes for best oral abstract

12.50 **Closing Remarks**

Professor Terry O’Neill, Conference Chair

13.00 **End of Conference** (no lunch provided)
All posters will be displayed in the Exhibition Hall and will remain on display for the whole conference. Posters will then be presented in two separate sessions.

**Poster Viewing Session A** will take place from 12.30 to 14.00 on Monday 2nd July 2012.

**Poster Viewing Session B** will take place from 12.30 to 14.00 on Tuesday 3rd July 2012.

Please refer to your supplement of *Osteoporosis International* for related poster abstracts.

### Premier Posters – Award Candidates

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<td>Nicholas Harvey</td>
<td>GROWTH IN UTERO AND EARLY CHILDHOOD PREDICTS HIP GEOMETRY AT SIX YEARS OLD</td>
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<td>Nicholas Harvey (1), Zoe Cole (1) Sarah Crozier (1) Georgia Ntani (1) Pam Mahon (1) Sian Robinson (1) Hazel Inskip (1) Keith Godfrey (1,2) Elaine Dennison (1) Cyrus Cooper (1); MRC Lifecourse Epidemiology Unit, Southampton, UK (1) NIHR Biomedical Research Unit in Diet, Lifestyle and Nutrition, Southampton, UK (2)</td>
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<td>P2</td>
<td>A</td>
<td>William WK To</td>
<td>COMPARISON OF QUANTITATIVE ULTRASOUND OF THE OS CALCIS AND STANDARD RADIOLOGICAL METHODS FOR MONITORING BONE MINERAL DENSITY CHANGES 18 MONTHS POSTPARTUM</td>
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<td>William WK To (1) Margaret WN Wong (2); United Christian Hospital, Hong Kong, Hong Kong (1) Prince of Wales Hospital, Hong Kong, Hong Kong (2)</td>
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<td>P3</td>
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<td>COMPARISON OF PATIENT POPULATIONS IDENTIFIED USING DIFFERENT FRAX THRESHOLDS: APPLICATION OF THE 20% MAJOR AND 5% HIP THRESHOLD IMPROVES TARGETING OF TREATMENT TO THOSE AT HIGH RISK</td>
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<td>Louise Carter (1), Jon Tobias (2,1); North Bristol NHS Trust, Bristol, UK (1) Academic Rheumatology, University of Bristol, Bristol, UK (2)</td>
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<td>P4</td>
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<td>Emma Clark</td>
<td>NHS RADIOGRAPH REPORTS OF VERTEBRAL FRACTURE (VF) COMPARE FAVOURABLY WITH STANDARDISED METHODS, BUT SOME NHS REPORTS ARE UNCLEAR</td>
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<td>Emma Clark, Virginia Gould, Leigh Morrison, Jon Tobias; University of Bristol, Bristol, UK</td>
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<td>Matthew Hamill</td>
<td>BONE AND BODY COMPOSITION IN HIV-POSITIVE AND HIV-NEGATIVE BLACK, SOUTH AFRICAN WOMEN</td>
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<td>Matthew Hamill (1,2) Kate Ward (1) John Pettifor (3) Shane Norris (2) Ann Prentice (1,4); MRC Human Nutrition Research, Cambridge, UK (1) Developmental Pathways for Health Research Unit, Johannesburg, South Africa (2) Department of Paediatrics, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa (3) MRC Keneba, Keneba, Gambia (4)</td>
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<td>P6</td>
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<td>Sylvia Edwards</td>
<td>THE RELATIONSHIP BETWEEN AORTIC STIFFNESS AND ABDOMINAL AORTIC CALCIFICATION MEASURED ON SPINE DXA VFA SCANS IN POSTMENOPAUSAL WOMEN</td>
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<td>Sylvia Edwards, Ignac Fogelman, Amelia Moore, Michelle Frost; King's College London School of Medicine, London, UK</td>
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<td>P7</td>
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<td>Stephen Pye</td>
<td>QUANTITATIVE HEEL ULTRASOUND PARAMETERS AND MORTALITY IN EUROPEAN MEN</td>
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<td>Stephen Pye (1) Dirk Vanderschueren (2) Steven Boonen (2) Evelien Gielen (2) Judith Adams (3) Kate Ward (4) David Lee (1) Gyorgy Bartfai (5) Felipe Casanueva (6) Joseph Finn (1) Gianni Forti (7) Aleksander Giwercman (8) Iipo Huhtaniemi (9) Krzysztof Kula (10) Margus Punab (11) Frederick Wu (1) Terence O'Neill (1); University of Manchester, Manchester, UK (1) Katholieke Universiteit Leuven, Leuven, Belgium (2) Radiology and Manchester Academic Health Science Centre in The Royal Infirmary, Manchester, UK (3) MRC Human Nutrition Research, Cambridge, UK (4) Albert Szent-Gyorgy Medical University, Szeged, Hungary (5) Santiago de Compostela University, Santiago de Compostela, Spain (6) University of Florence, Florence, Italy (7) University of Lund, Malmo, Sweden (8) Imperial College London, London, UK (9) Medical University of Lodz, Lodz, Poland (10) United Laboratories of Tartu University Clinics, Tartu, Estonia (11)</td>
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<td>Jennifer Thain</td>
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<td>Susan Hopkins (1) Chris Smith (1) Andrew Toms (2) Mary Brown (2) Joanne Welsman (3) Karen Knapp (1); College of Engineering Mathematics and Physical Sciences, University of Exeter, Exeter, UK (1) Princess Elizabeth Orthopaedic Centre, Royal Devon and Exeter Hospital, Exeter, UK (2) College of Life and Environmental Sciences, University of Exeter, Exeter, UK (3)</td>
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<td>Mette Juel Rothmann (1,2) Jette Amentorop (2) Mickael Bech (3), Kim Brixen (4) Anne Pernille Hermann (1,2); Department of Endocrinology, Odense University Hospital, Odense, Denmark (1) Institute of Regional Health Services Research, University of Southern Odense, Odense, Denmark (2) Research Unit of Health Economics, University of Southern Denmark, Odense, Denmark (3) Institute of Clinical Research, University of Southern Denmark, Odense, Denmark (4)</td>
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<td>P11 PP</td>
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<td>Alison Doyle</td>
<td>PREVENTING FALLS AND FRACTURES IN NURSING HOMES: A PILOT PROJECT</td>
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<td>Alison Doyle, Jane Beach, Neil Gittoes, Mark Cooper, Bola Ogunremi; Birmingham University, West Midlands, UK (1)</td>
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<td>DO HEAVY METALS IN MUNICIPAL DRINKING WATER INCREASE THE RISK OF HIP FRACTURES? THE NORWEGIAN EPIDEMIOLOGIC OSTEOPOROSIS STUDY (NOREPOS)</td>
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<td>Cecilie Dahl (1,2) Anne Johanne Søgaard (1) Grethe Tell (2) Geir Aamodt (1); Division of Epidemiology, Norwegian Institute of Public Health, Oslo, Norway (1) Department of Public Health and Primary Health Care, University of Bergen, Bergen, Norway (2)</td>
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<td>Judith Adams (1,2) Zulf Mughal (1,2) Steve Roberts (4) Kate Ward (3,2); Central Manchester University Hospitals NHS Foundation Trust, Manchester, UK (1) Manchester Academic Health Science Centre, University of Manchester, Manchester, UK (2) MRC Human Nutrition Research, Cambridge, UK (3) Health Sciences Methodology, University of Manchester, Manchester, UK (4)</td>
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<td>Natalie Bebbington</td>
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<td>Natalie Bebbington (1,2), Dee Chapman (2,1) Nick Shaw (2) Wolfgang Högl (2) Chris Bivin (1) Nicola Crabtree (2,1); Queen Elizabeth Hospital Birmingham, Birmingham, UK (1) Birmingham Children's Hospital, Birmingham, UK (2)</td>
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<td>Mark Edwards</td>
<td>MUSCLE SIZE, STRENGTH AND PHYSICAL PERFORMANCE AS PREDICTORS OF FALLS AND FRACTURES IN THE HERTFORDSHIRE COHORT STUDY</td>
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<td>Mark Edwards, Karen Jameson, Celia Gregson, Nicholas Harvey, Avan Aihie Sayer, Elaine Dennison, Cyrus Cooper; MRC Lifecourse Epidemiology Unit, Southampton, Hampshire, UK</td>
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<td>P16</td>
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<td>Evelien Gielen</td>
<td>SARCOPENIA AND BONE MINERAL DENSITY IN EUROPEAN MEN Sabine Verschueren (1) Evelien Gielen (1) Terence O’Neill (2) Stephen Pye (2) Judith Adams (3) Kate Ward (4) Frederick Wu (2) Pawel Szulc (5) Michaël Laurent (1) Frank Claessens (1) Dirk Vanderschueren (1) Steven Boonen (1); KU Leuven, Leuven, Belgium (1) University of Manchester, Manchester, UK (2) Radiology and Manchester Academic Health Science Centre in the Royal Infirmary, Manchester, UK (3) MRC Human Nutrition Research, Cambridge, UK (4) University of Lyon, Lyon, France (5)</td>
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<td>Marloes Bazelier</td>
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<td>Trish Davey</td>
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<td>PATIENTS ON STEROIDS – ARE THEIR BONES PROTECTED? DGH AUDIT</td>
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<td>Venkiah Kavuri, Anthony Egboh, Thushani Wickramaratne; Epsom General Hospital, Epsom, Surrey, UK</td>
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<td>Abhaya Gupta</td>
<td>A NEW MODEL OF CONSULTANT LED FRACTURE LIAISON SERVICE</td>
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<td>Abhaya Gupta, Srinivas Chenna, Allison Lorch; Glangwili Hospital, Carmarthen UK</td>
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<td>Vladyslav Povoroznyuk, Nataliia Dzerovych, Alla Palamarchuk, Anna Musienko; Institute of Gerontology AMS Ukraine, Kyiv, Ukraine</td>
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<td>Vladyslav Povoroznyuk</td>
<td>COMBINATION OF QUANTITATIVE ULTRASOUND AND FRAX® IN EVALUATION OF STRUCTURAL-FUNCTIONAL STATE OF BONE IN POSTMENOPAUSAL WOMEN</td>
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<td>Vladyslav Povoroznyuk, Nataliia Grygorieva, Vasy Povorozniuk; Institute of Gerontology AMS Ukraine, Kyiv, Ukraine</td>
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<td>Sonia Panchal</td>
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<td>Sonia Panchal, Yuthandar Aung, Peter Sheldon; University Hospitals of Leicester, Leicester, UK</td>
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<td>Monica Clarke</td>
<td>EFFECTIVENESS OF FRACTURE LIAISON SERVICES IN MIDDLESBROUGH</td>
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<td>Monica Clarke (1) Henry Waters (1) Stephen Tuck (1); Middlesbrough Primary Care Trust, Middlesbrough, UK (1) James Cook University Hospital, Middlesbrough, UK (2)</td>
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<td>P132</td>
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<td>Rebecca Sanders</td>
<td>INVESTIGATING LEVELS OF PRECISION IN GENE EXPRESSION MEASUREMENT BY DIGITAL PCR</td>
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<td>Rebecca Sanders, Carole Foy, Deborah Mason, Jim Huggett LGC, Teddington, UK (1) Cardiff University, Cardiff, UK (2)</td>
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<td>Veer Bahadur Singh</td>
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<td>Veer Bahadur Singh, Kusum Singh, Babu Lal Meena, Vijay Tundwal; SP Medical College, Bikaner, India</td>
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<td>Philipp Thurner</td>
<td>COMPOSITION AND STRUCTURAL HIERARCHY IN BONE - DETERMINANTS OF MECHANICAL COMPETENCE</td>
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<td>Philipp Thurner, Orestis Katsamenis, Orestis Andriotis; University of Southampton, Southampton, UK</td>
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<td>Tadeusz Jones</td>
<td>ORCHID – BONE: A NEW TOOL FOR BONE HEALTH CARE AND RESEARCH</td>
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<td>Tadeusz Jones (1) John Chelsom (2) Ian Gaywood (1) Ira Pande (1) presenting; Rheumatology Department, Queen’s Medical Centre, Nottingham University Hospitals, Nottingham, UK (1) Centre for Health Informatics, City University, London, UK (2)</td>
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Held every four years, the 8th World Congress on Active Ageing is to be hosted in Scotland from the 13 - 17 August 2012 and will celebrate the diversity of ageing. The benefits and availability of physical activity and exercise opportunities for those entering old age, to those in the transitional phase and, in particular, the needs of the oldest and frailest population will be highlighted. This event will be of interest to those working in the biological, behavioural and social sciences as well as the fields of medicine, physical and recreational therapy, health, sport and exercise sciences, health education, leisure and recreation and the social and caring services. Key one day congress themes will focus on the prevention and self management of conditions associated with old age, such as:

- Cognitive functioning and dementia
- Neurological and musculoskeletal conditions
- Falls, fractures and bone health
- Cardiovascular and respiratory conditions

These themes will be complimented by a series of conference strands which will include:

- The impact of the built, natural environment and technology upon physical activity participation
- Training and instruction in exercise leadership and safe and effective programming
- Motivation to take up and adhere to physical activity and exercise
- Measurement of physical activity and exercise outcomes
- Active Ageing and health promotion

The Active Ageing ‘Experience Zone’ will ensure that older people can actively engage with Congress delegates.

If you have an exciting idea for a Congress symposium, particularly relating to these themes, strands and the mental health, well-being, quality of life and needs of the frailest, oldest old, please contact us now at programme@wcaa2012.com

For information and to register your interest visit www.wcaa2012.com

Book these dates in your diary:
August 2011
31 August 2011
31 January 2012
30 March 2012

Programme Announcement
Online Registration
and Call for Papers Opens
Call for Papers Deadline
Early Registration Deadline

For any enquiries relating to the Congress, please do not hesitate to contact us at info@wcaa2012.com

Co hosted by:
GCU Glasgow Caledonian University
BHF National Centre for Physical Activity + Health

Congress Alliance Partners:
NHS Health Scotland
The Scottish Government

Supporter:
HUMAN KINETICS
Young Investigator Awards

O1 HABITUAL LEVELS OF HIGH, BUT NOT MODERATE OR LOW, IMPACT ACTIVITY ARE POSITIVELY RELATED TO HIP BONE MINERAL DENSITY AND STRENGTH: RESULTS FROM A POPULATION-BASED STUDY OF ADOLESCENTS

Kevin Deere (1) presenting; Adrian Sayers (1) Joern Rittweger (2) Jon Tobias (1); University of Bristol, Bristol, UK (1) Institute of Aerospace Medicine, Cologne, Germany (2)

O2 BRIEF HIGH IMPACT EXERCISE IMPROVED FEMORAL NECK BONE MINERAL DENSITY AND HIP STRUCTURAL PARAMETERS IN OLDER MEN: A RANDOMISED UNILATERAL INTERVENTION

Sarah J Allison (1) presenting; Jonathan P Folland (1) Winston J Rennie (2) Gregory D Summers (3) Katherine Brooke-Wavell (1); Loughborough University, Leicestershire, UK (1) University Hospitals of Leicester, Leicester, UK (2) Royal Derby Hospital, Derby, UK (3)

O3 THE RISK OF FRACTURE IN INCIDENT MULTIPLE SCLEROSIS PATIENTS: THE DANISH NATIONAL HEALTH REGISTERS

Marloes Bazelier (1) presenting; Joan Bentzen (2) Peter Vestergaard (3) Egon Stenager (2) Frank de Vries (1,4); Utrecht University, Utrecht, The Netherlands (1) University of Southern Denmark, Copenhagen, Denmark (2) Aarhus University Hospital, Aarhus, Denmark (3) University of Southampton, Southampton, UK (4)

O5 ACCRUAL OF LEAN MASS IN EARLY CHILDHOOD IS ASSOCIATED WITH HIP STRENGTH AND GEOMETRY AT SIX YEARS OLD: THE SOUTHAMPTON WOMEN’S SURVEY

Elizabeth Curtis (1) presenting; Zoe Cole (1) Sarah Crozier (1) Georgia Ntari (1) Sian Robinson (1) Keith Godfrey (1,2) Avan Sayer (1) Hazel Inskip (1) Cyrus Cooper (1) Nicholas Harvey (1); MRC Lifecourse Epidemiology Unit, University of Southampton, Southampton, UK (1) Southampton Nutrition Biomedical Research Unit, Southampton, UK (2)

O6 THE EFFECT OF OBESITY ON BONE MINERAL DENSITY MEASURED AT FOUR FRACTURE SITES IN OBESITY

Amy Evans (1) presenting; Jennifer Walsh (2) Richard Eastell (1); University of Sheffield, Sheffield, UK (1) Sheffield Teaching Hospitals Foundation NHS Trust, Sheffield, UK (2)

O8 RISK OF FRACTURE IN PATIENTS WITH BARIATRIC SURGERY AND MATCHED CONTROLS: A POPULATION-BASED COHORT STUDY IN THE UNITED KINGDOM

Arief Lalmohamed (1) presenting; Cyrus Cooper (2) Marloes Bazelier (1) Alun Cooper (2) Corinne Klop (1) Frank de Vries (1) Nick Harvey (2); Utrecht University, Utrecht, The Netherlands (1) Southampton General Hospital, Southampton, UK (2)
P16 SARCOPENIA AND BONE MINERAL DENSITY IN EUROPEAN MEN
Sabine Verschueren (1) Evelien Gielen (1) presenting; Terence O’Neill (2) Stephen Pye (2) Judith Adams (3) Kate Ward (4) Frederick Wu (2) Pawel Szulc (5) Michæl Laurent (1) Frank Claessens (1) Dirk Vanderschueren (1) Steven Boonen (1); KU Leuven, Leuven, Belgium (1) University of Manchester, Manchester, UK (2) Radiology and Manchester Academic Health Science Centre in Manchester Royal Infirmary, Manchester, UK (3) MRC Human Nutrition Research, Cambridge, UK (4) University of Lyon, Lyon, France (5)

O21 PLACENTAL SIZE AT 19 WEEKS PREDICTS NEONATAL BONE MASS: RESULTS FROM THE SOUTHAMPTON WOMEN’S SURVEY
Christopher Holroyd (1) presenting; Sarah Crozier (1) Pamela Mahon (1) Nicola Winder (1) Keith Godfrey (1,2) Hazel Inskip (1) Nicholas Harvey (1) Cyrus Cooper (1,3); MRC Lifecourse Epidemiology Unit, Southampton, Hampshire, UK (1) Southampton NIHR Biomedical Research Unit in Nutrition, Diet and Lifestyle, Southampton, UK (2) NIHR Musculoskeletal Biomedical Research Unit, Institute of Musculoskeletal Sciences, Oxford, UK (3)

O32 EFFECTS OF AGE ON GENETIC INFLUENCE ON BONE DENSITY AND BONE LOSS OVER 17 YEARS IN WOMEN: A LONGITUDINAL TWIN STUDY
Alireza Moayyeri presenting; Deborah Hart, Chris Hammond, Tim Spector; Department of Twin Research and Genetic Epidemiology, King’s College London, London, UK

O34 MUSCLE SIZE, STRENGTH AND PHYSICAL PERFORMANCE AS PREDICTORS OF BONE STRUCTURE IN THE HERTFORDSHIRE COHORT STUDY
Mark Edwards presenting; Karen Jameson, Celia Gregson, Nicholas Harvey, Avan Aihie Sayer, Elaine Dennison, Cyrus Cooper; MRC Lifecourse Epidemiology Unit, Southampton, Hampshire, UK

O36 META-ANALYSIS OF GENOME-WIDE SCANS FOR TOTAL BODY BMD REVEALS AN INTERACTION WITH WEIGHT BEARING AT THE WNT16 LOCUS
John P Kemp (1,2) presenting; Carolina Medina-Gomez (3,4) Karol Estrad (5,6) Joel Eriksson (7) Sju Reppe (8) David M Evans (1,2) Denise Heppe (4,5) Liesbeth van den Put (7) Lizbeth Herrera (3) Susann M Ring (2) Nicholas J Timpson (1,2) George Davey-Smith (1,2) Mattias Lorentzon (7) Andre G Uitterlinden (3,4) Claes Ohlsson (7) Rivadeneira Fernando (3,4) Jonathan H Tobias (1,9); MRC CAiTE centre, School of Social and Community Medicine, University of Bristol, Bristol, UK (1) ALSPAC, School of Social & Community Medicine, University of Bristol, Bristol, UK (2) Department of Internal Medicine, Erasmus University Medical Center, Rotterdam, The Netherlands (3) Generation R Study Group, Erasmus University Medical Center, Rotterdam, The Netherlands (4) Department of Epidemiology, Erasmus University Medical Center, Rotterdam, The Netherlands (5) Netherlands Genomics Initiative (NGI)-sponsored Netherlands Consortium for Healthy Aging (NCHA), Rotterdam, The Netherlands (6) Center for Bone and Arthritis Research, Institute of Medicine, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden (7) Department of Medical Biochemistry, Oslo University Hospital, Oslo, Norway (8) School of Clinical Sciences, University of Bristol, Bristol, UK (9)

O39 VITAMIN D LEVELS IN HIP FRACTURES: RATIONALE AND GUIDELINES FOR RAPID SUBSTITUTION THERAPY
Andy de Jong presenting; Matthew Porteous; West Suffolk Hospital, Bury St. Edmunds, UK
041  ESTABLISHING REFERENCE INTERVALS FOR BONE TURNOVER MARKERS IN HEALTHY POSTMENOPAUSAL WOMEN
Fatma Gossiel (1) presenting; Judith Finigan (1) Richard Jacques (1) David Reid (2) Dieter Felsenberg (3) Christian Roux (4) Claus Glueer (5) Richard Eastell (1); University of Sheffield, Sheffield, UK (1) University of Aberdeen, Aberdeen, UK (2) Charite Universitatsmedizin, Berlin, Germany (3) Rene Descartes Universite, Paris, France (4) Universitaetsklinikum Schleswig-Holstein, Kiel, Germany (5)

044  HIP FRACTURE PREDICTION USING ACTIVE SHAPE (ASM) AND APPEARANCE (AAM) MODELLING
S Goodyear (1) presenting; R Barr (1) R Aspden (1) D Reid (1) I Reid (2) J Gregory (1); University of Aberdeen, Aberdeen, UK (1) University of Auckland, Auckland, New Zealand (2)

045  EPIDEMIOLOGY OF ATYPICAL FEMORAL FRACTURES IN THE BRISTOL POPULATION
Cecilia Mercieca (1,2) presenting; Virginia Gould (1) Jon Tobias (1,2) Ashley Blom (1,2) Emma Clark (1,2); Academic Rheumatology, Musculoskeletal Research Unit, University of Bristol, Bristol, UK (1) North Bristol NHS Trust, Bristol, UK (2)

017  POWER COMPARISON OF DIFFERENT SCREENING TOOLS (FRAX WITHOUT BMD, OST, ORAI, ORISIS, SCORE AND AGE ALONE) TO IDENTIFY WOMEN WITH INCREASED RISK OF FRACTURE. ARE COMPLEX TOOLS BETTER
Katrine Hass Rubin (1,4) presenting; Bo Abrahamsen (3,1) Teresa Fris-Holmberg (2) Mickael Bech (5) Anne Pernille Hermann (4) Kim Brixen (1,4); Institute of Clinical Research, University of Southern Denmark, Odense, Denmark (1) Institute of Public Health, University of Southern Denmark, Copenhagen, Denmark (2) Department of Medicine F, Copenhagen University Hospital Gentofte, Gentofte, Denmark (3) Department of Medical Endocrinology, Odense University Hospital, Odense, Denmark (4) Research Unit of Health Economics, University of Southern Denmark, Odense, Denmark (5)

04  CAN FUNCTIONAL MUSCLE TESTING IMPROVE FRACTURE RISK ASSESSMENT IN AN AGEING FEMALE POPULATION?
Nicola Crabtree (1) presenting; Natalie Bebbington (1) Katie Stant (2) Helen Duffy (2) Jim Parle (2) Neil Gittoes (1); Queen Elizabeth Hospital, Birmingham, UK (1) Primary Care Clinical Sciences, University of Birmingham, Birmingham, UK (2)

0132 LUMBAR SPINE AND HIP BONE MINERAL DENSITY ARE IMPORTANT RISK FACTORS FOR STRESS FRACTURE IN ROYAL MARINE RECRUITS
Trish Davey (1,2) presenting; Susan A Lanham-New (2) Adrian J Allsopp (1) Pat Taylor (4) Cyrus Cooper (3) Joanne L Fallowfield (1); Institute of Naval Medicine, Gosport, UK (1) University of Surrey, Guildford, UK (2) University of Southampton, Southampton, UK (3) Southampton General Hospital, Southampton, UK (4)
Invited Plenary Speakers and Educational Update Speakers

●●● Professor Bo Abrahamsen is Consultant Endocrinologist at Hospital Gentofte, Copenhagen, Professor of Clinical Database Studies at the University of Southern Denmark and President of the Danish Bone Society. He attended medical school at Odense University, Denmark, and the BSc honours course in experimental pathology at St Andrews, Scotland, qualifying as a physician in 1990. He did the clinical work for his PhD at Odense University Hospital and the laboratory work with Sandy C Marks Jr. at UMASS Medical School, Worcester, MA. Professor Bo Abrahamsen was appointed Consultant Endocrinologist in 2005 and Professor in 2009. His main research interests are absolute fracture risk, fracture epidemiology, rational targeting of anti-osteoporotic therapy, adherence to therapy and safety of anti-osteoporotic drugs. Other research interests include the RANKL/OPG system, osteoporosis genetics, gene–environment interaction, male osteoporosis and personalised osteoporosis therapy. Professor Abrahamsen served on the ASBMR task force on atypical subtrochanteric fractures and on the editorial board for JBMR. He is the lead researcher for the DIPART vitamin D collaborative study and responsible for establishing a national osteodensitometry database on behalf of the Danish Bone Society and for register-based research under the auspices of the Odense Patient Exploratory Network (OPEN).

●●● Professor Juliet Compston is Professor of Bone Medicine and Honorary Consultant Physician at the University of Cambridge School of Clinical Medicine, a position she took up in 2003. Her research is focused on the pathophysiology of osteoporosis and the cellular and structural mechanisms by which pharmacological interventions preserve bone mass and reduce fracture risk. She has conducted studies into the pathophysiology of bone disease in a number of disorders, including post-menopausal osteoporosis, post-transplantation osteoporosis and cystic fibrosis. Recently, her research has focused on fractures in obese post-menopausal women.

Professor Compston is a past President of the Bone and Tooth Society of Great Britain, as well as a past Chairman and President of the International Society of Bone Morphometry. She is currently a member of the Board of the International Osteoporosis Foundation (IOF) and its Committee of Scientific Advisors, a Board Member of the International Society for Bone and Mineral Research and a member of the Clinical & Scientific Committee of the National Osteoporosis Society. She is Chair of the European Union Osteoporosis Consultation Panel and of the UK National Osteoporosis Guidelines Group. She is a member of the NICE Guideline Development Group for osteoporosis.

Professor Compston is Associate Editor of the Journal of Bone and Mineral Research and a member of the editorial board of several peer-reviewed journals including Bone, Osteoporosis International, Calcified Tissue International and the Journal of Clinical Densitometry. She has published over 300 original research papers and reviews.

In 2006, Professor Compston was awarded the National Osteoporosis Society Kohn Foundation Award, and in 2009, the International Bone and Mineral Society John G Haddad Jr Award and the ASBMR Frederic C Bartter Award.

●●● Dr Graham Davenport is a single-handed rural general practitioner and, having been taught by Frank Dudley Hart at Westminster Hospital, has maintained his interest in rheumatology through the Primary Care Rheumatology Society, of which he is a past President. Dr Davenport completed an MSc in rheumatology in 1999, and has been involved in post-graduate education for GPs as a lecturer, trainer, course organiser and appraiser. He has a particular interest in osteoporosis and is involved in the development of osteoporosis guidelines. He represents the RCGP on the Health Professional Partner Forum of the National Osteoporosis Society, the executive committee of the National Hip Fracture Database and the steering group of the National Audit of Falls and Bone Health in Older People. He is currently organising a primary-care-based research study into the management of carpal tunnel syndrome. He has chaired a working party to develop guidelines for the early management of inflammatory arthropathy (the ‘S-factor’) and is running a series of musculoskeletal educational days for GPs and registrars under the auspices of the RCGP and the Primary Care Rheumatology Society.

●●● Dr Alison Gartland studied Biomedical Technology at Sheffield Hallam University and following a period in industry studied for her PhD at the University of Liverpool on the ‘Expression and functional significance of the P2X7 receptor in skeletal tissue’. Her research interests are in understanding the basic cellular and molecular mechanisms responsible for musculoskeletal disease, with an emphasis on the role of extracellular ATP and P2 receptors. Dr Gartland is an elected member of the Bone Research Society.
committee, founder and Secretary of the UK Purine Club, and a long-standing member of the European Calcified Tissue Society and the American Society for Bone and Mineral Research.

Dr Nick Harvey trained at Oxford and Cambridge medical schools and after completion of his basic medical training he joined the Wessex Rheumatology Specialist Training Programme. During this time he was recruited to the MRC Epidemiology Resource Centre to undertake a period of doctoral research, culminating in a PhD thesis in which he confirmed that maternal factors such as diet, lifestyle, physical activity and 25(OH)-vitamin D concentration are associated with offspring bone mass. His post-doctoral work, as a NIHR Clinical Lecturer, investigated the relationships between early growth, assessed by high-resolution ultrasound scanning in pregnancy and anthropometric measurements post-natally, and offspring bone mass in childhood, together with examination of the role of post-natal factors such as childhood physical activity. Since his appointment as Senior Lecturer and Honorary Consultant Rheumatologist at the MRC Lifecourse Epidemiology Unit (www.mrc.soton.ac.uk), he has continued this line of research but now seeks to translate these epidemiological findings into potential public health interventions.

Professor David Hosking is a Consultant Physician in the Metabolic Bone Disease service at the City Hospital, Nottingham, UK and until recently was also Professor of Mineral Metabolism in the Department of Biochemistry at the University of Nottingham, UK. He is currently a Visiting Professor at the University of Zagreb, Croatia. He received his medical training at the University of Birmingham Medical School, Birmingham, UK and post-graduate training in Leiden, The Netherlands. His current research interests are in the long-term control of Paget’s disease, primary hyperparathyroidism and renal bone disease. He has published over 200 papers and book chapters on Paget’s disease, osteoporosis, calcium metabolism and bisphosphonates. He is a member of the editorial board of Osteoporosis International and of the Paget’s Foundation in the USA, from whom he received the J B Johnson Award for services to Paget’s disease.

Professor Sophie Jamal is an Associate Professor of Medicine at the University of Toronto and the Director of the Multidisciplinary Osteoporosis Research Program at Women’s College Hospital, Toronto. She is an endocrinologist and clinician scientist who specialises in the treatment of osteoporosis. She has a PhD in clinical epidemiology and is the Co-Director of the Toronto Centre for the Canadian Multicentre Osteoporosis Study. Her major research interests include identifying novel treatments for osteoporosis, determining the effects of kidney disease on bone and examining the relationship between osteoporosis and vascular calcification.

Professor David Marsh is Emeritus Professor of Clinical Orthopaedics, UCL, Chairman of the UK Arthritis and Musculoskeletal Alliance International Ambassador for the Bone and Joint Decade President of the Fragility Fracture Network. After studying medical sciences at Cambridge University he was awarded his MB BCh in 1975 and FRCS in 1980. In 1990 he was awarded his MD from the University of Cambridge. After working as Wellcome Research Fellow at the Physiological Laboratory at the University of Cambridge he became Lecturer in Orthopaedics at the University of Manchester. He then spent several years as Professor of Trauma and Orthopaedics at Queens University in Belfast before moving to Stanmore in 2005. His research interests focus on the biology of fracture healing and distraction osteogenesis; tissue engineering of bone; treatment of osteoporosis and fragility fractures; clinical trials of fracture treatment; and measurement of outcome in fractures and limb reconstruction.

Professor Tash Masud undertook his undergraduate training at Oxford University and St Bartholomew’s Hospital, London. After post-graduate training in Newcastle and London, he was appointed Consultant Physician at Nottingham City Hospital in 1994. He was the Clinical Sub-Dean at the Medical School, University of Nottingham from 2001 to 2007. He has a clinical and research interest in osteoporosis and falls, and heads the Clinical Gerontology Research Unit at Nottingham University Hospitals NHS Trust. He chairs the British Geriatrics Society’s Education and Training Committee and is a member of the British Geriatrics Society’s Steering Committee of the Special Interest Section of ‘Falls and Bone Health’ and also of the Organising Committee of the Annual International Conference on Falls and Postural Stability. He was previously a Scientific Advisor to the National Osteoporosis Society and was elected as the President of the International Society of Physical Activity for the Prevention of Osteoporosis, Falls and Fractures in 2004. In January 2005 he was appointed as a Visiting Professor of Musculoskeletal Gerontology at the University of Derby.

Professor Eugene McCloskey is Reader in Adult Bone Diseases in the Academic Unit of Bone Metabolism and WHO Collaborating Centre for Metabolic Bone Diseases at the University of Sheffield. He has published over 130 peer-reviewed publications, book chapters and reviews and is an acknowledged authority in the fields of vertebral fracture definition, osteoporosis epidemiology, fracture risk and bone health in cancer. He contributed to the development of the FRAX tool for fracture risk assessment and the subsequent guideline from the National Osteoporosis Guideline Group. He is on a number of editorial boards and is a member of committees within the IOF, the Bone Research Society and the ASBMR. He is also a member of the Clinical & Scientific Committee of the National Osteoporosis Society.
Dr Daniela Riccardi

Dr Daniela Riccardi obtained her BA in Zoology and MRes in Physiopathological methods from the University of Milan, Italy, where she investigated mechanisms of water transport in mammalian epithelia. She did her PhD in Physiology working between the University of Milan and the Harvard Medical School, Boston, USA, under the supervision of Prof. SC Hebert, in the Renal Division of the Brigham and Women's Hospital. Once she obtained her PhD in 1993, she returned to Prof. Hebert's lab, where she identified and characterised the CaR from mammalian kidney. Following her discoveries on the CaR, she was awarded the first prize in Excellence in Research from the American Society of Nephrology and National Kidney Foundation, a Research Fellowship from the National Kidney Foundation and the Wellcome Trust Prize for Excellence in Physiology.

Her current research centres on how cells sense and respond to perturbations in the extracellular milieu, particularly cations (Ca2+), nutrients (amino acids) and, in collaboration with Prof. PJ Kemp, gases (O2, CO and H2S).

In 1997 she moved to the UK, where she established her own research group at Manchester University, where she remained until 2004. In 2004, she moved to the School of Biosciences at Cardiff University.

Professor Nancy Rothwell

Professor Nancy Rothwell, DBE, FRS is a physiological and academic who became the President and Vice-Chancellor of the University of Manchester in July 2010.

Professor Stuart Ralston

Professor Stuart Ralston graduated in Medicine from Glasgow University in 1978 and underwent higher medical training in General Medicine and Rheumatology thereafter. He previously held the chair of Medicine and Bone Metabolism at the University of Aberdeen but moved to Edinburgh University in 2005 to take up the Arthritis Research UK Chair of Rheumatology. Professor Ralston has researched widely on the molecular and genetic basis of bone and joint diseases and has a special interest in the pathogenesis and management of Paget's disease of bone. He is joint editor-in-chief of Calcified Tissue International, and a member of the Commission for Human Medicines.

Dr Caroline Relton

Dr Caroline Relton is a Senior Lecturer in Genetic and Epigenetic Epidemiology at the Institute of Genetic Medicine, Newcastle University (with an honorary appointment at the MRC Centre for Causal Analyses in Translational Epidemiology, CAiTE, University of Bristol). Her research focuses upon the determinants and consequences of epigenetic variation in human populations. The primary aim of her programme of research is to identify novel epigenetic biomarkers, explore their causal relationship with common complex disease and pursue the translation and application of these biomarkers to clinically relevant scenarios. Much of this activity is conducted in collaboration with colleagues nationally and internationally.

Professor Socrates Papapoulos

Professor Socrates Papapoulos received his MD from the University of Athens, Greece, and was trained in internal medicine and endocrinology in Athens and at the Middlesex Hospital, London, UK. In 1984 he joined the Department of Endocrinology and Metabolic Diseases of the Leiden University Medical Center, where he is currently Professor of Medicine, Consultant Physician and Director of Bone and Mineral Research. Since 1974 he has been continuously engaged in basic and clinical research in disorders of calcium and bone metabolism.

Professor Avan Ahie Sayer

Professor Avan Ahie Sayer graduated in medicine from the University of London. She has an MSc in epidemiology from the London School of Hygiene and Tropical Medicine and received a PhD on the developmental origins of ageing from the MRC Lifecourse Epidemiology Unit. In 1998 she became a Consultant in Geriatric Medicine in Southampton, where her clinical work involved a special interest in sarcopenia, frailty and other geriatric syndromes. In 2000 she returned to the MRC as a Clinical Scientist and was appointed as Honorary Chair in Geriatric Medicine at the University of Southampton in 2007. She leads a multi-disciplinary Ageing and Health research group that focuses on sarcopenia and frailty by integrating clinical, epidemiological, basic and social science, with the ultimate goal of improving the health of older people. Since the loss of muscle strength is key in major geriatric syndromes, her research concentrates on the causes, clinical consequences and prevention of muscle loss in older people.

Professor Dawn Skelton

Professor Dawn Skelton is Professor of Ageing and Health and Deputy Director of the Scottish Centre for Evidence Based Care of Older People. She is a commissioned author for the World Health Organisation's Health Evidence Network and the UK's Department of Health, and is the Scientific Advisor for the International Society of Physical Activity for the Prevention of Osteoporosis, Falls and Fractures, and the British Heart Foundation National Centre for Physical Activity; and is Scientific Co-ordinator of ProFaNE (Prevention of Falls Network Europe), which has over 5000 members. She also runs training courses to move research into practice with allied health professionals and fitness instructors. Professor Skelton has won a number of awards (Evian Health Progress Award in 1995, Imperial College School of Medicine Research Into Ageing Prize in 1999, and a Distinguished Service Award in Clinical/Management/Educational Practice Certificate from the Chartered Society of Physiotherapists in 2004). She is a co-investigator on a number of MRC and NIHR grants and her particular interests lie in promoting physical activity and exercise to maintain health and independence. She is a Section Editor for the Journal of Aging and Physical Activity and sits on the National Osteoporosis Society's Research Board.
Pathophysiology of Osteoporosis

Professor Stuart Ralston, ARC Professor of Rheumatology, Molecular Medicine Centre, Rheumatic Diseases Unit, University of Edinburgh, Edinburgh, UK

Osteoporosis can occur as the result of reduced peak bone mass and/or accelerated bone loss. In normal individuals, bone mass increases during skeletal growth to reach a peak between the ages of 20 and 40 but falls thereafter. In women there is an accelerated phase of bone loss after the menopause that occurs as the result of oestrogen deficiency. This causes uncoupling of bone resorption and bone formation, such that the amount of bone removed during the bone remodelling cycle slightly exceeds that which is replaced. With increasing age there is a reduction in bone turnover but bone formation falls more than bone resorption and there is a tendency for fat cells (adipocytes) to accumulate in the bone marrow. This is thought to be due to that fact that, with increasing age, bone marrow stromal cells preferentially differentiate into adipocytes rather than osteoblasts. The physiological mediators of this process are incompletely understood but it has recently been shown that the type 1 cannabinoid receptor (CB1) regulates differentiation of marrow stem cells and protects against age-related osteoporosis in mice [11]. Conversely, drugs that are used in the treatment of diabetes (glitazones) promote adipocyte differentiation in bone marrow and this is thought to account for the increased rate of fractures in patients who are treated with these agents [2].

Bone remodelling and bone turnover are regulated by circulating hormones such as parathyroid hormone (PTH), oestrogen and 1,25 dihydroxyvitamin D, but these seem to act by regulating local production of mediators in the bone micro-environment such as cytokines and growth factors, which then regulate bone cell activity. Recent evidence suggests that osteocytes play a key role in coordinating bone remodelling at the tissue level by sensing and responding to mechanical loading and producing molecules such as RANKL and SOST, which influence differentiation and function of osteoclasts and osteoblasts [3,4]. The most important mediators of osteoclast activity are RANK, RANK ligand and Osteoprotegerin [5], whereas Lipopolysaccharide Receptor Related Protein 5 (LRP5) and Sclerostin (SOST) are important mediators of osteoblast activity. Bone resorption is stimulated by RANKL, which binds to RANK and causes osteoclast activation. This process is blocked by osteoprotegerin, which acts as a “decoy” receptor for RANKL. The importance of the RANK-RANKL-OPG system in skeletal homeostasis is emphasised by the fact that loss-of-function mutations affecting the genes that encode RANK and RANKL genes cause osteopetrosis [6], whereas mutations affecting the gene that encodes OPG causes juvenile Paget’s disease [7] and common polymorphisms at the RANK locus predispose to classical PDB [8]. Finally, a syndrome of severe osteoporosis has recently been described that is cause by the development of autoantibodies to OPG [9].

Bone formation is stimulated by signalling proteins that belong to the Wnt family. These cause activation of the LRP5 receptor, which stimulates bone formation and inhibits bone resorption [10]. A variety of inhibitors of LRP5 signalling have also been identified, including soluble frizzled related proteins (sFRP), Dickkopf (Dkk) and Sclerostin (SOST), and it is likely that regulation of bone formation depends on the balance between levels of the stimulatory Wnt molecules and levels of the inhibitors such as sFRP and SOST. Sclerostin is of particular interest since it is produced by osteocytes and probably acts as a mediator of the effects of mechanical loading on the skeleton [11]. Recent research has also shown that various other pathways play a role in regulating bone mass and bone turnover, including the sympathetic nervous system through production of catecholamines [12]; serotonin, acting on the Htrb1 receptor which is expressed on osteoblasts [13]; and cannabinoids acting on the type 1 (CB1) and type 2 (CB2) receptors [14,15]. Many of the molecules and pathways mentioned above have become clinically important because of the fact that new treatments for osteoporosis have been developed that target these pathways. Examples include Denosumab®, which inhibits RANK signalling [16], and Sclerostin antibodies, which show promise as anabolic agents [17].

Whilst fractures markedly increase in incidence with age, this is not fully explained by the age-related reduction in bone density since the risk of fracture is five times increased in people of aged 80 when compared with people of age 60 who have exactly the same level of BMD [18]. This indicates that age-related factors that influence the risk of falling (such as visual acuity, muscle strength, poor balance, postural hypotension and drug treatments that adversely affect balance) interact with BMD in a very important way to determine fracture risk.

Genetic factors play an important role in regulating peak bone mass and bone loss. Osteoporotic fractures also have a genetic component but the contribution to fracture is much less than for BMD and the heritability of fracture falls off markedly with age as environmental factors such as risk of falling become more important [19].
Natural variations (polymorphisms) have been identified in several genes that contribute to the pathogenesis of osteoporosis. Recent large-scale studies using the technique of genome-wide association (GWAS) have identified several variants that regulate bone mass \([20]\). They include genes in the RANK pathway (such as RANK, RANKL): and OPG and genes in the LRP5 pathway (such as LRP5 itself and SOST). The many other genes that have emerged through this approach include the oestrogen receptor gene (ESR1) and the SP7 gene, which encodes a transcription factor termed Osterix, which is important for osteoblast differentiation. At the present time, GWAS studies have identified 56 genes that predispose to osteoporosis, but together they account for only a small amount of the genetic variance in susceptibility to the disease \([21]\). This is likely because GWAS is geared towards identification of common variants of small effect rather than rare variants of large effect, and further work is in progress to determine to what extent rare (or less common) polymorphisms in other candidate genes contribute to the susceptibility to osteoporosis.

In summary, many advances have been made over recent years in identifying the factors that regulate bone turnover and in defining the role of genetic factors in the regulation of peak bone mass and bone loss. From a clinical viewpoint, the genes and regulatory factors that are involved in mediating bone turnover have importance as molecular targets for the design of new drugs to treat osteoporosis and as genetic markers for osteoporosis susceptibility.

References


Osteoporosis is a major public health issue through associated fragility fractures. At 50 years, the lifetime risk of any osteoporotic fracture has been estimated to be as high as one in two for women and one in five for men. Fracture incidence increases with age; at younger ages, fracture rates may be higher in men than women, because of trauma, but as age increases women overtake men and above 70 years the female rate is around double that for men. In addition to age and female sex, other risk factors include post-menopausal status in women, systemic inflammatory diseases such as rheumatoid arthritis, endocrine disorders such as hyperparathyroidism, Cushing's disease and medications such as glucocorticoids. A baseline osteoporotic fracture is associated with an increased risk of further fracture. This increased risk occurs quickly and thus any osteoporotic fracture should be treated as a reason for urgent assessment and possible treatment. Fracture incidence varies across the globe, and differential secular trends have been observed worldwide, although the reasons for these findings have not been fully elucidated. The net result is likely to be the major burden of osteoporosis shifting from the developed to the developing world. Over the last two decades it has become apparent that fracture risk might be usefully addressed many years before osteoporosis classically develops. Thus, poor early growth (in utero and infancy) is associated with increased risk of osteoporosis and hip fracture in later life; mothers who are poorly nourished, smoke in pregnancy, take vigorous physical activity, and, in particular, have low vitamin D levels during late pregnancy tend to have children with reduced bone mass who are likely to be more at risk of osteoporosis in later life. A randomised controlled trial (MAVIDOS Maternal Vitamin D Osteoporosis Study) is currently underway, testing whether maternal vitamin D supplementation during pregnancy will result in improved offspring bone mineral accrual. Such studies may inform novel public health strategies aimed at intervening early in life to reduce fracture risk in older age.

Pharmacological Therapy for Osteoporosis

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Most patients should start treatment with an oral bisphosphonate because of the drug's proven efficacy in reducing the incidence of both vertebral and non-vertebral fractures. The availability of generic preparations also makes bisphosphonates particularly cost-effective while the ability to give the drugs weekly or monthly by mouth or intravenously every 3–12 months increases patient convenience. Second-line drugs include strontium ranelate, selective oestrogen receptor modulators (SERMs: raloxifene, basodoxifene and lasofoxifene), denosumab (a human monoclonal antibody to RANK ligand) and teriparadate. Calcitonin and hormone replacement therapy need to be added for completeness but are little used currently for long-term therapy in the UK.

All the major drug types, with the exception of teriparadate, which stimulates bone formation, have their major effect through the inhibition of bone resorption. An inevitable consequence is that there is a coupled inhibition of bone formation that limits the gain in bone. Odanacatib, a Cathepsin K inhibitor that also inhibits bone resorption, seems to be associated with relative preservation of bone formation and is currently in a pivotal fracture trial. Strontium ranelate (2g daily) has been shown to reduce vertebral fractures by 41% with a small (16%) decrease in non-spine fractures. However in a post-hoc analysis in women at high risk of hip fracture (age ≥74 years and T score ≤ -3.0) that included 1977 women representing 39% of the total population, there was a 36% reduction in hip fractures.

Selective oestrogen receptor modulators (SERMs), of which raloxifene was the first example, also reduce the risk of vertebral fractures (by 30%) but not non-vertebral or hip fractures. The anti-oestrogen effect in the breast also reduces the risk of ER-positive breast cancer and the drug is licensed for this indication in some countries. Basodoxifene has similar effects to raloxifene but lasofoxifene (0.5mg/day) reduced the risk of non-vertebral fractures by 24% and ER-positive breast cancer by 81%.

Denosumab in a dose of 60mg administered subcutaneously every six months reduced the risk of all major fracture types (vertebral by 68%, non-vertebral by 20% and hip by 40%). An interesting finding with denosumab is that, in contrast to other anti-resorptive drugs, there is a preservation of forearm BMD but
there is no current evidence that this provides better protection against non-spine fractures. However, one of the great defects in the current evidence base of osteoporosis treatment is the lack of head-to-head studies of the effect of different drugs on fracture risk because of the enormous cost of such an undertaking.

Teriparatide (PTH 1-34) is the first anabolic agent and in a dose of 20 mcg daily by subcutaneous injection reduced the risk of vertebral fractures by 65% and non-vertebral fractures by 35%. Treatment is limited to two years and should be followed by an anti-resorptive agent to prevent loss of the new bone and allow completion of mineralisation.

As with many other treatments for disease it seems reasonable to change therapy in the face of failure, but in the case of osteoporosis the definition of 'failure' is not always easy. The occurrence of drug side effects such as upper gastrointestinal symptoms with oral bisphosphonates is relatively straightforward and can be managed by bypassing the gut through the use of an intravenous preparation or by changing the class of drug. The occurrence of a new fracture while on treatment erodes the confidence of both patient and doctor but may not necessarily indicate that the drug is not affecting bone turnover. Firstly, it is well recognised that compliance with medication for chronic diseases is poor and for fracture protection patients need to take about 80% of their tablets. Very few patients will admit to poor compliance but a fall in bone turnover after a change to an intravenous bisphosphonate will give a strong indication of the true state of affairs. Secondly we have to recognise that in clinical trials with high patient compliance new fractures are not completely prevented (vertebral fractures reduced by 50–70%, non-vertebral by about 25% and hip fractures by about 40%). The development of a new fracture means that compliance needs to be assessed and new causes of secondary osteoporosis excluded, but a change of therapy may be an emotional rather than objective decision.

Bisphosphonates are excreted by the kidney and are only licensed for patients with a GFR > 30–35 ml/min. If the decision is to change from an oral to a parenteral preparation then denosumab is the preferred alternative to an intravenous bisphosphonate in patients with this level of renal impairment (late CKD stage 3) because the monoclonal antibody is catabolised in the reticulo-endothelial system rather than the kidney. For the elderly patient at high risk of hip fractures, there is a good case for the use of strontium ranelate as a drug of second choice. At the other end of the age range, the relatively young woman at risk of vertebral fractures, particularly if concerned about breast cancer risk, may choose to change to a SERM. The high cost of teriparatide means that its role as a second-line drug is predominantly reserved for those at very high risk of fracture.

All therapies should be supplemented by calcium and vitamin D where the natural supply is insufficient. The function of vitamin D is to optimise upper gut calcium absorption; it is primarily obtained from sunlight rather than the diet. Recent recommendations are for an intake of 800–1000 IU/day and since vitamin D is stored in the fat it can be conveniently given as a weekly or monthly supplement. Calcium has to be given daily and may be associated with poor long-term compliance but will be required in vitamin-replete individuals with a very low dietary intake.

Even though the prolonged retention in bone of bisphosphonates means that their effect wears off more slowly than other drugs, all patients will eventually start to lose bone again when treatment is stopped. There is currently concern about whether long-term treatment may be associated with rare complications such as osteonecrosis of the jaw or atypical femoral fractures, even though a causal link to treatment has not been established. In the present state of knowledge, it seems reasonable to review the need for treatment after 3–5 years and only continue in those patients where the risk of harm is significantly outweighed by benefit.

Non-pharmacological Interventions

Professor Tahir Masud, Nottingham University Hospitals NHS Trust, Nottingham, UK

A holistic approach to managing osteoporosis patients requires interventions other than drugs aimed at improving bone quality. This talk embraces the management of acute pain caused by vertebral fractures by analgesics, back supports, physical therapy and vertebral augmentation procedures. The use of hip protectors, whole body vibration (WBV) and exercise is also discussed. Fall prevention and dietary and vitamin D interventions are covered elsewhere.

The traditional conservative approach to managing acute vertebral fracture (AVF) pain includes bed rest, analgesia, bracing and physiotherapy. Bed rest should be as short as possible to enable early mobilisation, thereby avoiding problems associated with prolonged immobility including thrombo-embolic disease, muscle wasting, deconditioning and bone loss. The WHO analgesic ladder is appropriate for treating AVF pain (Step 1: non-opioids e.g. paracetamol, non-steroidal ± adjuvants; Step 2: mild opioids e.g. codeine, tramadol ± non-opioids ± adjuvants; Step 3: strong opioids e.g. morphine, oxycodone and fentanyl or buprenorphine patches ± non-opioids ± adjuvants). A ceiling effect occurs with opioids and frail older individuals are particularly prone to side-effects including drowsiness, delirium, constipation and increased falls risk.
A systematic review of 5 RCTs suggested that salmon calcitonin (intramuscular and intranasal) can reduce AVF pain, and this approach could allow earlier mobilisation [Knopp et al Osteoporos Int 2005]. Similarly, some evidence exists for analgesic effects of bisphosphonates in AVF, particularly for pamidronate [Armingeat et al. Osteoporos Int 2006].

The aim of bracing in AVF is to stabilise the spine, limit progression of deformity and allow earlier mobilisation. Its use in osteoporotic AVF, however, is largely opinion-based rather than evidence-based. Good evidence exists for orthoses in acute non-osteoporotic vertebral fractures (‘burst fractures’) and some orthopaedic specialists extrapolate this to the osteoporotic AFV scenario. Examples include the Jewett, CASH and Taylor orthoses. The only orthosis that has RCT evidence in osteoporotic vertebral fracture individuals is the SpinoMed brace (and related SpineMed Active), which has been shown to improve back extensor strength and other parameters including pain, kyphotic angle and activities in daily living [Pfeifer et al. Am J Phys Med Rehabil 2011].

The vertebral augmentation procedures vertebroplasty (VP) and balloon kyphoplasty (BKP) are used as other methods for pain relief following AVF. Large retrospective and prospective case series and prospective comparative trials had demonstrated encouraging results with dramatic pain relief in appropriately selected patients. However, 2 RCTs published in the NEJM in 2009 failed to show a benefit for VP versus a sham procedure, generating much debate in the field [Kallmes et al, Buchbinder et al.]. Some methodological shortcomings were identified in these trials that may have introduced bias. A recent systematic review of randomised and non-randomised controlled trials identified 9 studies comparing VP with non-surgical management (NSM), 6 studies comparing BKP with NSM and 12 studies comparing VP with BKP [Papanastassiou et al. Eur Spine J 2012]. Meta-analyses showed that VP and BKP had better pain reduction than NSM. Both groups also had fewer subsequent fractures, which was reassuring as some previous reports had suggested that there may be an increased risk of adjacent vertebrae collapsing following VP and BKP. There was no difference in pain reduction between VP and BKP groups although the BKP group had less cement extravasation, better height restoration, kyphotic angle reduction and quality of life improvement compared to the VP group.

Studies on the effects of exercise on bone and fractures have been inconsistent over the last 4 decades. This is partly because many factors influence the effectiveness of exercise interventions, including adherence, type and duration of exercise and intensity, frequency and length of exercise programmes. The Cochrane review ‘Exercise for preventing and treating osteoporosis in postmenopausal women’ identified 43 studies [Howe et al. 2011]. The main summary findings were: At the spine people who exercised had on average 0.85% less bone loss than those who didn’t exercise. People who engaged in combinations of exercise types had on average 3.2% less bone loss than those who did not exercise. At the hip, people who exercised had on average 1.03% less bone loss than those who didn’t exercise. In terms of fractures, 4 fewer women out of 100 who did exercise had a fracture (7/100 compared to 11/100). Compared to a control group, back flexion exercises increased the risk of recurrent spine fractures whereas back extension exercises decreased the risk [Sinaki et al. Arch Phys Med Rehab 1984]. Appropriate advice therefore must be given to osteoporotic women regarding safe exercise (e.g. avoid certain moves such as abdominal curls, sit-ups and certain yoga moves associated with flexion).

Low adherence to exercise is an important issue limiting its benefits. A proposed alternative or adjunctive treatment is whole-body vibration therapy (WBV), in which energy produced by a forced oscillation is transferred to an individual from a mechanical vibration platform. A recent review found that the designs of WBV platforms and protocols for their use vary widely, and the optimal target populations are not yet defined. There is confusion between low-intensity vibration platforms intended for osteoporosis therapy and high-intensity platforms intended for exercise. Although there are theoretical advantages (e.g. they can be used in people who cannot undertake conventional exercise), it has yet to be established whether WBV therapy can lead to clinically important increases in bone mineral density and reduce the risk for fractures [Wysocki et al. Ann Intern Med 2011].

The rationale for using hip protectors to reduce the risk of hip fractures is that, after a fall, either the force is distributed more widely and the energy is shunted away from the greater trochanter (hard shell type) or the energy is absorbed, thus reducing the impact of the force (soft type). Some of the earlier studies in care homes that suggested a benefit have subsequently been criticised; e.g. the results were not adjusted for cluster randomisation. A systematic review identified 11 RCTs in care homes (5 individually randomised and 6 cluster randomised) and the meta-analysis showed a 23% reduction in hip fractures in the hip protector compared to the control groups (RR 0.77; 95%CI 0.62–0.97). In the 3 RCTs in community-dwelling populations, however, there was no benefit seen in each study and in the meta-analysis [Parker et al. BMJ 2006]. Poor adherence is thought to be the reason for these negative results. Currently, a rational approach, therefore, is to consider using hip protectors in those individuals at the highest risk of hip fractures including care home residents, the cognitively impaired, frequent fallers and the severely osteoporotic. Major advantages for hip protectors are their low relative cost and immediate benefit when compared to other interventions such as anti-osteoporotic drugs and exercise.
Prevention and Management of Falls

Professor Dawn Skelton, Professor of Ageing and Health, School of Health and Life Sciences, Glasgow Caledonian University, Glasgow, UK.

Over a third of over-65-year-olds fall each year and this rises with increasing age. Injuries and changing activity levels, fear of falling and consequent isolation and reduced quality of life affect not only older people but also health and social care providers. Research over the past 3 decades has advanced our understanding of common risk factors predisposing to falling and has provided important insights for the prevention of falls1. Well over 400 studies have considered different risk factors that differentiate fallers from non-fallers and predict falls over time. Recently, considering only the most robust studies, a systematic review has produced combined Odds Ratios (OR) from data from 74 studies for 31 risk factors2. The most important risk factors for falls include prior history of falls (OR 2.8), gait problems (OR 2.1), Parkinson’s Disease (OR 2.7) and fear of falling (OR 1.6)2. ORs were generally higher for recurrent fallers than for fallers with a history of one fall. Not all risk factors assessed in the literature could be combined (e.g. balance and muscle weakness) as there was wide heterogeneity in design and in outcome measures. Other risk factors identified include: urinary continence and nocturia, foot health and foot pain, vision impairment, multiple medications (as an indicator of multiple medical conditions), alcohol, and of course, the environment3. There is still considerable debate about which are the best risk factor assessment tools for clinical practice3.

There is now a whole suite of Cochrane Reviews covering falls prevention interventions in community-dwelling older people4, those in nursing care or in hospital5 and population-based approaches to falls prevention6. Multi-factorial interventions delivered to those living in the community reduce rate of falls but not risk of falling. In hospitals, such multi-factorial interventions reduce rate of falls and risk of falling and may do so in nursing care facilities. Group and home-based exercise programmes delivered by trained professionals to those living in the community reduce rate of falls and risk of falling, and Tai Chi reduces risk of falling. In sub-acute hospital settings, exercise appears effective but its effectiveness in nursing care facilities remains uncertain. Home safety interventions, delivered by an occupational therapist, reduce rate of falls and risk of falling for those living in the community. Finally, vitamin D supplementation in nursing care facilities reduces the rate of falls. There is some evidence that a home-based exercise programme can be cost-saving within one year in over-80s and group exercise is cost-effective for over-65s. Similarly, home safety assessment and modification in those with a previous fall, and one multi-factorial programme targeting eight specific risk factors have been shown to be potentially cost-effective57. There is still a need for research studies to rationalise the outcome measures they use and to include cost analysis. The growing number of exercise-only interventions has meant we can now better understand the importance of dose (greater than 50 hours), the need for standing balance and strength programmes, the need for trained professionals to deliver the exercise over the transition from rehabilitation to self-managed interventions and, of course, the perils of brisk walking in fallers6.

Best practice guidelines on the assessment of management of falls have been provided for clinicians9,10,11,12. Indeed, the strength of evidence that falls in later life can be prevented has grown to the point where it can no longer be ignored by health policy makers and providers13. The challenge now is to improve provision of effective interventions within practice, both in terms of linking falls prevention with bone health14 and in terms of exercise delivery15.

References:

1. Skelton DA, Todd C. What are the main risk factors for falls amongst older people and what are the most effective interventions to prevent these falls? How should interventions to prevent falls be implemented? 2004. World Health Organisation Health Evidence Network, Denmark.
Altered bone remodelling – excessive bone resorption and/or impaired bone formation – is a key risk factor for osteoporotic fracture and, to date, the majority of pharmacologic agents developed for the prevention and treatment of osteoporosis act by inhibiting bone resorption (hormone replacement therapy, bisphosphonates and selective oestrogen receptor modulators) or by stimulating bone formation (parathyroid hormone). Currently available agents have some limitations; they only target one part of the bone remodelling cycle; there are limited long-term safety data (<10 years); and generally speaking these agents are costly and not available worldwide. An optimal agent would be one that decreased bone resorption while increasing bone formation to have maximum effects on BMD, was inexpensive and was available worldwide. One potential agent is nitric oxide (NO).

NO is a short-lived free radical involved in the regulation of many physiological processes, including bone remodelling. One source of NO is the organic nitrates (e.g. nitroglycerin (NTG), isosorbide mononitrate (ISMO) and isosorbide dinitrate) that can act as NO donors. In vitro studies report that NO decreases bone resorption by decreasing osteoclast formation, motility and function. Administration of organic nitrates has been shown to prevent bone loss in oophorectomised rats and can alleviate bone loss induced by corticosteroid administration in rats.

Nitrates may also have clinical utility in post-menopausal osteoporosis. We reported on the effects of NTG on bone turnover, bone density, bone geometry and strength in 144 post-menopausal women followed for 2 years. We found that, compared with placebo, NTG increased spine, femoral neck and total hip BMD, as well as cortical thickness, cortical area and periosteal circumference. NTG also increased indices of bending and twisting strength: polar section modulus and polar moment of inertia at the radius and tibia respectively. We also found that NTG ointment uncoupled bone formation from bone resorption: it increased a marker of bone formation and decreased a marker of bone resorption. The only significant adverse event associated NTG use was headaches.

Nitric oxide donors appear to have a unique mechanism of action: increasing bone formation and decreasing bone resorption – this may translate into greater anti-fracture efficacy. Further, nitrates are inexpensive and widely available with no known long-term side effects. The efficacy of nitrates for reducing risk of fracture remains to be tested in a randomised controlled trial.

Learning Objectives
1. To briefly review:
   a. The pathophysiology of osteoporosis – with a specific focus on bone turnover
   b. The role of nitric oxide in the regulation of bone turnover.
2. To focus on: The clinical data concerning nitric oxide donors and effects on bone turnover, bone mineral density and fractures.
3. To discuss: Future research directions.
BRS2
The Role of Purinergic Signalling in Bone
Dr Alison Gartland, The Mellanby Centre for Bone Research, University of Sheffield, Sheffield, UK

Purinergic signalling, in its simplest of explanations, is the most primitive cell-to-cell signalling system that exists. It involves the energy molecule Adenosine Triphosphate (ATP) and its breakdown products are used outside the cell to bind specific receptors, called purinoceptors, which then direct the action and fate of cells. Just as every living organism uses ATP for energy, nearly every cell, including bone cells, will express purinoceptors and use purinergic signalling to control cellular functions. Purinergic signalling in bone, in a more elaborate explanation, is a ubiquitous signalling system that involves extracellular purines and pyrimidines binding specific cell surface purinoceptors expressed by the different cell types in bone to trigger intracellular calcium and the respective signalling cascades that direct the fate of bone cells and ultimately help control bone homeostasis. Regulation of purinergic signalling in bone involves the co-ordinated actions of 1) controlled release of purines and pyrimidines from bone cells; 2) expression and activity of enzymes on the surface of bone cells that can break down or interconvert ATP and other nucleotides; 3) purinoceptor expression by bone cells which is tightly controlled in a temporal and spatial manner. Purinergic signalling in bone was first proposed in the early 1990s with the observation that extracellular ATP could raise intracellular calcium and induce secondary messenger activation – events crucial to the normal functioning of osteoblasts. Since then the expression of nearly all the P2Y and P2X receptors by osteoblasts and osteoclasts (of one species/type or another) has been reported, mediating multiple processes including proliferation, differentiation and cell death. My lab’s research has been concerned with trying to decipher the contribution that ATP release, receptor activation and genetics play in co-ordinating the actions of ATP and purinergic signalling on bone homeostasis. By taking a multi-disciplinary approach using in vitro osteoblast cell models, in vivo mouse models and human cohort data, we have found out which specific types of mechanical loading stimulates ATP release to activate downstream signalling pathways and what affect deletion of P2 receptors has on bone homeostasis, and that polymorphisms in the P2X7 receptor gene are associated with increased bone loss in post-menopausal women. Taken together these data highlight three potential targets that may help to detect and treat bone disease such as osteoporosis.


BRS3
Antagonizing the Calcium-Sensing Receptor: Towards New Bone Anabolics?
Dr Daniela Riccardi
School of Biosciences and Cardiff Institute of Tissue Engineering and Repair, Cardiff University, Cardiff, UK

Abstract: With a rise in the aging population, the global osteoporosis market represents a major unmet need and one of the greatest challenges for the pharmaceutical companies. Currently, bisphosphonates constitute the mainstay anti-osteoporotic treatment. They inhibit osteoclast-dependent bone resorption, and substantially reduce the risk of vertebral and non-vertebral fractures. However, bisphosphonates are only marginally effective in subjects with significant loss of bone mineral density. Furthermore, safety concerns have recently been raised due to an increased risk of low-energy fractures associated with long-term bisphosphonate treatment; hence the need for new osteoanabolic drugs. Transient fluctuations in plasma parathyroid hormone (PTH) are a well-established bone anabolic stimulus and efforts have aimed at identifying new medical therapies that can reduce the risk of vertebral and non-vertebral fractures and increase bone mineral density through modifications of circulating PTH. Two approaches have recently emerged in the search for new bone anabolics: a) administration of exogenous PTH, and b) administration of compounds, that evoke transient release of endogenous PTH, namely calcilytics. This review will focus on the potential use of PTH modifying agents as the new osteoanabolics.

Keywords: Osteoporosis, bisphosphonates, osteoblast, osteoclast, parathormone (PTH), calcium-sensing receptor (CaSR), calcilytics.
Workshop: M1
A Debate: “This house believes that surgically induced osteoporosis in rodents is an excellent model for human disease!”

Convenor:
Dr Tonia Vincent, University of Oxford, Oxford, UK

Additional Workshop Participants:
Andrew Pitsillides, Royal Veterinary College, London, UK
David Walsh, Nottingham University, Nottingham, UK
Rose Maciewicz (reserve), Astra Zeneca, Macclesfield, UK

Pre-clinical models of osteoarthritis have been used for more than a century but pathogenic mechanisms are only just emerging with the recent use of genetically modified mice. Much of this advance has come from surgical models but are they really good models for human disease? We will debate the pros and cons of using such models, covering what we have learnt from recent studies and whether these findings are ever going to improve the human condition.

Workshop: M2
Fracture Liaison Services: All You Ever Wanted to Know but Were Afraid to Ask!

Convenor:
Dr Gavin Clunie, Ipswich Hospital NHS Trust, Ipswich, UK

Additional Workshop Participants:
Dr Alun Cooper,
Sonya Stephenson,
Mary Elliot,

Fracture Liaison Services (FLSs) are now part of the national commissioning landscape. The process of identifying patients who have osteoporosis and/or high fracture risk at the time of their first or ‘signal’ fracture, and intervening with fracture prevention management, is well established.

FLSs have different forms: secondary- or primary-case-based, led by specialists or general practitioners, integrated across care boundaries and operating at different thresholds of patient age and presenting pathology.

In this workshop we will present two illustrative models of FLS working – models of care that have grown and succeeded in a somewhat resource-poor and sometimes unfacilitative funding environment. The strengths and essentials of certain aspects of FLS development will be discussed, as will potential pitfalls.

The workshop will importantly involve two specialist nurses who have forged services from ‘the sharp end’. Accordingly, the presenters will be able to show how issues of FLS development, implementation and operation are dealt with from day to day. The presenters will allow a lengthy interactive period to discuss how to overcome barriers to effective integrated care when dealing with processes operating through different healthcare areas (e.g. A&E, orthopaedics, GP surgeries, geriatrics).

Specifically, the presenters will encourage participants, who may already run FLSs, to share their successes and comment on pitfalls, but also will use such examples to illustrate how to overcome problems of day-to-day FLS operation.

Workshop: M3
FRAX to the Future

Convenor:
Professor Jon Tobias, Academic Rheumatology, University of Bristol, Bristol, UK

Additional Workshop Participants:
Professor Eugene McCloskey

The NICE short clinical guideline on assessment of the risk of fragility fracture, due to be published imminently, recommends the use of FRAX [1] and QFracture [2] as web-based fracture risk calculators to assess fracture risk. These generate estimates for ten-year fracture probability (FRAX) or risk (QFracture) based on combinations of risk factors entered into the calculator. Both provide clinically useful information, but, as with any tool including the measurement of BMD, it is important to understand their benefits and limitations so that clinical judgement can be exercised when using them. On the one hand, adjustments may need to be made to the estimated fracture risk in light of other information about risk factors that is not adequately captured by these risk calculators. On the other, decisions need to be taken about what fracture risk should be used to guide treatment, with individualised decisions on a case by case basis, according to the particular intervention under consideration.
This workshop will discuss factors that may influence fracture risk in ways that are not well captured by FRAX and/or QFracture. As part of this discussion, strategies for adjusting fracture risk estimates in different contexts will be reviewed, including the application of further refinements to FRAX based on recent research findings. For example, an adjustment to FRAX for patients on high-dose steroids has recently been published [3]. Other situations that will be discussed include patients with low DXA scores at the spine or forearm, multiple and/or vertebral fractures, a history of falls, and secondary osteoporosis due to type II diabetes mellitus or treatment with aromatase inhibitors for breast cancer.

This workshop will also discuss what intervention thresholds should be used to guide treatment, by comparing different strategies. The National Osteoporosis Guideline Group (NOGG), whose recommendations are accessed via a link from the UK version of the FRAX website, are based on an intervention threshold that increases with age, which tends to increase the proportion of younger patients in whom treatment is recommended. For other countries, such as the US, intervention thresholds are independent of age, increasing the proportion of older patients recommended for treatment. The impact of these different strategies on the number and case mix of patients recommended for treatment will be discussed, by reviewing results of a study comparing the effect of applying a range of intervention thresholds to an NHS DXA referral population.

References

Workshops

Workshop: M4
Management of Vertebral Fractures

Convenor:
Dr Emma Clark, University of Bristol, Bristol, UK

Additional Workshop Participants:
Rachel Lewis

Secondary fracture prevention
A vertebral fracture identifies a person at the highest risk of further fractures. Future fracture risk should therefore be reduced by:

A usual lifestyle advice
- regarding diet, smoking and alcohol

B medications
- according to national guidelines [1] or according to treatment thresholds e.g. as assessed by FRAX or QFracture (i.e. treat irrespective of bone density)

Management of Symptoms
Symptoms may include pain, poor posture (kyphosis), shortness of breath, poor balance, loss of confidence and reduced quality of life.

A medications
- pain killers, NSAIDs, gabapentin/amitriptyline, calcitonin, teriparatide

B physiotherapy [2–4]
- muscle strengthening
- posture and trunk control

These can be improved by chair-based, standing, kneeling and prone exercises, which will be demonstrated during this workshop. The starting level will change on an individual patient basis, and intensity can be increased by holding each position for longer, and by use of a back-pack.

Hydrotherapy can be helpful for pain relief, posture, balance and muscle strengthening.

Heat, acupuncture and TENS can also be useful for pain relief.

A recent RCT [5] has shown that exercise can improve mobility, balance and health-related quality of life.

C vertebroplasty/kyphoplasty [6,7]
This can be used for pain relief, but it is only helpful for a few select patients.

D bracing
This will be demonstrated during the workshop

Patient education
A on general osteoporosis
B on medication adherence and what to do if there are tolerability issues
C on pain relief
D on posture
- particularly ergonomic advice for ADLs and employment
E on getting in and out of bed
- this will be demonstrated during the workshop
F on ongoing exercise
- at levels appropriate to the patient; e.g. Tai Chi, swimming, gym
G advice on prevention in family members

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- regarding diet, smoking and alcohol

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- this will be demonstrated during the workshop
F on ongoing exercise
- at levels appropriate to the patient; e.g. Tai Chi, swimming, gym
G advice on prevention in family members
References
1. NICE TA161 for secondary fracture prevention in osteoporosis.

Workshop: M5
Paediatric Osteoporosis – Assessment and Management

Convenor:
Dr Nicola Crabtree, Principal Clinical Scientist
Bone Densitometry, Department of Endocrinology,
Birmingham Children’s Hospital, Birmingham, UK

Additional Workshop Participants:
Dr Zulf Mughal

This workshop will focus on the challenges of diagnosing and managing paediatric osteoporosis. The session will cover topics including current and future imaging techniques and their role in the paediatric arena, concentrating on the potential pitfalls of using these techniques in children. Also covered will be the current and future management of childhood osteoporosis, leading on to a discussion concerning the application of currently available paediatric guidelines in the clinical setting.

The workshop will take the format of a basic introduction to paediatric bone densitometry followed by a series of case studies presented by Dr Zulf Mughal. There will be an opportunity to discuss case studies presented and other cases brought to the workshop.

Suggested Reading

Books
- Bone Densitometry in Clinical Practice: Application and Interpretation (Current Clinical Practice) by Sydney Lou Bonnick
- Bone Densitometry in Growing Patients: Guidelines for Clinical Practice (Current Clinical Practice) by Aenor Sawyer
- Calcium and Bone Disorders in Children and Adolescents (Endocrine Development) by J Allgrove & N J Shaw

Guidelines
- NOS Publication: A practical guide to bone densitometry in children
(www.nos.org.uk)
- ISCD2007 official pediatric positions
www.iscd.org/Visitors/positions/OfficialPositionsText.cfm

Workshop: M6
Nutrition and Bone Disease

Convenor:
Dr Helen Macdonald, Senior Lecturer in Nutrition and Translational Musculoskeletal Research, University of Aberdeen, Musculoskeletal Research, Health Sciences Building, Foresterhill, Aberdeen, UK

Additional Workshop Participants:
Niki Gonty

How do we assess whether someone has an adequate diet for healthy bones? This workshop will examine current evidence and particularly focus on how we can estimate calcium intakes to determine whether people are getting enough calcium from their diet or whether they need to take supplements. We wish to encourage discussion around possible pitfalls of short questionnaires and whether they can be overcome to allow us to recommend a simple calcium assessment tool.
You may find it helpful to look at an example of a calcium intake questionnaire (e.g., http://www.rheum.med.ed.ac.uk/calcium-calculator.php) prior to attending the workshop.

The reference nutrient intake (RNI) for adults in the UK is 700 mg calcium a day [1]. It is defined as the estimated average intake for the population plus 2 standard deviations. This is considered sufficient to cover the needs of 97.5% of the population. The lower reference nutrient intake is 400mg calcium a day, and below this amount 97.5% of the population would be considered at risk of calcium deficiency. When re-examining the evidence for bone health, the committee on medical aspects of nutrition (COMA) could not find any evidence to support higher intakes in the general population [2].

For those with osteoporosis, it is recognised that higher daily intakes of calcium may be required. This would be around 1000mg calcium a day as indicated in SIGN guidelines [2003] [3], or sometimes 1200mg will be suggested. This is mainly because for most osteoporosis treatments the patients in the studies testing the treatments were also given calcium and vitamin D. This was to ensure that everyone in the study was replete in these nutrients, as there was a possibility if someone was deficient in calcium or vitamin D that the bone would not be adequately mineralised, and this could result in osteomalacia.

Most clinicians agree that if someone has sufficient calcium in the diet they do not need a supplement. However, can you be certain whether someone has adequate dietary calcium? It is easier to meet average daily intakes of 700mg calcium than to meet 1000mg or more. If higher intakes are required, the patient might have to think more carefully about what they are eating to make sure they are getting enough, or take a supplement.

How do we assess if someone is getting enough calcium? Measuring calcium intake is a challenge. There is not a good biomarker for calcium – serum calcium is tightly controlled and urinary calcium as 24-hour urine would be burdensome to collect, and the result may not be an accurate reflection of intake as it is influenced by other factors. Bone mineral density (BMD) could not be used as a proxy for calcium stores as BMD depends on many factors, not just dietary calcium.

We can ask people what they eat, and work out how much calcium they are getting from the foods they are eating. This can range from (1) simply asking the patient ‘how much milk or dairy products do you have a day?’ to estimating calcium intakes from (2) a short questionnaire given at clinic (3) food frequency questionnaire, the method used in large epidemiological studies (3) food diary, where the participant has to record everything they eat either with estimates of portion size or weighing the food. The latter method is considered the gold standard but places a considerable burden on the patient or participant and the clinician or researcher.

It is also important to consider how the patients or participants interpret the questions – when asked about how much milk they have a day, do they count milk in tea or coffee? Do they know much milk they add? People do not eat the same things every day and you are asking them to think about this in a clinical setting. If they are unprepared for the question, they might give a different answer from if they had more time to think about it, in less stressful surroundings. They might overestimate calcium-rich foods, as they would like their doctor or nurse to think they have a good diet.

What about the validity of each of method? We can only compare different methods, and even with the gold standard we cannot be certain that the result is accurate. People can change their eating habits during the period they are completing a food diary (unintentionally or for convenience). In epidemiological research, we rank people according to low and high intakes so the exact amount eaten is not as important compared to an individual’s assessment, where we want to know whether calcium intake is above the RNI.

The UK National Diet and Nutrition Survey [3] shows that dairy products are the main source of calcium in the diet.

The workshop will show data on what foods are contributing to calcium intake, compare results from food diaries, FFQ and the short questionnaire. It will be followed by an open discussion around what tool is best used to estimate calcium intakes, and, knowing what factors could affect the response, what measures could be employed to ensure that the assessment is as accurate as it can be. Is it better to ask about intakes of many foods or focus on a few key foods?

References:
Workshop: M7
DXA Reporting for Professionals Allied to Medicine

Convenor:
Dr Karen Knapp, Senior Lecturer, Medical Imaging, College of Engineering, Mathematics and Physical Sciences, University of Exeter, Exeter, UK

Additional Workshop Participants:
Jill Griffin
Dr Nicola Peel

Role extension of allied health professionals has become a necessity in some areas and reporting of scans by professions allied to medicine has become standard practice in some departments. While radiographer-reported radiographs has become relatively widespread in many hospitals, there are fewer radiographers reporting DXA scans [1]. However, with the appropriate education, training, protocols and mentoring, this is an appropriate area for the role extension of radiographers and other professions allied to medicine who are undertaking dual energy X-ray absorptiometry (DXA) scanning and working within osteoporosis services.

This workshop will cover the medico-legal requirements of non-medical reporting, the educational and training considerations, the scope of practice and protocols for DXA reporting, clinical governance and what to include in an optimum report.

A report undertaken by a professional allied to medicine must be conducted by a competent person who possesses the appropriate knowledge, expertise and education to provide this service, resulting in a report of the same reasonable standard of care as the professional who would have ordinarily have reported the scan [2]. As such, the educational underpinning to reporting DXA scans is essential. Professionals reporting DXA scans must have an in-depth understanding of osteoporosis and other metabolic bone diseases, DXA scanning and DXA interpretation. This should include knowledge and understanding of the pathophysiology of bone, including hormonal actions and the effect of aging on bone [3, 4]. They should have an awareness of the cortical and trabecular content of different skeletal sites and the differing metabolic activities of these two types of bone [5]. The influence of trabecular and cortical bone content on disease progress and response to therapeutic intervention should be well understood. The definitions of osteoporosis and the epidemiology of osteoporosis, including the incidence, pattern of fracture, contribution of bone mineral density (BMD) and previous osteoporotic fracture to fracture risk, and the morbidity and mortality related to fractures [6, 7]. Knowledge of clinical risk factors for osteoporosis is of the utmost importance for both identifying which patients should undergo a DXA scan [8, 9] and for assessing overall fracture risk [10]. Understanding of the pharmacological action of common osteoporosis therapies is also useful but, in general, specific recommendations for treatment would not be included in the report beyond recommending that a treatment is commenced in accordance with NICE guidelines or other local guidance.

Once a professional allied to medicine is deemed to be at a level of expertise to commence reporting, a period of mentorship is required to ensure the reports are of an appropriate standard and accuracy. Protocols for the scope of reporting must be agreed at a local level and adhered to. Competency logs can be used to document the development and competence of an individual until they are signed off to report independently. Regular audits of reports are important to ensure continuing accuracy and competence. Attendance at appropriate conferences and keeping up to date with current literature is important in underpinning continuing professional development and ensuring the service is up to date and fit for purpose.

Further information and recommendations on reporting can be found in the National Osteoporosis Society’s practical guide ‘Reporting dual energy X-ray absorptiometry scans in adult fracture risk assessment (2011) UPDATED’, which can be found at: www.nos.org.uk/document.doc?id=854.

In conclusion, reporting of DXA scans by professionals allied to medicine is achievable with appropriate education, experience and expertise, mentorship, protocols and monitoring. This can create a valuable asset to the service and help staff to enhance their roles.

References:
Workshop: M8
Cancer Therapies and Osteoporosis

Convenor:
Professor Roger Francis, Emeritus Professor of Geriatric Medicine, Institute for Ageing and Health, Newcastle University, Newcastle upon Tyne, UK

Additional Workshop Participants:
Dr Terry Aspray
Dr Stephen Tuck

The latest data from Cancer Research UK suggest that over 48,000 women and 40,000 men develop breast cancer and prostate cancer respectively each year. The age-specific incidence of breast cancer has increased by 18% over the past 15 years, whereas prostate cancer has risen by 53% during this period of time. This is probably due in part to earlier diagnosis because of the mammography screening programme and the increased measurement of prostate-specific antigen. Early identification of these cancers and the use of treatments such as aromatase inhibitors in cancer and androgen-deprivation therapy in men have improved prognosis, such that the overall five-year survival after the diagnosis of breast cancer has increased from 78.4% in 1996–2000 to 85.1% in 2005–09, whilst survival after prostate cancer has improved from 53.6% in 1991–95 to 77.0% in 2001–05. Unfortunately, although aromatase inhibitors and androgen-deprivation therapy have revolutionised the management of breast and prostate cancer, they are associated with accelerated bone loss and an increased risk of fractures.

In this workshop, Terry Aspray will outline the effect of breast cancer treatments, including gonadotrophin-releasing hormone agonists, tamoxifen and aromatase inhibitors, on bone loss and fracture risk. He will review the use of osteoporosis treatment in the prevention of bone loss in women with breast cancer and discuss guidelines on the management of breast-cancer-treatment-induced bone loss, including intervention thresholds based on fracture risk assessment. Stephen Tuck will then highlight the role of orchidectomy and androgen deprivation therapy in the development of bone loss, sarcopenia and fractures in men with prostate cancer, before reviewing the effect of osteoporosis treatments and outlining approaches to patient management. Finally, Roger Francis will outline plans for the development of UK guidelines on the management of bone loss in men with prostate cancer.

References

Workshop: T1
Bone Markers – Laboratory Issues and Clinical Value

Convenor:
Bill Fraser, Norwich Medical School, University of East Anglia, Norwich, UK

The knowledge base gained using bone markers has given physicians confidence in the therapeutic effects of treatments for osteoporosis. Bone markers accurately reflect bone cell function and bone metabolism. It is important to collect the correct sample type at the appropriate time and store samples correctly. Optimal conditions for collection and storage and the implications of poor pre-analytical processes will be discussed.

Baseline markers give information on secondary causes of osteoporosis. Combining bone markers with other risk factors can improve the ability to predict the
likelihood of future fracture. Treatments that target the
osteoclast (anti-resorptive) result in a significant decrease
in markers such as CTX or collagen crosslinks. The
greater the reduction in markers, the greater the fracture
efficacy. A lack of response can be readily observed,
by measuring bone markers giving confidence at a
consultation to discuss concordance or adherence
with therapy that may result in a change of therapeutic
approach. Markers of type 1 collagen synthesis, P1NP
and P1CP can supply valuable information regarding the
therapeutic efficacy of expensive anabolic treatments in
terms of the likely BMD response or non-concordance
with treatment.

Workshop: T2
Vertebral Fracture Assessment

Convener: Professor Judith Adams, Consultant
Radiologist, Manchester Royal Infirmary, Manchester, UK
Email: judith.adams@manchester.ac.uk

Additional Workshop Participants:
Speaker: Dr Nicola Peel
Email: Nicola.Peel@sth.nhs.uk

Vertebral Fracture Assessment

Introduction: vertebral fractures (VF) are the most
common osteoporotic fracture[1], and may occur with
no, or minimal, trauma. In patients over 50 years,
VF prevalence is 20% in women, with strong age
dependency[2]. In men the prevalence is 20%, but
more are present at an earlier age, so traumatic events
are presumed to be aetiological[2-3]. Clinical symptoms
include back pain, height loss, deformity, disability
and limited spinal mobility[4-5], and there is associated
increased mortality[6]. With a single VF there is a 12%
increased risk of a future VF within 12 months; with
multiple VFs this risk rises to 22%[7]. Patients with VF
are at increased risk of fracture (X5 VF; X2 hip fracture)
[8]. A patient with VF and low bone mineral density (BMD)
has a 25-fold higher risk for subsequent VF than does
one with no fracture and high BMD[9]. Consequently,
accurate identification of VF is vital in diagnosis &
management of osteoporosis.

Assessment: VF may be symptomatic, but 30%-75%
may be asymptomatic, and not come to clinical attention
[10]. Spinal radiographs are currently the best method
for confirming the presence and severity of VF. They are
classified as end-plate, wedge and crush[11,12] and can
be assessed visually, or by the semi-quantitative (SQ)
method[13], in which the vertebrae are graded according
to their altered shape. If a VF is present it must be
reported clearly and unambiguously as ‘fractured’ so that
there is no misunderstanding of the significance of the
feature; their presence influences the WHO FRAX 10-year
fracture risk prediction (www.shef.ac.uk/FRAX). There
is evidence that significant numbers (30%-50%) of VF
are not reported or their significance is not recognised
[13]. Consequently, patients with VF who would benefit
from fracture-reducing therapies are not receiving such
therapies[13,14]. Consequently, there has been a VF
initiative by the International Osteoporosis Foundation
and educational material on this topic is available at
www.osteofound.org. There are important features
that differentiate osteoporotic VF from normal variants
and other aetiologies that alter vertebral shape & the
algorithm-based approach for qualitative identification of
VF (ABQ) method is helpful[15].

Although currently diagnosis of VF is made from
radiographs, technical improvements in dual-energy
X-ray absorptiometry (DXA) scanners (fan beam
scanners provide faster scanning and improved spatial
resolution (1.0mm to 0.5mm)) have made it possible
to make vertebral fracture assessment (VFA) from DXA
images[16,17]. Such images can be obtained in dual-
(better visualisation of thoracic vertebrae) or single-energy
mode, and are acquired with lower radiation
doses (1/10th to 1/100th) than radiographs[18], include
the entire spine on a single image (without endplate
distortion of the divergent X-ray beam of radiographs)
and have potential for computer automation[19].
Guidelines for the use of VFA have been published:

All fractures cause vertebral deformities, but not
all deformed vertebrae are due to fractures[20].
Other pathologies (trauma, myeloma, metastases,
Scheuermann’s disease, Schmorl’s nodes, infection,
degenerative disease, congenital anomalies) may cause
deformities and must be differentiated from osteoporotic
VF. Other imaging methods such as computed
tomography (CT), magnetic resonance imaging (MRI)
and radionuclide scanning (RNS) can be helpful in this
differentiation[21].

References:
1. Melton LJ et al. Prevalence and incidence of vertebral
2. O’Neill T et al. The prevalence of vertebral fracture in
European men and women: the European Vertebral
Osteoporosis Study. J Bone Miner Res 1996;11:1010-
1018.
3. Samuelson EJ et al. Risk factors for the incidence of
vertebral fracture in men and women: 25 year follow-up
results from the Framingham Osteoporosis Study. J Bone
Miner Res. 1999;14(S1):S147.
4. Nevitt MC et al. The association of radiographically
detected vertebral fractures with back pain and function: a
5. Ettinger B et al. Contribution of vertebral deformities
to chronic back pain and disability. The Study of
Osteoporotic Fractures Research Group. J Bone Miner
6. Ensrud KE. Prevalent vertebral deformities predict mortality
and hospitalization in older women with low bone mass.
Fracture Intervention trial Research Group. J Am Geriatr
7. Lindsay R et al. Risk of new vertebral fracture in the year
8. Black DM et al. Prevalent vertebral fractures predict

for a breakfree future


Most recently, the excellent depiction of bone with CT has led to the development of imaging software that can estimate cortical thickness with sub-millimetre resolution (lower than the inherent system resolution), while high field MRI can now be used to quantitatively and qualitatively assess cortical bone and trabecular microarchitecture. Cross-discipline collaboration (e.g. with engineering and physics) is yielding significant advances in image analysis, particularly in harnessing the 3D capability of these newer volumetric techniques. The resurgence of 18F-Fluoride as a radiolabel in positron emission tomography (PET) is also bringing molecular imaging capabilities to the assessment of skeletal kinetics in bone disease. The ability of imaging techniques to simultaneously assess the skeleton, articular cartilage, muscle and adipose tissue also delivers the potential to assess multiple disease states from a single acquisition: for example, muscle and bone in sarcopenia and osteoporosis, and cartilage and bone in osteoarthritis and osteoporosis.

A critical evaluation of existing technologies is important to fully determine their potential, appropriate use (clinical vs research) and interpretation. Imaging the skeleton in volumetric cross-section has the potential to become a new standard in future clinical practice.

This workshop will be in two parts and aims to:

1. Describe the status of imaging as a tool to assess bone strength and discuss why measurement of multiple aspects of bone strength is important in understanding an individual/population risk of fracture.

2. Discuss emerging imaging techniques and their potential for clinical application. We also introduce their value in the assessment of other bone-related diseases, namely osteoarthritis.

References:

General Overview

This review is an excellent introduction to the role that imaging has to play in the assessment of bone microarchitecture, particularly with respect to monitoring response to therapy, with some background on the relevant bone biology.

This review discusses the definition of a healthy bone phenotype and how bone and muscle interact, and how QCT has furthered our understanding of the multiple
components of bone strength. DXA and QCT are critically evaluated as the most common tools currently available.


**Computed Tomography**

- Treece GM, Gee AH, Mayhew PM, Poole KES (2010) High resolution cortical bone thickness measurement from clinical CT data. Med. Image Anal. 14, 276–290. This paper describes a novel analysis technique for measuring cortical bone thickness on standard clinical CT acquisition with accuracy below the inherent resolution of the CT imaging system.

- Poole KES, Treece GM, Ridgway GR, Mayhew PM, Borggreve J, and Gee AH (2011) Targeted regeneration of bone in the osteoporotic human femur. PLoS ONE 6, e16190. This study uses the novel cortical thickness mapping analysis technique (Treece et al., 2010) on qCT data to show that recombinant human PTH therapy can cause significantly increased thickness in patches of cortical bone in the proximal femur of women with osteoporosis.

- Turmezei TD and Poole KES (2011) Computed tomography of subchondral bone and osteophytes in hip osteoarthritis: the shape of things to come? Front. Endocrin. 2:97. This review discusses the strength of CT in imaging bone for the assessment of hip osteoarthritis, including the use of computational analysis techniques such as cortical thickness mapping (Treece et al., 2010) and shape deformation modelling.


- Lang T et al. (2010) Computed tomographic measurements of thigh muscle cross-sectional area and attenuation coefficient predict hip fracture: the health, aging, and body composition study. J Bone Miner Res. 25(3) 513. This paper describes the measurement of muscle from QCT scans, to move forward the assessment of bone and muscle in QCT; this is important in terms of the recognition of the importance to consider both compartments when assessing fracture risk.


**Magnetic Resonance Imaging**

- Chang G, Pakin SK, Schweitzer ME, Saha PK, Regatte RR (2008) Adaptations in trabecular bone microarchitecture in olympic athletes determined by 7T MRI. J. Magn. Reson. Imaging 27, 1089–1095. This study describes a high-field (7T) MRI approach to measuring trabecular bone microarchitecture in Olympic fencers, suggesting that high-impact activity may lead to increased marrow volume and improved trabecular bone structure. The paper also touches on novel post-processing techniques in MRI relevant to bone imaging.


**Molecular Imaging**

- Li Y, Schiepers C, Lake R, Dadparvar S, Berenji GR. (2012). Clinical utility of 18F-fluoride PET/CT in benign and malignant bone diseases. Bone 50, 128-139. This review covers the application of 18F PET/CT to assess skeletal kinetics in bone disease.

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**Workshop: T4**

**Bone Biology – The Essentials**

**Convenor:**
Professor Tim Arnett, Professor of Mineralised Tissue Biology, University College, London, UK

Bone can be thought of as a relatively simple structural tissue. Basic knowledge of the ‘stuff’ from which bone is made and the behaviour of the main cell types found in bone can give a useful understanding of the processes of bone growth and turnover in health and disease. Some key facts are briefly reviewed here.

**Bone Composition**

Bone is made up of a mineralised protein ‘matrix’ and cells. The protein matrix consists of about 90% type I collagen, the tough fibrous material also found under the skin and in many internal organs. The remaining 10% is a complex assortment of smaller, non-structural proteins, most notably osteocalcin (which can serve as a useful marker for bone turnover). This collagenous matrix is impregnated with tiny crystals of an almost insoluble mineral (hydroxyapatite, a form of calcium phosphate). These mineral crystals envelop the collagen fibres to form a composite material with the right properties of stiffness, flexibility and strength (similar in principle to plaster casts for fracture). The 3 main types of bone cell are considered below.

**Osteoblasts, Osteocytes and Osteoclasts**

**Osteoblasts** are specialised bone forming cells, related to fibroblasts (the cells that make collagen in skin and internal organs). They work together in groups to secrete new collagenous bone matrix. This protein (called osteoid) is then mineralised. In adults, it is mineralised after a few days, provided sufficient calcium is available. Vitamin D is needed to ensure correct uptake of calcium from the gut; vitamin D deficiency results in soft bone (rickets in children, osteomalacia in adults). Osteoblasts use the enzyme alkaline

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phosphatase to promote mineralisation (it breaks down pyrophosphate, the body’s natural ‘water softener’). Some osteoblasts become incorporated into the matrix they are making and develop into osteocytes, a sparse but regular, interconnecting network of cells that runs throughout the interior of living bone. Osteocytes may act to detect the small amount of bending that occurs in bone when it is heavily loaded, transmitting some form of message to the bone surface that instructs osteoblasts to add new layer(s) of matrix to strengthen the bone. This adaptation to loading is extremely important. Osteoblasts are very busy during growth but in adult life they tend to become flattened and less active, covering bone surfaces in a near-continuous single layer often referred to as “bone-lining cells”. These cells can be re-activated, as required (e.g. by osteocyties?). Osteoblasts and osteocytes communicate with osteoclasts (see below) and other cells using a variety of local messenger molecules.

**Osteoclasts** are large, mobile cells with several nuclei that possess the unique ability to resorb (destroy) bone by excavating pits in bone surfaces. This involves removal of both the mineral and the fibrous collagen matrix. Osteoclasts are formed from the fusion of white blood cells related to monocytes. Osteoclasts work much more rapidly than osteoblasts (analogly: it is quicker to demolish a house than to build it). In adult bone, osteoclasts are relatively uncommon. Bone loss in osteoporosis is mainly due to the presence of increased numbers of active osteoclasts. Osteoclast formation is controlled mainly by a messenger protein (RANKL) produced by osteoblasts and other cells; blocking this protein (e.g. with an antibody) reversibly stops bone resorption.

**Bone Remodelling**

Bone is normally renewed throughout life by the co-ordinated action of osteoblasts and osteoclasts (and osteocytes). This usually takes place in limited zones as part of a rolling programme of replacement – or because damage has occurred. In later life, and after the menopause, osteoclast formation/activity tends to outstrip the bone-forming action of the osteoblasts, leading to net bone loss. Bone can also be rapidly destroyed by osteoclasts if the skeleton experiences insufficient loading (presumably also detected by osteocyties), due to bedrest or lack of exercise.

**Websites with a good range of teaching materials on bone / osteoporosis**

http://courses.washington.edu/bonephys
www.iofbonehealth.org/?id=576
www.brsoc.org.uk
www.asbmr.org
www.ucl.ac.uk/cdb/research/arnett/gallery

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**Workshop: T5**

**Bone-Unfriendly Drugs**

**Convenor:**
Dr Nicholas C Harvey, Senior Lecturer and Honorary Consultant Rheumatologist, MRC Lifecourse Epidemiology Unit, University of Southampton, Southampton, UK

**Additional Workshop Participants:**
Dr Chris Holroyd
Dr Mark Edwards

Historically, several classes of therapeutic agent have been known to be deleterious to bone quality and to increase risk of fracture. Thus, use of corticosteroids has been shown in many studies to adversely affect fracture risk even at low daily doses. The aim of this workshop, however, is to explore the potential bone-related adverse effects of other medications including proton pump inhibitors, H2R-blockers, glitazones, anti-depressants, aromatase inhibitors and androgen-deprivation therapy.

In recent years, several observational studies have suggested an increased fracture risk with PPI and H2R-blocker use for acid suppression. Whilst these results are reasonably consistent, a more holy contested issue is whether the use of bisphosphonates modifies this relationship. In the workshop we will try to pick our way through these data and give some guidance on what is clinically reasonable given the current evidence base. The finding of increased fracture risk as an adverse event in randomised controlled trials of glitazones for the treatment of type II diabetes has added another drug with bone-unfriendly features. Large population-based studies have replicated these results and we will discuss the clinical approach to diabetic patients and fracture risk. Anti-depressants are widely used in the general population and there is increasing evidence from epidemiological studies that SSRIs and tricyclics are associated with increased fracture risk. These data will be reviewed.

One of the major emerging fields in the area of osteoporosis is the concept of cancer-treatment-induced bone loss (CTIBL). Thus newer treatments for breast cancer (that is, the aromatase inhibitors), have been shown to increase bone loss and fracture risk. Recent guidelines have been developed for the risk assessment and treatment of bone loss associated with these hormonal therapies. Similarly, androgen-deprivation therapies for prostate cancer are also associated with increased fracture risk and bone loss. During the workshop we will discuss practical approaches to these types of patients.

By the end of the session, participants should have a clearer idea of the evidence base for the adverse effects of these treatments and a better understanding of the implication of these data for daily clinical practice.
References


Workshop: T6

Treatment Conundrums

Convenor:
Professor Juliet Compston, Professor of Bone Medicine, University of Cambridge, School of Clinical Medicine, Cambridge, UK

Additional Workshop Participants:
Professor Socrates Papapoulos

In recent years there have been significant advances in the management of osteoporosis. However, there is still some uncertainty in some areas, particularly with respect to the duration of treatment, monitoring of treatment response and use of combination therapy. The workshop will focus on the following topics.

Duration of Treatment

For how long should anti-resorptive therapy be prescribed? Do drug holidays have a place in bisphosphonate therapy and, if so, which criteria should be used in decision-making? What is the incidence of adverse effects of long-term treatment such as atypical fractures and osteonecrosis of the jaw and how does this affect the overall benefit/risk ratio of treatment?

Monitoring of Treatment

How should treatment response be monitored and how should non-response be defined? Does a fracture during treatment indicate failure to respond? How often should BMD be measured and do biochemical turnover markers have a place in monitoring treatment response?

Combination Therapy

Is there a place for combination therapy, either with the simultaneous use of two anti-resorptives or an anabolic and anti-resorptive agent? Does prior anti-resorptive treatment impair the response to PTH peptide or strontium ranelate therapy? How should the effects of PTH be maintained after treatment is withdrawn?

References


Workshop: T7
How Do We Really Manage Paget’s Disease?

Convenors:
Anne Sutcliffe, Healthcare and Education Officer, the Paget’s Association, Manchester, UK
Professor David Hosking, Consultant Physician, City Hospital, Nottingham, UK

Through a series of interactive case histories, this workshop will cover the clinical presentation of Paget’s Disease and its complications. It will discuss the means of diagnosis, exploring the rationale for using X-rays, isotope bone scans and biochemical investigations. The workshop will outline appropriate treatment options and offer guidance on timing and efficacy of therapeutic interventions and the need for monitoring.

By the end of the session, it is anticipated that participants will be able to:

- Understand the clinical implications of Paget’s Disease
- Diagnose it by appropriate methods
- Recommend appropriate therapeutic interventions

References:

Workshop: T8
Novel In Vitro Cell Culture and Signalling Approaches

Convenor:
Dr Agi Grigoriadis

Additional Workshop Participants:
Dr Lynne Hocking,
Dr Alison Gartland

Many different in vitro cell culture models are available for the study of bone cell biology – which is fundamental to understanding the factors that govern bone remodelling and for developing new therapies for musculoskeletal disorders.

This workshop will provide practical advice and guidance about available in vitro cell culture models related to musculoskeletal biology. This will include mouse and human osteoblast, osteoclast and chondrocyte cell types (both clonal and primary) as well as pluripotent stem cells. In addition, current novel approaches to analysis of gene and protein processing as well as intra- and extra-cellular signalling will be discussed.

During the workshop we will provide an overview of the existing methodology followed by a discussion that will be participant-focused – you bring the questions and we will bring the answers.

Suggested Reading Material:

Supporting health professionals in the field of osteoporosis and bone health

The National Osteoporosis Society supports a growing network of health professionals in a number of ways:

- Osteoporosis Resources for Primary Care website (www.osteoporosis-resources.org.uk)
- Leading scientific journal Osteoporosis Review
- Helpline staffed by nurses with a specialist knowledge of osteoporosis
- A wide range of free information leaflets for use in clinical settings and practical guides
- Study days and professional training courses
- Free Allied Health Professional Network online community

Find out more today at: www.nos.org.uk/professionals
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<thead>
<tr>
<th>Time</th>
<th>Monday</th>
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<tr>
<td>9.00–9.25</td>
<td>Educational update</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>8.30–9.00</td>
<td>Update from the National Osteoporosis Society</td>
<td>Claire Severgnini and Professor Terry O'Neill</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>9.25–9.50</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>9.00–10.20</td>
<td>Parallel Session: NOS Presentations from Submitted Abstracts</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<tr>
<td>10.15–10.45</td>
<td>Refreshments</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>10.45–11.00</td>
<td>Official Opening: Professor Nancy Rothwell, University of Manchester</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<tr>
<td>9.30–11.00</td>
<td>Parallel Session: BRS Presentations from Submitted Abstracts</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>11.00–11.30</td>
<td>Educational Update EU4 Non-pharmacological Interventions: Professor Tash Masud</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>11.00–11.30</td>
<td>IS3 Osteoporosis – A Primary Care Perspective: Dr Graham Davenport</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>11.00–11.30</td>
<td>IS4 Epigenetics: Dr Caroline Relton</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>11.30–12.00</td>
<td>EU5 Pharmacological Therapy for Osteoporosis: Professor David Hosking</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<tr>
<td>11.30–12.00</td>
<td>BRS Symposium on Extracellular Signalling BRS1 – The Role of Nitric Oxide on Bone: Dr Sophie Jamal</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>11.30–12.30</td>
<td>IS5 Sarcopenia: Causes and Consequences: Professor Avan Aihie Sayer</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>11.30–12.30</td>
<td>Parallel Session: BRS Presentations from Submitted Abstracts and Oral Posters</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>11:00–11:30</td>
<td>Refreshments</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>11:30–12:00</td>
<td>Linda Edwards Memorial Lecture IS7 Legends of Osteoporosis: Bone: A Target for Systemic Disease: Professor Juliet Compston</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>12.00–12.30</td>
<td>EU6 Prevention and Management of Falls: Professor Dawn Skelton</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>12.00–12.30</td>
<td>BRS Symposium on Extracellular Signalling BRS2 – The Role of Purinergic Signalling in Bone: Dr Alison Gartland</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>12.00–12.50</td>
<td>Annual General Meeting of the Bone Research Society</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>12:00–12.30</td>
<td>Poster Viewing Session A and Lunch</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
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<td>12:30–14:00</td>
<td>Poster Viewing Session B and Lunch</td>
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<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>14.00–14.30</td>
<td>IS1 Fracture Healing: Professor David Marsh</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
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<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>14.40–15.40</td>
<td>Workshops M1–M8</td>
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<td>15.45–16.00</td>
<td>Break</td>
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<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>17.00–17.30</td>
<td>IS2 Paget’s Disease Symposium: Professor Socrates Papapoulos &amp; Professor Stuart Ralston</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>17.30–18.00</td>
<td>Break</td>
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<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>18.00–19.00</td>
<td>Conference Session: Putting Patients First</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
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<td>19:00–20:00</td>
<td>Conference Dinner</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
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<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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<td>20:00–20:30</td>
<td>End of Day 1</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
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<td>20:30–21:00</td>
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<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
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<td>21:00–22:00</td>
<td>End of Day 3</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
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<td>22:00–23:00</td>
<td>End of Conference</td>
<td>EU2 Epidemiology and Impact of Osteoporosis: Dr Nick Harvey</td>
<td>8.00–8.30</td>
<td>EU1 Pathology of Osteoporosis: Professor Stuart Ralston</td>
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**Registration Opens**

*Conference Dinner*