Methods: There are no trials comparing different steroid dosing regimens in either GCA or PMR. The recommendations are based on BSR guidelines on GCA and PMR and are mostly based on level 3 evidence.

Results: GCA: The key issues are early recognition, and prompt institution of corticosteroid therapy and TA biopsy. Delay in recognition and treatment may cause ischemic vision loss. Uncomplicated GCA (no jaw claudication or visual disturbance) should be given 40-60 mg prednisolone daily; evolving visual loss (recent onset visual symptoms over 6-12 h) or amaurosis fugax (complicated GCA): intravenous methylprednisolone 500-1000 mg for 3 days before oral steroids; established visual loss: 60 mg prednisolone daily, to protect the contralateral eye; large-vessel disease: treatment along the lines of systemic vasculitis protocols is recommended, although this needs to be individualized. Initial steroids should be continued for 3-4 weeks and steroids tapered gradually after gaining disease control. Relapses: Return of headache - treat with previous higher CS dose; Jaw claudication with 40-80 mg prednisolone; recurrent eye symptoms either 60 mg prednisolone or intravenous methylprednisolone; symptoms of large-vessel disease need investigation with MRI/PEF and systemic vasculitis treatment protocols.

PMR: Low-dose initial corticosteroids, initial dose 15 mg prednisolone, with gradual tapering over 1-2 years is advised. The dose is adjusted according to disease severity, comorbidities, fracture risk, patient wishes or adverse events. Relapses are treated on principles similar to GCA. Isolated elevated ESR/CRP does not need CS dose adjustment.

Conclusions: Adjunct agents: These are currently recommended following 2 or more relapses. Methotrexate has been shown in a meta-analysis to have a modest disease modifying effect. Azathiprine is also used and a small open case series has shown benefit of leflunomide in both GCA and PMR.

Biologics: Neither infliximab nor etanercept have been found to be effective in controlled trials although there are case reports of success. Rituximab has been reported effective in 2 cases. A trial of abatacept in large vessel vasculitis is ongoing. IL-6 levels are elevated in both PMR and GCA and cytokine blockade with tocilizumab may be a future treatment target. Newer formulations and modes of CS delivery may also be candidates for future studies.

Disclosures: The author has declared no conflicts of interest.

References

The era of biologics for children and young people with rheumatological disease

IP98. UNDERSTANDING THE MECHANISM OF DISEASE AND RESPONSE TO THERAPY IN CHILDHOOD ARTHRITIS
Lucy Wedderburn
Department of Rheumatology, Institute of Child Health, University College London and Great Ormond Street Hospital, London, United Kingdom

Health care professionals caring for children with arthritis are working in an era of increasing number of biologic treatments for arthritis, yet with a lack of evidence base for how to predict those who will respond, to any individual drug, or those who will not. Without such biomarkers of prediction, we cannot yet practice ‘personalized’ medicine. Instead, we step through disease modifying agents, and then biologics, with choices led largely by availability or cost rather than what is ideal for each child. In this talk we will consider recent data that throw light on mechanisms of different disease subtypes in JIA review the current evidence base for biomarkers that identify mechanisms which underlie clinical heterogeneity of childhood arthritis, and then consider ongoing studies that aim to generate biomarkers for use in predicting response to therapy and ultimately to aid clinical choice for each child.

Disclosures: The author has declared no conflicts of interest.

IP99. THE YOUNG ADULT WITH JUVENILE IDIOPATHIC ARTHRITIS AND BIOLOGICS
Helen Foster
Paediatric Rheumatology, Musculoskeletal Research Group, Institute of Cellular Medicine, Newcastle University, Newcastle upon Tyne, United Kingdom

Over one third of patients with Juvenile Idiopathic Arthritis (JIA) continue to have active disease into their adult years and it is not uncommon for de novo flares of disease to occur after years of remission. Many such patients warrant treatment with biologics and indeed many young people transfer to adult care having started their biologic treatment under the care of paediatric rheumatologists. Outcome studies and Registries are yet to report the long term impact and safety of biologics in JIA into adulthood and there is a paucity of studies reporting use of biologics in young adults with JIA. Current UK national guidance for the use of biologics in JIA does not extend into the adult years and there is no consensus as to the optimal use of different biologic agents in this age group. Indeed there are no data on any of these subtypes of JIA. In many instances the use of biologics is based on the extrapolation of studies in adults with rheumatoid arthritis and clearly for many patients their lives are transformed by the introduction of biologic agents. In this talk, we describe the need for Guidance on the optimal use of biologics in adult JIA subtypes, Guidance on the information needs for young people and their clinicians to inform decision making and Consensus policy statements to facilitate equitable access to these important group of drugs.

Disclosures: H.F. has received honoraria from Pfizer, Schering Plough and Abbott.

The era of biologics for children and young people with rheumatological disease

IP100. GENETICS AND BIOLOGIC USE IN AUTOINFLAMMATORY DISEASE
Paul Brogan
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The autoinflammatory diseases were previously referred to as periodic fever syndromes, and are disorders of innate immunity. They are characterized by recurring episodes of fever and constitutional upset usually beginning very early in life and persisting into adulthood. There is often normal health between attacks. Systemic inflammatory symptoms typically affect the serosal surfaces, joints, skin, brain and eyes. Symptoms are always accompanied by biochemical markers of
inflammation such as raised ESR, CRP, serum amyloid A and leucocytosis. With the notable exception of severe CAPS (see below) most are compatible with near normal life expectancy except for the risk of developing AA amyloidosis in later life. Despite similarities in symptoms, they have differing aetiologies, inheritance, duration and clinical features.

The hereditary autoinflammatory diseases are associated with mutations in genes involved in innate immunity and generally the onset is early in childhood affecting both sexes equally. Seven major syndromes (below) are currently recognized, although novel autoinflammatory diseases are rapidly being discovered as a direct result of advances in genetic technologies.

- Familial Mediterranean fever (FMF)
- TNF receptor associated periodic syndrome (TRAPS)
- Mevalonate kinase deficiency (MKD) (also known as Hyperimmuno-globulin D and periodic fever syndrome (HIDS))
- Cryopyrin associated periodic syndrome (CAPS) (subdivided into Familial cold autoinflammatory syndrome (FCAS), Muckle Wells Syndrome (MWS) and chronic infantile, neurological, cutaneous, and articular syndrome/neonatal onset multisystem inflammatory disease (CINCA/NOMID))
- Pyogenic arthritis, pyoderma gangrenosum and acne (PAPA) syndrome
- Deficiency of IL-1 receptor antagonist (DIRA)
- Blau syndrome/early onset sarcoidosis (EOS)

There are now effective treatments for most patients with FMF, CAPS and DIRA and although not always quite as effective, good treatments are available for the remainder. Table 1 provides a summary of the genetics and treatments currently used for the hereditary autoinflammatory disorders.

This talk will provide a clinical overview of these major hereditary autoinflammatory diseases, current therapies, and will summarize an approach for the discovery of new genetic fever syndromes using emerging genetic technologies.

**Disclosures:** The author has declared no conflicts of interest.

<table>
<thead>
<tr>
<th>Autoinflammatory disease</th>
<th>Gene</th>
<th>Mode of inheritance</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>FMF</td>
<td>MEVF</td>
<td>Autosomal recessive</td>
<td>Colchicine</td>
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<tr>
<td>TRAPS</td>
<td>TNFRSF1A</td>
<td>Autosomal dominant</td>
<td>Etanercept? anti-IL1, High dose corticosteroids</td>
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<td>HIDS</td>
<td>MVK</td>
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<td>NLRP3</td>
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<tr>
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</tr>
<tr>
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<td>IL1RN</td>
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<td>IL-1Ra</td>
</tr>
</tbody>
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**Table 1.**

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**Clinical musculoskeletal research in the UK (Arthritis research UK and BSR joint session)**

**IP101. CURRENT AND FUTURE ACTIVITY IN RHEUMATOLOGY TRIALS**

Alan Silman
Rheumatology, Arthritis Research UK, Manchester, United Kingdom

Abstract not provided.

**Disclosures:** The author has declared no conflicts of interest.

**IP102. BUILDING A UK CLINICAL RESEARCH NETWORK IN RHEUMATOLOGY**

David Scott
Rheumatology, King’s College London, London, United Kingdom

**Background:** A “National Arthritis Research Network” is needed to encourage and enable most rheumatology and orthopaedic units to participate in clinical research studies. Increasing recruitment to clinical studies will be of crucial benefit to our specialty.

**Methods:** Rheumatologists and orthopaedic surgeons need to enrol patients under their care into clinical research studies. Both randomised controlled trials and observational studies are important in furthering our understanding of the effects of treatment and therefore providing optimal clinical care. This presentation will consider ways in which the enrolment of patients into clinical research studies could be enhanced.

**Results:** Clinical research is challenging to deliver. Much of the current administrative work involved in setting up individual studies is repetitive. There are currently substantial delays involved in the process. By establishing a national research network it will be possible to reduce these delays to a minimum. This is because the network could ensure all relevant data is held in a secure on-line database and would not need to be collected repeatedly. It will also be possible to identify any specific causes of delay and to ensure these are overcome by concerted action.

The network will simplify the enrolment of patients into national clinical studies by:

a. Liaising with Chief Investigators and their research staff to both assist in overcoming the various regulatory issues involved in initiating research and providing information about individual units wishing to participate in research studies.
b. Interacting with Research and Development Departments and the local Clinical Research Networks to ensure research studies are started and completed in a timely manner.
c. Provide advice and training for clinicians involved in research.

**Conclusions:** The network should involve all the main constituents involved in promoting research in arthritis. This includes rheumatologists, orthopaedic surgeons, allied health professionals and funders. It should also take into account the needs of patient groups.

Although the concept of a network is under development, it has the potential to be a key method of helping specialists deliver their research goals. The views of BSR members are vital in defining how a register should be organised and function. There is a unique opportunity to streamline clinical research by acting in unison as a community and engagement of a range of BSR members, spanning senior consultants, trainees and allied health professionals, will prove invaluable.

**Disclosures:** The author has declared no conflicts of interest.

**IP103. WHAT ARE THE PRACTICAL ISSUES?**

Deborah Symmons
Rheumatology, University of Manchester, Manchester, United Kingdom

Abstract not provided.

**Patient and public involvement in musculoskeletal research**

**IP104. PATIENT AND PUBLIC INVOLVEMENT IN MUSCULOSKELETAL RESEARCH: THE NATIONAL AGENDA**

Sarah Buckland
INVOLVE Coordinating Centre, University of Leeds, Eastleigh, United Kingdom

INVOLVE is a national advisory group which supports active public involvement in NHS, public health and social care research (www.involve.org.uk). INVOLVE was established in 1996 and is funded by the National Institute for Health Research (NIHR). We use the term ‘public’ to include: consumers, patients and potential patients, service users, carers and parents, long term users of health and social services, disabled people and organisations representing the public. Active involvement is an active partnership between the public and researchers, where research is carried out ‘with’ the public rather than ‘to’, ‘for’, or ‘about’ the public. For example, advising on a research project, assisting in the design of a project, or involvement in undertaking the research.

Members of the public bring perspectives and skills that are not always the same as those of researchers and health and social care professionals. Their involvement helps to ensure that the research process is focused on what is important to people and is therefore more relevant and acceptable to the users of services. We believe that