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)
- 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 1.**

<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>MEFV</td>
<td>Chromosome 16</td>
<td>Colchicine</td>
</tr>
<tr>
<td>TRAPS</td>
<td>TNFRSF1A</td>
<td>Chromosome 12</td>
<td>Etanercept? anti-IL1, high dose corticosteroids</td>
</tr>
<tr>
<td>HIDS</td>
<td>MVK</td>
<td>Chromosome 12</td>
<td>Anti-TNF and anti-IL-1 therapies</td>
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<tr>
<td>CAPS</td>
<td>NLRP3</td>
<td>Chromosome 1</td>
<td>Anti-IL1 therapies</td>
</tr>
<tr>
<td>PAPA</td>
<td>PSTPIP1 (CID2BP1)</td>
<td>Chromosome 15</td>
<td>Anti-IL or Anti-TNF therapy</td>
</tr>
<tr>
<td>DIRA</td>
<td>IL1RN</td>
<td>Chromosome 2</td>
<td>IL-1ra</td>
</tr>
<tr>
<td>Blau Syndrome</td>
<td>NOQ2 (CARD15)</td>
<td>Chromosome 16</td>
<td>Corticosteroids</td>
</tr>
</tbody>
</table>

**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 delivers 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.