common cortical network that, when activated, leads to the perception of pain. Activation of this circuit can result from direct stimulation of peripheral nociceptors or from the disruption of endogenous modulatory circuits in the CNS that allows normally nonpainful stimuli to access the cortical pain system. Using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), we find that a network of cortical regions is reliably activated by experimental pain and chronic clinical pain states, including the primary and secondary somatosensory cortices (S1 and S2), the anterior cingulate cortex (ACC) and the insular cortex (IC). This network is activated during pain produced by stimuli as divergent as cutaneous heat, visceral pain produced by esophageal distension, or tactile allodynia produced by capsaicin application. Our imaging data show that allodynia related to peripheral or CNS damage activates the same cortical pain system as that activated during acute pain states. Other experiments suggest that these various cortical regions have a differential role in processing the sensory and affective dimensions of pain. Experimental manipulations that selectively alter the affective component of pain predominantly modulate activity in ACC, whereas those that alter the sensory component of pain modulate activity in the other cortical regions, particularly S1 cortex. Finally, whether the cortical areas are activated by experimental pain or by aberrant chronic pain states, the pain-evoked activation can be modulated by cognitive factors, such as direction of attention.

Molecular genomics – Thursday 14 June, 14.00-15.30/South Hall

SP0037 WHAT HAS BEEN ACHIEVED AND WHAT CAN BE EXPECTED IN RHEUMATOLOGY?

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Major advances have already been made in Rheumatology, many of which have translated into improved clinical care and management. These have largely centred around:

Descriptive studies. These have resulted in:

- a better classification/diagnosis and thus treatment of rheumatic conditions
- a clearer understanding of the natural history of diseases
- epidemiologically defined disease parameters (e.g. prevalence, incidence, geographical distribution, age at onset, gender ratios)

Imaging and definition of relevant laboratory tests

- higher resolution imaging of hard and soft tissues for appreciating both early disease processes and disease progression/severity.
- Laboratory investigations, as useful aids for diagnosis (autoantibody profiles, rheumatoid factor) and disease activity (e.g. CRP)

Treatments and interventions

- The development of effective and lasting joint replacement prostheses
- The introduction of better treatments (e.g. Cox2 inhibitors) and biological based therapies to antagonise inflammatory cytokines and processes (e.g. TNF-alpha antagonists)

What can be expected in the future for rheumatology remains speculation but exciting prospects are likely for clinical medicine in the post human genome sequencing era. A primary objective will be the identification of the genetic and environmental components to disease aetiopathogenesis. These should lead to:

- a better understanding of disease processes and classification resulting in more appropriate treatments.
- The identification of novel targets for pharmaceutical intervention.
- An ability to predict disease risk and disease outcome, permitting early screening and a more rapid or aggressive treatment plan.
- The characterisation of gene-environment interactions, resulting in health education and changes in life style or in possible prophylactic intervention.
- The identification of the pharmacogenetic basis for response to drugs and why there is variation in efficacy or adverse drug reactions. This should lead to personalised medicine.

New developments in molecular biology and cell biology are also likely to impinge on rheumatology in the areas of:

- Tissue Engineering
- Gene therapy
- Stem cell transplation

Genomics, genetics basis of disease and HLA/T cell recognition

AB0001 T-CELL IMMUNITY IN HLA-B27+ AND HLA-B27- PATIENTS WITH REACTIVE ARTHRITIS (ReA) OF DIFFERENT AETIOLOGY

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Background

Objectives Estimation of T-cell immunity peculiarities in HLA B27+ and HLA B27- patients with reactive arthritis (ReA) of different aetiology.

Methods We observed 67 pts with ReA: male -62 pts., female 5 pts., mean age 27 ± 3 , 2 yrs., men duration of illness 24 ± 6 months. In all patients triggering infections were detected: in 36 pts. high levels of chlamydia trachomatis IgG and IgM together with HSV I IgG and IgM were observed, in 16 pts. serological activation of enterobacterial infection and in 10 pts. triggering agent was not found. Specific monoclonal antibodies were used to detect CD3⁺, CD4⁺, CD8⁺, CD16⁺, CD19⁺, CD22⁺ and presence of HLA-B27 antigen.

Results It was estimated that all patients with chlamydial infection were HLA-B27 positive. This pts had higher levels of CD4⁺ ($p < 0,001$), CD3⁺ ($p < 0,01$), CD22⁺ ($p < 0,01$), CD19⁺ ($p < 0,001$) and increased level of IgG ($p < 0,001$) and CIC ($p < 0,01$) in comparison to HLA-B27 negative pts. Presence of HLA-B27 correlated with duration and activity of ReA.

Conclusion Chlamydial infection often associates with presence of HLA-B27 antigen. This pts. have more expressed changes in T-cell immunity that leads to the longer duration and severe activity of the disease.