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Immunology  
Bibliography

Brand D. The Mouse Model of Collagen-Induced Arthritis. Methods in Molecular Medicine, Vol 102: Autoimmunity: Methods and Protocols

-This paper goes in depth into how to actually go about inducing CIA in mice, with a description of the protocol and methods to do so. It has little to do with the actual immunopathology of CIA. It mentions the structure, problems with experimentation, how to inject, various levels of inflammation, and everything else needed to understand how to go about handling these experiments in the lab.


-This paper goes in depth into the various process at play in CIA, focusing on both B and T cell response to CII. It is an in depth look at the pathogenisis of the process, focusing in how the various MHC II molecules function to begin CIA in various strains of mice. It is the best paper I encountered thus far on the topic.


-This paper looks into development of transgenic reagents to study various autoimmune diseases, with just a brief discussion on RA, and thus is not that useful of a paper.

Holm B.C. et al Adjuvent oil induces waves of arthritogenic lymph node cells prior to arthritis onset. Clinical Experiment Immunology 2004; 137, 59-64

-This paper is one that I will present for discussion. It looks into the development of T helper one cells in the lymph nodes, and at what time point do they become capable of actually being able to transfer arthritis to irradiated mice post injection of squalene. It looks at cells originating from both draining lymph nodes and also cells which originate from non-draining lymph nodes. Both are able to transfer arthritis, but cells from draining lymph nodes are much more effective, and sooner.

Patten C. et al Characterization of Pristane-Induced Arthritis, a Murine Model of Chronic Disease. 2004; vol 50: 3334-3345

-This paper reveals that it is the presence of certain microbes which allows pristane to be effective in producing arthritis. Also, it reveals that the arthritis is not due to a single antigen, but a broad response, to many antigens present in the joint.

-The SCID mouse is the central focus for studying SA cells, which are revealed to release certain matrix degrading proteins, with the use of various RNA probes, demonstrating that it could be the balance between MMPs and TIMP that could be a focus for future research, as far as degradation of the synovium.

Jirholt J. The genetics of rheumatoid arthritis and the need for animal models to find and understand the underlying genes. Arthritis Research 2000; vol 3, No. 2

-I took just a piece from this paper, because I was interested in a general description of human models of RA. This paper briefly mentions the use of linkage analysis, but a more common method of looking for alleles which maybe shared between relatives who have developed RA, and a brief sentence about twins who share the disease, leading to the conclusion that its not just a result of environment, but genetics.


-This paper goes into a general description of what we have learned from all studies with RA models. It is a short paper, describing the cytokines which are responsible for joint destruction, and later the cytokines which may oppose this forces. Leading to the conclusion that the overall balance of cytokines in the synovium is the key to understanding the process. Also, gives a brief note to the RANK/RANKL/OPG balance which is effected by TNF.

Wooley. The usefulness and the limitations of animal models in identifying targets for therapy in arthritis. Best Practice and Research Clinical Rheumatology; vol 18, 47-58

-I took from this paper a discussion of transgenic models of RA, with an emphasis on the viral model, cytokines, KBXNT, and also various HLA transgenics. It has a brief paragraph about each one. The point of the discussion is to show that these transgenics allow us to study a single gene event in a mouse, and that these mice become victims of RA without induction using antigen, or adjuvant (some of them).