

Dr. L.A. Trouw

Leendert Trouw was born in Rotterdam, The Netherlands, and studied biology at Leiden University. Following his PhD training with Prof.Dr. Moh Daha in Leiden, he moved to Sweden to work as a post-doc with Prof. Dr. Anna Blom, at Lund university, in Malmö. He currently holds a position as an assistant-professor at the Department of Rheumatology of the LUMC, in Leiden.



His main research interest are autoantibodies and complement. Over the years he and his team have studied the occurrence and pathological role of several autoantibodies in systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) and their capacity to activate complement.

During his PhD training he studied anti-C1q autoantibodies in murine models of lupus nephritis and established why such antibodies can be found in healthy people without pathological consequences and why they are pathogenic in lupus patients. During his postdoc in Malmö, Sweden, he mainly focused on the interaction between complement and autoantigens such as DNA and apoptotic cells again mostly in the context of SLE. Now in his current position at the Dept. of Rheumatology at the LUMC, in Leiden the Netherlands, his team focuses on anti-citrullinated protein antibodies (ACPA) and their biochemical and biological properties in relation to clinical presentation of RA as well as genetic variants of complement components in relation to RA. Some of the work is highlighted in the papers as listed below:

Anti-C1q autoantibodies in murine lupus nephritis.

Trouw L.A., Seelen M.A., Visseren R., Duijs J.M.G.J., Benediktsson H., De Heer E., Roos A., Van Kooten C., Daha M.R.

Clinical and Experimental Immunology 2004; 135: 41-48

Anti-C1q autoantibodies deposit in glomeruli but are only pathogenic in combination with glomerular C1q-containing immune complexes.

Trouw L.A., Groeneveld T., Seelen M.A., Duijs J.M.G.J., Bajema I.M., Prins F.A., Kishore U., Salant D.J., Verbeek J.S., Van Kooten C., Daha M.R.

Journal of Clinical Investigation 2004 sept. 114 (5) 679-88

C4b-binding protein binds to necrotic cells and DNA, limiting DNA release and inhibiting complement activation.

Trouw L.A., Nilsson S.C., Goncalves I., Landberg G., Blom A.M.

Journal of Experimental Medicine 2005 Jun 20;201(12) 1937-48

C4b-binding protein and factor H compensate for the loss of membrane-bound complement inhibitors to protect apoptotic cells against excessive complement attack.

Trouw LA, Bengtsson AA, Gelderman KA, Dahlback B, Sturfelt G, Blom AM.

Journal of Biological Chemistry 2007 Sep 28;282(39):28540-8.

Anti-cyclic citrullinated peptide antibodies from rheumatoid arthritis patients activate complement via both the classical and alternative pathways.

Trouw LA, Haisma EM, Levarht EW, van der Woude D, Ioan-Facsinay A, Daha MR, Huizinga TW, Toes RE.

Arthritis Rheum. 2009 Jul;60(7):1923-31.