Hilda de Jong
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HJI de Jong, RIVM, Laboratory for Health Protection Research; Maastricht University Medical Centre, Toxicogenomics; Utrecht University, Pharmacoepidemiology and Clinical Pharmacology

As a PhD-student in Pharmacoepidemiology and Immunology, I attended the 27th International Conference on Pharmacoepidemiology and Therapeutic Risk Management 2011 in Chicago, IL, US. This conference focus mainly on drug exposure or methodological issues in pharmacoepidemiology but this year the organisation included a special session about rheumatic condition therapies in the program. This session draw my attention because of all the concerns I had using rheumatoid arthritis (RA) as an outcome in my studies. In the session "rheumatic condition therapies" six researchers presented their work. One of the presenters discussed the role of statins and the risk of acute myocardial infarction in a population-based cohort of RA patients. Because of the population-based study design, I was particularly interested in the way they defined RA in their study. In this study, RA was not confirmed by physicians but the researchers defined RA as individuals with an RA diagnostic code (ICD-9) and at least two physician visits >= 2 months. Furthermore, they excluded the patients with at least two visits subsequent to the second RA visit with diagnoses of other inflammatory arthritis (e.g. psoriatic arthritis, systemic lupus erythematosus), or patients with a RA-coded visit by a non-rheumatologist that was not confirmed on subsequent rheumatologist visit or patients with no subsequent RA-coded visits during at least 5 years of follow-up in order to improve the specificity of RA. The number of prevalent RA cases included in this cohort was consistent with the RA prevalence estimates in general. The researchers used another definition of RA than I did in my study, resulting in almost the same prevalence rate. In all the other presented studies the outcome RA was confirmed by GPs or specialists.
Furthermore, I was interested in the effectiveness and the adverse drug reactions of biologics in RA patients. An analysis in the British Society for Rheumatology Biologics Register showed that switching to rituximab may be of more benefit to switching to an alternative anti-TNF therapy after failing the first anti-TNF in RA patients. Another presentation in the same session discussed the adverse drug reactions of abatacept therapy among patients with rheumatoid arthritis. Abatacept initiation was associated with a higher incidence of claims of tuberculosis.

I had the opportunity to present my own study during the poster walks. The poster presented results of a very specific subject, i.e. statins and the occurrence of polymyalgia rheumatica. Researchers working in the field of pharmacovigilance were interested in the results. With these researchers I had valuable conversations about the increasing number of adverse drug reactions, especially the increasing numbers of autoimmune-related adverse drug reactions.

In conclusion, attending this conference was a great chance for me to discuss my results and see them in a broader perspective, and to get to know more people working in the field of pharmacoepidemiology with a special focus on RA. I want to thank the NVVI for the financial support, allowing me to participate in this international conference.