Table 3. Research Agenda

1. Do we have enough data to recommend a specific treatment in patients with pre-RA at high risk to develop RA?
2. Is the application of a TNF-inhibitor after abatacept, tocilizumab, rituximab or a Jak-inhibitor has failed, safe and efficacious?
3. How safe and efficacious are abatacept, tocilizumab and rituximab after any of the other non-TNF-inhibitor-bDMARDs or a tsDMARD has failed?
4. How safe and efficacious is the use of an IL-6 pathway inhibitor if another IL-6 pathway inhibitor/a JAK-inhibitor has failed?
5. How safe and efficacious is the use of a JAK-inhibitor after another Jak-inhibitor has failed?
6. How safe and efficacious is the combination of a JAK-inhibitor with a bDMARD, such as a TNF-inhibitor?
7. Does the risk stratification for bDMARD/tsDMARD initiation based on presence of good or bad prognostic factors as recommended by EULAR translate into improved outcomes for both prognosis groups?
8. Do patients who lack poor prognostic factors benefit as much from a switch or addition of a csDMARD as from the addition of a bDMARD?
9. Is tapering of bDMARD monotherapy possible?
10. Will RCTs on tapering of bDMARDs and tsDMARDs designed to following predefined predictors for maintenance of good outcomes after withdrawal of bDMARDs show success?
11. How good is patient adherence to a bDMARD or tsDMARD and can non-adherence explain secondary loss of efficacy?
12. How can refractory RA be best defined, and what is the optimal treatment approach?
13. Can we identify new biomarkers to stratify patients and to predict therapeutic response and pending lack of response?
14. Which other factors, e.g. life-style characteristics, treatment history, allow to make the best possible therapeutic decisions?
15. Do JAKi confer specific safety signals of concern?
16. Can the identification of disease phenotypes inform tailored therapeutic use?
17. Do the different bDMARD/tsDMARD lead to comparable improvements in co/multimorbidities?
18. Does the concomitant use of glucocorticoids at very low doses (1-3mg prednisone equivalent) increase therapeutic success without producing unacceptable side effects?
19. Will therapeutic drug monitoring improve disease course and outcome and support decisions about switching within or between drugs?
20. Is leflunomide equivalent to MTX as first line csDMARD therapy?
21. For active RA patients who have failed multiple drugs, are there combinations that may be more successful such as JAK-inhibitor with bDMARD?
22. Is secondary loss of efficacy dues to non-adherence or a consequence of true loss of efficacy of a given drug and if the latter, what is the reason for this loss of efficacy?
23. Can taxonomy of RA be improved to guide therapeutic decisions?