**Objectives:** To assess in a real-world, observational study whether treatment with ABA had a similar malignancy risk as other biologics, with or without MTX, when used as the initial bDMARD for RA.
**Methods:** The Truven MarketScan® Commercial and Supplemental Medicare databases were used to identify adult pts diagnosed with RA who initiated bDMARD treatment with ABA or another bDMARD between Jan 2007 and Dec 2014. Other bDMARDs included adalimumab, anakinra, certolizumab, etanercept, golimumab, infliximab, rituximab and tocilizumab. Pts were required to have ≥6 months (M) of continuous health plan enrolment before bDMARD initiation (index date) and deemed to have initiated a treatment if there was no claim for any bDMARD in the limited 6M period before bDMARD initiation. Pts who had a malignancy in the baseline 6M period were excluded. Pts were followed up from the date of the first bDMARD prescription initiation, either ABA or another bDMARD, until occurrence of a malignancy (identified by ICD-9 diagnosis code), end of enrolment in the database or end of data collection, whichever occurred first. A 6M latency period was included. Propensity scores of ABA initiation were estimated from the baseline covariates using a logistic regression model, and trimmed to include only pts with ranges common to both ABA-exposed and comparator bDMARD cohorts. The Cox proportional hazard regression model was used to provide an estimate of the hazard ratio (HR) of malignancy associated with ABA initiation compared with initiation of another bDMARD, adjusted for age and deciles of the propensity score after trimming.
**Results:** A total of 5391 pts were identified as above as having initiated bDMARD therapy with ABA and 74,315 initiated with another bDMARD, with follow-up of <8 yrs (mean 2.1 yrs). Pts who initiated ABA vs other bDMARDs were older (mean 55 vs 52 yrs), had more co-morbidity, used less MTX (49 vs 57%) and more other non-bDMARD (41 vs 36%) at baseline. After trimming on propensity scores, 565 pts developed a malignancy after ABA (incidence rate 5.0 per 100/yr) compared with 5750 after another bDMARD (incidence rate 3.6 per 100/yr). The adjusted HR (95% CI) of any malignancy with ABA initiation relative to other bDMARDs was 1.18 (1.06, 1.30), while for any malignancy excluding non-melanoma skin cancer it was 1.17 (1.02, 1.34). The risk (HR; 95% CI) was not significantly elevated for lung cancer (1.11; 0.70, 1.76), female breast cancer (1.21; 0.91, 1.62) and lymphoma (1.21; 0.77, 1.90).
**Conclusions:** In this large, real-world study of pts treated for RA, the incidence of the most common malignancies of breast, lung and lymphoma were not significantly increased in pts using abatacept as first-line bDMARD treatment compared with other bDMARDs, though the confidence intervals were wide. The slight increase in the risk of overall malignancy with abatacept needs further investigation, particularly to assess the potential for residual confounding and the impact of the short baseline period.
**References:**
[1] Mercer LK, et al. Ann Rheum Dis 2015;74:1087–93.
**Disclosure of Interest:** S. Suissa Consultant for: Bristol-Myers Squibb, S. Dell'Aniello: None declared, T. Simon Shareholder of: Bristol-Myers Squibb, Employee of: Bristol-Myers Squibb
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## FRI0130 RATES AND RISK FACTORS OF NEW-ONSET PSORIASIS UNDER DIFFERENT BIOLOGIC AGENTS AND CONVENTIONAL SYNTHETIC DMARD TREATMENT

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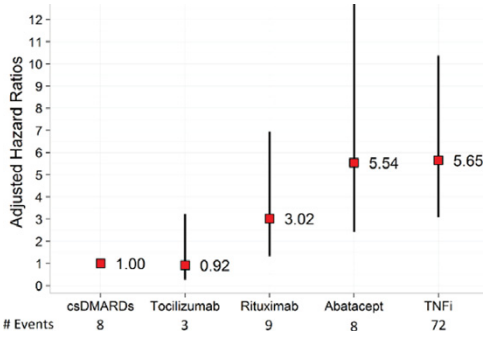
**Background:** Psoriatic skin disease is a burdensome, sometimes painful, dermatologic condition which was reported to occur as an adverse event (AE) during TNF-inhibitor (TNFi) treatment of rheumatoid arthritis (RA). Single case reports revealed the occurrence of psoriasis also during treatment with non-TNFi, but the magnitude under those agents remains unclear.
**Objectives:** To compare incidence rates of psoriasis in RA under treatment with different biologic and conventional synthetic (b/cs)DMARDs and to investigate risk factors.
**Methods:** We used data of 12,722 patients (53,585 patient years (py)) enrolled with the start of a b/csDMARD in the German biologics register RABBIT. Patients were required to have no psoriasis at baseline and at least one follow-up. All psoriatic events (PsE) reported until 30 April 2016 were selected and assigned to treatments administered within 3 months prior to the event. Crude incidence rates (IR) of PsE were calculated per 1,000py. Cox regression was applied to investigate risk factors for the occurrence of PsE with and without inverse probability weights (IPW) to adjust for confounding by indication.
**Results:** 96 PsE were reported, with only 6 of them categorized as being serious. The median time between enrollment in the cohort and onset of psoriasis was 19 months (IQR:11–45 months). 21 of all PsE (22%) were palmoplantar manifestations of which 9 were reported as pustular type.
Compared to csDMARD treatment with a crude IR of 0.44/1,000py (95% CI 0.2;0.9), the IRs found under TNFi (IR 2.99 (95% CI 2.3;3.8)) and abatacept (IR 3.99 (95% CI 1.7;7.9)) were significantly higher. In patients treated with rituximab (IR 1.8 (95% CI 0.8;3.4)) or tocilizumab (IR 0.7 (95% CI 0.1; 2.0)) IRs for PsE were not significantly different from csDMARD patients. Across TNFi, the IR varied insignificantly.
Adjusted regression analysis showed higher risk for PsE with TNFi, abatacept and rituximab (graph). Female sex (adjusted hazard ratio (HR) 1.8 (1.0;3.3)) and being rheumatoid factor negative (HR 1.6 (1.0;2.6)) were additional significant risk factors. Smoking (HR 1.6 (1.0; 2.5)), age (HR 1.0 (0.98;1.01)), glucocorticoids per 5 mg/d increase (HR 1.1 (1.0;1.2)), and prior (≤6months) skin infections (HR 2.2 (0.5;9.7)) were not significantly associated. Replacing glucocorticoids with DAS28 did not show differing results. Adjustment with IPW attenuated the effect of rheumatoid factor (p=0.4) but smoking was significantly associated with a higher risk (p<0.01).




**Conclusions:** This is the first analysis comparing the incidence of psoriasis under biologics with different modes of action within one cohort. Our results confirmed a higher risk for TNFi [1] and showed a similar result for abatacept. A lower but still significant increased risk was found for rituximab, whereas there was no difference for tocilizumab compared to csDMARDs. New onset psoriasis is a rare and most often non-serious event. The number needed to harm is 334 patients treated with TNFi for one year to observe one PsE.
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**References:**
[1] Hernandez et al., Arthritis Care Res 2013; 65:2024–31.
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## FRI0131 THE 2010 ACR/EULAR CRITERIA ARE INSUFFICIENTLY ACCURATE IN THE EARLY IDENTIFICATION OF AUTOANTIBODY-NEGATIVE RHEUMATOID ARTHRITIS: RESULTS FROM THE LEIDEN-EAC AND ESPOIR COHORTS

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**Background:** The 2010-ACR/EULAR criteria were derived to classify RA earlier in time. Previous studies indeed observed that the 2010-criteria were fulfilled earlier than the 1987-criteria. This study determined whether the 2010-criteria perform equally in the early detection of autoantibody-positive and autoantibody-negative RA.
**Objectives:** To compare the performance of the 2010-criteria between autoantibody-positive and autoantibody-negative RA within two different early arthritis cohorts.
**Methods:** From the total Leiden-EAC (n=3448) and ESPOIR (n=813) RA-patients who fulfilled the 1987-RA criteria at 1-year but not at presentation were selected (n=515 and n=53, respectively). These RA-patients were studied on the presence of ACPA and RF, and on fulfilling the 2010-criteria at baseline, as 2010-positivity indicated that these RA-patients were earlier identified.
**Results:** In the EAC, 67% of the selected RA-patients did already fulfil the 2010-criteria at baseline. In ESPOIR this was 57%, indeed demonstrating early classification with the 2010-criteria. Among the selected autoantibody-positive RA-patients of the EAC, 85% was identified at baseline already with the 2010-criteria. Within autoantibody-negative RA this was 45% (p<0.001). Similarly within autoantibody-positive RA-patients in ESPOIR 92% was 2010-positive at baseline, whereas this was only 25% within autoantibody-negative RA (p<0.001).
**Conclusions:** The 2010-criteria perform well in the early identification of autoantibody-positive RA, but autoantibody-negative RA-patients are still frequently missed with these criteria. As it has been demonstrated that early