Other factors associated with higher IRR were sex (being woman), type of biological treatment (TNF inhibitors), concomitant therapy (methotrexate), smoking, comorbidities (Charlson Index) and time of evolution of rheumatic disease. No differences were found between pathologies.

Table. Results of the Poisson regression model. Factors associated with risk of incidence of a first adverse event since biological therapy initiation.

	CRUDE IRR (IC95%)	P	ADJUSTED IRR (IC95%)	P
AGE AT THE BEGINNING OF BIOLOGICAL TREATMENT (REF. ADULTS)				
YOUNG	0.78 (0.55-1.11)	0.175	1.04 (0.71-1.53)	0.829
ELDERLY ADULTS	1.61 (1.36-1.91)	<0.001	1.47 (1.20-1.80)	<0.001
ELDERLY	1.99 (1.44-2.76)	<0.001	1.90 (1.32-2.75)	0.001
WOMEN	1.32 (1.19-1.47)	<0.001	1.41 (1.23-1.61)	<0.001
TNF INHIBITOR	0.84 (0.74-0.96)	0.009	0.67 (0.57-0.79)	<0.001
DIAGNOSIS (REF. RA)				
AS	0.80 (0.70-0.91)	0.001	1.04 (0.87-1.26)	0.651
PSA	0.97 (0.86-1.10)	0.642	1.08 (0.92-1.28)	0.327
CORTICOSTEROIDS	1.31 (1.18-1.47)	<0.001	1.10 (0.96-1.26)	0.186
METHOTREXATE	1.42 (1.27-1.59)	<0.001	1.35 (1.19-1.54)	<0.001
OTHER DMARDS	1.16 (1.04-1.30)	0.009	1.16 (1.01-1.32)	0.038
SMOKING (REF. NO)				
SMOKER	1.16 (1.02-1.32)	0.028	1.19 (1.02-1.39)	0.029
FORMER SMOKER	1.61 (1.32-1.97)	<0.001	1.59 (1.23-2.07)	<0.001
COPD	1.37 (0.97-1.94)	0.072	1.12 (0.76-1.66)	0.571
DIABETES	1.31 (1.07-1.59)	0.008	1.30 (1.02-1.65)	0.034
CHARLSON INDEX	1.15 (1.08-1.21)	<0.001	1.10 (1.02-1.19)	0.009
TIME OF EVOLUTION OF DISEASE	1.01 (1.00-1.01)	0.074	1.01 (1.00-1.02)	0.021

Conclusion: A mix of clinical and patient factors seem to explain the appearance of a first AE after biological therapy initiation, with age being one of those explanatory variables.

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SAT0169 RISK OF SERIOUS INFECTIONS IN OFFSPRING ACCORDING TO TNFI SUBTYPES

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Background: TNFi subtypes have differential trans-placental passage; infliximab and adalimumab (both monoclonal immunoglobulins) have the highest transfer, reaching higher fetal than maternal blood levels, while certolizumab (a pegylated Fab fragment) and etanercept (a fusion protein) display the lowest passage (less than 0.25% and 4-7% respectively). Thus, some TNFi subtypes could cause immunosuppression in the offspring. However, to date, there is no data on the risk of serious infections according to TNFi subtypes.

Objectives: We evaluated serious infections in a large group of children born to mothers with chronic inflammatory diseases who used TNFi during pregnancy. Our comparators were unexposed offspring born to affected and unaffected mothers. We determined if the risk of serious infections differed according to TNFi subtypes.

Methods: We identified all women with ≥ 1 hospitalization for delivery after a diagnosis of rheumatoid arthritis (RA), ankylosing spondylitis (AS),

psoriasis (PsO), psoriatic arthritis (PsA), or inflammatory bowel diseases (IBD), and a randomly selected group of unaffected mothers, matched $\geq 4:1$ for age, year of delivery, and state of residence, using MarketScan database (2011-2016). Only women continuously enrolled within MarketScan for ≥ 12 months prior to delivery and with available child linkage were included. We defined TNFi exposure based on ≥ 1 filled prescription and/or infusion procedure code during pregnancy and/or the preconception period. We further refined TNFi exposure based on potential for high (infliximab, adalimumab, golimumab) versus low (certolizumab, etanercept) placental transfer. We ascertained serious infections in the offspring based on ≥ 1 hospitalization with infection as a primary diagnosis, in the first year of life. We performed multivariate analyses, adjusting for maternal demographics, disease type, co-morbidities, pregnancy complications, and drugs (corticosteroids, DMARDs, non-TNFi biologics).

Results: We identified 16,490 offspring of mothers with RA (4,142), AS (381), PsO/PsA (5,743), and IBD (6,731), as well as 164,553 children born to unaffected matched mothers. Among offspring whose mothers had inflammatory diseases, 1,611 (9.8%) were exposed to TNFi during pregnancy, 338 (2.0%) were unexposed during pregnancy but exposed in the preconception period, and 14,541 (88.2%) were unexposed both during the pregnancy and preconception periods. The percent of serious infections in offspring of inflammatory disease mothers with no TNFi exposure (2.1%; 95% CI 1.9, 2.3) was similar to those with TNFi exposure (2.3%; 95% CI 1.6, 3.0), while the percent of serious infections in children born to unaffected mothers was 1.6% (95% CI 1.6, 1.7). In offspring exposed to TNFi with high placental transfer, the percent of serious infections was 2.3% (95% CI 1.6, 3.3), while for TNFi with low transfer, it was 1.7% (95% CI 1.0, 3.1).

In multivariate analyses, we did not observe an increased risk of serious infections in TNFi exposed offspring versus unexposed offspring of mothers with inflammatory diseases (OR 1.0; 95% CI 0.7, 1.5). Results were similar when we restricted TNFi exposure to the third trimester (OR 1.1; 95% CI 0.7, 1.7). In multivariate analyses of children exposed to TNFi with high versus low placental transfer, our point estimate was consistent with increased risk, but the confidence interval was wide, including the null value (OR 1.43; 95% CI 0.66, 3.08).

Conclusion: When we compared the risk of serious infections in offspring exposed to TNFi with high versus low placental transfer, we observed a potential trend for an increased risk, although the confidence interval was wide and included the null value. Our findings stress the need to further study this issue.

Disclosure of Interests: None declared

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SAT0170 A NOVEL FORMULATION OF CT-P13 FOR SUBCUTANEOUS ADMINISTRATION: 30 WEEK RESULTS FROM A PART 2 OF PHASE I/III RANDOMIZED CONTROLLED TRIAL IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: CT-P13 subcutaneous (SC) formulation showed comparable efficacy and safety with CT-P13 intravenous (IV) formulation in rheumatoid arthritis (RA)¹ and Crohn's disease² preliminary studies (Part 1).

Objectives: The purpose of this study was to demonstrate non-inferiority (NI) of efficacy and compare safety profiles of CT-P13 SC to CT-P13 IV in RA patients over 30 weeks of Part 2.

Methods: In this randomized, controlled, double blinded, phase I/III study, RA patients received CT-P13 IV 3 mg/kg at Weeks 0 and 2 and were randomized at Week 6 to receive CT-P13 SC 120 mg every 2 weeks or CT-P13 IV 3 mg/kg every 8 weeks. From Week 30, all patients received CT-P13 SC 120 mg every 2 weeks. The primary efficacy endpoint, change of DAS28 (C-reactive protein [CRP]) from baseline to Week 22, was analyzed by using an analysis of covariance (ANCOVA). Non-inferiority is to be concluded if the lower bound of the 95% CI for the