healthy donors: 0.0015(0.001–0.003) vs 0.003(0.001–0.007) and 0.01(0.005–0.02) vs 0.02(0.01–0.04), respectively, p<0.01 for both cases. At baseline, a significant correlation was found in RA pts between absolute counts of B cells (CD19+CD27+) and CRP (r=0.50, p<0.05); the percentage and absolute counts of plasmablasts (CD19+CD38++CD27+IgD–CD20–) and RF (r=0.41 and r=0.52, p<0.05). After 12 mo of TCZ therapy, 54% of pts were categorized as good responders, 46% of pts – as moderate responders according to the EULAR response criteria. Reductions in the percentages and absolute counts of plasmablasts (CD19+CD38++CD27+IgD–CD20–) were documented after 12 mo of TCZ therapy: 0.15%(0.1–0.3) vs 0.1%(0.01–0.1) and 0.0003(0.00007–0.004) vs 0.001(0.0001–0.0003), respectively, p<0.05. The median percentages/absolute counts of switched memory B cells (CD19+CD27+IgD–) were 6.8%(3.6–11.6)/0.01(0.005–0.02) at baseline; and 3.1%(1.1–4.2)/0.003(0.002–0.006) after 12 mo of TCZ therapy, p<0.05. After 12 mo of TCZ therapy, the median percentages and absolute counts of memory B cells (CD19+CD27+IgD–) became lower in RA pts than in the controls: 1.0%(0.7–1.2) vs 2.2%(1.1–3.0); 0.001(0.006–0.03) vs 0.003(0.001–0.007); 3.1%(1.1–4.2) vs 12.8%(9.3–17.0); 0.003(0.002–0.006) vs 0.002(0.01–0.04), respectively, p<0.05, respectively, for all cases. Other B-cell subpopulations did not change after 12 mo of TCZ therapy as compared to baseline values.

Conclusions: Immuno-phenotyping in pts with active RA showed the decrease in the absolute counts of memory B cells (CD19+CD27+), switched memory B cells (CD19+CD27+IgD–) as compared to healthy subjects. Positive correlation between the counts of memory B cells and plasmablasts and values of laboratory indicators of RA (CRP, RF) suggests that B-lymphocytes may be involved in RA pathogenesis. The reduction in the levels of plasmablasts after 12 mo of TCZ therapy was observed.

Disclosure of Interest: None declared


SAT0180 TWO-YEAR CONSOLIDATED SAFETY DATA FOR ABP 501 IN PATIENTS WITH MODERATE TO SEVERE RHEUMATOID ARTHRITIS

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Background: Biosimilars are expected to have similar long-term safety profiles as originator products.

Objectives: To describe the consolidated, 2-year safety data on ABP 501, an approved biosimilar to adalimumab.

Methods: We combined individual patient data from a 26-week randomized controlled head-to-head study (parent study) comparing ABP 501 with adalimumab (NCT01970475) and its 72-week open-label extension (OLE) study (NCT02114931) in which all patients received only ABP 501. Safety data were reported by exposure-adjusted incidence rate as the number of subjects with the specified adverse events (AEs) per 100 patient-years (100 * n/E).

Adverse Event Category Exposure-adjusted incidence rate n/E (r) ABP 501/ABP 501 (N = 264) Adalimumab RP/ABP 501 (N = 262)

Any AE 187/187.6 (99.7) 197/192.2 (102.5)
Any grade ≥3 AE 32/405.2 (7.9) 30/410.6 (7.3)
Any treatment-related AE 72/345.3 (20.9) 79/344.3 (22.9)
Any grade ≥3 treatment-related AE 6/427.2 (1.4) 5/433.6 (1.2)
Any serious AE 34/407.6 (8.3) 32/410.1 (7.8)
Any treatment-related serious AE 6/427.9 (1.4) 2/434.3 (0.5)
Any events of interest 141/691.5 (53.9) 154/694.7 (66.5)
Infections and infestations 125/289.9 (43.1) 130/287.4 (45.2)
Liver enzyme elevations 25/401.4 (6.2) 20/415.6 (4.8)
Hypersensitivity 19/407.4 (7.7) 22/412.2 (5.3)
Injection site reactions 6/417.3 (1.4) 2/431.8 (0.5)
Hematological reactions 6/421.8 (1.4) 7/426.1 (1.6)
Malignancies 6/423.9 (1.4) 3/433.6 (0.7)
Heart failure 1/428.4 (0.2) 2/435.6 (0.5)

n = number of subjects with the specified AE; E = total subjects exposure-time (patient-years); r = exposure-adjusted incidence rate per 100 patient-years (100 * n/E).

Conclusions: Over the 2-year observation period, there were no meaningful differences in AEs between adalimumab reference product and ABP 501.


SAT0181 EFFECT OF ANTI-IL-6 THERAPY ON SERUM LEVELS OF METABOLIC SYNDROME-RELATED BIOMARKERS IN RHEUMATOID ARTHRITIS PATIENTS

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Background: Metabolic syndrome (MeS) is a pathologic state that encompasses metabolic anomalies such as hyperglycemia, dyslipidemia, obesity and hypertension and that, apart from being a cardiovascular risk factor, it has been associated with chronic inflammatory diseases such as rheumatoid arthritis (RA)1,2. Ghrelin and retinol binding protein-4 (RBP-4) are two biomarkers associated with MeS, and are also linked to different cardiometa-bolic risk factors. In this regard, it is known that ghrelin exerts an anti-inflammatory role, while RBP-4 has a pro-inflammatory role3.

Objectives: Since a beneficial effect on endothelial function has been reported for anti-IL-6 therapy2, we aimed to evaluate the effect of a single infusion of anti-IL-6 on the serum levels of ghrelin and RBP-4 in patients with RA.

Methods: Ghrelin and RBP-4 levels were measured in serum samples from 50 Spanish individuals with RA that fulfilled the 2010 classification criteria4, and that were under treatment with the anti-IL-6 monoclonal antibody Tocilizumab. Patients with diabetes mellitus or plasma glucose levels >110 mg/dL were excluded. Blood samples were taken in the fasting state, immediately before (time 0) and after (time 60 minutes) Tocilizumab infusion.

Results: A significant increase in serum levels of ghrelin was observed after a single infusion of Tocilizumab (mean-standard deviation: 72.99±58.43 μg/mL versus 134.02±225.93 μg/mL, before and after Tocilizumab infusion, p=0.04). Serum levels of RBP-4 were not affected by the administration of Tocilizumab (mean standard deviation: 23.48±13.99 μg/mL versus 20.90±15.54 μg/mL, before and after Tocilizumab infusion, p=0.42).

Conclusions: Our results show that ghrelin levels increase after a single infusion of Tocilizumab, supporting the hypothesis that IL-6 blockade has a rapid beneficial effect on factors associated with MeS and cardiovascular risk in RA patients. Hence, long-term treatment with anti-IL-6 may reduce the risk of developing cardio-vascular disease in RA.

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Disclosure of Interest: None declared

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