**Methods:** We studied the serum from 30 apparently healthy individuals, and 108 rheumatoid arthritis patients. Antibodies to CP were determined by enzyme immunoassay using immobilized granulated antigen preparations (modification by Gontar et al, 2002). The amount of CP was determined by enzyme immunoassay according to the method of I.S. Kuzmina et al (1991) using commercial diagnostic agent manufactured by Mechnikov Research Institute for Vaccines and Sera.
**Results:** Enzyme immunoassay showed a mean level of CP antibodies in donor sera of 0,020±0,006 optical density units. The level of normal values of specific antibodies determined as M±2σ included an extinction value in the range 0 – 0,086. The mean value of oxidase activity and the amount of CP in healthy people was 716±26,3 and 921±32 ng/ml, correspondingly. In the process of study we revealed a reliable increase in CP antibody count, the activity and amount of CP in patients with rheumatoid arthritis while in all cases the parameters under study correlated with the degree of disease activity ($p<0,05$): at activity degree I CP antibodies were 0,098±0,011; CP activity was 954±48,1; CP amount was 1292±73,4. At activity degree II CP antibodies were 0,138±0,007; CP activity was 1163±39,6; CP amount was 1763±69,3. At activity degree III, CP antibodies were 0,182±0,015; CP activity was 1368±89,5; CP amount was 1794±102,8. After a course of hospital treatment was completed, we noted a reliable decrease in the activity and amount of CP (at degree I of rheumatoid arthritis activity $p<0,001$, at degree II of rheumatoid arthritis activity $p<0,01$for both parameters; at degree III, $p<0,05$) compared with baseline findings. A decrease in CP antibodies shows decelerated dynamics, especially in patients with pronounced disease activity, which indicates severe disorders in the immunity that cannot be cured completely within 30 – 40 days of hospital treatment course.
**Conclusion:** Determination of CP antibodies, as well as quantitative content of CP and its oxidase activity can serve as indicators of the activity of rheumatoid arthritis, as well as an accessory criterion of the effectiveness of administered therapy.
**Disclosure of Interests:** None declared
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## AB0111 FAMILIAL RHEUMATOID ARTHRITIS WITH LATE ONSET (50 YEARS AND OLDER) IN CLINICAL PRACTICE ACCORDING TO THE ALL-RUSSIAN ARTHRITIS REGISTER (OREL)

A. Satybaldyev[1], O. Rumyantseva[2], N. Demidova[2], E. Gerasimova[1], E. Strebkova[3], J. Murtazalieva[4], E. Koltsova[4], M. Nikolenko[4], G. Lukina[4], E. Zhilyaev[4], E. Nasonov[5]. *[1]V A Nasonova Research Institute of Rheumatology, Department of Early Arthritis, Moscow, Russian Federation*; *[2]V A Nasonova Research Institute of Rheumatology, Clinical Department, Moscow, Russian Federation*; *[3]V A Nasonova Research Institute of Rheumatology, Osteoarthritis Laboratory, Moscow, Russian Federation*; *[4]Moscow Research and Practical Center, Moscow Healthcare Department Clinical Moscow, Moscow, Russian Federation*; *[5]V A Nasonova Research Institute of Rheumatology, Scientifical Department, Moscow, Russian Federation*

**Background:** Relatives of the 1st degree of relationship (RFDR) of a patient with rheumatoid arthritis (RA) have the higher risk for the development of RA, associated with a 2-5 fold increase.
**Objectives:** Clinical characteristics of probands with RA onset at an older age (50 yrs and older) and their RFDR, which had RA, other rheumatic diseases (RD) and other autoimmune diseases (AID).
**Methods:** 2766 RA pts with onset of the disease at the age of 50 yrs and older, residents of Moscow and the Moscow region, were included in the Russian register of arthritis OREL in the period from 01.06.2012 to 31.12.2018. 67 probands with RA (2.4%) had RFDRs with RA, other RDs, other AIDs (86 RFDRs in total). Disease activity (by DAS-28), X-ray stage - (by modified Steinbrocker), functional disorders (by functional class - FC), immunological characteristics (RF, ACPA), etc., characterizing the clinical manifestations of RA were evaluated in accordance with the requirements of the current national working RA classification. RFDRs were divided into groups depending on relation to proband father, mother and siblings with RA, RFDRs withr other RD and RFDR with other AID; serological affiliation to RF and ACPA; association with concordant and discordant RFDR, as well as on number of RFDRs (1 and 2 or more).
**Results:** 67 probands with RA were investigated: 9 men, 58 women, average age in debut (m ± δ) 58.78 ± 6.23 yrs; the average age at the time of the last study was 68.8 ± 6.38 yrs, average duration of RA was 10.14 ± 5.69 yrs. 6 (9%) probands had FC1, 45 (69%) - FC2, 13 (20%) - FC3, 1 (2%) - FC4, 2 - without data; 34 (53%) had high activity, 23 (36%) -medium, 6 (9%) - minimal, 1 (2%) - remission, 3 without data; RF-positive (RF+) - 77.6%, ACPA-positive (ACPA+) - 71.7%. 19 probands (32%) had 1-2a X-ray stage, 27 (46%) - stage 2b, 10(17%) - stage 3, 3 (5%) - stage 4, 8 - without data. 61 (91%) had advanced clinical stage, 6 (9%) - late. Among 86 RFDRs (67 - RF+, 19 - RF-) 10 had father with RA (8 RF+ probands, 2 - RF-), 2 - father with other RDs (all RF+), 10 - mother with RA (all RF+), 7 - mother with other RDs (4 - RF+, 3 - RF-), 26 - siblings with RA (20 - RF+, 6 - RF-), 11 - siblings with other RDs (9 - RF+, 2 - RF-), 20 - RFDR with other AIDs (14 - RF+, 6 - RF-). Probands with concordant RFDRs had more pronounced changes: high activity (56% versus 32%), X-ray erosive stages (67% versus 60%) and stages III and IV (32% versus 6%); FC3 and FC4 (23% versus 12%) and a tendency towards more frequent development of the late clinical stage (11% versus 2%). Comparison of clinical signs in probands with 1 RFDR (49 cases) and probands with 2 or more RFDRs (17 cases) showed a significant predominance of high inflammatory activity (61% versus 41%), erosive arthritis (77% versus 57%), and a tendency to development of a late clinical stage (10% versus 6%) in probands with 1 RFDR. In probands with RF-positive RA, the predominance becomes more significant: high inflammatory activity (69% versus 43%), erosive arthritis (90% versus 54%), late clinical stage (13% versus 7%), and a tendency to severe functional impairment - FC3 and FC4 (22% vs. 15%). ACPA-positive probands showed the same tendency as RF-positive.
**Conclusion:** The clinical picture of probands with RFDRs with RA, other RDs, other AIDs is generally characterized by more pronounced changes in probands with RA who have RFDRs with concordant diseases in the form of more often high inflammatory activity, less often moderate, erosive arthritis, more frequent development of advanced 3 and 4 X-ray stages (every fifth). Also, more pronounced changes develop in probands with RA who have only 1 RFDR compared to probands with 2 or more RPSRs. In this group, the differences are even more pronounced in the case of RF-positive probands. In cases with ACPA-positive probands, the same trends persist. Careful interview of a proband with RA about the presence of RFDRs with RA, other RDs and other AIDs will make it possible to predict its course more correctly and facilitate planning of rational therapy.
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## AB0112 TNF INHIBITOR MONOTHERAPY IN RHEUMATOID ARTHRITIS: IS THERE REALLY A DIFFERENCE IN COMPARISON WITH COMBINATION THERAPY WITH CSDMARDS IN REAL-LIFE?

B. M. Fernandes[1], S. Garcia[1], F. Oliveira Pinheiro[1], M. Rato[1], D. Fonseca[2], D. Santos Oliveira[1,3], A. Martins[1], F. R. Martins[4], M. Bernardes[1,5], L. Costa[1]. *[1]Centro Hospitalar Universitário São João, Rheumatology, Porto, Portugal*; *[2]Centro Hospitalar Vila Nova de Gaia/Espinho, Rheumatology, Vila Nova de Gaia, Portugal*; *[3]Faculty of Medicine, University of Porto, Center for Health Technology and Services Research (CINTESIS), Porto, Portugal*; *[4]Centro Hospitalar Universitário do Algarve, Rheumatology, Faro, Portugal*; *[5]Faculty of Medicine, University of Porto, Departamento de Medicina, Porto, Portugal*

**Background:** In Rheumatoid Arthritis (RA), tumor necrosis factor inhibitors (TNFi) in combination with conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) has shown advantages concerning efficacy and immunogenicity in comparison with monotherapy. However, in clinical practice, up to 40% of patients under biological DMARDs (bDMARDs) are on monotherapy.
**Objectives:** To compare the efficacy outcomes of TNFi in monotherapy and in combination therapy in a RA monocentric cohort.
**Methods:** Retrospective, cross-sectional study including all the RA patients under TNFi followed at our Rheumatology Department and registered in the national database. Demographic, clinical and laboratorial data and disease activity measures were collected at the last visit of 2019 from each patient. Mann-Whitney U and chi-square tests were used to the comparison analysis between the two groups (continuous and categorical variables, respectively).
**Results:** A total of 144 patients were included: 84% were females; at the last visit of 2019, the mean age was 56.3±10.9 years and the mean disease duration was 18.3±10.2 years; 73.6% were positive for rheumatoid factor (RF), 81.9% for anti-citrullinated protein autoantibodies (ACPA) and 45.1% had erosive disease. There were no statistically significant differences in these variables between the monotherapy and the combination therapy groups (table 1).

**Table 1. Demographic and disease-related variables in the monotherapy and the combination therapy group.**

| | Monotherapy (n=31) | Combination therapy (n=113) |
|---|---|---|
| Age - mean±SD | 59.1±14.0 years | 55.5±9.8 years |
| Disease duration - mean±SD | 20.5±11.2 years | 17.7±9.7 years |
| RF positive - n (%) | 20 (60.4%) | 86 (76.8%) |
| ACPA positive - n (%) | 25 (80.6%) | 93 (85.3%) |
| Erosive disease - n (%) | 15 (48.4%) | 50 (44.6%) |

Thirty-one patients (21.5%) were under monotherapy with TNFi and etanercept was the most frequent TNFi in both groups (54.8% vs 50.0%; monotherapy and combination therapy groups, respectively). At the start of the first bDMARD, the monotherapy group had a higher disease activity score 28 - 4 variables (DAS 28 4V; 6.083±0.930 vs 5.605±1.043, p=0.039) and a higher simple disease activity score (SDAI; 36.12±11.77 vs 28.76±9.98, p=0.035); also, in the monotherapy group more patients had already started the bDMARD in monotherapy (22.6% vs 2.7%, p<0.001), less patients were under (38.7% vs 73.2%, p=0.001) or had already been treated with (77.4% vs 93.8%, p=0.007) methotrexate, in comparison with the combination group therapy.

At the last visit of 2019, the monotherapy group had a higher mean years of duration of iTNF treatment (5.5±5.8 vs 3.4±4.5, p=0.048), a higher mean patient global assessment - visual analogue scale (PGA-VAS; 49±18 vs 39±25, p=0.023), a higher mean prednisolone equivalent dose in mg/day (7.6±6.3 vs 5.6±3.2, p=0.045) and a lower proportion of American College of Rheumatology 50 and 70 responses (ACR 50: 12.9% vs 17.0%, p=0.023; ACR 70: 3.2% vs 10.7%, p=0.045) in comparison with the combination therapy group.

**Conclusion:** In line with the literature, our real-life results demonstrate some direct (higher PGA-VAS and lower ACR 50 and 70 responses) and indirect (higher current prednisolone equivalent dose) data that suggest that patients with TNFi monotherapy may have a worst disease activity control in comparison with combination therapy.

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AB0113

## ANTI-CARBAMYLATED PROTEIN ANTIBODIES POSITIVITY AND DISEASE ACTIVITY IN HISPANIC PATIENTS WITH ESTABLISHED RHEUMATOID ARTHRITIS: AN OBSERVATIONAL STUDY

D. Vega-Morales[1], M. A. Garza Elizondo[1], L. A. Trouw[2], K. I. González Márquez[1], E. Torres-Lopez[3], M. Eguia Bernal[1], S. A. Loredo Alanis[1], T. S. Gracia-Arechiga[1], B. R. Vázquez Fuentes[1], D. D. Castañeda Martínez[1], M. M. Castañeda-Martínez[1], C. V. Solis[1], A. Mendiola-Jimenez[1], M. C. Salinas-Carmona[3], P. Herrera-Sandate[1], A. Cárdenas[1], G. E. Rodriguez-Sanchez[1], D. Á. Galarza-Delgado[1]. [1]*Hospital Universitario Dr. José Eleuterio González, Rheumatology Service, Monterrey, Mexico*; [2]*Leiden University Medical Center (LUMC), Department of Immunohematology and Bloodtransfusion, Leiden, Netherlands*; [3]*Hospital Universitario Dr. José Eleuterio González, Immunology Department, Monterrey, Mexico*

**Background:** Clinically relevant anti-carbamylated (anti-CarP) antibodies are detected in up to 45% of rheumatoid arthritis (RA) patients and are associated with severe radiological progression, higher disease activity, and significantly more disability when studied in early phases of arthritis.

**Objectives:** We aimed to determine the prevalence of anti-CarP antibodies in Mexican Hispanics with established RA and to assess their relationship with disease activity.

**Methods:** A cohort study was conducted in 278 patients with established RA during an 18-month follow-up. We measured IgG/IgM/IgA rheumatoid factor (RF), IgG anticitrullinated protein antibodies (ACPA) and IgG/IgM/IgA anti-CarP antibodies using enzyme-linked immunosorbent assay (ELISA). For disease activity, we performed the 28-joint disease activity score with erythrocyte sedimentation rate (DAS28-ESR). Repeated measures one-way ANOVA was used to test the association between anti-CarP IgG antibody status and longitudinal DAS28-ESR scores. Patients were evaluated at baseline and at 6, 12, and 18 months during follow-up.

**Results:** Anti-CarP IgG antibodies were positive in 47.8% of patients and, accounting for all isotypes, in 9.5% of patients with negative RF and ACPA. Triple antibody positivity was present in 42.6% of patients in our sample. Anti-CarP IgG antibody positivity did not show statistically significant differences in mean DAS28-ESR when compared to anti-CarP IgG antibody negative patients at baseline, 6, 12 or 18 months.

**Conclusion:** Anti-CarP IgG antibodies are present in almost 50% of RA patients and, accounting for all isotypes, in 9% of RF and ACPA negative patients. Anti-CarP IgG antibody positivity was not associated to a higher disease activity measured by DAS28-ESR in Hispanic patients with established RA.

References: Shi J, Knevel R, Suwannalai P, Van Der Linden MP, Janssen GMC, Van Veelen PA, et al. Autoantibodies recognizing carbamylated proteins are present in sera of patients with rheumatoid arthritis and predict joint damage. Proc Natl Acad Sci U S A. 2011;108:17372–17377.

**Table 1. Anti-CarP antibody status by isotype in a cohort of 278 patients with established RA.**

| | Mean (SD) | Antibody positivity, n (%) | 95% CI |
|---|---|---|---|
| RF IgA[a] | 266.9 (460.5) | 155 (58.9) | 53.0 to 64.9 |
| RF IgM[a] | 406.8 (611.9) | 188 (71.5) | 66.0 to 77.0 |
| RF IgG[a] | 36.1 (249.6) | 44 (16.7) | 12.2 to 21.3 |
| ACPA IgG[a] | 191.01 (411.1) | 144 (54.8) | 48.7 to 60.8 |
| Anti-CarP IgA[b] | 212.9 (464.2) | 74 (26.6) | 21.4 to 31.8 |
| Anti-CarP IgM[b] | 381.6 (762) | 89 (32) | 26.5 to 37.5 |
| Anti-CarP IgG[b] | 227.5 (402.5) | 133 (47.8) | 41.9 to 53.8 |

[a]Data were available for 263 patients. Units are RU/mL. [b]Data were available for 278 patients. Units are AU/mL. RF, rheumatoid factor; ACPA, anticitrullinated protein antibodies; Anti-CarP, anti-carbamylated protein antibodies; IgG, immunoglobulin; SD, standard deviation; 95% CI, 95% confidence intervals.

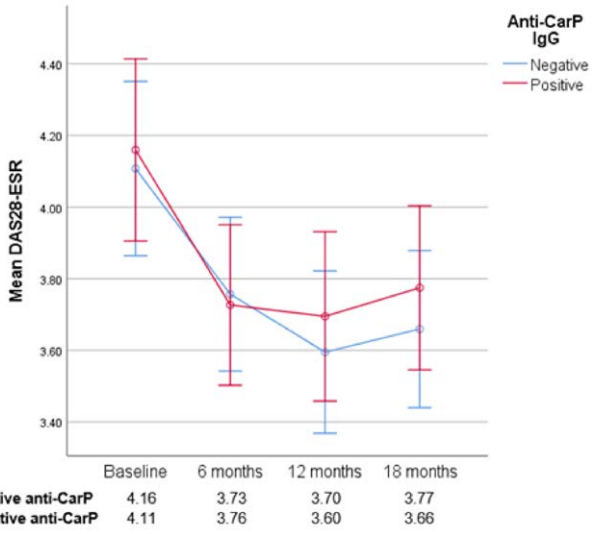


**Figure 1.** Mean DAS28-ESR scores over an 18-month follow-up by anti-CarP antibody status with error bars corresponding to 95% CI. With the non-sphericity assumed by Greenhouse-Geisser analysis, a non-significant statistical difference was found among groups in the repeated measures one-way ANOVA (p = 0.829). DAS28-ESR, 28-joint disease activity score with erythrocyte sedimentation rate; Anti-CarP, anti-carbamylated protein antibodies; Ig, immunoglobulin.

**Figure 1.**

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