

# BIOLOGICAL DRUGS: PERSISTENCE RATE IN PATIENTS WITH RHEUMATOID ARTHRITIS AND SPONDYLARTHROSIS

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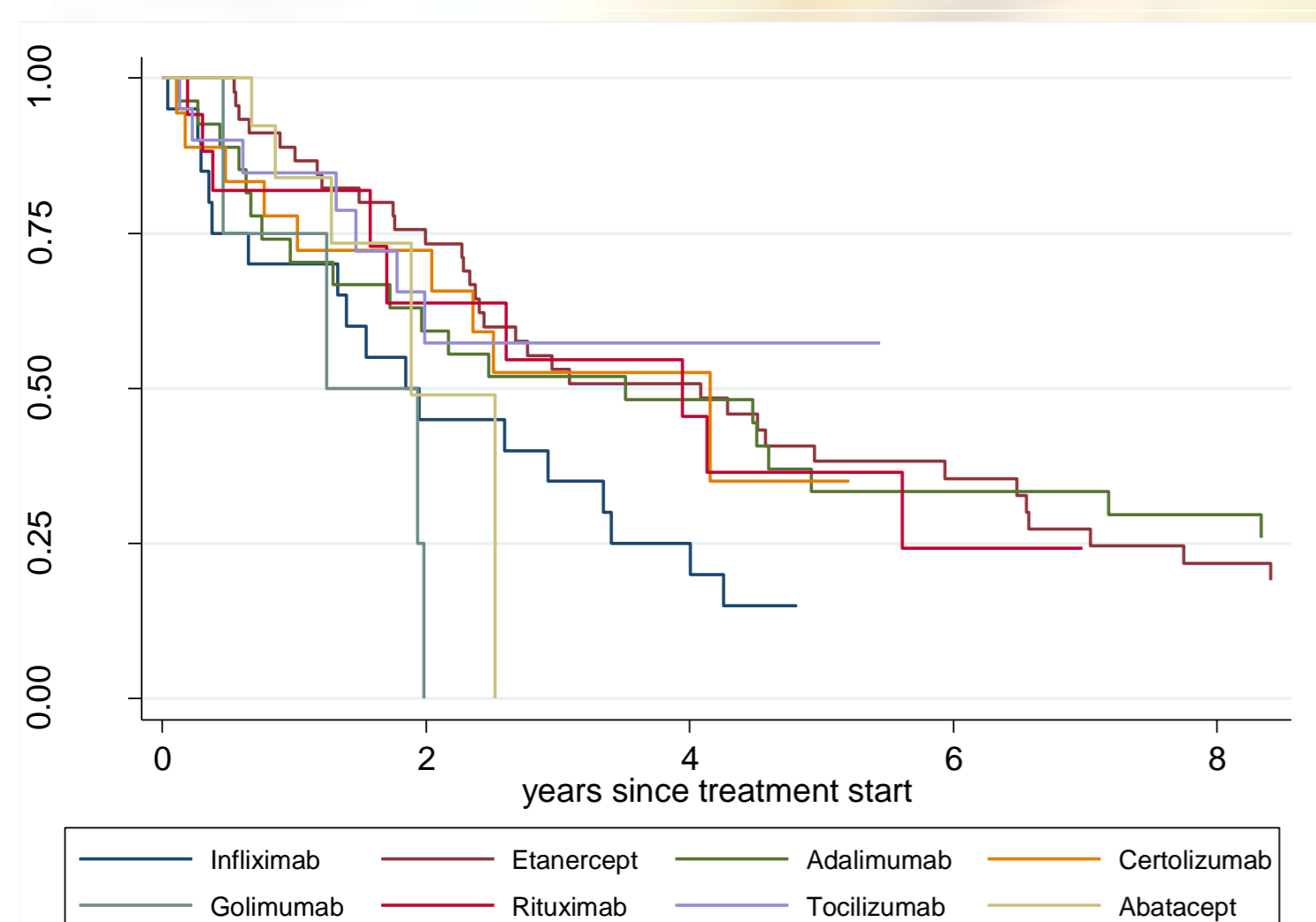
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## Objectives:

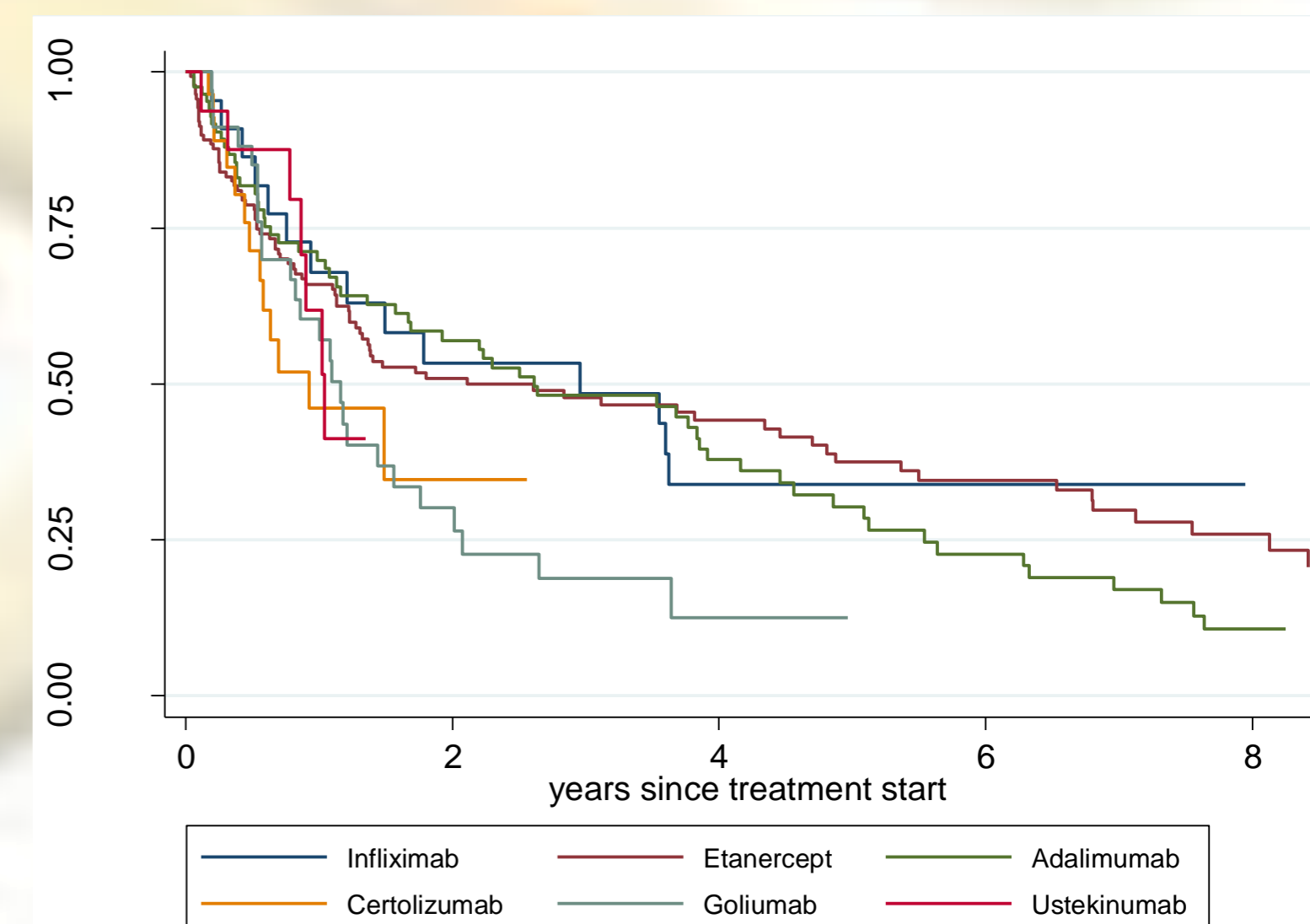
Rheumatic diseases, such as Rheumatoid Arthritis and Spondylarthritis, are chronic autoimmune diseases that can be treated with new biological drugs. The purpose of this study is to evaluate the drug retention, as surrogate of effectiveness of biologic therapy.

## Methods:

In a rheumatological center of Apulia (Italy) 210 patients with Rheumatoid Arthritis (RA) and 336 patients with Spondylarthritis (SpA) were evaluated, treated with "biologics", from 1 January 2001 to 31 May 2016. It was valued the effectiveness of biological drugs using the therapy through the Kaplan Meier drug retention curves.



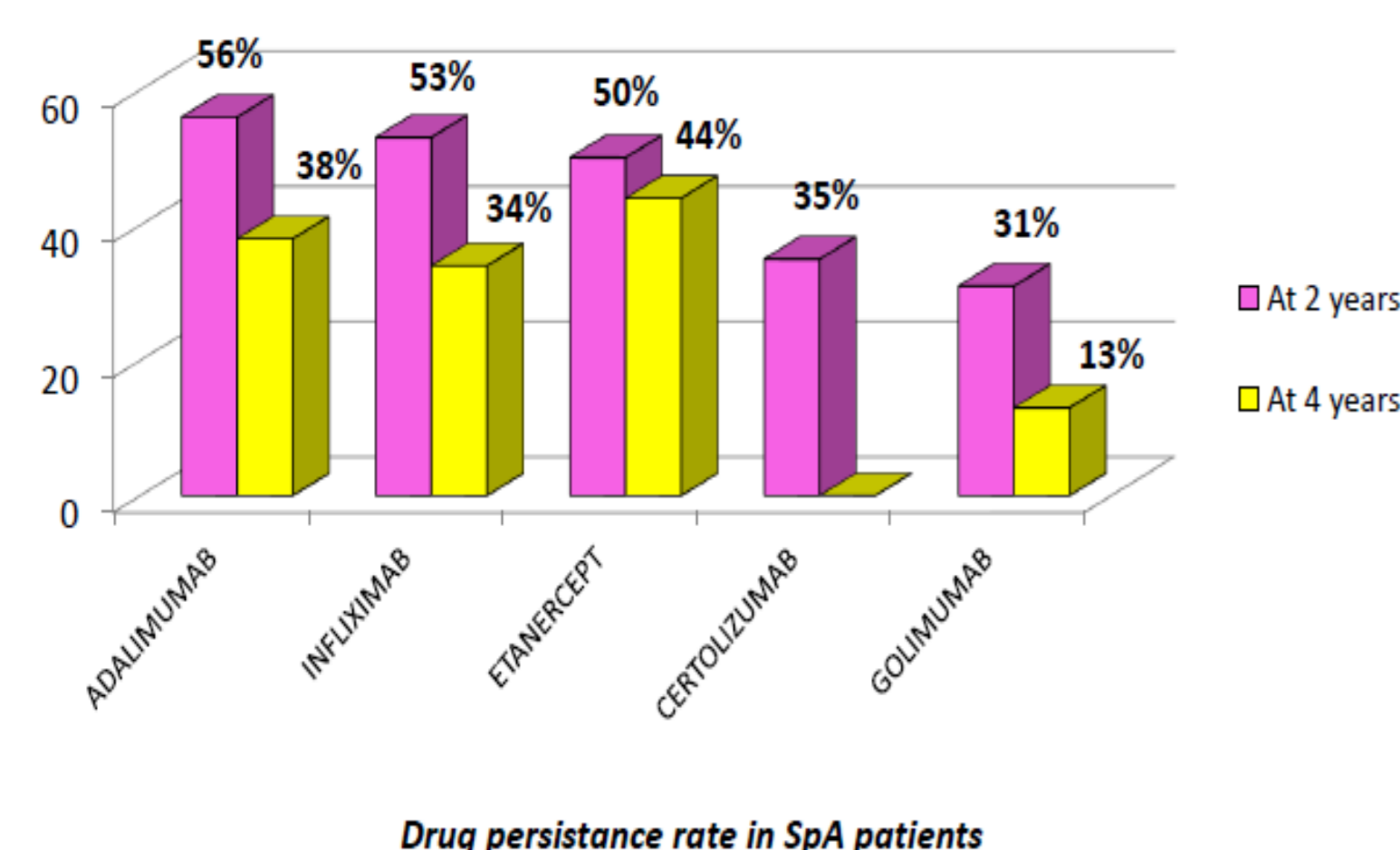
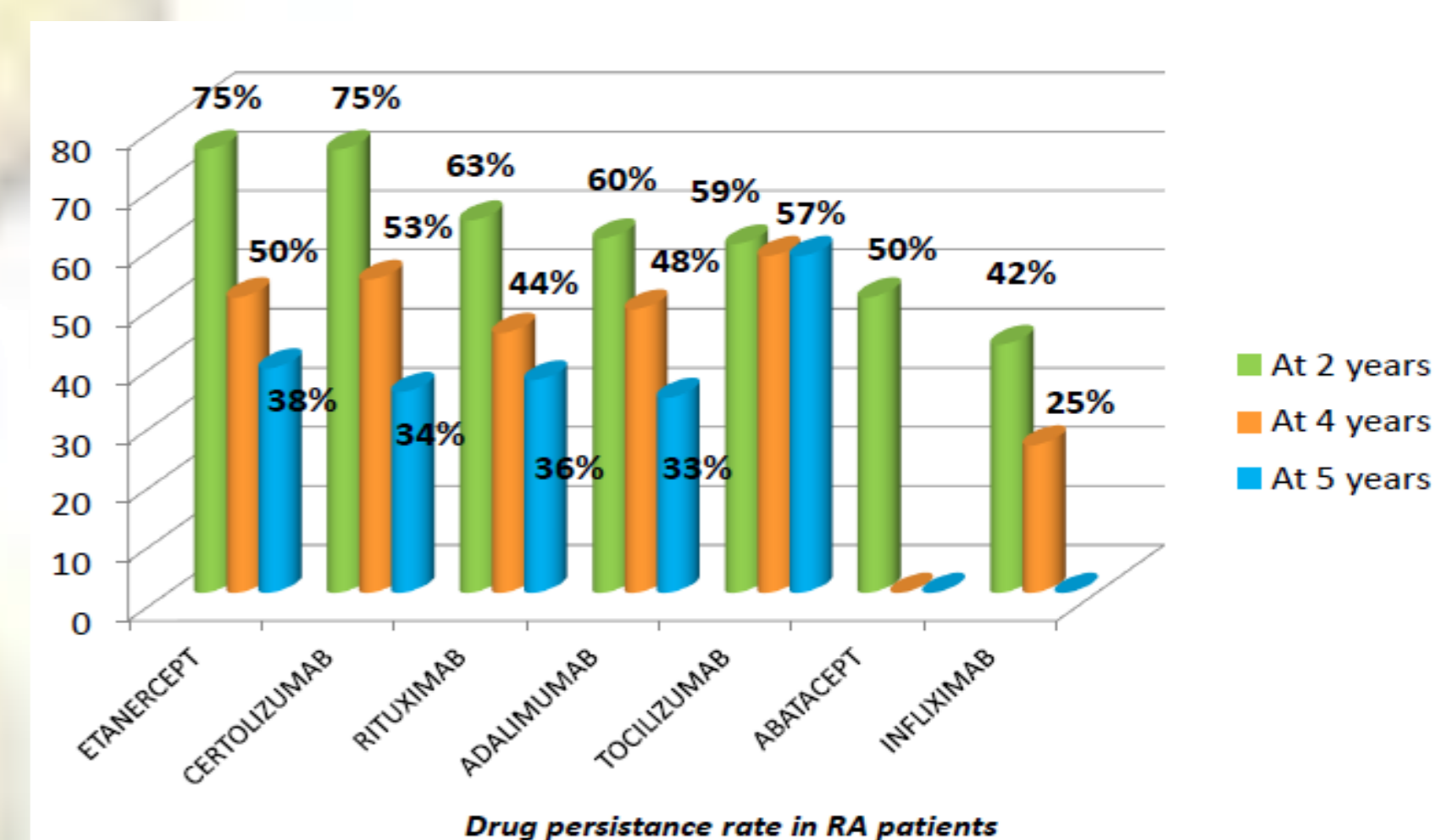
Kaplan Meier drug retention curves RA patients



Kaplan Meier drug retention curves SpA patients

## Results:

RA patients: we have analyzed the data at 2, 4 and 5 years of therapy. At 2-years, the drug persistence rate: 75% etanercept, rituximab 63%, 60% adalimumab. Abatacept 50% with rapid decrease during the continuation of the therapy; infliximab 42% at 2 years, 25% at 4 years. At 5 years: 57% tocilizumab; 35% rituximab, adalimumab, etanercept, certolizumab.



SpA patients: we analyzed the data at 2 and 4 years of therapy. At 2-years, the drug persistence rate: 50-56% etanercept, and infliximab adalimumab; 35% certolizumab, golimumab 31%. Ustekinumab not statistically evaluable because it has marketing authorisation in less than 2 years. At 4 years: 44% etanercept, adalimumab 38%, 34% infliximab, golimumab 13%. Certolizumab not statistically evaluable because it is commercially in less than 4 years for Psoriatic Arthritis.

## Conclusions:

A comparison between RA and SpA allows to note that in RA patients will have a higher persistence in 2 years treatment (75-60% vs. 55-50%) than in SpA patients; at 4 years the drug persistence: 50% for RA compared to 35% of SpA. In this study it is observed that RA patients probably respond better to "biological drugs" because the biological targets are defined; in SpA targets are less known, having recently discovered the IL23/IL17 (interleukin-23/interleukin-17) etiopathogenetic way. Therefore the different clinical response and the frequent change of drugs shows that it can't predict neither therapeutic response nor the appearance of adverse events.

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