Conclusion: In our study, LEF was comparable to RTX for achievement of ACR20/50/70 responses at 24 weeks. LEF can be considered as an add-on option to MTX instead of more expensive biologic agents in MTX refractory RA. Larger studies are needed to confirm this hypothesis.

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AB0400 A SYSTEMATIC LITERATURE REVIEW AND META-ANALYSIS INTO THE SUCCESS RATE OF GLUCOCORTICOID DISCONTINUATION AFTER THEIR USE AS INITIAL BRIDGING THERAPY IN RHEUMATOID ARTHRITIS PATIENTS IN OBSERVATIONAL COHORTS AND CLINICAL TRIALS

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Background: Glucocorticoids (GC) are widely used for the initial treatment of rheumatoid arthritis (RA), to induce rapid suppression of inflammation and clinical symptoms and thereby limit radiographic damage progression. There are concerns that GC use in the long term is associated with a dose and duration dependent risk of serious side effects. Therefore, international guidelines have recommended to start GC when initiating a csDMARD, but to discontinue GC as rapidly as clinically feasible, preferably within 3 months (bridging therapy). In contrast, due to the concerns of GC side effects, the ACR guidelines published in 2021 conditionally recommend to start csDMARD monotherapy without GC bridging therapy.

Objectives: We aim to evaluate the success rate of GC discontinuation after using temporary GC as part of initial therapy ('bridging') both in observational cohorts and clinical trials in newly diagnosed RA patients.

Methods: Systematic literature searches were conducted to identify observational cohorts (scoping search) and clinical trials (in-depth search) that included RA patients who were treated with initial GC bridging therapy. GC bridging was defined as oral or intramuscular GC treatment that was discontinued within one year, alongside conventional DMARD therapy. Patient percentages still or again using GC were considered to represent the reverse of successful discontinuation. Random-effects meta-analyses were performed stratified by time point.

Results: The literature search on observational cohort studies could not identify any study answering the research question, since it remained unclear which patients had received GC as part of the initial treatment. The literature search for clinical trials identified 7160 abstracts, resulting in 10 included studies, with varying type and dose of GC and varying tapering schedules (Table 1). Of these included studies, 4 reported sufficient data on GC discontinuation or GC use after the bridging phase. The pooled proportion of patients who were still using GC was 22% (95% Confidence Interval (CI) 8; 37, based on 4 trials) at 12 months and 10% at 24 months (95% CI -1; 22, based on 2 trials) (Figure 1). Thus, the vast majority had stopped GC. Heterogeneity was substantial ($I^2 \geq 65\%$).

Conclusion: The success rate of GC discontinuation after bridging as part of initial treatment of RA has been described in a limited number of studies. Reports on observational cohorts did not answer the research question and in clinical trials reports, GC (dis)continuation data were also scarce. However, the available data show that GC can be discontinued successfully in a large majority of patients. The paucity of data also reveals that more efforts are needed to provide data towards identifying the optimal GC bridging and discontinuation strategy, combining Treatment to Target with Starting to Stop.

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Table 1. Overview of included clinical trials.

Study (publication year)	Tapering schedule (mg/day)
COBRA (1997)	In 7 weeks to 7.5. Stop after 28 weeks.*
BeSt (2005)	In 7 weeks to 7.5. Stop in 8 weeks after week 28 if DAS persistently ≤2.4
IDEA (2014)	N.A.
COBRA-light (2015)	arm 1: in 7 weeks to 7.5 arm 2: in 9 weeks to 7.5 Stop after 32 weeks if DAS<1.6.
IMPROVED (2014)	In 7 weeks to 7.5. Stop after 20 weeks if DAS <1.6 at 4 months.
ARCTIC (2016)	In 7 weeks to 0 if DAS <1.6 and no swollen joints present.
tREACH (2013)	In 10 weeks to 0.*
CareRA (2017)	- in 7 weeks to 7.5, further tapered from week 28, stop after 34 weeks.
- *Classic*	- in 6 weeks to 5, further tapered from week 28, stop after 34 weeks.
- *Slim*	- in 6 weeks to 5, further tapered from week 28, stop after 34 weeks.
- *Avant garde*	All if DAS28(CRP) ≤3.2.
Hua et al. (2020)	Tapering after 4 months to 5, stop after 6 months.*
NORD-STAR (2020)	In 9 weeks to 5. Stop after 9 months.*
- *arm 1A (oral prednisolone)*	

DAS=disease activity score; mg=milligram; N.A.=not applicable. *GC tapered and stopped according to protocol, not depending on disease activity score.

AB0401 THE BASELINE SERUM SOLUBLE TNF RECEPTOR LEVELS ARE ASSOCIATED WITH THE RESPONSE OF RHEUMATOID ARTHRITIS PATIENTS TO JAKINIBS

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Background: Rheumatoid arthritis (RA) is a chronic autoimmune disease that primarily affects the multiple joints.

The elucidation of the pathogenesis of RA has progressed dramatically in recent decades, and among the many cytokines involved in the pathogenesis of RA, interleukin (IL)-6 and TNF-α are known to be the major pro-inflammatory cytokines that are abundant in the bloodstream and synovial tissue. JAK inhibitors (JAKinibs) such as tofacitinib and baricitinib are used in the treatment of RA by inhibiting JAK, which in turn inhibits the signaling of various cytokines including IL-6. However, predictors of the response to JAKinibs are still required.

Objectives: We aimed to combine soluble TNF receptor (sTNFR) I, sTNFR II, IL-6, soluble IL-6R (sIL-6R) and soluble gp130 (sgp130) levels to identify groups of JAKinibs responses in RA patients.

Methods: This research is a retrospective study. We reviewed medical records of RA patients initiating JAKinibs between July 2013 and July 2021 in our hospital. The Simplified Disease Activity Index (SDAI) was evaluated at baseline and 3, 6 months after JAKinibs administration. Clinical remission was defined when SDAI decreased ≤ 3.3. Of the 125 patients treated with JAKinibs, 89 patients with 6 months follow-up, valid SDAI and serum available were enrolled. Serum samples were tested for IL-6 (Human IL-6 Quantikine ELISA Kit, R&D systems), sIL-6R (Human soluble IL-6R alpha Quantikine ELISA Kit, R&D systems) and sgp130 (Human soluble gp130 Quantikine ELISA Kit, R&D systems), sTNFR I (Human TNF RI/TNFRSF1A Quantikine ELISA Kit DRT100) and sTNFR II (Human sTNF RII/TNFRSF1B Quantikine ELISA Kit DRT200) using specific ELISAs according to the manufacturer's instructions. The statistical analyses were performed with EZR 1.55, and p values less than 0.05 were considered significant.

Results: The median age of patients was 62 (IQR: 51 - 72) years and the median of disease duration was 6.0 (2.0 - 16.0) years. Twenty-seven (30.3%) patients were biologics and Jakinibs naive. The baseline SDAI was median 18.9 (12.7 - 27.9). When comparing SDAI-remission group (clinical remission: CR) and non-remission group, there were no significant differences in any of the baseline clinical parameters. There was no significant difference in the serum levels of IL-6, sIL-6R and sgp130 between the CR and non-CR groups, but the serum levels of sTNFR I and sTNFR II in the CR group were significantly lower than non-CR group. Univariate logistic regression analysis suggested Biologics and JAKinibs naive (odds ratio (OR) 3.58, p = 0.015), baseline Log sTNFR II levels (OR 0.013, P=0.034)