safety profile consisting of events occurring in unexposed infants of similar age.
**Conclusions:** CZP was below the lower limit of quantification in 56% of the milk samples. When detectable, CZP concentrations were less than 3x LLOQ (<1% of expected plasma concentration of a therapeutic dose[4]), indicating no to minimal transfer of CZP from plasma to breast milk. RID was below 0.5% of maternal dose; <10% is unlikely to be of clinical concern.[3] CZP absorption via breast milk is unlikely, due to low bioavailability and its Fc-free molecular structure. These findings support continuation of CZP treatment during breastfeeding.
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## FRI0180 MULTIPLE SCLEROSIS RISK-ALLELES STUDY IN PATIENTS WITH DEMYELINATING SIDE EFFECTS ON ANTI TNF ALPHA THERAPY

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**Background:** Tumor Necrosis Factor alpha (TNFα) is a key cytokine in inflammatory rheumatic diseases. TNF inhibitors (TNFi) has revolutionized treatment of rheumatic diseases, but may cause flares of multiple sclerosis (MS). Two Single Nucleotide Polymorphisms, (SNPs) *rs1800693* and *rs4149584*, located within *TNF receptor superfamilly 1* (*TNFRSF1A*) locus have been shown to increase the risk of developing MS [1]. The rs1800693*G allele leads to a dysfunctional TNFα soluble receptor that inhibits TNFα signaling while *rs4149584* is involved in TNF receptor associated periodic syndrome.
**Objectives:** The aim of this study was to look for a possible the association between *TNFRSF1A* polymorphisms and demyelinating complications occurring during TNFi therapy.
**Methods:** Patients who presented with a demyelinating disorder (central or peripheral involvement) while treated with TNFi (cases), were recruited between March 2013 and December 2015, through the physicians involved in the CRI ("Club Rhumatismes et Inflammation") a nationwide network of the French Society of Rheumatology. Rheumatoid arthritis patients treated with TNFi, from the French ReAct cohort, and who did not develop demyelinating complication constituted the control population (n=294). The frequency of *rs1800693* and *rs4149584* TNFRSF1A SNPs were compared between cases and controls.
**Results:** Twenty-four cases with demyelinating disorders, recruited from 11 centers with a median age of 39.7 years (range 30.4–75.8); of which 16 (67%) were females were included in the study. Neurological symptoms occurred after a median of 18.3 (1–66) months of anti TNFi; 15 (62.5%) had central neurologic involvement and 9 (37.5%) had peripheral involvement. The median follow-up was 26 (4–54) Months. No significant difference in the frequency of the *rs1800693* MS risk-alleles (39,5% for cases vs 38,6% for controls) was observed. Similarly no difference was observed between cases (2%) and controls (4%) for *rs4149584.*
**Conclusions:** This study was unable to show an association between MS-associated SNPs within *TNFRSF1A* locus and the occurrence of demyelination while taking TNFi, suggesting that demyelination might be linked to other genetic factors or other pathways.
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## FRI0181 TAPERING OR CESSATION OF ANTIVIRAL AGENT IN HEPATITIS B VIRUS-INFECTED PATIENTS CONCOMITANTLY TREATED WITH BIOLOGIC AGENTS

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**Background:** Clinicians generally prescribe antiviral agents to patients with chronic hepatitis B (CHB) or to inactive carriers of the virus until 6–12 months after the cessation of biologic agents. However, the current antiviral prophylaxis regimen, in addition to biological therapy, is expensive and poses an economic burden to both patients and societies. We have had patients in our medical center quit or reduce antiviral prophylaxis due to economic reasons.
**Objectives:** To assess the outcome of tapering or discontinuation of antiviral agents in patients who were infected with hepatitis B virus (HBV) during biologic therapy.
**Methods:** We identified 45 patients who were infected with HBV and treated with biologic agents concomitantly from January 2005 to December 2016. They were diagnosed with rheumatoid arthritis (n=20), Crohn's disease (n=13), ankylosing spondylitis (n=8), ulcerative colitis (n=3), and psoriatic arthritis (n=1). The criteria of HBV reactivation was a 10-fold rise in HBV DNA compared with previous HBV DNA titers, resulting in HBV DNA of greater than 20,000 IU/ml (HBeAg-positive patients) or 2,000 IU/ml (HBeAg-negative patients), and an increase in AST or ALT to more than twice the upper normal limit (40 IU/l).
**Results:** Sixteen CHB patients and 29 inactive carriers were treated with biologic agent for 4.1±2.7 years. No reactivation case was observed in 23 patients (10 of CHB and 13 of inactive carrier) who maintained antiviral prophylaxis for 4.0±2.3 years. Among them, 4 patients (3 treated with infliximab and 1 with adalimumab) taking antiviral prophylaxis regimen on alternate days did not experience HBV reactivation for 28–42 months of follow-up period. In the discontinuation group (n=9), no reactivation case was observed in all inactive carrier patients (2 treated with etanercept, 1 with infliximab, and 1 with rituximab) after discontinuation of antiviral prophylaxis for 6–33 months of follow-up period. In contrast, 3 patients (2 treated with etanercept and 1 with adalimumab) among the 5 patients with CHB experienced reactivation after discontinuation of antiviral prophylaxis for 3–29 months of follow-up period.
**Conclusions:** During biologic therapy, HBV reactivations were frequently found in CHB patients who ceased to take antiviral prophylaxis. However, no reactivation after the cessation of prophylaxis was found in inactive carrier patients who were previously treated with prophylaxis. Based on our experience, tapering or discontinuation of antiviral agent in inactive carriers with economic problem undergoing concomitant biologic agent therapy could be considered viable, albeit with caution.
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## FRI0182 DISEASE WORSENING AND SAFETY IN PATIENTS SWITCHING FROM ORIGINATOR INFLIXIMAB TO BIOSIMILAR INFLIXIMAB (CT-P13) IN THE NOR-SWITCH STUDY: EXPLORATIVE ANALYSIS OF RA PATIENTS

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**Background:** The NOR-SWITCH study was a 52-week randomized, double-blind, non-inferiority, phase IV switch trial in patients with Crohn's disease (CD), ulcerative colitis (UC), spondyloarthritis (SpA), rheumatoid arthritis (RA), psoriatic arthritis (PsA) and plaque psoriasis (Ps) on stable treatment with originator infliximab (Remicade®, INX) and was funded by the Norwegian government. Previously, the primary analyses of the pooled indications have been published[1].
**Objectives:** To investigate efficacy, safety and immunogenicity in RA patients treated with continous INX vs patients switched to CT-P13 (biosimilar infliximab, Remsima®) in the NOR-SWITCH study (explorative analyses).
**Methods:** Patients were randomized 1:1 to continued INX or switch to CT-P13. Serum drug levels were analysed in automated in-house assay. The primary