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INTRODUCTION

A growing number of medicines are based on biological molecules such as proteins and monoclonal antibodies. These novel drugs have resulted in new, more effective treatments for a number of serious conditions. Yet sometimes these medicines trigger a response from the patient's immune system, which can decrease the effectiveness of the drug or cause severe side effects.

The aim of the IMI-founded ABIRISK project **"Anti-Biopharmaceutical Immunization: Prediction and Analysis of Clinical Relevance to Minimize the Risk",** is to shed new light on the factors behind this immune response. The project, which represents the first concerted effort to solve this problem, officially kicked off March 1st, 2012. ABIRISK project will aid in the creation of new, safer **biopharmaceuticals (BPs)** and also generate tools to determine how individual patients are likely to respond to them both in clinical trials and after release to the market.

The ABIRISK consortium (presently made up of thirty-five partners, twenty-four of which are academic institutions, nine are EFPIA member companies and two are small and medium enterprises, with thirteen countries represented), has been designed to meet all of these requirements in order to target three types of disorders: **Hemophilia A, Multiple sclerosis and Inflammatory diseases: inflammatory rheumatisms (including rheumatoid arthritis) and inflammatory bowel diseases.**

ABIRISK Project will collect data both retrospectively from patients suffering from various types of diseases and treated with various BPs at European centers with a high level of experience in clinical research and will prospectively recruit additional patients in dedicated studies during the 5 years of this program. Guidelines and Standard Operating Protocols for the study of anti-drug immunization will be established and used to standardize the collection of prospective data from these patients.

ABIRISK Project thus represents a unique opportunity to create an interdisciplinary task force of clinical centers especially designed to study immune responses against biopharmaceuticals.

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WELCOME

Dear Reader,

We would like to welcome you to the JULY 2013 of the **ABIRISK Scientific Newsletter**. The Scientific Newsletter gives you a monthly update on the most relevant literature related to ABIRISK topics published around the globe, both inside and outside ABIRISK consortium.

This month, we highlighted a publication in the biomarker field. *Mathews et al.* found evidence of modulation of the NLRP3-inflammasome in patients with rheumatoid arthritis prior to receiving biotherapy and some evidence of inflammasome genetic variants association with rheumatoid arthritis susceptibility and response to anti-TNFα treatment.

In addition, we chose this month to also highlight some pieces of regulatory news from the EMA, as they mark the first market authorization of anti-TNFα biosimilars in Europe.

We look forward to your visit on **ABIRISK** website for more information and updates on the program.

Enjoy reading !

Best wishes

The ABIRISK management team

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LITERATURE

This month's selected article

Inflammasomes are a set of intracellular protein complexes that enable autocatalytic activation of inflammatory caspases, which drive host immune responses by releasing cytokines and alarmins into circulation and by inducing pyroptosis, a proinflammatory cell death mode. The NLRP3-inflammasome (or caspase-1 inflammasome) senses damage-associated molecular pathogens and is involved in proteolytic maturation of proIL-1β and proIL-18.

Several cytokines have been implicated in rheumatoid arthritis (RA) pathogenesis and persistence among which tumour necrosis factor (TNF) and interleukin-1β (IL-1β). In fact, secretion of IL-1β and IL-18 subsequent to NLRP3-inflammasome activation may be destructive to tissues and is known to play an important role in bone resorption and cartilage destruction in RA. Genetic variation in two proteins, namely NLRP3 and CARD8 of the NLRP3-inflammasome complex, have been reported to influence susceptibility and severity of RA. Furthermore, a number of pharmacogenetic studies have reported single nucleotide polymorphisms (SNPs) in various genes associated with good response/resistance to biologics therapies in RA patients.

In this context, the present study by Mathews et al*. was undertaken to examine the contribution of NLRP3-inflammasome components to active RA and the effects of anti-TNF therapy. Hence, expression of six NLRP3-inflammasome components (ASC, pyrin, NLRP3-FL, NLRP3-SL, CARD8 and caspase-1, encoded by *ASC, MEFV, NLRP3, CARD*8 and *CASP1*) was investigated in peripheral blood mononuclear cells (PBMCs) of 29 active RA patients at baseline, and at week 14 into treatment with infliximab. In parallel, whether genetic variation within genes encoding constituent proteins of the NLRP3-inflammasome (*NLRP3*, *MEFV* and *CARD8*,) influenced the response to anti-TNF therapy was investigated in the Biologics in Rheumatoid Arthritis Genetics and Genomics Study Syndicate (BRAGGSS) cohort.

Evidence of modulation of the NLRP3-inflammasome at the transcriptional level in PBMC of patients with RA prior to receiving infliximab therapy was observed : at baseline, gene expression of *ASC, MEFV, NLRP3*-FL, *NLRP3*-SL and *CASP1* were significantly higher compared with controls whereas *CARD8* was lower in the patients. Caspase-1 and interleukin-18 levels were significantly raised in patients with RA. However, expression levels of these inflammasome components were not significantly altered after 14 weeks of therapy in either responders or non-responders to infliximab.

Analyses conducted in the BRAGGSS cohort showed that genetic variation at SNPs within two different components of the NLRP3-inflammasome (NLRP3 and CARD8) influences disease susceptibility and response to anti-TNF therapy in patients with RA. SNPs in *NLRP3* showed association with RA susceptibility and EULAR response to anti-TNF, in monocytes but not B cells, in expression quantitative trait loci (eQTL) analysis of 283

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healthy controls. *CARD8* SNPs were associated with RA susceptibility and DAS28 improvement in response to anti-TNF and eQTL effects in monocytes and B cells. The authors underline that the SNPs associated with susceptibility/response are not the main eQTL variants for either locus, and the associations with treatment response require replication in an independent cohort.

*Evidence of NLRP3-inflammasome activation in rheumatoid arthritis (RA); genetic variants within the NLRP3-inflammasome complex in relation to susceptibility to RA and response to anti-TNF treatment.
Mathews RJ, Robinson JI, Battellino M, Wong C, Taylor JC; Biologics in Rheumatoid Arthritis Genetics and Genomics Study Syndicate (BRAGGSS),, Eyre S, Churchman SM, Wilson AG, Isaacs JD, Hyrich K, Barton A, Plant D, Savic S, Cook GP, Sarzi-Puttini P, Emery P, Barrett JH, Morgan AW, McDermott MF.
Ann Rheum Dis. 2013 May 17

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Immunogenicity

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Methods

Immunogenicity testing strategy and bioanalytical assays for antibody-drug conjugates.
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Rheumatoid Arthritis

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Multiple Sclerosis

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Hemophilia

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Opinions/Commentaries

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Ann Rheum Dis. 2013 Apr;72 Suppl 2:ii2-34

Anti-TNF antibodies in Inflammatory Bowel Disease: do we finally know how it works?
Oikonomopoulos A, van Deen W, Hommes DW.
Curr Drug Targets. 2013 Apr 26.

ECCO position statement: The use of biosimilar medicines in the treatment of inflammatory bowel disease (IBD).
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J Crohns Colitis. 2013 Apr 25.

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[When do we dare to stop biological or immunomodulatory therapy for Crohn's disease? Results of a multidisciplinary European expert panel.]

Pittet V, Froehlich F, Maillard MH, Mottet C, Gonvers JJ, Felley C, Vader JP, Burnand B, Michetti P, Schoepfer A; the EPACT-II Update Panellists.

J Crohns Colitis. 2013 May 9

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REGULATION

Selected news of the month

EMA
Pending EC decision: Inflectra, infliximab
Opinion
June 2013
Inflectra *is a biological medicinal product similar to the reference product Remicade (infliximab) authorised in the European Union (EU) since 13 August 1999. Studies have shown Inflectra to have a comparable quality, safety and efficacy profile to Remicade (infliximab).*
A pharmacovigilance plan for Inflectra will be implemented as part of the marketing authorisation.

EMA
Pending EC decision: Remsima, infliximab
Opinion
June 2013
Remsima *is a biological medicinal product similar to the reference product Remicade (infliximab) authorised in the European Union since 13 August 1999. Studies have shown Remsima to have a comparable quality, safety and efficacy profile to Remicade (infliximab).*
A pharmacovigilance plan for Remsima will be implemented as part of the marketing authorisation.

EMA

Pending EC decision: Lemtrada, alemtuzumab
Opinion
June 2013

Opinion/decision on a Paediatric Investigation Plan (PIP): Humira, adalimumab
Therapeutic area: dermatology
June 2013

Human medicines European Public Assessment Report (EPAR): Cimzia, certolizumab pegol
Revision 7 authorised
June 2013

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Human medicines European Public Assessment Report (EPAR): Simponi, golimumab
Revision 13 authorised
June 2013

Human medicines European Public Assessment Report (EPAR): Remicade, infliximab
Revision 39 authorised
June 2013

Scientific guideline: Draft guideline on similar biological medicinal products containing biotechnology-derived proteins as active substance: non-clinical and clinical issues
Draft, consultation open
June 2013
End of consultation : 30 November 2013

EMA draft guideline.pdf

Announcement

Workshop on the clinical investigation of new medicines for the treatment of multiple sclerosis
European Medicines Agency, London, UK, 17-Oct-2013

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