**Conclusion:** In addition to inducing p75NTR up-regulation, inflammatory stimuli increase the release of proNGF in arthritis SFs. Autocrine proNGF binds to p75NTR and further enhances pro-inflammatory cytokine production, creating a vicious circle that amplifies the inflammatory response. Blocking the binding of endogenous proNGF to its receptor p75NTR strongly reduces the functionality of inflammatory mediators and prospects the use of p75NTR inhibitors as a new therapeutic approach to chronic arthritis.

**REFERENCES**


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**AB0063**

**A SYSTEMS APPROACH TO INVESTIGATE INFLAMMATION RESOLUTION BY MULTICOMPONENT MEDICINAL PRODUCT TR14**

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**Disclosure of Interests:** Patrick Schopohl: None declared, Suchi Smitsa: None declared, Faiz Khan: None declared, Tom Gebhardt: None declared, Math Hoch: None declared, David Brauer: None declared, Konstantin Cesnulevicius Employee of: Biologische Heilmittel Heel GmbH, Myron Schultz Employee of: Biologische Heilmittel Heel GmbH, Olaf Wolkenhauer Grant/research support from: The research project was financially supported by Biologische Heilmittel Heel GmbH (Heel), Consultant for: I have received consultancy honoraria from Heel, Shahiendra Gupta Grant/research support from: The research project was financially supported by Biologische Heilmittel Heel GmbH (Heel), Consultant for: I have received consultancy honoraria from Heel


**AB0064**

**SERUM TENASCIN-C LEVELS ARE ELEVATED IN PATIENTS WITH AXIAL SPONDYLOARTHROPATHIES**

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**Background:** Tenascin-C (TNC) is a pro-inflammatory extracellular matrix glycoprotein that is involved in various pathological conditions. TNC induces inflammatory activity and promotes damage of joints. Its expression in adults is restricted to sites of tissue injury, particularly during phases of inflammation and active tissue remodelling. Only a few studies have described elevated serum TNC levels in ankylosing spondylitis (AS) compared to healthy controls (HC).

**Objectives:** The aim of this study was to examine the levels of serum TNC among different axial spondyloarthritis (axSpA) subsets and whether TNC levels are related to disease activity measures or other clinical features.

**Methods:** Sixty-one patients who fulfilled the Assessment of SpondyloArthritis international Society (ASAS) classification criteria for axSpA and twenty age and sex-matched HC were included in this study. Based on imaging, patients were further classified as AS (n = 45) and as nr-axSpA (n = 16). Patients with AS were further divided into two subsets based on the absence (n=22) or presence of syndesmophytes (n=23).

**TNC serum levels were determined using ELISA.** The following data were collected: clinical and laboratory disease activity measures; demographic status; disease-related factors such as the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) and CRP levels. Statistical analyses were performed with GraphPad Prism 5.1. The data are presented as the median and interquartile range.

**Results:** TNC serum levels were elevated in axSpA patients (535.3 (457.7-677.2) ng/mL) compared to HC (432.1 (329.1-565.9) ng/mL, p = 0.007). Diving axSpA into nr-axSpA and AS subsets the difference was observed only between AS patients and HC (535.3 (434.5-677.2) vs. 432.1 (329.1-565.9) ng/mL, p = 0.022). TNC serum levels did not correlate with disease activity biomarkers (serum CRP or BASDAI) in patients with axSpA. Although we have not observed a correlation between TNC and mSASSS radiographic score, weak correlation with disease subsets was found (r=0.25, p=0.025).

**Conclusion:** We demonstrated here elevated serum TNC levels in patients with axSpA, particularly in those with syndesmophytes, which may suggest its role in bone formation during radiographic stage of the disease.

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