Background: Inflammatory mediators and prospects the use of p75NTR inhibitors as a new therapeutic approach to chronic arthritis.

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-inflammation cytokine production, creating a vicious circle that amplifies the inflammatory response. Blocking the binding of endogenous proNGF to its receptor p75NTR strongly reduces the production 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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AB0064 SERUM TENASCIN-C LEVELS ARE ELEVATED IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS
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