6 THE SPECIFIC INHIBITOR OF RORγT TRANSCRIPTIONAL ACTIVITY AND TH17 POLARISATION DIGOXIN REDUCES THE SEVERITY OF ADJUVANT-INDUCED ARTHRITIS

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Background The authors have previously reported an increase of interleukin (IL)-17 levels during the first weeks of rheumatoid arthritis onset.

Objectives Our main goal here was to investigate whether administration of digoxin, a specific inhibitor of Th17 cells polarisation, is able to attenuate inflammation in a rat model of adjuvant-induced arthritis (AIA).

Materials and methods Digoxin was administered to AIA rats in the early phase (4 days after disease induction) or in the established phase of arthritis (11 days after disease induction). The inflammatory score, paw perimeter and body weight were

evaluated during the period of treatment. Rats were killed after 19 days of disease evolution and paw samples were collected for histological and immunohistochemical evaluation.

Results The authors found that digoxin administration significantly suppressed joint inflammation if administrated in the early phase of disease course. The histological and immunohistochemical evaluation revealed that digoxin treatment was not efficient in inhibiting the infiltration of immune cells within the joint, but it was able to reduce local immune cells proliferation, if administrated in the early phase of arthritis.

Conclusions Early inhibition of Th17 polarisation ameliorates AIA but does not inhibit immune cell infiltration into the joints.