adjuvant and boosted 35 days later (Stockholm ethical committee license number N66/10). At the end of the experiment mice were killd and investigated by histopathology and for T and B cell responses to CII.

Results Naïve Ncf1 mutant mice did not have noticeable differences in the composition of leucocytes nor did they have signs of activation in lymph nodes and spleen as judged by flow cytometry. After CIA boost both male and female Ncf1 mutant mice developed arthritis with higher incidence compared to wild type controls. CII reactive T cells produced both interferon-γ and interleukin-17 in an enzyme-linked immunosorbent spot assay and titres of anti-CII antibodies were detected in the serum.

Conclusion The arthritis susceptibility in ROS deficient mice is not dependant on persistent bacterial infections but occurs also in an aseptic environment. This indicates that ROS has an important immune regulatory role on T cell priming and/or the inflammatory response in the joint.

4 ARTHRITIS DEVELOPMENT IN GERM FREE MICE DEFICIENT FOR REACTIVE OXYGEN SPECIES

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Background The Ncf1 protein is a subunit of the nicotinamide adenine dinucleotide phosphate oxidase (NADPH) oxidase complex, which is responsible for producing the precursors of reactive oxygen species (ROS). ROS is mainly used to kill invading pathogens and a defect in the NADPH oxidase complex therefore leads to a diminished resistance to bacterial and fungal infections in humans, a disorder called chronic granulomatous disease. Interestingly, mice and rats with a mutated Ncf1 gene are more sensitive to arthritis induction compared to wild type littermates. However, Ncf1 mutant mice are also more prone to infections, which could affect arthritis incidence and severity. To dissect the immune regulatory role of ROS from its role in pathogen control, Ncf1 mutant mice were bred in germ free condition and investigated for collagen induced arthritis (CIA).

Methods Ncf1 mutant mice and wild type controls both on B10Q background were bred in the germ free facility at Karolinska Institute. 10–14 week old mice were immunised with rat collagen type II (CII) emulsified in complete freunds