cocktail containing four antibodies of $\gamma 2a$ and $\gamma 2b$ isotypes. Interestingly, IgG subclass dependency of *Cia9i* gene segment in arthritis development was ascertained using CII-specific $\gamma 2a$ or $\gamma 2b$ subclass antibodies. Furthermore, the significance of Fc γ R locus controlling CIA could be confirmed by analysis of the Northport heterogeneous stock cohort, a cross between eight inbred strains involving higher recombinant resolution.

Conclusions The data from congenic approach and HS cross thus suggest increased susceptibility to arthritis of *Cia9* locus is due to polymorphisms in the Fc γ R region.

23 IDENTIFICATION OF ARTHRITIS PROMOTING NON-OBES DIABETIC GENES IN THE *CIA9* LOCUS USING DIFFERENT GENETIC STRATEGIES

Dorota Kłaczowska, Diana Ekman, Rikard Holmdahl, Kuttyselva Nandakumar *Division of Medical Inflammation Research, Department of Medical Biochemistry and Biophysics, Karolinska Institute, Stockholm, Sweden*

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Background In the gene segregation experiment between susceptible B10.Q and protective NOD.Q mice strains, two strong loci associated with collagen-induced arthritis (CIA) were found, disease-promoting locus on chromosome 1 named *Cia9* and a disease-protective locus on chromosome 2, named *Cia2*. NOD allele on *Cia9* locus promotes arthritis whereas on *Cia2* locus it inhibits arthritis development.

Materials and methods The authors used both the classic congenic approach and heterogeneous stock mice to identify arthritis promoting genes in the *Cia9* locus. The authors dissected the original *Cia9* locus into three minimal fragments namely, *Cia9b* (165–172.6 Mbp), *Cia9c* (173–175 Mbp) and *Cia9i* (170.9–173.4 Mbp) by generating three congenic lines.

Results Fragments *Cia9b* and *Cia9c* had no effect on CIA and collagen-antibody induced arthritis (CAIA). On the other hand, *Cia9i* fragment containing a cluster of Fc γ R genes promoted CIA and, CAIA induced by a monoclonal antibody