Zhou et al. Vol. 188, No. 5, September 7, 1998. Pages 877–886.

The authors deeply regret to report here that they now believe some of the data published in their paper are not valid, and apologize to their colleagues for any difficulties caused by inclusion in the paper of these results.

In the paper, they described rats transgenic for a minigene construct, NP1, expressing the peptide SRYWAIRTR in the endoplasmic reticulum. The main conclusion of the paper remains valid, that the presence of the NP1 transgene construct reduces the prevalence of arthritis in B27 rats. Moreover, the validity and usefulness of the HLA-B27 transgenic rat disease model remain unimpaired. However, the results shown in Fig. 2 C, Fig. 5, and Fig. 6, A and B, must now be considered invalid. Moreover, although the mass spectrometry analysis described in Fig. 4 and in the text is valid and accurate, the source of the samples that were analyzed is questionable. The purpose of the experiments that are now in question was to confirm and quantitate the expression of the NP1 peptide in $B27^+NP1^+$ transgenic rats. Despite removal of these results from the data set, the basic conclusion of the paper is not altered, namely, that the NP peptide is expressed from the NP1 minigene and that this expression influences the disease phenotype of HLA-B27 transgenic rats by reducing the prevalence of arthritis. Updated data confirming the influence of the NP transgene constructs on B27-associated arthritis in rats have recently been published (Taurog, J.D., S.D. Maika, N. Satumtira, M.L. Dorris, I.L. McLean, H. Yanagisawa, A. Sayad, A.J. Stagg, G.M. Fox, A. Le O'Brien, M. Rehman, M. Zhou, A.L. Weiner, J.B. Splawski, J.A. Richardson, and R.E. Hammer. 1999. *Immunol. Rev*. 169:209–223).