TCR, also develop spontaneous uveitis [22,23,44]. However, some findings in these mice must be interpreted with caution, because responses may be affected by integration effects, by the level of expression and tissue distribution of the neo-self Ag and by the affinity of the specific TCR, and therefore may be quite different from responses to the native Ag. Thus, the R161 TCR transgenic mice offer some specific advantages over the previously available models that make them attractive for particular types of studies, e.g., study of natural triggers of uveitis or cell migration, under "amplified" conditions.

In conclusion, TCR transgenic mice specific to native self-antigens are available for only a few autoimmune disease models, namely, type 1 diabetes [13,14], experimental autoimmune gastritis [15,16] and experimental autoimmune encephalomyelitis [17–21]. There is no doubt that they have contributed tremendously to unraveling the basic mechanisms involved in the pathogenesis of the diseases represented by these models. It is of note that some of them develop autoimmune diseases spontaneously under certain environmental conditions (e.g. "dirty" housing) and/or genetic backgrounds (crossed to a specific MHC, or in a lymphopenic situation). Our new model of spontaneous uveitis in R161 TCR Tg mice now joins these other disease models as a valuable tool to study the pathogenesis of potentially blinding uveitic diseases.

Acknowledgment

We thank NEI Genetic Engineering Core for microinjection of TCR transgenic constructs and assistance in maintaining the Tg mouse colonies, NEI Histology Core for processing the slides and NEI Flow Cytometry Core for assistances in cell sorting and analysis. We are grateful to Drs. Hidehiro Yamane and William E. Paul for critical reading of the manuscript. This work was supported by NEI intramural funding, project # EY000184.

Appendix A. Supplementary data

Supplementary data related to this article can be found online at http://dx.doi.org/10.1016/j.jaut.2013.06.003.

Supplementary Table 1
Vα and Vβ primer sequences used in this study.

Vα	Primer sequence (5′ -> 3′)	Vβ	Primer sequence (5′ -> 3′)
1	GCACTGATGTCCATCTTCTC	1	CTGAATGCCCAGACAGCTCCAAGC
2	AAAGGGAGAAAAAGCTCTCC	2	TCACTGATACGGAGCTGAGGC
3	AAGTACTATTCCGGAGACCC	3.1	CCTTGCAGCCTAGAAATTCAGT
4	CAGTATCCCGGAGAAGGTC	4	GCCTCAAGTCGCTTCCAACCTC
5	CAAGAAAGACAAACGACTCTC	5.1	CATTATGATAAAATGGAGAGAGAT
6	ATGGCTTTCCTGGCTATTGCC	5.2	AAGGTGGAGAGAGACAAAGGATTC
7	TCTGTAGTCTTCCAGAAATC	6	CTCTCACTGTGACATCTGCCC
8	CAACAAGAGGACCGAGCACC	7	TACAGGGTCTCACGGAAGAAGC
9	TAGTGACTGTGGTGGATGTC	8.1	CATTACTCATATGTCGCTGAC
10	AACGTCGCAGCTCTTTGCAC	8.2	CATTATTCATATGGTGCTGGC
11	CCCTGCACATCAGGGATGCC	8.3	TGCTGGCAACCTTCGAATAGGA
12	TCTGTTTATCTCTGCTGACC	9	TCTCTCTACATTGGCTCTGCAGGC
13	TGAGGCCGAGTTTAGGAAGA	10	ATCAAGTCTGTAGAGCCGGAGGA
14	GAGTCCTCAGTCCCTGGTTG	11	GCACTCAACTCTGAAGATCCAGAGC
15	AACGATTCTCCCTGCACATC	12	GATGGTGGGGCTTTCAAGGATC
16	CTGTAGTGCAGAGCCCTTCC	13	AGGCCTAAAGGAACTAACTCCCAC
17	TTCCATCGGACTCATCATCA	14	ACGACCAATTCATCCTAAGCAC
18	AGAAGCGCAGTGGAAGACTC	15	CCCATCAGTCATCCCAACTTATCC
19	TGCCTCATACCTCTGTGCTG	16	CACTCTGAAAATCCAACCCAC
20	TTCTCACTGCACATCACAGC	17	AGTGTTCCTCGAACTCACAG
		18	CAGCCGGCCAAACCTAACATTCTC
		19	CTGCTAAGAAACCATGTACCA
		20	TCTGCAGCCTGGGAATCAGAA
Cα	TGGCGTTGGTCTCTTTGAAG	Cβ	CTTGGGTGGAGTCACATTTCTC

Vα1-12 and Cα from Casanova et al. [31], Vα13-20 from DiRienzo et al. [32], and Vβ1-20 from Pannetier et al. [33].

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Please cite this article in press as: Horai R, et al., Breakdown of immune privilege and spontaneous autoimmunity in mice expressing a transgenic T cell receptor specific for a retinal autoantigen, Journal of Autoimmunity (2013), http://dx.doi.org/10.1016/j.jaut.2013.06.003