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Poster Abstract

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### ABSTRACT

Allymphoplasia ( *aly/aly* ) mice, a natural strain with a mutant NF- $\kappa$ B-inducing kinase ( NIK ) gene, manifest a unique phenotype lacking lymph nodes and Peyer's patches. *Aly/aly* mice have been well known to be as a model of Syögren syndrome because of the autoimmune inflammation in exocrine glands. However, the mechanisms of the autoimmunity in *aly/aly* mice have not been well understood. CD25<sup>+</sup>CD4<sup>+</sup> T cells were reported to suppress immune responses and play an important role in peripheral tolerance of autoreactive T cells as immune regulatory T cells. To clarify the mechanisms of the autoimmunity in *aly/aly* mice, we analyzed CD25<sup>+</sup>CD4<sup>+</sup> T cells in these mice. Numbers of CD25<sup>+</sup>CD4<sup>+</sup> T cells in thymus and spleen from *aly/aly* mice were significantly lower than those from *aly/+* or C57BL/6 mice. However, the suppressive function of CD25<sup>+</sup>CD4<sup>+</sup> T cells from *aly/aly* mice in in vitro proliferation assay was comparable with that of CD25<sup>+</sup>CD4<sup>+</sup> T cells from *aly/+* or C57BL/6 mice. *Aly/aly* mice adaptively transferred with CD25<sup>+</sup>CD4<sup>+</sup> T cells from C57BL/6 mice showed less inflammatory cell infiltration in salivary gland than non-transferred *aly/aly* mice. The number of CD25<sup>+</sup>CD4<sup>+</sup> T cells in the transferred *aly/aly* mice was higher than that of those T cells in non-transferred mice at 12 weeks after the transfer. These results indicate that the autoimmune inflammation in *aly/aly* mice seems to be resulted from the decreased number of CD25<sup>+</sup>CD4<sup>+</sup> T cells in these mice. Thus, survival and/or generation of CD25<sup>+</sup>CD4<sup>+</sup> T cells may be impaired in *aly/aly* mice.

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