



ELSEVIER

Contents lists available at ScienceDirect

Data in Brief

journal homepage: www.elsevier.com/locate/dib

Data Article

Data on the TGFβ response of CD4⁺ T cells in the absence of *Eed*Taku Naito^{a,*}, Sawako Muroi^b, Ichiro Taniuchi^b, Motonari Kondo^a^a Department of Molecular Immunology, Toho University School of Medicine, Tokyo, Japan^b Laboratory for Gene Regulation, Institute for Medical Sciences, RIKEN, Yokohama, Japan

ARTICLE INFO

Article history:

Received 24 January 2018

Received in revised form

8 February 2018

Accepted 14 February 2018

Available online 17 February 2018

Keywords:

TGFβ

PRC2

Eed

CD4⁺ CD8α⁺ T cells

ABSTRACT

The data presented here are related to the research article entitled “Loss of *Eed* leads to lineage instability and increased CD8 expression of mouse CD4⁺ T cells upon TGFβ signaling” [1]. The cited research article investigates the molecular mechanism of CD8α upregulation observed in *Eed*-deficient (ΔEed) CD4⁺ T cells upon activation in the presence of TGFβ. This data report describes the effect of retinoic acid (RA) and/or anti-interferon-gamma (IFNγ) antibody supplementation on up-regulation of CD8α and Foxp3 in ΔEed CD4⁺ T cells, the effect of dose or timing of TGFβ treatment on CD4⁺ T cell identity of ΔEed , adding further information regarding the conditions that induces CD8α, and mRNA expression changes of genes encoding polycomb repressive complex 2 (PRC2) subunits by TGFβ treatment.

© 2018 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>).

Specifications Table

Subject area	<i>Immunology and Molecular Biology</i>
More specific subject area	<i>Differentiation of T-helper subsets</i>
Type of data	<i>Graphs and flow cytometry plots.</i>

* Corresponding author.

E-mail address: taku.naitou@med.toho-u.ac.jp (T. Naito).<https://doi.org/10.1016/j.dib.2018.02.045>2352-3409/© 2018 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>).

How data were acquired	– Flow cytometry (FACSCanto II and FACSaria III, BD Biosciences)
Data format	– Quantitative PCR (qPCR) (ABI 7500 Fast, ABI and QuantStudio3, Thermo Fisher)
Experimental factors	Analyzed – The <i>Eed</i> gene was specifically deleted in T cells by crossing mice carrying the floxed <i>Eed</i> alleles [1] with the CD4-Cre transgenic mouse [2]. – Naïve (CD25 ⁻ CD62L ^{high} CD44 ^{low}) CD4 ⁺ T cells of wild type or ΔEed mouse were cultured in the presence of anti-CD3/anti-CD28 supplemented with cytokines, neutralizing antibodies, and/or retinoic acid. – RNA samples were recovered using TriZOL (Life Technologies), reverse transcribed using Super Script III (TaKaRa) with oligo-dT primers, and then subjected to qPCR analysis using ExTaq II SYBR Green Reagent (TaKaRa). – Expression of proteins and cell surface markers was assessed by flow cytometry. – Various concentrations or timings of TGF β treatment were tested.
Experimental features	– Expression of <i>Foxp3</i> in activated wild type or ΔEed T cells treated with TGF β , RA and anti-IFN γ antibody. – Expression of CD4 and CD8 α on ΔEed T cells treated with varying doses, or differing time windows, of TGF β . – Expression of genes encoding PRC2 subunits after TGF β treatment.
Data source location	Toho University School of Medicine, Tokyo, Japan
Data accessibility	Data are within this article.

Value of the data

- The data show the limited capability of ΔEed CD4⁺ T cells to differentiate toward the Foxp3⁺ Treg lineage by TGF β treatment, regardless of the presence of RA or the inhibition of IFN γ .
- The data reveal how ΔEed CD4⁺ T cells upregulate CD8 α in response to different dose of TGF β , or how the addition or withdrawal of TGF β at different time points affects CD8 α induction of ΔEed CD4⁺ T cells.
- The data show that the mRNA levels of *Eed* and *Ezh2* changes in the presence of TGF β upon activation.

1. Data

The expression of Foxp3 in TGF β -stimulated wild type and ΔEed CD4⁺ cells in the presence of RA and/or anti-IFN γ are shown in Fig. 1. The response of ΔEed CD4⁺ T cells to different concentrations of TGF β , or to TGF β added in different time windows after anti-CD3/anti-CD28 activation, are shown in Fig. 2. The changes in the expression level of *Eed* and *Ezh2* after anti-CD3/anti-CD28 activation in the presence or absence of TGF β , measured by qPCR, are presented in Fig. 3.

2. Experimental design, materials and methods

2.1. Cell isolation, cell culture, and flow cytometry

Detailed procedures and used reagents are as previously described [1].

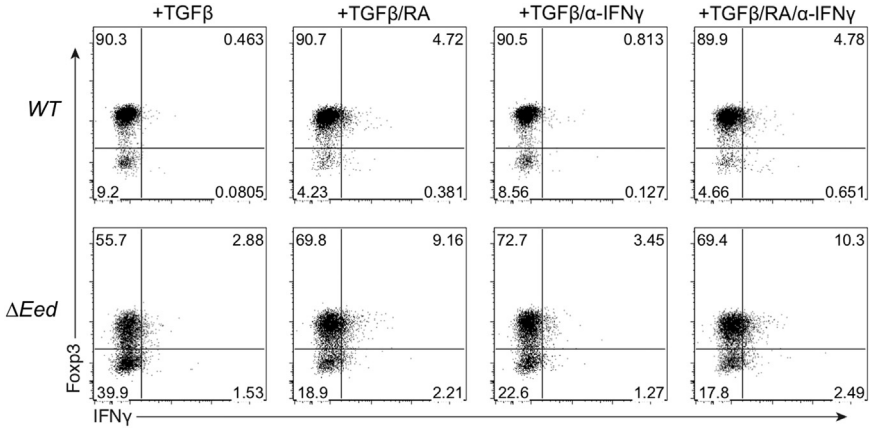


Fig. 1. Effects of RA and anti-IFN γ antibody on Treg differentiation in Δ Eed T cells. T cells were activated with anti-CD3/anti-CD28 antibody in the presence of TGF β , supplemented with RA, anti-IFN γ antibody, or both. Profiles of Foxp3 and IFN γ expression under each condition are shown.

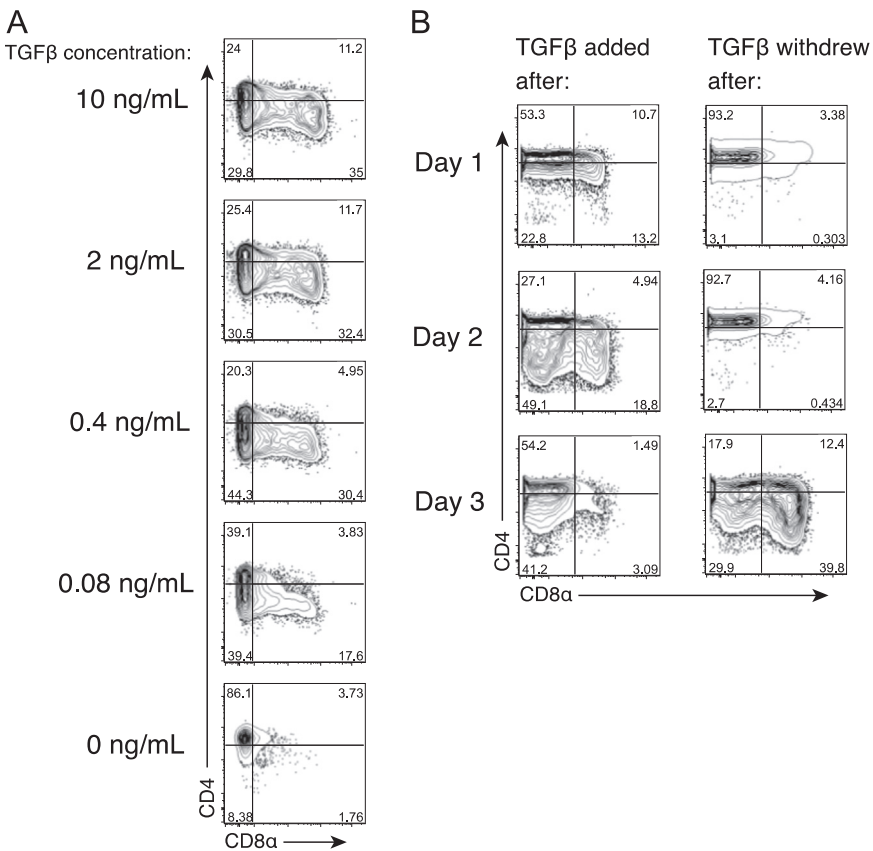


Fig. 2. Effect of TGF β concentration and timing on CD8 α induction. (A) Δ Eed T cells were activated with anti-CD3/anti-CD28 antibodies in the presence of the indicated amount of TGF β . The CD4/CD8 α profiles of 6 days post-activation are shown. (B) Δ Eed T cells were activated with anti-CD3/anti-CD28. Ten ng/mL of TGF β was added to the culture one, two or three days after activation (left column). Alternatively, TGF β was included in the culture at the time of activation and then withdrew one, two or three days after activation (right column). The CD4/CD8 α expression profiles of 6 days post-activation are shown.

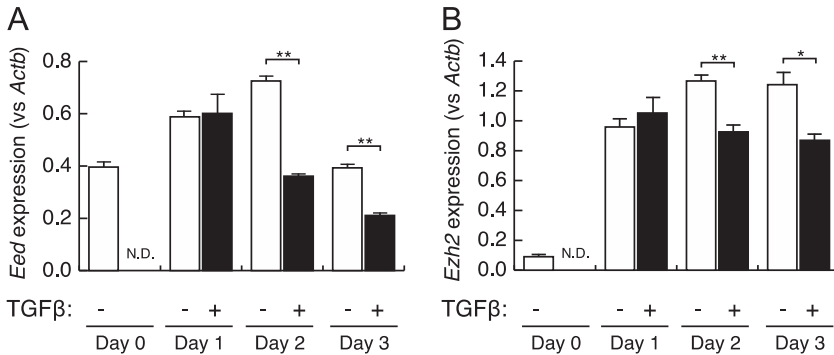


Fig. 3. Expression changes of PRC2 component by TGF β treatment. Naïve WT CD4⁺ cells were activated with anti-CD3/anti-CD28 in the presence or absence of TGF β , and expression of *Eed* or *Ezh2* was examined by qPCR, normalized by *Actb* expression. Mean \pm S.E.M. are shown. * $P < 0.05$ and ** $P < 0.01$ by Student *t*-test. N.D.: no data.

2.2. Quantitative PCR

Detailed procedures of RNA isolation, cDNA synthesis and qPCR are as previously described [1]. The sequences of primers used in this study are: *Eed* forward; gttgagcagcgacgagaacag, *Eed* reverse; gtgcactctcaactgacag, *Ezh2* forward; actgctggcaccgtctgatg, *Ezh2* reverse; tcctgagaataatctccccag.

2.3. Statistical analysis

Data were analyzed using a two-tailed, paired Student *t*-test where appropriate.

Acknowledgements

This work was funded by the Ministry of Education, Culture, Sports, Science and Technology Grants-in-Aid for Scientific Research (C) #25460599, and by Toho University School of Medicine Grants for Project Research #25-5, awarded to T.N., and a Grant-in Aid for Private University Research Branding Project from the Ministry of Education, Culture, Sports, Science and Technology awarded to M.K. The final manuscript was checked by R.J. Turner.

Transparency document. Supplementary material

Transparency data associated with this article can be found in the online version at <https://doi.org/10.1016/j.dib.2018.02.045>.

References

- [1] T. Naito, S. Muroi, I. Taniuchi, M. Kondo, Loss of *Eed* leads to lineage instability and increased CD8 expression of mouse CD4⁺ T cells upon TGF β signaling, *Mol. Immunol.* 94 (2018) 140–152. <http://dx.doi.org/10.1016/j.molimm.2017.12.021>.
- [2] P.P. Lee, D.R. Fitzpatrick, C. Beard, H.K. Jessup, S. Lehar, K.W. Makar, et al., A critical role for Dnmt1 and DNA methylation in T cell development, function, and survival, *Immunity.* 15 (2001) 763–774.