# Histone deacetylases 1 and 2-mediated control of intestinal CD4+ T cell immunity during bacterial infection

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**Type selection**

**Choose your preferred presentation format**: Oral presentation

**General data**

**Track**: Adaptive Immunity

**Topic**: Transcriptional and epigenetic control of immune cell function

**Would the presenting author be interested in applying for a travel grant?** No

**Abstract text**

**Text**: Some effector CD4$^+$ T cell subsets display cytotoxic activity, thus breaking the functional dichotomy of CD4 $^+$ helper and CD8$^+$ cytotoxic T lymphocytes. However, molecular mechanisms regulating CD4$^+$ cytotoxic T lymphocyte (CD4 CTL) differentiation are poorly understood. We previously showed that the combined activity of histone deacetylases 1 and 2 (HDAC1 and HDAC2) is crucial for maintaining CD4 lineage integrity and that HDAC1-HDAC2 levels are key determinants of CD4 CTL differentiation in lymph nodes and spleen during CD4$^+$ T activation and viral infection. However, it is not known whether the combined activity of HDAC1/HDAC2 is also required for the proper differentiation, regulation and function of intestinal CD4 CTLs as well as for proper CD4$^+$ T cell responses during bacterial infection.

In order to address this question, we have been performing a comprehensive analysis, including flow cytometry, mucosal infection with *Citrobacter rodentium* and scRNA-seq approaches, of intestinal T cells from mice with a deletion of both Hdac1 and one Hdac2 alleles (HDAC1 $^{cKO}$-HDAC2$^{HET}$).

Our results indicate alterations in intestinal CD4$^+$ and CD8$^+$ TCRab$^+$ T cell composition within small intestinal intraepithelial lymphocyte (SI-IEL) and lamina propria (SI-LPL) populations of HDAC1$^{cKO}$-HDAC2$^{HET}$ mice at steady-state. Upon *Citrobacter rodentium* infection, colonic CD4$^+$ and CD8$^+$ TCRab$^+$ T cells from HDAC1$^{cKO}$-HDAC2$^{HET}$ mice displayed impaired T cell responses in comparison to wild-type mice accompanied with an upregulation of cytotoxic markers.

Our data suggest that HDAC1$^{cKO}$-HDAC2$^{HET}$ mice have a distinct intestinal T cell composition compared to wild-type mice, which might result in a compromised function of intestinal immunity in homeostasis and during infection with *Citrobacter rodentium*. Data from our ongoing study will be presented.

**Conflict of Interest statement**

**Do you have any conflict of interest to declare?**: No

**Affirmation**

**1. Consent to participate and present onsite**: Yes

**2. Consent to publication publication**: Yes

**3. Consent on treatment of contact details saved in this system**: Yes