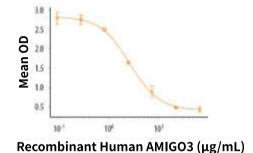
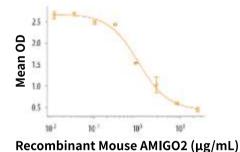
## Immune checkpoint: AMIGO Family

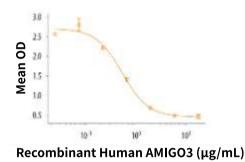
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## AMIGO2 modulates T cell functions and its deficiency in mice ameliorates experimental autoimmune encephalomyelitis

The immune function of AMIGO2 is currently unknown. Here, we revealed novel roles of AMIGO2 in modulating T-cell functions and EAE using Amigo2-knockout (AMG2KO) mice. Amigo2 was abundantly expressed by murine T helper (Th) cells. Its deficiency impaired transplanted T-cell infiltration into the secondary lymphoid organs and dampened Th-cell activation, but promoted splenic Th-cell proliferation and abundancy therein. AMG2KO Th cells had respectively elevated T-bet in Th1- and GATA-3 in Th2-lineage during early Th-cell differentiation, accompanied with increased IFN-γ and IL-10 but decreased IL-17A production. AMG2KO mice exhibited ameliorated EAE, dampened spinal T-cell accumulation, decreased serum IL-17A levels and enhanced splenic IL-10 production. Adoptive transfer of encephalitogenic AMG2KO T cells induced milder EAE and dampened spinal Th-cell accumulation and Tnf expression. Mechanistically, Amigo2-overexpression in 293T cells dampened NF-kB transcriptional activity, while Amigo2-deficiency enhanced Akt but suppressed GSK-3β phosphorylation and promoted nuclear translocations of NF-kB and NFAT1 in Th-cells. Collectively, our data demonstrate that AMIGO2 is important in regulating T-cell functions and EAE, and may be harnessed as a potential therapeutic target for multiple sclerosis.





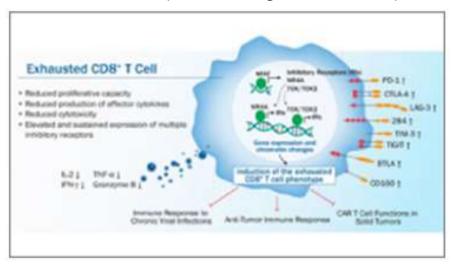


Mouse CD3+ T cell에서 IL-2의 분비가 AMIGO family protein에 의하여 저해되는 것을 측정

## \* R&D systems의 AMIGO proteins

Proteins	Species	Catalog No.
AMIGO	Human	2565-AM
AMIGO2	Human	2080-AM
AMIGO2	Mouse	9424-AM
AMIGO3	Human	9508-AM
AMIGO3	Mouse	9425-AM

이외의 Immune checkpoint 와 Next generation checkpoint 정보를 확인해보세요



More Information >>