

OX40L-Jagged-1-Fc, for the In Vivo Expansion of Treg Cells

► Asset Overview

Product Type	Chimeric Fusion Protein
Indication	Immunology
Current Stage	Preclinical
Target(MoA)	Targeting both Notch3 and OX40 receptors on Tregs
Brief Description	<ul style="list-style-type: none"> • Tregs downregulation has been implicated in the pathogenesis of many autoimmune diseases • Selectively expands functional Tregs <i>in vitro</i> and <i>in vivo</i>, and not pathogenic T effector cells (Teff) • Restores homeostatic balance to the immune system • Does not cause general immune suppression • Current research validates both OX40 and Notch3 as targets • Next plan: Determine pathway for product type (separate Notch3 and OX40 ligands, a linked Notch3-OX40 ligand, or Notch3-OX40 bi-specific antibody) and indication for the best regulatory path
Organization	University of Illinois at Chicago

► Differentiation

□ Tregs in autoimmune disease

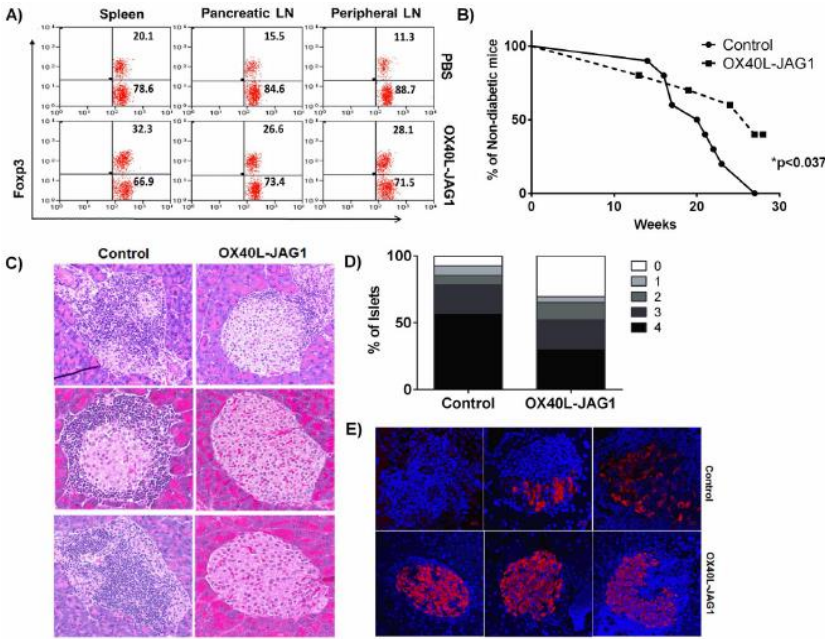
- Tregs down regulation causes the self-reactive lymphocytes to escape natural control, thereby triggering the immune system to attack self
- Current immunosuppressive therapies non-specifically suppress the body's defense/immune system, resulting in debilitating side effects and a very poor quality of life
- Currently available approaches are not curative, are nonspecific, are non-targeted, and have a narrow therapeutic window (efficacy–toxicity trade-off)
- There is a dire need for a more targeted, safe & accepted approach

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► Key Data

Preliminary diabetes results by combination of OX40L and Jagged1

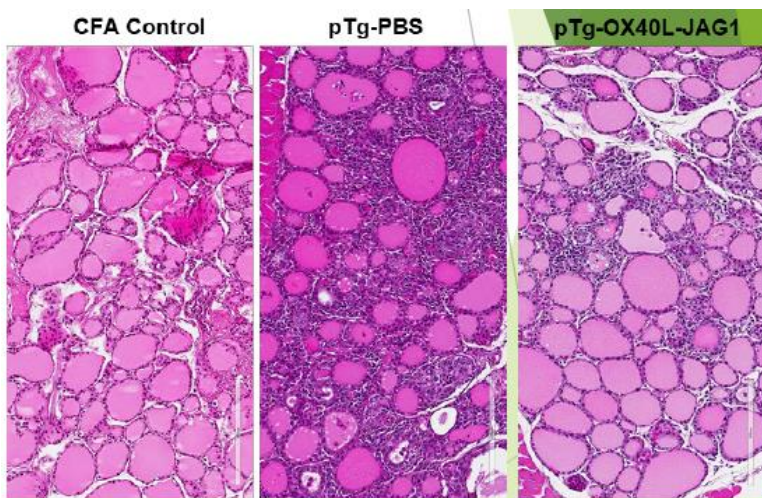
1-NOD mice treated with soluble OX40L and Jagged1



- ✓ Significantly delays the onset of diabetes
- ✓ Substantially reduces insulitis
- ✓ Arrests insulin producing islet beta cell destruction
- ✓ Increases anti-inflammatory cytokines (e.g., IL10, TGF-β)
- ✓ Decreases pro-inflammatory cytokines (e.g., IL1β, IFNγ)

Preliminary diabetes results by combination of OX40L and Jagged1

1-NOD mice treated with soluble OX40L and Jagged1



- ✓ Significantly delays the onset of Thyroiditis
- ✓ Arrests Thyroid hormone producing thyrocyte destruction

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► Intellectual Property

Patent No.	PCT-US2016-060349
Application Date	2016.11.03
Status	Application Pending
Country	US, EP, IN

► Contact Information

Contact Person	Kim, Hyunjin
Email	hkim227@otm.uic.edu
URL	https://uic.flintbox.com/#technologies