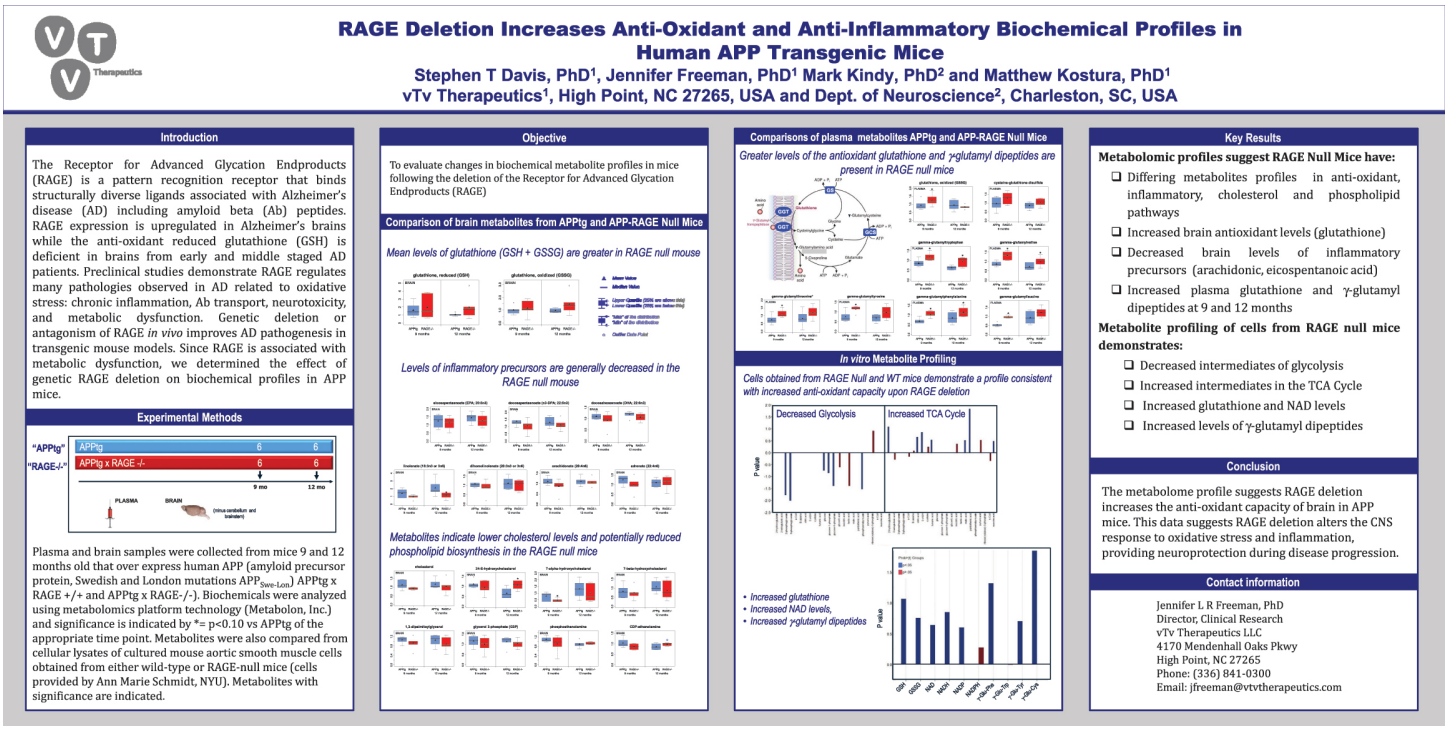
Explanatory Note: The following clinical posters regarding the Receptor for Advanced Glycation Endproducts (RAGE) and *azeliragon* (TTP488) have been prepared by vTv Therapeutics, Inc. (the "Company") or with the Company's participation. The Company expects that the posters regarding RAGE and TTP488 will be posted at the Alzheimer's Association International Conference on or about July 20, 2015 and July 21, 2015, respectively. These posters are being provided to supplement certain information in the Company's preliminary prospectus filed by the Company with the United States Securities and Exchange Commission on July 20, 2015, and are thus deemed to be a free writing prospectus. For additional information regarding the Company and the offering to which this free writing prospectus relates, please see the information below under the caption, "Free Writing Prospectus."



RAGE Deletion Increases Anti-Oxidant and Anti-Inflammatory Biochemical Profiles in Human APP Transgenic Mice

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Introduction

The Receptor for Advanced Glycation Endproducts (RAGE) is a pattern recognition receptor that binds structurally diverse ligands associated with Alzheimer's disease (AD) including amyloid beta (Ab) peptides. RAGE expression is upregulated in Alzheimer's brains while the anti-oxidant reduced glutathione (GSH) is deficient in brains from early and middle staged AD patients. Preclinical studies demonstrate RAGE regulates many pathologies observed in AD related to oxidative stress: chronic inflammation, Ab transport, neurotoxicity, and metabolic dysfunction. Genetic deletion or antagonism of RAGE *in vivo* improves AD pathogenesis in transgenic mouse models. Since RAGE is associated with metabolic dysfunction, we determined the effect of genetic RAGE deletion on biochemical profiles in APP mice.

Experimental Methods

"APPtg" APPtg 6 6
"RAGE-/-" APPtg x RAGE -/- 6 6
9 mo 12 mo
PLASMA BRAIN (Hindbrain (cerebellum and brainstem))

Plasma and brain samples were collected from mice 9 and 12 months old that over express human APP (amyloid precursor protein, Swedish and London mutations $APP_{Swe-Lon}$) APPtg x RAGE +/+ and APPtg x RAGE-/-). Biochemicals were analyzed using metabolomics platform technology (Metabolon, Inc.) and significance is indicated by *= p<0.10 vs APPtg of the appropriate time point. Metabolites were also compared from cellular lysates of cultured mouse aortic smooth muscle cells obtained from either wild-type or RAGE-null mice (cells provided by Ann Marie Schmidt, NYU). Metabolites with significance are indicated.

Objective

To evaluate changes in biochemical metabolite profiles in mice following the deletion of the Receptor for Advanced Glycation Endproducts (RAGE)

Comparison of brain metabolites from APPtg and APP-RAGE Null Mice

Mean levels of glutathione (GSH + GSSG) are greater in RAGE null mouse

glutathione, reduced (GSH) glutathione, oxidized (GSSG)

Levels of inflammatory precursors are generally decreased in the RAGE null mouse

Metabolites indicate lower cholesterol levels and potentially reduced phospholipid biosynthesis in the RAGE null mice

Comparisons of plasma metabolites APPtg and APP-RAGE Null Mice

Greater levels of the antioxidant glutathione and γ-glutamyl dipeptides are present in RAGE null mice

In vitro Metabolite Profiling

Cells obtained from RAGE Null and WT mice demonstrate a profile consistent with increased anti-oxidant capacity upon RAGE deletion

Decreased Glycolysis Increased TCA Cycle

- *Increased glutathione*
- *Increased NAD levels,*
- *Increased γ-glutamyl dipeptides*

Key Results

Metabolomic profiles suggest RAGE Null Mice have:

- ❑ Differing metabolites profiles in anti-oxidant, inflammatory, cholesterol and phospholipid pathways
- ❑ Increased brain antioxidant levels (glutathione)
- ❑ Decreased brain levels of inflammatory precursors (arachidonic, eicospentanoic acid)
- ❑ Increased plasma glutathione and γ-glutamyl dipeptides at 9 and 12 months

Metabolite profiling of cells from RAGE null mice demonstrates:

- ❑ Decreased intermediates of glycolysis
- ❑ Increased intermediates in the TCA Cycle
- ❑ Increased glutathione and NAD levels
- ❑ Increased levels of γ-glutamyl dipeptides

Conclusion

The metabolome profile suggests RAGE deletion increases the anti-oxidant capacity of brain in APP mice. This data suggests RAGE deletion alters the CNS response to oxidative stress and inflammation, providing neuroprotection during disease progression.

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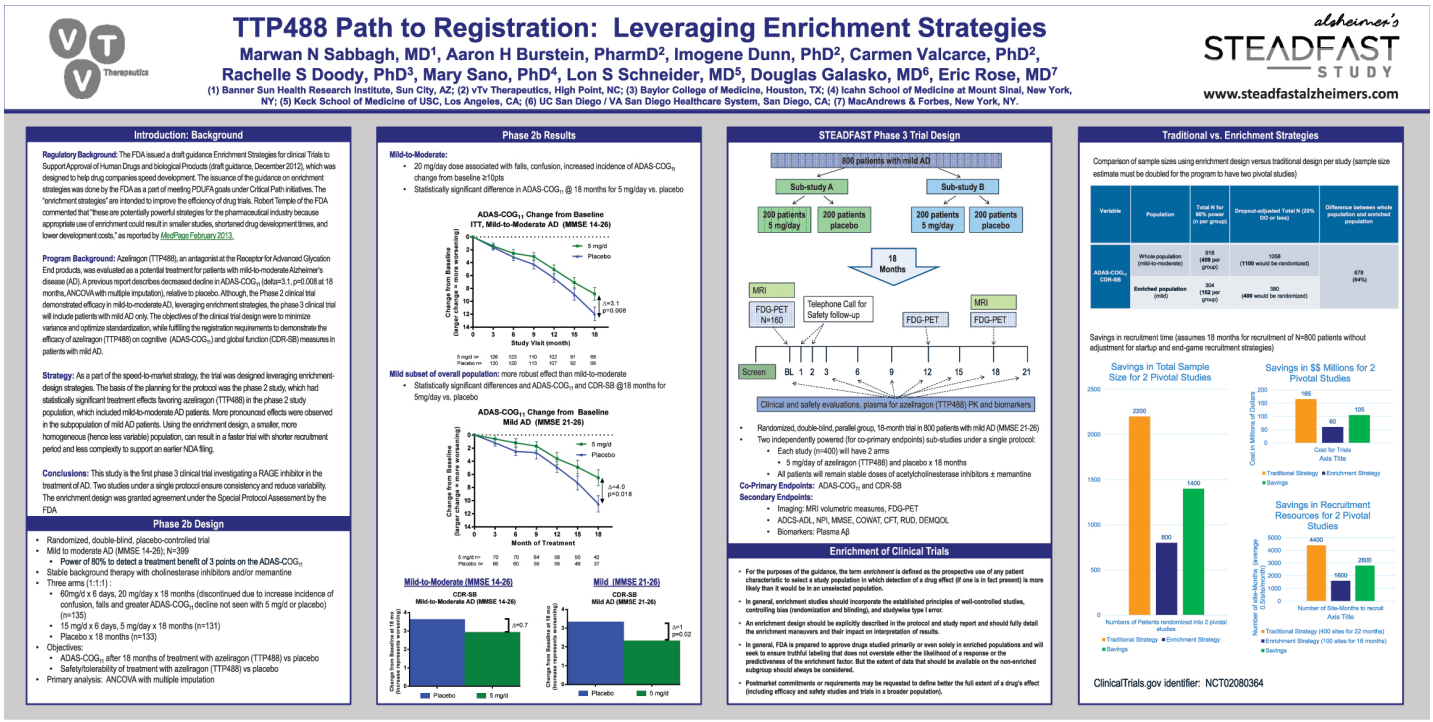

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