Solanezumab's interim phase II data suggests an attractive safety profile (vs. Bapineuzumab) and pharmacodynamic effect (CSF and plasma biomarkers); Efficacy and dose response data is indeterminate; Phase III trials are not prospectively stratified by ApoE4 status.

	Clinical Results (Phase II)
Efficacy:	• Demonstrated increased beta amyloid levels in CSF and plasma, suggest Solanezumab mobilizes beta amyloid in plaques and normalizes soluble CSF beta amyloid - Subsequently, a correlation was established between plasma beta-amyloid amyloid burden using single PET with IMPY - No effect was shown on CSF t-tau and p-tau • No clinical efficacy or dose response has been shown, given the 12 week duration of the initial interim phase II data
Safety:	• No reports of treatment-related vasogenic edema, infusion reactions or meningoencephalitis
Dose Response:	• Indeterminate
Lumleian Commentary:	• Interim phase III data reads out in Q1 '12 with top line phase III results in H2 '12; Lack of ApoE4 stratification

	Phase II Program (Completed)	Phase III Program (Ongoing)
Patient Segment:	• Mild to Moderate AD	• Mild to Moderate AD
Stratification:		• Post-hoc: ApoE4 carrier; ApoE4 non-carrier
Studies: (Target Enrollment)	• Single phase II (N=52), Sub-Study (N=24) • Open label study in Japan (N=33)	• EXPEDITION 1 (N=1,000); 1:1 arms; Global • EXPEDITION 2 (N=1,000); 1:1 arms; Global
Comparator:	• Placebo	• Placebo
Dosing:	• 100; 400 mg/kg IV every week • 100; 400 mg/kg IV every month	• 100; 400 mg/kg IV every month
Duration:	• 12 weeks	• 18 months
Primary End-Points:	• Cognition: ADAS-cog • Function: NA	• Cognition: ADAS-cog • Function: ADCS-ADL

Notes: In January '11 Lilly announced that one patient in phase III trials had developed vasogenic edema, but did not specify if this was in the Solanezumab or placebo arm

Sources: IACD presentation (2008); Company press releases; 3rd party equity research reports; Bio-Pharma Insight; Clinical Trials.gov; Centerwatch; Siemers, E. R., S. Friedrich, and R. A. Dean, et al. "Safety and Changes in Plasma and Cerebrospinal Fluid Amyloid Beta after a Single Administration of an Amyloid Beta Monoclonal Antibody in Subjects with Alzheimer Disease." *Clinical Neuropharmacology* 33.2 (2010): 67-73.; Siemers , E. R., R. B. Demattos, F. Stuart et al., "Use of a Monoclonal Anti-A Antibody with Biochemical and Imaging Biomarkers To Determine Amyloid Plaque Load in Patients with Alzheimer's Disease (AD) and Control Subjects," American Academy of Neurology 61st Annual Meeting; April 25-May 2, 2009; Seattle, WA. Abstract IN3-2.009.

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