About AlzForum

- Launched in 1996, Alzforum reports on the latest scientific findings, from basic research to clinical trials; creates and maintains public databases of essential research data and reagents, and produces discussion forums to promote debate, speed the dissemination of new ideas, and break down barriers across the numerous disciplines that can contribute to the global effort to cure Alzheimer's disease.
RESEARCH NEWS

- **Wake Up and Smell the...Neurodegeneration? AD Model Causes Cell Loss**
  3 October 2011. Researchers have created a mouse model that selectively and reversibly overexpresses a mutant form of human amyloid precursor protein (APP) in the olfactory epithelium...

- **Too Many “Burdensome Transitions” for Nursing Home Residents?**
  3 October 2011. According to a study in the New England Journal of Medicine, the final days for nursing home residents in this situation are often far from peaceful...

- **ALS: Sticky Tryptophan Achilles' Heel for Superoxide Dismutase?**
  3 October 2011. Researchers report on their use of a truncated enzyme and conformation-specific antibodies to analyze the folding and misfolding of superoxide dismutase 1 (SOD1)...

- **Does Deep-Brain Stimulation Spark Neurogenesis, Enhance Learning?**
  30 September 2011. Some preliminary studies hint that deep-brain stimulation (DBS) could help improve memory in people with dementia,
We Continue to Grow

- 3,000 news articles
- 86,000 citations
- 10,000 comments
- 7,300 members, including 3,200 researchers from 104 countries
- 700 genes/3,000 polymorphisms in AlzGene
- 30,000 antibodies
- 300 research models…
- ….1.4 millions page views from 231,000 unique visitors last year
New γ-Secretase Modulators Reduce Aβ42, Avoid Notch

12 September 2010. Despite recent setbacks on the clinical front (see ARF related news story), the hunt for small molecules that can cleanly tweak γ-secretase to slow Alzheimer disease seems to be alive and well. In the September 9 Neuron, researchers led by Steven Wagner, University of California, San Diego, report on a new class of γ-secretase modulators that reduces β amyloid deposition in an AD mouse model without affecting Notch or other γ-secretase substrates that mediate critical functions. Originally identified at Neurogenetics, Inc., a San Diego-based firm that became TorreyPines Therapeutics, Inc., which has since folded, the compound penetrates the brain, reduces levels of toxic Aβ more efficiently than do other γ-secretase modulators, and was safe over a six-month treatment in mice. “Overall, I thought this was a very nice drug discovery paper,” said Michael Wolfe of Brigham and Women’s Hospital in Boston. “This is an important new class of molecules that is definitely worth following up with more preclinical studies.”

Early efforts to target γ-secretase for AD focused on inhibiting the enzyme, which cuts a C-terminal piece of amyloid precursor protein (APP) to release the Aβ peptides that can glom together to form the hallmark AD plaques. However, because γ-secretase has numerous substrates besides APP, most notably Notch, the wholesale blockade of the enzyme is not a viable approach. The new class of molecules seems to be a promising alternative.
Relationship of this News Story to other AlzForum Content

- protein complex (1)
- protein (10)
- research news (5)
- paper (1)
- conference news (1)
- people (5)
- conference (1)
- research model (1)
- cell line (3)
- drug (5)
- company (5)
Content/Data Relationships, Potential Knowledge Discovery

- **BMS-708163** is a drug that mentions the News Article with News ID:2557.
- The News Article is a News Article.
- The Protein Complex is a protein complex.
- **Gamma-secretase** is a protein complex that has target BMS-708163.
- **Amyloid Precursor Protein** is a protein complex.
- **APP695 Transgenic Mouse** is a mouse.
- **APP695** is a protein.
- **Abeta40** is a protein.
- **Abeta42** is a protein.
- Protein Complex has target gamma-secretase.
- Amyloid Precursor Protein cleave into Abeta40 and Abeta42.
- Abeta40 is a protein.
- Abeta42 is a protein.