Erratum

In the article "Energy Inhibition Elevates β-Secretase Levels and Activity and Is Potentially Amyloidogenic in APP Transgenic Mice: Possible Early Events in Alzheimer's Disease Pathogenesis," by Rodney A. Velliquette, Tracy O'Connor, and Robert Vassar, which appeared on pages 10874–10883 of the November 23, 2005 issue, a low-resolution version of Figures 3, 4, and 5 was used in the printed issue. These figures and their legends are reprinted in this issue.

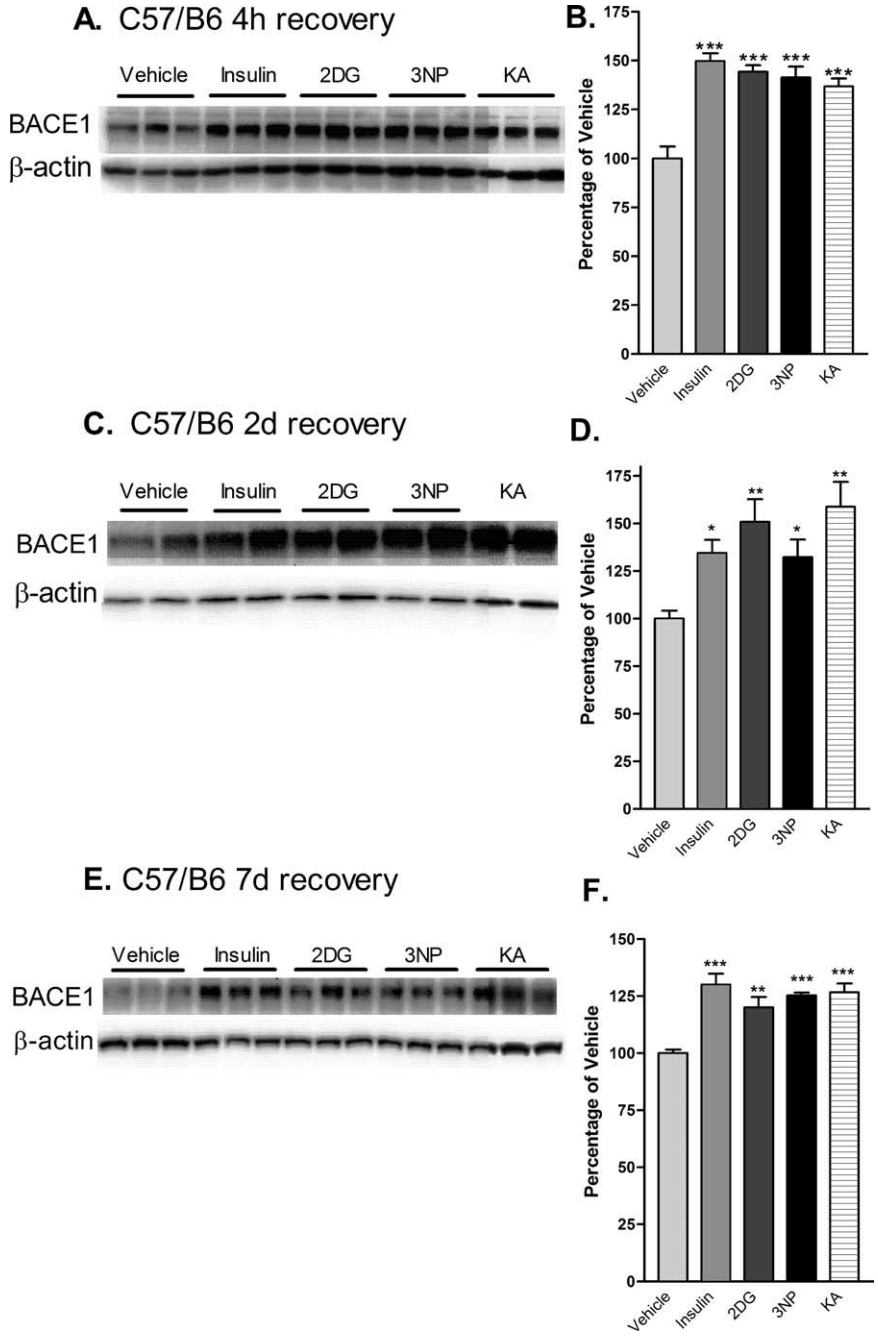

Figure 3. Acute energy inhibition increases BACE1 protein levels in the brains of C57/B6 mice. ***A–F***, Two- to 3-month-old C57/B6 mice were given a single intraperitoneal injection of 18 U/kg insulin, 1 g/kg 2DG, 100 mg/kg 3NP, 30 mg/kg KA, or vehicle and were then allowed to recover for 4 h (***A, B***), 2 d (***C, D***) or 7 d (***E, F***). Brains were then isolated and homogenized, and samples (15 μg/lane) were subjected to immunoblot analysis for BACE1 protein using anti-BACE1 antibody PA1–757. Blots were stripped and reprobed with anti-β-actin antibody as a loading control. ***A, C, E***, Representative BACE1 (top panels) and β-actin (bottom panels) immunoblots for the various treatments and recovery times are shown. ***B, D, F***, The intensities of BACE1 band signals were quantified on a PhosphorImager (Eastman Kodak), normalized against the β-actin immunosignals for each sample, and then expressed as percentages of the mean of the vehicle control for a given recovery time. Note that the energy-inhibitor treatments elevated cerebral BACE1 protein levels to 125–150% of vehicle control values for all recovery times ($^{*}p < 0.05$, $^{**}p < 0.01$, and $^{***}p < 0.001$, one-way ANOVA with Newman-Keuls multiple-comparison test). ***A–D***, Data represent mean ± SEM; $n = 9$ mice/treatment (***A, B***), $n = 5$ mice/treatment (***C, D***), and $n = 4$ mice/treatment (***E, F***).

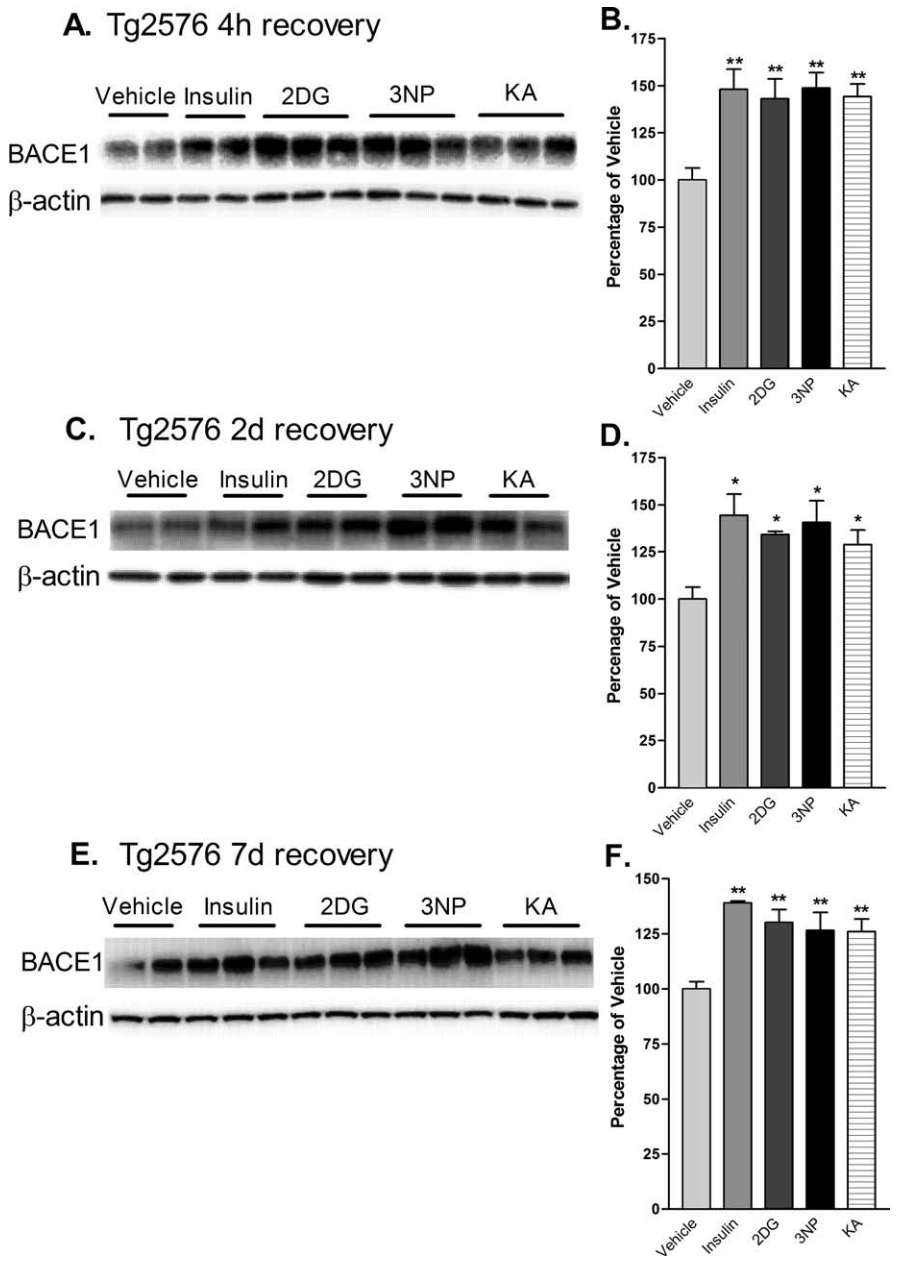

Figure 4. Acute energy inhibition increases BACE1 protein levels in the brains of Tg2576 mice. ***A–F***, Two- to 3-month-old Tg2576 mice were injected with 18 U/kg insulin, 1 g/kg 2DG, 100 mg/kg 3NP, 30 mg/kg KA, or vehicle and were allowed to recover for 4 h (***A, B***), 2 d (***C, D***), or 7 d (***E, f***). ***A, C, E***, Representative immunoblots of brain samples for BACE1 (top panels) and β-actin (bottom panels). ***B, D, F***, BACE1 immunosignals were quantified, normalized against β-actin signals, and expressed as percentages of vehicle controls, as before. Similar to the effects observed in C57/B6 mice, energy-inhibitor treatments in Tg2576 mice caused cerebral BACE1 levels to increase to 125–150% of vehicle controls for all recovery times ($^{*}p < 0.05$ and $^{**}p < 0.01$, one-way ANOVA with Newman-Keuls multiple-comparison test). Data represent mean $\pm$ SEM; $n = 6$ mice/treatment (***A, B***), and $n = 4$ mice/treatment (***C–F***).

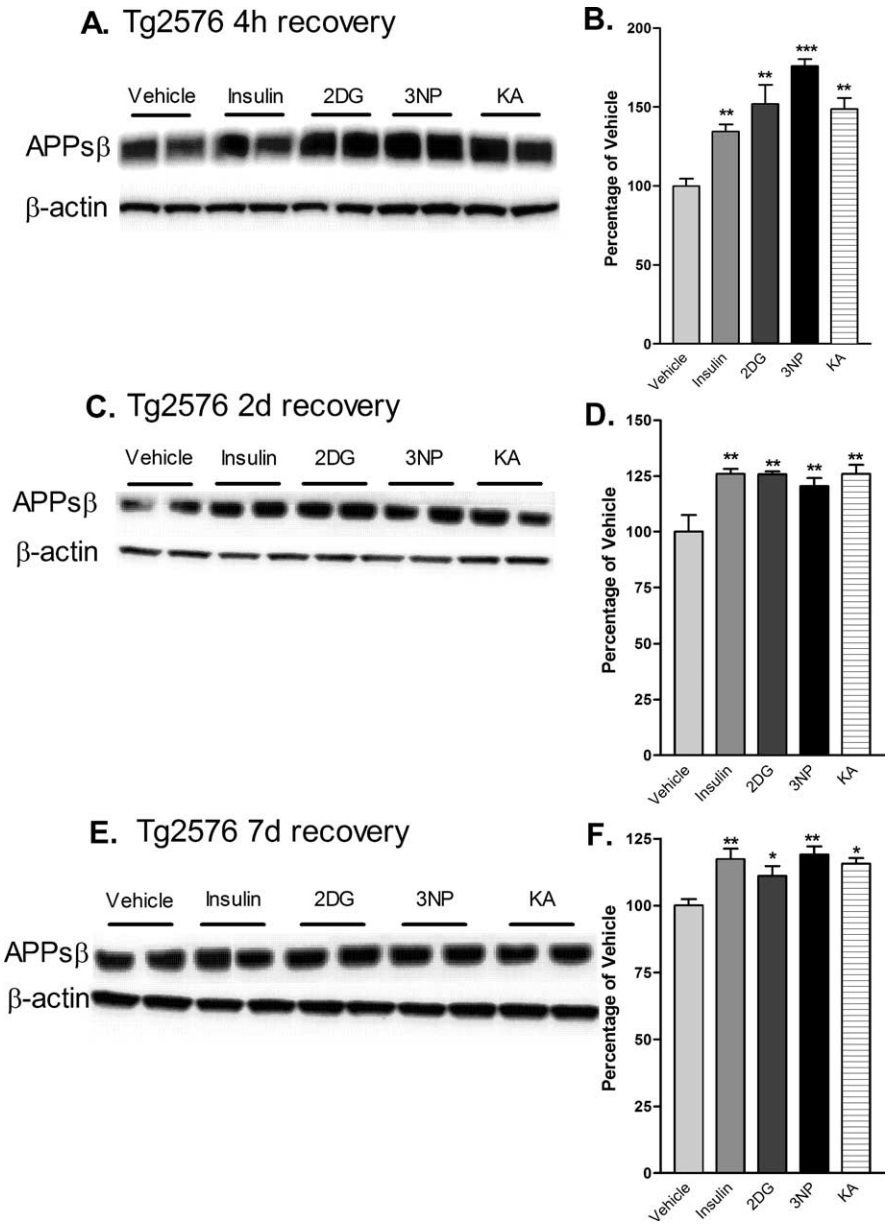

Figure 5. Cerebral levels of APPs(sw) are elevated after acute inhibition of energy production in Tg2576 mice. Brain homogenates from Tg2576 mice treated with 18 U/kg insulin, 1 g/kg 2DG, 100 mg/kg 3NP, 30 mg/kg KA, or vehicle were subjected to immunoblot analysis using an antibody raised against the C-terminal neoepitope generated by BACE1 cleavage of APPsw, which recognizes APPsβ(sw) (Seubert et al., 1993; Vassar et al., 1999; Cole et al., 2005). ***A, C, E***, Representative APPsβ(sw) (top panels) and β-actin (bottom panels) immunoblots of brain samples from treated Tg2576 mice. Recovery times were for 4 h (***A, B***), 2 d (***C, D***) or 7 d (***E, F***). ***B, D, F***, APPsβ(sw) immunosignals were normalized against β-actin signals and expressed as percentages of vehicle controls. Note that cerebral APPsβ(sw) levels were increased to ~125–175% of vehicle controls after 4 h of recovery from energy inhibition (***B***), and they continued to be significantly elevated after 2 d (***D***) and 7 d (***F***) of recovery ($^{*}p < 0.05$, $^{**}p < 0.01$, and $^{***}p < 0{,}001$, one-way ANOVA with Newman-Keuls multiple-comparison test). Data represent mean ± SEM; $n = 6$ mice/treatment (***A, B***), and $n = 4$ mice/treatment (***C–F***).