



**Supplementary Figure 1. Tat-GluR2<sub>3Y</sub>, but not Tat-GluR2<sub>3A</sub>, prevents the insulin-induced reduction in cell-surface AMPA receptor expression in cultured hippocampal neurons.** The wild type GluR2<sub>3Y</sub> and mutant GluR2<sub>3A</sub> peptides were rendered cell-permeable by fusing each to the cell-membrane transduction domain of the human immunodeficiency virus-type 1 (HIV-1) Tat protein (Schwarze et al, 1999). Cultured hippocampal neurons were pretreated with either Tat-GluR2<sub>3Y</sub> or Tat-GluR2<sub>3A</sub> (1  $\mu$ M) for one hour and then subjected to insulin (10 min; 0.5  $\mu$ M) treatment. The transduction of these peptides into neurons was confirmed by visualization, under fluorescent microscopy, of the fluorophore, dansyl chloride, which was conjugated to each of the peptides (data not shown). Cell surface expression of native AMPA receptors was measured by a colorimetric cell-ELISA assay using an antibody against the N-terminal extracellular domain of native AMPA receptor GluR2 subunits (n = 6). While neither peptide had an effect on the basal level of cell-surface AMPA receptor expression, Tat-GluR2<sub>3Y</sub>, but not Tat-GluR2<sub>3A</sub>, prevented the insulin-induced reduction in the cell-surface AMPA receptors.