Supplemental Data

Excess Protein Synthesis in FXS Patient Lymphoblastoid Cells Can Be Rescued with a p110β-Selective Inhibitor

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Figure S1. Increased PI3K activity in LCLs from FXS patients. PI3K activity was detected with a radioactive assay using phosphoinositide (P) and radiolabeled ATP as substrate, followed by thin layer chromatography and autoradiography. An example autoradiography is shown for two different control LCLs (Ctr: cell line GM10851, Ctr-b: cell line J1), as well as LCLs from a patient with a deletion in the fmr1 gene (Fdel: cell line DM316, no FMRP detectable by western blot, as shown below) and a patient with a full trinucleotide expansion (FXS: cell line GM03200, residual FMRP levels detectable).

Figure S2. Protein levels of the PI3K catalytic subunit p110β (A) are increased in LCLs from FXS patients compared to a healthy control (Fdel, FXS and Ctr-b, as described in Figure S1), whereas p110α (B) and p110δ (C) levels were highly variable, but did not show any significant changes in the same protein samples, in which p110β was increased (n=3, separate 1-way ANOVAs for A, B and C, Tukey’s posthoc tests: *p=0.021, #p=0.048). Protein levels of p110 subunits were quantified by densitometry of western blots and normalized to tubulin on the same blots. Example western blots are shown on top.