

Supplemental Data

Excess Protein Synthesis in FXS Patient Lymphoblastoid Cells Can Be Rescued with a p110 β -Selective Inhibitor

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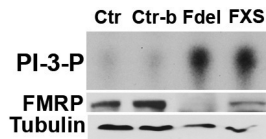


Figure S1. Increased PI3K activity in LCLs from FXS patients. PI3K activity was detected with a radioactive assay using phosphoinositide (PI) and radiolabeled ATP as substrate, followed by thin layer chromatography and autoradiography. An example autoradiography is shown for two different control LCLs (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).

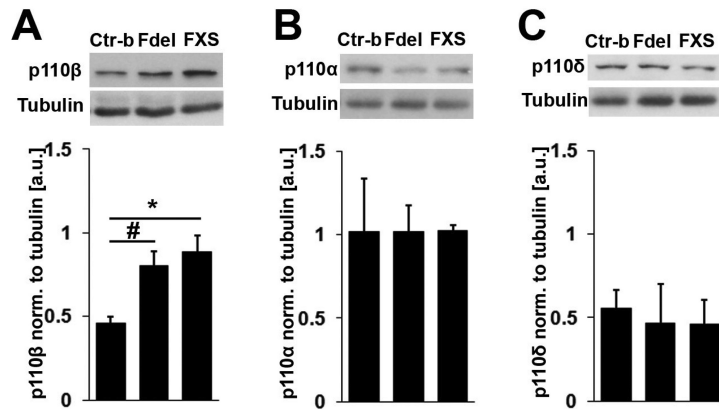


Figure S2. Protein levels of the PI3K catalytic subunit p110 β (A) are increased in LCLs from FXS patients compared to a healthy control (Fdel, FXS and Ctr-b, as described in Figure S1), whereas p110 α (B) and p110 δ (C) levels were highly variable, but did not show any significant changes in the same protein samples, in which p110 β 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.