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Alterations of mGluR5 and mGluR5 signaling partners in schizophrenia

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Presentation Title: Alterations of mGluR5 and mGluR5 signaling partners in schizophrenia

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Abstract: Animal and genetic studies point towards a role of metabotropic glutamate receptor subtype 5 (mGluR5) in the pathophysiology of schizophrenia, but direct evidence from postmortem schizophrenia studies have been inconsistent. We thus assessed mGluR5 protein levels in samples of human post-mortem dorsolateral prefrontal cortex (DLPFC; n=37) and hippocampal cornu ammonis 1 (CA1; cohort subset: n=20) from schizophrenia and matched controls. We also measured the mGluR5 signaling partners, Norbin (neurochondrin), Tamalin (GRASP1), and Preso1 (FRMPD4), which modulate mGluR5 trafficking, localization and recycling. To determine if current antipsychotics influence mGluR5 and mGluR5 signaling partners, lifetime antipsychotic history was correlated with protein measures in postmortem samples. Protein levels were additionally analyzed in rats chronically treated with haloperidol or olanzapine. mGluR5 protein levels were consistently increased in both the DLPFC (22%; $p<0.001$) and CA1 (42%; $p<0.001$) of schizophrenia subjects compared to controls. mGluR5 signaling partners exhibited brain-region dependent alterations, with reductions in the DLPFC (Norbin 37%, $p<0.001$; Tamalin 30%, $p=0.040$; Preso1 29%, $p=0.001$) and increases in CA1 (Norbin 47%, $p<0.001$; Tamalin 34%, $p=0.009$; Preso1 83%, $p<0.001$). There were no effects of current antipsychotics on mGluR5, Norbin, Tamalin or Preso1 in humans or rats. In this study, we provide the first evidence that mGluR5 is increased and mGluR5 signaling partners are differentially altered in two highly important brain regions for schizophrenia. The present findings thus support that mGluR5 regulation is altered in schizophrenia, and that the identified changes in mGluR5, Norbin, Tamalin and Preso1 expression are unaffected by current therapeutics.