PW01-185 - **GENETIC PROFILE OF SCHIZOPHRENIC PATIENTS IN PAKISTAN**

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Background: Schizophrenia is a brain disease that has distressed human kind since the beginning of the written history. Firm knowledge about this illness is limited to certain areas including cognitive, risk genes etc. Basic question remains unanswered about the diagnostic heterogeneity and tissue neurochemistry. Several lines of evidence focus on direct involvement of glutamergic system in the pathophysiology of psyhosis.

Methods: The pilot study measured the difference between the plasma serine recemase level of normal and schizophrenic patients and estimated the D-isomers excreted in the urine using gas chromatography and Gas chromatography and mass selectivity (GCMS) respectively.

Results: Plasma and urine samples of normal and schizophrenic patients from UAE shows that the level of serine recemase and D-serine respectively is lower in schizophrenic pateints than that of the normal subjects.

Discussion: The hypofunction of the glutamate N-methyl-D-Aspartate receptor (NMDAR) has been proposed as a model of schizophrenia in humans using molecular marker and also due to evidence suggesting modulation of glutamate cicuitries after antipyschotic administration. In this regard there is increasing evidence from pharmacological and genetic studies that suggest that D-serine an endogenous co agonist to the NMDA subtype glutamate receptor, may be implicated in schizophrenia (SCZ). Although an association of genes for D-serine degradation such as D-amino acid oxidase and G72 has been reported, a role of recemase in SCZ is unclear.

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