

AFFECTIVE DISORDERS (BIPOLAR/UNIPOLAR) - Free Communications
FC-34-005
Serotonin Transporter gene polymorphisms and its correlation with depressive patients

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Objectives: Study the correlation between Serotonin Transporter gene polymorphisms 5HTTLPR and depressive patients.

Methods: Study the correlation between Serotonin Transporter gene polymorphisms 5HTTLPR and depressive patients.

Results: Significant differences were found among the patients carrying a short allele in the genotype (ss-ll = n.70) compared with the homozygotes (ll = n.30) depressive patients. The "s" carriers presented earlier onset of depression (aver. 25 years vs. 33.8 years) and more:

- Difficulties in development academic skills (36.5% vs. 14.8%)
- Number of suicidal attempts (rates 10,2 vs. 2,2), in violent ways (57.% vs. 33%.)
- Comorbidity with personality disorders (37 % vs 15,%)
- Anxiety disorders (48% vs 37%)
- Substance abuse (21% vs. 15%)
- Autoaggressivity (62 % vs 33%)
- Impulsivity (67% vs 19%)
- Number of psychiatric hospitalization (52% vs 26 %)
- Past history of stressors events (particularly abandon experiences in early adulthood with lack of attachment figures and sexual abuse (30 % vs 7 %)
- Family background of alcohol abuse history (38 % vs 15 %). Homozygotes "ll" group presented more history of depression in relatives (70 % vs 50%) and successful suicide attempts (26% vs 15%). Better response to antidepressant treatment (61% vs 44,3) was found in this group of patients.

Conclusions: Patients carrying a short allele in the genotype have presented more vulnerability to depression and comorbidity, associated with a reduced response to antidepressant treatment. These factors increased the risk to have a worst development in the course and prognosis of the mental disorder.

FC-34-006
Meta-analytic evidence for familial coaggregation of schizophrenia and bipolar disorder

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Objectives: Several sources of data suggest a link between schizophrenia and bipolar disorder (BD), however family studies have not revealed coaggregation of these disorders. We systematically reviewed family studies of probands with schizophrenia and BD to determine whether these disorders coaggregate in families.

Methods: Prospective studies were identified by searching Medline and PsycInfo databases from January 1, 1980 to December 31, 2006. All family studies reporting morbid risk or raw counts of schizophrenia or BD in first-degree relatives (FDR) of a proband group with DSM-III or later, ICD-9 or 10, or research diagnostic criteria schizophrenia or BD were included. A total of 38 studies were used to investigate rates of BD in FDR of probands with schizophrenia, while 39 studies were used to examine rates of schizophrenia in FDR of BD probands, out of the original 2326 studies identified by the database search. Data were analyzed with a novel random-effects bootstrapping technique that allows for the inclusion of studies lacking a patient or control group, which made up a substantial portion of the available data. Data were also blindly weighted for methodological quality.

Results: The FDR of probands with schizophrenia showed significantly ($p = 0.01$) increased rates of BD relative to control FDR, with an odds ratio (OR) of 2.08. The FDR of probands with BD showed marginally ($p = 0.06$) increased rates of schizophrenia relative to control FDR, with an OR of 2.10—this analysis was significant ($p = 0.02$) when studies not reporting morbid risk estimates were excluded, in which case the OR was 3.49.

Conclusions: The present study provides the first direct evidence for familial coaggregation of schizophrenia and BD, a finding that argues against a view of these disorders as entirely discrete diagnostic entities. Rather, a continuum or overlapping disease entity model is supported.