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EV0589

Genetic determinants of psychic resilience after a diagnosis of cancer

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Introduction Co-morbidity between cancer and psychiatric disorders including adjustment disorder, depressive disorders or angst can seriously influence the prognosis and the quality of life of patients.
Aim The identification of the psychological and biological profile of patients at risk for such co-morbidity is not yet available. Classical candidate genes such as the BDNF, the 5-HTLPR and genes whose products are involved in inflammatory events have received some attention, but results are inconclusive.
Object and methods In the present review the association between cancer and psychiatric disorders is reviewed, a focus on the investigation of the Gene X environment and the epigenetic control over the activation of the HPA axis is proposed as a tool to refine the definition of the biologic profile at risk for co-morbidity between psychiatry and cancer.
Results and conclusion A number of genes and socio-demographic variables that may influence risk to suffer from a psychiatric disorder after a diagnosis of cancer is identified and discussed. The identification of such biologic and socio-demographic profile is instrumental in the identification of subjects at risk of a double diagnosis, both somatic and psychiatric. An early identification of such profile risk would pave the way to the implementation of early intervention strategies.
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Is 22q11.2 deletion syndrome a genetic subtype of schizophrenia?

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Introduction 22q11.2 deletion syndrome is a primary immunodeficiency due to micro-deletion on the large arm of chromosome 22. Patients suffer from several anomalies, including metal illness, that such the case we present, mean a warning sign for further study.
Methods Twenty-one years-old male, with psychotic symptoms, typical of schizophrenia, behavioral disorders and mental confusion, plus epileptic episodes and psychomotor agitation. Two previous incomes with the diagnosis of psychotic disorder not otherwise specified. Treated with anti-psychotics at low doses with inter-episode stability.
Background Prematurity, low birth weight, neonatal asphyxia, generalized seizures, otitis and recurrent urinary tract infections, hypernasal voice, poor academic performance, difficulty relating. Physical examination: hypernasal voice, furred tongue, dysmorphic faces, scoliosis, hipotanía, stereotypes, delusions, auditory hallucinationsd negative symptoms.
Results We considered the possibility of a neurodevelopmental disorder, with a multidisciplinary approach, resulting in the diagnosis of paranoid schizophrenia and velocardiofacial syndrome, which had gone unnoticed. Mean doses of clozapine, haloperidol and topiramate were used. He accepted psychiatry and other specialties follow-up, since it requires a complex and multidisciplinary approach.
Conclusions Definition of velocardiofacial Syndrome and lack of consensus on terminology:
– syndrome 22q11.2 DS as genetic subtype of schizophrenia? Opportunity to study the pathogenesis of schizophrenia;
– the importance of a comprehensive approach to early diagnosis, clinical improvement and preventing complications.
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EV0591

The genetic study of computer game addiction

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Introduction Addiction to computer games (CA) is growing with a lightning speed in whole world. Very few studies are focused in the genetic basis of this disorder.
Objectives To study the COMT and MAOA polymorphism in addicts to computer games.
Methods Totally 42 persons were included in this study, 22 of them had CA and 20 were totally healthy. Out of 22 gamers, 10 persons had only CA. The rest of 12 patients suffered from another psychiatric disorder besides of CA (Schizotypal disorder, depression, bipolar disorder). Their mean age was 16 years (15; 17) and all of them were males.
Results The total frequency of alleles 3R and 5R of MAOA gene in patients with CA was 30.0%, which doesn't have any statistical difference with the healthy persons. The genotype frequency of Val158Met of COMT gene is high in CA rather than in healthy persons ($\chi^2 = 6.85$, $P = 0.03$). Also, the homozygotes Val are much more in CA patients (59.1%) than in healthy persons (25%). On the other hand, the Val/Met combination is lower in CA patients (18.2%) than in healthy persons (55.0%).
Conclusion The Val158Met polymorphism of gene COMT may lead to CA formation.
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Family-based association study between the brain derived neurotrophic factor (bdnf) gene and the attention deficit hyperactivity disorder in a Mexican population

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