BRO E ALTERAÇÃO EM TESTE DE COGNIÇÃO PRÉ-FRONTAL

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Introdução: O fator neurotrófico derivado do cérebro (BDNF) é uma neurotrofina e tem se mostrado um potente modulador da transmissão sináptica e plasticidade no sistema nervoso central, participando dos processos cognitivos como o aprendizado e a memória. Recentemente, o gene do BDNF tem se mostrado um gene candidato para estudo da patogenia em doenças psiquiátricas. Atualmente, dois polimorfismos deste gene já foram identificados: o polimorfismo de dinucleotídeos em repetição e o polimorfismo de um único nucleotídeo Val66Met. Recentes estudos demonstram uma associação entre ambos os tipos de polimorfismos e o transtorno bipolar. Déficit de desempenho cognitivo no pré-frontal têm se mostrado como um possível marcador na doença bipolar. Objetivos: O presente estudo teve por objetivo avaliar a associação do polimorfismo do gene do fator neurotrófico derivado do cérebro (BDNF) e a performance cognitiva através do Teste Wisconsin de Classificação de Cartas (WCST) em pacientes bipolares Materiais e métodos: Foram avaliados 58 pacientes, 14 do gênero masculino e 44 do gênero feminino, sendo a idade média 40 anos (de18 a 68 anos de idade). Foi analisada a associação entre a presença do alelo Met do polimorfismo (val66met) do BDNF e o número de erros perseverativos (WCST-P), número de erros não perseverativos (WCST-NP), resposta de nível conceitual (WCST-%CONC), número de categorias completadas (WCST-CC) e ensaios para completar a primeira categoria (WCST- 1st CAT). Resultados e conclusões: O percentual de indivíduos Val/Val, Val/Met e Met/Met foi respectivamente 48,4%, 24,2% e 4,8%. Não houve diferença entre os grupos portadores e não portadores do alelo Met em relação a sexo, idade, início da doença, números de anos estudados nem tempo de evolução da doença. O desempenho do grupo de portadores do alelo Met (Val/Met e Met/Met) apresentou um pior desempenho no domínio de erros não perseverativos (p

PANIC DISORDER AND SEROTONINERGIC GENES (5-HTTLPR, HTR1A AND HTR2A): ASSOCIATION AND INTERACTION WITH CHILDHOOD TRAUMA AND PARENTING.

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Background: Panic disorder (PD) has been related genetic and environmental risk factors. However, no study has evaluated a gene-environment interaction for this disorder. The aim of this study is to evaluate the association between HTR1A, HTR2A and 5-HTTLPR and PD. We also ought to evaluate the interaction between these genes and two environmental factors previously associated with PD: childhood trauma and parental bonding. **Methods**: This is a case-control candidate gene study (107 PD patients and 125 controls). Diagnoses were confirmed by M.I.N.I and clinical interview. Childhood trauma was evaluated by the Childhood Trauma Questionnaire (CTQ) and Parental Bonding Instrument (PBI) was used to evaluate parenting. Genes were screened using a set-based test in PLINK software followed by single marker association tests and haplotype test for genes that reached experiment-wide significance. Logistic regression was used to model gene-environment interaction. We addressed multiple comparisons at two levels of significance correction: gene-wide (p_1) and experiment-wide (p_2). **Results**: Only HTR1A was experiment-wide associated with PD in set-based test (p_2=0.027). Regarding interaction analysis with optimal father parenting, interaction terms HTR2A SNPs (rs6311 and rs6313) were nominally associated with PD and rs6311 remained significant at gene-wide level of correction. Among subjects with TT/TC genotype in rs6311 the protection effect of fathers with high care and low overprotection was higher than the protection effect among subjects with CC genotype (β=0.134, t=-2.678, p_0=0.007, p_1= 0.042). **Conclusion**: We replicated association between the HTR1A promoter SNP (rs6295) and PD, but did not observe association with HTR2A or 5-HTTLPR. We also reinforce evidence of gene-environment interaction in HTR2A gene with parenting, maybe influencing the capacity of subjects to use familiar experiences as environmental support.

VARIANTS IN A GENE ENCONDING A REGULATOR OF G PROTEIN SIGNALING 4 (RGS4) ARE ASSOCIATED WITH THE COMORBIDITY BETWEEN PANIC DISORDER (PD) AND SOCIAL ANXIETY DISORDER IN PD PATIENTS

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Background: Recent evidences suggest that the genes encoding regulators of G proteins (RG) as RGS2 (RG signaling 2) and RGS4 (RG signaling 4) are implicated in childhood temperament as behavioral inhibition, an intermediate phenotype tightly related with Social Anxiety Disorder in adulthood. The aim of this study was to examine whether variants in RGS2 and RGS4 genes are associated with the comorbidity between Panic Disorder (PD) and Social Anxiety Disorder (SAD) in PD patients. **Methods**: This is a candidate-gene association study with 127 PD patients diagnosed by M.I.N.I. (106 without comorbidity with SAD and 21 with comorbidity with SAD). We have examined 22