Annals of General Psychiatry

BioMed Central

Poster presentation

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Pain-related comorbidity in depression: the association with 5-HTTLPR and STin2

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from International Society on Brain and Behaviour: 2nd International Congress on Brain and Behaviour
Thessaloniki, Greece. 17–20 November 2005

Published: 28 February 2006

Annals of General Psychiatry 2006, **5**(Suppl 1):S165 doi:10.1186/1744-859X-5-S1-S165

Background

Response to selective serotonin reuptake inhibitors (SSRIs) in major depressive disorder has been suggested to be influenced by variations in the serotonin transporter gene (such as 5-HTTLPR and STin2) [1-3]. In addition, the presence of concomitant physical complaints, particularly pain-related complaints, also increases the risk on SSRI non-response [4]. Since the serotonin transporter is thought to be involved in pain development, an observed association between serotonin transporter genotype and SSRI non-response could be partially due to mediation of the risk of comorbidity in depression. This study evaluates the association between the presence of comorbidity (with or without pain) and serotonin transporter genotype in major depressive disorder.

Materials and methods

For this study, 164 patients meeting the DSM-IV criteria for major depressive disorder were included in the analyses. Blood samples or buccal swabs were taken from all participants to determine the 5-HTTLPR an STin2 genotype. Additional information was gathered through interviews and general practitioners' files. Reported comorbidity was categorised according to level of associated pain (no pain, moderate pain and strong pain).The association between genotype and comorbidity was assessed using logistic regression.

Results

More comorbidity was reported by patients with the 5-HTTLPR s/s genotype compared to patients with the s/l and l/l genotype (65% versus 44.4 and 48.3%). These patients also reported more comorbidity associated with strong pain (52.5% versus 38.1 and 36.2%). For STin2, patients with the 10/10 genotype only reported more comorbidity not associated with pain compared to the 10/12 and 12/12 genotype (8.0% versus 3.0 and 2.9%). Patients with the 5-HTTLPR s/s genotype appeared to have an increased risk of developing physical complaints with painful symptoms; Odds Ratio (OR) = 2.33 (95% CI 0.92-5.89). For STin2, patients with the 10/10 genotype seemed to have an non-significant increased risk of developing comorbidity without painful symptoms; OR = 2.27 (95% CI 0.27-19.32). No elevated risks were found in patients heterozygote for 5-HTTLPR or STin2.

Discussion

Our findings point to a slightly increased risk of developing comorbidity (with or without painful symptoms) with the 5-HTTLPR s/s and the STin2 10/10 genotype, these finding are not sufficient to exclude serotonin transporter genotype as a possible risk factor for SSRI non-response.

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