Tues-P101

^{123}I-IOMAZENIL SPET REVEALS INCREASED PREFRONTAL BENZODIAZEPINE RECEPTOR DENSITY IN PANIC DISORDER

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Some evidence points to an involvement of the benzodiazepine receptor complex in the pathogenesis of panic disorder. Since the introduction of ^{11}C-Flumazenil for PET and of ^{123}Iodine-Iomazenil for SPET imaging, the in vivo measurement of the regional receptor density in vivo has become possible. Two studies using the SPET method revealed contradictory results.

Therefore we investigated 12 outpatients (6 M, 6 F; 22–53 y) with panic disorder according to DSM-III-R criteria without any benzodiazepine medication before. Six of them got antidepressants, mostly SSRI, and TCA. In addition 9 healthy controls (4 M, 5 F; 22–47 y), who also never had taken benzodiazepines, were included in the investigation. Besides a neuropsychiatric workup, patients and volunteers got ratings with the Hamilton depression rating scale (HAMD) and Spielberger's State Trait Anxiety Inventory (STAI). All patients got an intravenous injection of 185 MBq ^{123}I-Iomazenil followed by Single Photon Emission Tomography (SPET) with an interval of 90 minutes. Eight regions of interest (ROI) were defined each on the right and left hemisphere according to the stereotactic system of Talairach and Tournoux. Right/left ratios and ratios to an "internal standard" were calculated. Computerised statistical analysis was performed using SAS software.

The results showed a significant ($p < 0.05$) increase of benzodiazepine receptor density in the right prefrontal cortex and a trend for higher values in the right temporal cortex in the patient group. The other ROIs (frontal, parietal, occipital, medial and lateral hippocampal) showed no differences compared to the control group.

The increase of benzodiazepine receptor density in the prefrontal cortex points to an involvement of this receptor system in the disease process.

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PROLACTIN RESPONSE TO TRH IN PATIENTS WITH PANIC DISORDER

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The aim of this study is to assess prolactin response to thyrotropin-releasing hormone (TRH) in patients with panic disorder (PD).

The effects of TRH administration on the release of prolactin were examined in 15 patients who met DSM-III-R criteria for PD and compared their test results with those of 15 normal control subjects. Blood samples were taken before (baseline) and at 15, 30, and 60 minutes after TRH (400 µg) IV injection.

Baseline prolactin levels were similar in PD patients (8.12 ± 9.50 ng/ml) and control subjects (9.60 ± 7.53 ng/ml). No significant group effect was observed on the prolactin responses ($F = 2.14$, $df = 1.26$). Although the Δ_{max}prolactin levels were higher in the PD group (47.41 ± 37.10 ng/ml) than the control group (29.79 ± 13.51 ng/ml), the difference between them was not statistically significant. When men and women were evaluated separately, Δ_{max} prolactin levels were found to be higher but not significant in women of the PD group (71.72 ± 35.74 ng/ml) than the women of the control group (34.88 ± 16.39 ng/ml), ($p < .1$).

In conclusion, the results demostrate that prolactin responses to TRH did not differ between PD patients and normal control subjects. When only women were evaluated, the findings indicate that women with PD tend to show excessive prolactin responses to TRH.

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A CROSS-SECTIONAL SURVEY OF SEXUAL DYSFUNCTION IN PATIENTS TAKING ANTIPSYCHOTIC MEDICATION

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Background: Conventional antipsychotics prevent the suppression of prolactin by dopamine. The resultant hyperprolactinaemic state is associated with menstrual irregularities, erectile and ejaculatory failure and infertility. Therefore, sexual dysfunction may be a common side effect of neuroleptic medication and thus a cause of patient non-compliance with the resultant risk of relapse.

Method: Sexual functioning was assessed in 75 outpatients taking antipsychotic medication and compared with patients from a Sexual Dysfunction Clinic ($n = 55$) and normal controls ($n = 60$).

Results: 50.7% of the subject group scored highly on the Sexual Dysfunction Questionnaire (SDQ), indicating moderate to severe dysfunction, compared with 18.2% of normal controls and 56.0% of the sexual dysfunction group. The mean SDQ score (99.95) for the patient group was significantly higher than that of the normal controls (81.45); $p < 0.001$, (95% CI 14.2 to 22.7), but no different to that of patients with known sexual dysfunction (99.69); $p = 0.903$, (95% CI −3.99 to 4.52).

Conclusion: Patients taking antipsychotic medication have a level of sexual dysfunction comparable to patients attending a sexual dysfunction clinic. Most subjects were convinced that medication was the cause of their sexual difficulties and felt it affected their ability to comply with medication regime. Sexual history prior to the prescription of neuroleptic medication will allow baseline estimation of sexual function and follow-up should include regular inquiry with regard to sexual function.

Tues-P104

TESTESTERONE & PITUITARY HORMONES IN SEXUAL DYSFUNCTION

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Background: sex is organized within a genetically determined neuro-endocrinal framework (Fuller, 1960).

Methods: 200 patients with erectile disorder were compared to 100 controls. Middlesex Hospital Questionnaire was introduced in addition to measurement of plasma levels of testesterone, prolactin, follicle stimulating hormone (F.S.H.) and leutinizing hormone (L.H.).

Results: 72% of the patients had obsessions, anxiety was found in 50.5% of the patients while depression was detected in 52% of the patients.

No difference in hormonal levels was found except that testesterone was lower in the patients group ($P < 0.05$). Prolactin was increased in the age group 50–60 years ($P < 0.05$), F.S.H. was lower in the age group 20–30 years ($P < 0.05$), while testesterone

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