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PLASMA CRH CHANGES DURING THE FEELING OF INDUCED EMOTIONS

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BACKGROUND

Neuropeptides like Corticotropin-Releasing Hormone (CRH), are widespread within the Central Nervous System (CNS) and modulate behaviour. Peripheral CRH may reflect CNS production. We explored emotive modulation of plasma CRH levels.

PATIENTS AND METHODS

60 female subjects were studied. Emotion was induced by the projection of a 20 min movie fragment – Sadness (Schindler's List) [Group A-20 subjects] or Joy (Mr. Bean) [Group B-20 subjects]. No film was presented in Group C – 20 subjects. Venous blood samples were collected at 0, 30 and 90 min (samples 1, 2 and 3). Personallity was evaluated with the MMPI. CRH was measured by ELISA. SPSS was used to analyse results.

RESULTS

Without movie projection, CRH levels did not change significantly. During Sadness induction, CRH increased: sample 1- 394 ± 147 pg/mL; sample 2- 791 ± 636 pg/mL and sample 3- 803 ± 771 pg/mL, $F(2,57)=4.399$, $p<0.05$, pos-hoc t-test, $p<0.05$. During Joy induction, CRH levels increased slightly and non-significantly: sample 1- 364 ± 138 pg/mL, sample 2– 486 ± 260 pg/mL, sample 3- 483 ± 228 pg/mL, $F(2,57)=2.680$, $p<0.10$. Baseline CRH levels were significantly related to the neurotic triad –r=-0.328- and to the psychotic dyad –r=+0.267– $p<0.05$.

DISCUSSION

We have previously shown in mice, that a specific and modulated brain to blood transport of CRH, allows central CRH to act at the periphery and totally account for plasma CRH levels. We now show in man, that acute emotions specifically modulate plasma CRH levels and that baseline plasma CRH levels are related to classic personality dimensions.

PLASMA CRH AND β-ENDORPHIN IN DEPRESSED PATIENTS BEFORE AND AFTER SHORT TERM TREATMENT WITH SEROTONIN RE-UPTAKE INHIBITORS

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BACKGROUND

Depression is generally associated with low central serotonin and increased cortisol production. Both abnormalities may be casually related, since the midbrain raphe serotoninergic system regulates hypothalamic CRH. We measured plasma CRH and β-endorphin in neurotic depression and evaluated the response to serotonin re-uptake inhibitors.

PATIENTS AND METHODS

15 non-depressed – Group A – and 15 depressed subjects – Group B – were studied. Subjects filled several mood evaluation scales: POMS, GMS, HADS and SDS. A baseline venous blood sample was collected. Treatment with fluoxetine 20mg, *bid, po*, was then initiated in Group B. After one month all subjects were again evaluated as before. CRH, β-endorphin, ACTH, cortisol, DHEAS and PRL were measured in each sample. Statistical analysis used SPSS.

RESULTS

At the first visit subjects from group B were significantly more depressed – (SDS) 29 ± 11 *vs* 13 ± 12, (POMS) 31 ± 40 *vs* –11 ± 13, (GMS) 4 ± 14 *vs* 9±13, $p<0.05$. They also presented lower DHEAS – 70 ± 47 *vs* 143 ± 71 ng/mL, $p<0.05$ – and lower CRH levels – 360 ± 174 *vs* 576 ± 376 pg/mL, $p<0.08$ – with non-significantly lower β-endorphin levels. CRH was directly related to β-endorphin – $r=0,579$, $p<0.005$. After one month with fluoxetine treatment, no significant changes on psychometric measures or on CRH and β-endorphin were observed.

DISCUSSION

Because of brain to blood transport systems for several peptides, plasma levels may reflect central levels. In this study we showed that neurotic depression is associated with decreased CRH and β-endorphin plasma levels. These changes are not modified by short term psychotropic treatment with serotonin re-uptake inhibitors.