

SES06.3

Hypoestrogenism in schizophrenic women: implications for research

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Estrogens seem to have a neuroleptic-like effect in schizophrenia and therefore a beneficial influence on the course of illness in schizophrenic patients. Therefore, the impact of hypoestrogenism in women with schizophrenia is of high clinical interest. While the precise mechanism of low estrogen levels in schizophrenic women is unclear, there is evidence that hypoestrogenism in schizophrenic women is not exclusively the consequence of hyperprolactinemia-inducing neuroleptics, but connected with schizophrenia itself.

To test this hypoestrogenism hypothesis the levels of estradiol, prolactin, LH, FSH, progesterone, and testosterone of 75 women with schizophrenia were determined in the follicular, periovulatory and luteal phases of the menstrual cycle.

Hypoestrogenism was found in about 60% of the patients and anovulatory cycles were assumed in most of the women. To rule out a possible effect of hyperprolactinemia on the gonadal axis and the subsequent effect on the estrogen level due to conventional neuroleptics, estradiol serum levels of patients treated with atypical neuroleptics known to induce only a mild increase in prolactin, or no increase at all, were compared with those from patients treated with conventional neuroleptics. The data clearly indicate high prolactin levels in the latter, but low levels in the group treated with atypical neuroleptics. In both groups, however, low levels of estradiol were measured.

The findings provide evidence that hypoestrogenism in schizophrenia occurs in women with and without neuroleptic-induced hyperprolactinemia. Because of the clinical consequences of hypoestrogenism in schizophrenic women further research should be conducted to clarify the cause of this endocrinological abnormality.

SES06.4

Estrogen therapy in women with depression

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Objectives: Summarize the present evidence regarding the impact of estrogen decline on mood disorders in the postpartum period, in peri and postmenopausal women and in women suffering from PMDD. Develop a practical model of integrated care for patients with depressive disease

Methods: Review of the literature regarding biological, epidemiological, clinical evidence. Review of the methods applied. Critical evaluation of research concepts. Qualitative longitudinal studies performed at our department.

Results: Biological evidence supports the link of estrogens with affective disorders. Epidemiological data are however controversial. The methodological difficulties include the lack of longitudinal studies, endocrine status, variation in measures etc. Theories include the domino effect of estrogen induced vegetative disturbances on affective symptoms, the developmental crisis theory and the psychosocial stress hypothesis. Pathogenetic studies point to the importance of psychosocial stressors. Intervention studies have frequently shown the positive effect of estrogens on non psychiatric classified mood disorders. Our own longitudinal studies show the intraindividual variation of effective interventions.

Conclusion: An individualized clinical model emerges, in which the patient with a mood disorder during specific life span changes (endocrine and psychosocial) is evaluated not only using psychiatric diagnostic scales but also determining the

gynecological-endocrine status including somatic symptoms and risks as well as psychosocial stressors. The model of practice in Basel will be presented.

SES06.5

Cerebrospinal fluid estradiol and β -amyloid levels in female patients with Alzheimer's disease

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Objective: Recent epidemiological studies demonstrated that estrogen replacement therapy may delay onset and progression of AD. Experiments in cell cultures indicated that estrogens such as 17 β -estradiol (E2) may decrease the production of β -amyloid 1–42 (A β 42), a peptide central for the formation of senile plaques in Alzheimer's disease (AD).

Method: To test this finding in a clinical study, cerebrospinal fluid levels of E2 were investigated in 33 female AD patients and 14 patients with depression with respect to β -amyloid 1–40 (A β 40) and A β 42 levels. In a second step, we compared E2 levels in a larger sample of 59 AD patients with 13 healthy controls.

Results: E2 levels were significantly ($p < 0.05$) lower in the AD group compared to both depressed and healthy controls. Within the AD group, low E2 levels were inversely correlated with A β 42 concentrations.

Conclusions: Our study revealed a slight E2 deficit in female AD patients which may influence A β 42 metabolism. This observation corresponds to the beneficial effects of estrogen replacement therapy on the development and course of AD.

SES07. AEP Section Epidemiology & Social Psychiatry – Gender differences in major mental disorders – Part II

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SES07.1

Psychosis after childbirth

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The rate of psychosis after childbirth is about 1–2/1000 and has been remarkably consistent over 150 years and across countries. Most illnesses are affective in nature and have an onset within 2 weeks of childbirth.

In bipolar women the occurrence of episodes in the puerperium has recently been shown to be partially genetically determined. It is likely therefore that the rapid steroid decline after childbirth can highlight an otherwise latent neurotransmitter dysfunction in predisposed subjects. Interactions of oestrogens and progestins with neurotransmitter systems are numerous and complex. Recent findings have suggested several possible sites for such an interaction, for example within the serotonin and the dopamine systems.

Although pregnant women with a history of severe affective disorder are at a high risk of recurrence after delivery there are no