

**LS04.4**

Escitalopram – in the treatment of mood and anxiety disorders

D. Baldwin. UK

No abstract was available at the time of printing.

**SAL07. Work related depression and exhaustion (burn-out syndrome)****SAL07**

Job-stress, exhaustion and depression

M. Åsberg\*. *Department of Clinical Neuroscience, Psychiatry Section, Karolinska Institutet/Karolinska hospital, S-17176 Stockholm, Sweden*

Since 1997, rates of long-term sick-leave have increased dramatically in Sweden. The costs to society have more than doubled over the same time period. Evidence from databases kept by the two Swedish insurance companies who together insure the majority of employed Swedes (about 3 million people) suggest that the increase is mainly due to depressive illness, which is particularly prevalent among middle management employees and school and health care personnel.

The diagnostic assessment used in the insurance databases is that of the doctor who writes the sick-leave certification. Few of these doctors are psychiatrists, and doubt has therefore been expressed about the validity of the diagnoses. We have performed structured diagnostic interviews with 250 consecutive individuals on sick-leave for any affective disorder, and found that 80 per cent fulfilled DSM-IV criteria for major depressive disorder. In about 50 per cent of the cases, no other cause for the depressive illness could be identified except job stress (particularly repeated reorganizations at the workplace, and an increased workload). Although the patients often described a very abrupt onset of the illness that forced them to seek medical help, a closer scrutiny of the case histories suggests that low-back pain and other musculo-skeletal symptoms, sleep disturbance, stomach trouble, tiredness and concentration difficulties often precede the depression by several years. The clinical picture as assessed in SCID-interviews and depression rating scales was very similar to that of major depressive disorder from other causes. Personality disorder was rare, however, and suicidal behaviour, although it did exist in some patients, was less common than in patients with major depression treated at an outpatient psychiatric clinic.

Some possible reasons for the Swedish epidemic of job stress related depressions will be discussed in the lecture. Randomized controlled studies of different treatment and preventive strategies are currently under way and will be described, and preliminary data from studies of the natural history of the conditions, of personality features of affected individuals, and of their work environment, will be presented.

Increasing rates of job stress related psychiatric conditions appears to be an international phenomenon. Interestingly, the situation in Sweden was relatively favourable until 1997, when the current epidemic started. The dramatic increase over such a short period suggests that eliciting factors may be indentifiable, and hopefully also amenable to intervention.

**SES06. AEP Section Women's Mental Health – Estrogen effects in mental disorders***Chairs: A. Riecher-Rössler (CH), N. Bergmann (D)***SES06.1**

Neuroprotective effector mechanisms of estrogens

H. Vedder\*, J.-C. Krieg. *Department of Psychiatry and Psychotherapy, Philipps-University, Germany*

Clinical data support beneficial functions of estrogens in health and disease. At the moment, a certain gap still exists between the likely clinical effects on diseases such as Alzheimer's and Parkinson's as well as schizophrenia and the large number of preclinical findings. The basic neurobiological effector mechanisms include genomic and non-genomic effects as well as interactions with free-radical detoxifying systems and the inhibition of the cellular lipid peroxidation (LPO). We here present an overview on several of the neuroprotective mechanisms of actions of estrogens showing that a pre- and even a postincubation results in neuroprotective effects after a challenge with hydrogen peroxide. Additionally, these compounds inhibit the iron-induced LPO of cellular compounds and affect the anti-oxidative state of the cell as reflected by increased glutathione levels. Therefore, it is hypothesized that estrogens exert their beneficial clinical effects by increasing the cellular resistance to various toxic challenges resulting in a decreased vulnerability to different types of neuronal cell damage.

**SES06.2**

Estrogen effects in schizophrenia – brief update and potential therapeutic implications

A. Riecher-Rössler\*. *Kantonsspital Basel, University Psychiatric Outpatient Department, Switzerland*

There is increasing evidence from clinical as well as from epidemiological and basic research that estrogens have a protective effect in schizophrenia. Psychotic symptomatology seems to exacerbate/deteriorate in low estrogen states of women (premenstrual, postpartum, peri-/postmenopausal phase). Estrogens seem to act similarly to neuroleptics in animal studies, seem to save neuroleptics and improve psychosis in women. The effects are probably not specific for schizophrenia, but rather reflect a general "mental stabilization" by estrogens. The underlying mechanisms probably mainly lie in the modulation of different neurotransmitter systems in the limbic system of the brain like dopamine, serotonin, GABA and also the MAO.

A brief review of the different hints on a protective effect of estrogens in schizophrenia will be given, and potential therapeutic implications will be discussed, as if this effect could be confirmed, this could have interesting consequences for prophylaxis and therapy: We would e.g. have to reevaluate strategies like estrogen replacement therapy in postmenopausal schizophrenic women, adjunct estrogen therapy in women with estrogen deficiency syndromes, cycle modulated neuroleptic therapy in women with frequent perimenstrual relapses and/or the switch to atypical neuroleptics without hyperprolactinaemia in women with hypoestrogenism (secondary to neuroleptic induced hyperprolactinaemia). Further research is urgently needed, as there could evolve direct therapeutic benefits for women.