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Letters to the editor

A contentious issue in dementia research

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Until now we do not know very much about the processes in the central nervous system that initiate cognitive decline in Alzheimer's dementia.

The risk of Alzheimer's dementia is generally considered to depend on the number and impact of the contributing risk factors rather than on the presence of a single polymorphism or exposure to a single risk factor [3]. The hypothesis that most Alzheimer's dementia-type patients suffer from a pathologic processes that involves the neurons can be debated [1]. Furthermore, the main risk factor of real importance is age [4].

This raises the question as to whether the cause of cognitive decline in Alzheimer's dementia might be a manifestation of normal age-dependant neuron loss instead of a manifestation of excessive neuron loss evoked by a disease.

Ageing biological systems show often alterations that can be described merely as random processes.

Neuron loss can be considered, as well as a random, and, at the level of the individual, unpredictable age-dependant normal process. As age increases, all human beings will lose neurons. Probably it is the location of the random scattering of these normal losses that determines the moment of cognitive decline and the onset of Alzheimer's dementia.

This can be illustrated as follows: imagine the simple sequence of information processing: Input → Neuron (or neuro-element).A → Neuron.B → Neuron.C. A,B,C... are considered to be "information elements" [2] of one ore more coupled neurons. Suppose the random loss of A or B or C occurs. In the case of loss of A the sequence B and C will be hampered, resulting in very severe cognitive disturbances. In the case of loss of C the sequence A–B will not be disrupted, causing probably no, or minor, changes in cognition. In this example the location and not the number of neuron losses determines the supposed clinical manifestations. This location of losses is a random process. Exerting influence on this process, and thus on the cause of cognitive decline, will only be possible if ageing can be delayed.

The present brain research in Alzheimer's dementia is almost exclusively aimed at the various aspects of excessive neuron loss. There are arguments to bring this to discussion.

The random location of this normal age-dependant neuron loss is proposed here to be the cause of cognitive decline. If so, the present research in Alzheimer's dementia could not be considered as most appropriate. Research on ageing in more accessible human tissue than brain tissue is probably more suitable.

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Venlafaxin for the treatment of psychotic depression

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DSM-IV psychotic depression is a severe major depressive episode with delusions or hallucinations. Some authors suggest that major depressive disorder with psychotic features should be regarded as a diagnostic entity on its own, separated from non-psychotic major depressive disorder [6].

For the treatment of psychotic depression, a combination of a tricyclic antidepressants and an antipsychotic has been shown to be superior to any of these treatment modalities used alone [7]. Various selective serotonin reuptake inhibitors have been associated with a rather favorable response rate in patients suffering from psychotic depression [3, 5, 8]. Sachetti et al [5] suggested that predominant serotonergic activity may be a prerequisite for an antidepressant to be efficacious in the treatment of psychotic depression.

Venlafaxine is a selective serotonin and norepinephrine reuptake inhibitor with well documented antidepressant properties [2]. However, there have been no previous reports on the treatment of psychotic depression with venlafaxine. We hereby present a case in which venlafaxine alone appeared to be successful in treating psychotic depression in a young woman.

A 37-year-old woman with depressive and psychotic symptoms for 6 months was hospitalized for treatment. At no time during her illness did she receive any medical treatment. There was no history of previous mood disorders or of any other psychiatric condition including abuse of alcohol or drugs. There was no family history of psychiatric illness. She was highly educated and had been socially well-functioning until the onset of symptoms.

On admission, she presented with moderately depressed mood, psychomotor retardation and impaired attention. She complained of weight loss (approximately 10 kg), lack of