# Correspondence

## What is the relevance of the term "functional" in psychiatric disorders in the era of functional brain imaging?

**Dear Editor,**

Traditionally mental illnesses are classified as organic (biological) or functional (non-biological) mental illness depending on the presence or absence of biological changes in the brain respectively (1). Sims (2) wrote about "organic and functional" terminology as follows: "I have not contrasted organic with the conventional functional, because functional is a most misleading term. It causes conceptual fog rather than enlightenment. A logical person who is innocent of medical jargon would be baffled to know why disturbance of human function by psychological mishap should be called functional, while similar disturbance of function from organic disease is not." However, this classification has lost it original meaning with the increase in neurobiological findings in psychiatry.

Traditionally, imaging techniques are divided into structural and functional neuroimaging, despite some modalities containing a mixture (3). "Structural neuroimaging" involves techniques that map the anatomy of the brain, whereas "functional" neuroimaging (in the broadest sense) denotes electrophysiological assessment of brain functions. Current research in imaging in psychiatry is a combination of both structural and functional imaging. In general, structural imaging research in psychiatry is focused on non-specific structural abnormalities (e.g., ventromegaly in schizophrenia, abnormal amygdala and hippocampus in PTSD) and indirectly focuses on the functions of those abnormal areas, whereas, functional imaging research focuses on functional abnormalities of a particular brain area, which is relatively specific to a finding (e.g., increased activation of amygdala in response to fearful face in functional imaging, whereas there will be no changes in structural imaging). In view of low yield and low predictive power of structural imaging research these many years to find reliable structural markers for psychiatric disorders (4), the authors presume that functional imaging in psychiatric research will have more chances to provide new insights into the etiology of psychiatric disorders in the near future. Undoubtedly the rapid growth in functional brain imaging techniques will increasingly offer more detailed diagnostic information of psychiatric disorders.

In the current clinical practice of psychiatry, structural brain imaging is being routinely used to exclude organicity especially in atypical presentations before making a definite diagnosis of a particular psychiatric disorder. The authors feel that the time may come in the near future when functional imaging modalities are routinely used in the clinical practice of psychiatry to make a definite diagnosis of psychiatric disorders because of the presence of comparatively specific findings of functional neuroimaging. This was also predicted in the editorial of Acta Psychiatrica Scandinavica in 2006 (5): "Brain imaging in psychiatry: from a technique of exclusion to a technique for diagnosis" and American Journal of Psychiatry in February 2009 issue (6): "Connecting brain structure and function in schizophrenia."

Traditionally the functioning of the body is studied in the branch of "physiology." The branch of "neurophysiology" deals with the function of brain. Current functional imaging research in psychiatry is heavily focused on different aspects of functional components of the brain (biochemicals as diagnostic biomarkers, neuron functions in molecular imaging and neuroimaging genomics) in the belief that brain functioning is associated with psychiatric disorders. Hence, future psychiatric disorders may be termed functional psychiatric disorders, meaning, neurophysiological disorders.

The authors are in favor of the revival of the term "functional" in psychiatry with a new meaning for an old term, "functional" meaning functional changes of the brain associated with psychiatric disorders and in favor of abandoning the literal meaning of the term "functional" as non-biological. We would like to revive the term "functional" to its logical meaning rather than the medical meaning to avoid confusion. Hence, the authors feel that the term "functional" in psychiatry still has relevance now and in the future.

### CONCLUSION

The future of classification of psychiatric disorders heavily depends on neurophysiologic factors, which in turn depend more on functional neuroimaging research. The contributions of functional neuroimaging to the biological basis of psychiatric disorders are enormous and still evolving. In the future functional neuroimaging research needs to establish more specific

biological findings of psychiatric disorders. Once the more specific biological basis of psychiatric disorders is known, future psychiatric disorders may be referred to as functional psychiatric disorders denoting neurophysiological disorders.

The time may come in the future when psychiatrists routinely use functional imaging to diagnose functional psychiatric disorders whenever patients comes with psychiatric symptoms and to guide the choice of appropriate treatments from the findings of the functional imaging of patients.

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## Is disturbed sleep a clinically useful marker to determine the suicide risk in patients with post-traumatic stress disorder?

**Dear Editor,**

Sleep abnormalities are prevalent in the general population and among patients with psychiatric illnesses. A growing body of research has associated sleep disorders such as primary insomnia, nightmares, sleep terrors, parasomnias and breathing-related sleep disorder (sleep apnea) with other psychiatric conditions including posttraumatic stress disorder (PTSD), major depression, substance use disorders, schizophrenia and suicide.

For example, it has been reported that nightmares were associated with suicidality after controlling for depressive symptoms (1). Similar results were obtained by another group: it was found that adolescents who experienced frequent nightmares had a higher risk for suicide attempts or suicidal ideation (2). They also reported that adolescents sleeping less than eight hours per night had a higher risk to make a suicide attempt than the adolescents who slept nine hours per night or more. Changes in rapid eye movement (REM) sleep have also been reported, including a shorter mean REM latency, a higher mean REM percentage and different within-night distribution of dream quality in a sample of depressed patients with suicidal tendencies when compared with non-suicidal depressed patients (3). Despite these findings, it remains unclear whether the appropriate treatment of insomnia reduces the risk of suicide. Current knowledge already points out insomnia as a putative warning sign of future suicidal behavior among depressed patients.

We want to draw particular attention to the association of suicidality with disturbed sleep among patients with posttraumatic sleep disorder (PTSD). To date, there is still no clear understanding of this relationship. Are sleep disturbances a consequence of the disorder or they are a risk factor for the development of PTSD? Spoormarker and Montgomery (4) proposed three possible links between disturbed sleep and PTSD: a) disturbed sleep acts as a risk factor for PTSD; b) they are separate disorders that share a common origin in trauma; c) disturbed sleep acts as a mediating factor, based on a common vulnerability and its influence on the development of PTSD. Of note, Spoormarker and Montgomery (4) also suggested that insomnia may be underdiagnosed and undertreated.

Several lines of evidence suggest that PTSD is associated with suicidal behavior (5). Several links between PTSD, sleep abnormalities and suicidal behavior may exist. For example, 1) suicidal behavior in PTSD may be mostly related to sleep abnormalities not to the diagnosis of PTSD per se; 2) suicidal behavior in PTSD may be mostly related to the diagnosis of PTSD and sleep abnormalities may be a contributing factor; 3) suicidal behavior in PTSD may be mostly related to psychological trauma not to the diagnosis of PTSD and sleep abnormalities may be a contributing factor; 4) the same neurobiological mechanisms may underlie PTSD, sleep problems, and suicidal behavior; for example, seroton-

ergic disturbances may play a role in the pathophysiology of all three conditions.

Clinicians may give more importance to the primary disorder, while considering sleep complaints as secondary symptomatology. However, it is reasonable to suggest that disturbed sleep may be a clinically useful marker to determine the suicide risk in PTSD patients and further research on this issue is warranted.

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## Affective disorder and polycythaemia vera

**To the Editor**

We are reporting on three siblings diagnosed as affective disorder with polycythaemia vera during the episodes. Polycythaemia disappearing with the remission of affective disorder is the focus of attention of these cases.

A 28-year-old single woman presented at our outpatient clinic for treatment of a manic episode. Her psychiatric history revealed that she had had manic and subclinical depressive episodes for almost ten years. Three days after polycythaemia vera developed, a manic episode began with abnormally elevated, irritable mood, grandiosity, decreased need for sleep, more talkative than usual, increase in goal directed activity such as spending too much money to solve troubles, anxiety. On physical examination there was a remarkable redness on her face. She complained of sensations of shortness of breath, smothering and choking. She feared closed spaces. She was treated with typical antipsychotics. The manic episode disappeared within two months. Physical examination was performed in an internal medicine clinic. One week after the remission of manic episode her polycythaemia disappeared. The patient was given phlebotomies of 500cc weekly during the manic episode.

Our patient told us that her two brothers also suffered from similar psychiatric and hematological problems. One brother was 26 years old, single, with psychotic depression developing rapidly after a stressful life event. At the same time polycythaemia was present. Phlebotomy was performed four times during one month. Polycythaemia and depression remitted at the same time.

Another brother was 24 years old, single, with polycythaemia and nonpsychotic depression developing after his brother's depression. Phlebotomy was performed three times during three weeks of treatment. Again polycythaemia and depression remitted at the same time.

The siblings had no internal disease. Only our patient was on lithium treatment, 600mg/day for chronic affective disorder but her first polycythemia attack was prior to lithium treatment.

### DISCUSSION

Polycythaemia signifies an increase above the normal in the number of red corpuscles in the circulating blood. Relative polycythaemia occurs when, through loss of blood plasma, the concentration of the red corpuscles becomes greater than normal in the circulating blood. This may be the consequence of abnormally lowered fluid intake, of the marked loss of body fluids such as vomiting or sweating (1). The term stress erythrocytosis has been applied to polycythaemia that is seen occasionally in active, very hard-working persons in a state of anxiety (1). These conditions may be suitable for our patient due to motor excitation.

Erythrocytosis develops as a consequence of a variety of factors and represents a physiologic response to conditions of hypoxia (2). Erythrocytosis, hypercapnia and somnolence may present in very obese individuals. Erythrocytosis is found in Cushing's Syndrome and can be produced by the administration of large amounts of adrenocortical steroids. Our patient was neither obese nor suffered from Cushing's Syndrome.

In our case polycythaemia is not related with lithium treatment (3).

We found only one report on the relationship between affective disorder and polycythaemia vera in which mania, delirium and polycythaemia rubra vera were documented (4).

There is a letter to the editor reporting polycythaemia vera presenting as sudden-onset cognitive impairment in a 67-year-old woman with clinical dementia. Cognitive dysfunction was due to watershed infarction caused by polycythaemia vera (5).

This case history presents polycythaemia that developed just before the onset of each manic episode and disappeared after the remission of the episode.

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