Development of Depression Profile: a new psychometric instrument to selectively evaluate depressive symptoms based on the neurocircuitry theory

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Background: Although we have several self-report instruments available to assess depression, they yield a composite score and thus do not allow for the differential examination of major symptom clusters associated with depression. However, such an instrument would be a useful tool in subtyping depression and selecting the most appropriate pharmacotherapy for each patient. The neurocircuitry theory describes the biochemical and neuroanatomic background associated with the major symptoms of depression. Based on the neurocircuitry theory, our team has developed a new instrument, the Depression Profile, to selectively assess depressive symptom clusters associated with different neurotransmitter systems and neuroanatomic structures. The aim of our study was to investigate the psychometric characteristics of Depression Profile. **Methods:** 339 patients consecutively admitted with DSM-IV major depression in our hospital completed the Depression Profile in the first two weeks of their hospitalisation. 81 patients in an adult outpatient unit also completed the Zung Self-rating Depression Scale. Internal consistency of Depression Profile was tested with item analysis. The external validity of Depression Profile against the Zung Self-rating Depression Scale was tested using Pearson correlations. **Results:** The internal consistency of Depression Profile proved to be excellent. The Cronbach alpha values of the scales met the expectable minimum level derived from the number of items in the scales. In testing for convergent validity, all Pearson correlation coefficients between Depression profile subscales and the Zung Self-rating Depression Scale were significant and moderate to high which indicates the good external validity of our instrument. **Discussion:** The initial psychometric evaluation of Depression Profile indicates that our instrument has good reliability and internal and external validity. The instrument also proved to be useful in clinical work to aid the choice of medications and determine the subtype of depressive episodes. Further studies, possibly with biochemical and neuroimaging methodology are needed to validate the 9 main symptom clusters of the Depression Profile subscales with respect to their neuroanatomical and neurochemical bases.

Keywords: depression, psychometric validation, depressive symptoms, neurocircuitry theory

Although there are several self-report instruments to measure depression for clinical and research purposes, most of them measure only one aspect, or only part of all symptoms associated with this disorder, yielding one condensed depression score. However, depression may present with several distinct and very different clinical pictures, and important core symptoms may be missing (Carragher et al., 2009). Different subtypes of depression presenting with different symptom clusters probably have different neuroanatomical and neurochemical, and also possibly different genetic background, and these subtypes behave differently also pharmacologically (Asnis et al., 1995; Kendler et al., 2009). In order to select the most appropriate pharmacological treatment, depressive symptoms within each episode should be evaluated separately.

Our understanding of depression and of the biochemical and genetic background of this disorder has been vastly increasing in the last years, and evidence indicates that there are different neurochemical processes and neuroanatomical structures underlying the several different symptom clusters associated with depression. The course of development of antidepressive drugs also reflects these new results. After the transition from tricyclic antidepressants which acted on several neurotransmitter systems simultaneously to the SSRI group which exerted its effect by influencing the serotonergic system more selectively, now the focus increasingly once more turns to drugs that act not exclusively on the serotonergic system but also on the noradrenergic and dopaminergic systems as well (Stahl, 2008). Since, besides their highly beneficial effect, antidepressants have several possible undesirable side effects related to their effect on multiple neurotransmitter systems, it would be crucial to identify depressive symptoms in such a way that they could be mapped to their neurochemical background. This would make it possible to select a drug with the right neurochemical action profile that most closely matches the neurochemical profile of symptoms thus maximizing the effects and minimising the side effects. However, current self-report depression scales do not provide for the separate in-depth screening of depressive symptom clusters associated with the different neurotransmitter systems playing a role in depression.

Based on DSM-IV criteria, out of 9 possible symptom clusters associated with major depression at least five should be present and out of those five at least one should be one of two major core symptoms related to depressed mood and loss of interest or pleasure (APA,

2000). This approach puts mood symptoms in the focus of attention, although recognising the importance of other affected areas. However, we know that other disorders that are part of the depressive spectrum, such as subthreshold forms of depression, or atypical forms of depression tend to present without the priority of mood symptomatology (Judd, 1997). Therefore to be able to grab depressive episodes and syndromes in their wholeness, attention should equally be paid to all major symptom clusters not only those dealing with phenomena related to mood. Therefore the mood symptoms of depression should not have priorities over other distressing phenomena associated with depression especially that mood improvement is not the sole goal of treatment and different depressive symptoms do not resolve at an equal rate (Stahl et al., 2003). The adequate psychopharmacological approach therefore in the treatment of depression is to examine symptoms individually considering their neuroanatomical and neurochemical nature. The neurocircuitry theory postulates that in the background of each depressive symptom there is a hypothetical dysfunction of well-defined brain neural circuits. This may provide an approach which is both scientifically and clinically useful in developing and selecting treatment regimens to selectively target depressive symptoms present in case of a given patient.

NEUROCHEMISTRY THEORIES OF DEPRESSION

Although the classical neurochemical approach to depression was the monoamine hypothesis (Schildkraut, 1995; Schildkraut, 1965), our view is much more complex today, ranging far beyond noradrenaline and serotonin deficit, which seems not to be fully able to account for all phenomena associated with depression (Hindmarch, 2001). In spite of this, studies indicate that currently used antidepressive agents prominently exert their action via influencing monoaminergic pathways (Madaan and Wilson, 2009) which may also in part explain the several residual symptoms encountered in clinical practice (Owens, 2004; Lanni et al., 2009). The monoamines are still thought to play a profound role in the etiopathology of depression, but monoamines are increasingly viewed as playing a regulatory role in depression regulating neural circuits responsible for the individual symptoms of depression (Stahl et al., 2003). The theory is based on the fact that monoamines originate from the brain stem but are released in various projection areas throughput the brain, therefore influencing the function of only one or two monoamines can have an effect on various

different symptoms (Stahl et al., 2003). It is a well known clinical fact that several residual symptoms of depression remain by selective manipulation or boosting serotonergic or noradrenergic neurotransmission in the brain which could be targeted by other pharmacological agents affecting other neurotransmitter systems (Lanni et al., 2009) which gives rise to the vision of applying pharmacotherapies designed for a given patient's neurotransmitter deficiencies based on his unique depressive symptoms profile. For this we should be able to understand the neurochemical background of each symptom and we should also be able to detect and measure these symptoms.

Currently there are 9 main symptoms clusters associated with depression.

I. Depressed mood is thought to be the chief symptom. The emergence of depressed mood and sadness is linked to an abnormal activity in the medial prefrontal cortex, primarily in the anterior cingulate cortex and the orbitofrontal cortex, which are innervated by serotonergic projections from the raphe nucleus and by noradrenergic projections from the locus coeruleus, and by ventral tegmental dopaminergic projections (Stahl et al., 2003; Drevets, 2001; Steketee, 2003; Soares and Mann, 1997; Gelenberg, 2010).

II. Loss of interest and pleasure is one of the classical core symptoms of depression. These symptoms are linked to disturbances in the hypothalamus with noradrenergic and serotonergic input and the nucleus accumbens with dopaminergic innervation (Stahl et al., 2003; Gorwood, 2008).

III. Psychovegetative changes related to major depression include sleep and appetite disturbances. Different types of sleep disturbances, such as insomnia, hypersomnia and disturbed sleep-wake cycle are another important symptom cluster associated with depression. Those areas of sleep regulation which are relevant to sleep problems associated with depression include neural circuits connecting the hypothalamus, the brain stem and the cortex (Stahl et al., 2003). A profound disruption in sleep-wake cycle regulations is often present in depression, and this disruption involves several mechanisms. The regulation of sleep is however much more complex and besides the brain stem monoaminergic projections to the cortex several other complex systems, such as histaminergic neurotransmission play a role (Steiger, 2007; Nofzinger, 2005; Nofzinger, 2008). Appetite change and weight alterations are another frequent symptom of depression, generally linked to hypothalamic functions receiving noradrenergic and serotonergic input.

These projections may not only play a role in weight and appetite control but also, as mentioned earlier, may play a role in other symptoms such as loss of interest and pleasure where the hypothalamus is also thought to play a role (Stahl et al., 2003; Overstreet et al., 2005; Ressler and Nemeroff, 2000).

IV. Psychomotor changes, such as either agitation or retardation are also a frequent phenomena in depression and are probably regulated by the striatum and the cerebellum and their incoming monoaminergic projections, with the role of an abnormal activity in the basal ganglia and the left prefrontal cortex as well as the neurocircuitry involving these areas (Stahl et al., 2003).

V. Tiredness and feeling of loss of energy and fatigue is another common but little understood symptom associated with major depressive episodes. It was suggested that these symptoms are more related to psychomotor problems or somatic symptoms, or even be in fact a lack of mental energy related to cognitive dysfunction (Stahl et al., 2003). We know little about the neurobiological basis of the feeling of loss of energy in depression in spite of the fact that it contributes to significant suffering and dysfunction in affective disorder patients. Candidate brain areas include those responsible for motor function such as the striatum and the cerebellum with serotonergic, dopaminergic and noradrenergic projections, whereas areas possibly associated with mental fatigue, such as cortical projections or noradrenaline, dopamine and acethycholine are equally though to play a role (Stahl et al., 2003).

VI. Guilt and worthlessness feelings and the appearance of suicidal tendencies are also associated with depression along with thoughts concerning suicide. These phenomena are less understood in terms of neurobiology, however it is thought that the mesolimbic dopaminergic pathway and serotonergic and noradrenergic projections to the amygdala and the anterior cingulate cortex may play a role in the background of suicide (Stahl et al., 2003; Arango et al., 2002; Oquendo et al., 2003).

VII. Cognitive disturbances and problems of decision and execution are also easily observable in depression and comprise a third main symptom cluster. Especially concentration problems and executive dysfunction seem to be dominant, which are associated with dorsolateral prefrontal cortex hypoactivity (MacDonald et al., 2000; Ernst and Paulus, 2005). There are several neurotransmitter systems playing a role in executive functions including noradrenaline, dopamine, acethylcholine and

histamine besides other systems (Stahl et al., 2003). The most important neurocognitive functions associated with major depression include diminished problem solving and concentration capacity, slowed thinking or inability to think, and difficulty in making decisions which cause significant distress to patients, and are characteristic not only of major depression but several other psychiatric states and disorders as well (McClintock et al., 2010).

VIII. Somatic pains and complaints are frequently associated with major depression (Wise et al., 2007; Muller et al., 2008). Pathways responsible for sensory input from the periphery to the spine may be likely candidates, just as descending serotonergic and noradrenergic pathways, which have a role in determining the perception of stimuli as painful (Stahl and Briley, 2004; Fava, 2003).

IX. Anxiety is frequently associated with depression as well and anxiety and depressive disorders are often comorbid conditions (Nutt, 2010). The noradrenergic projections form the locus coeruleus to the amygdala as well as serotonergic innervations from the raphe nuclei to the limbic system are thought to play a crucial role in the modulation of fear and anxiety also in depression (Charney, 2003).

THE DEVELOPMENT OF DEPRESSION PROFILE

Based on the neurocircuitry theory, several studies investigated the possible neuroanatomical and neurochemical substrates of major depressive symptoms. Our team has developed a new scale, the Depression Profile, which incorporates all symptoms associated with depression forming such symptom clusters that can be mapped to different neurotransmitter activity in different neuroanatomical structures. Currently available depression self-report scales used in clinical practice yield one condensed depression score and do not provide subscales scores for different symptom clusters. Depression Profile, however, decomposes depressive illness to 9 such symptom clusters and provides a profile of symptoms.

After reviewing several depression screening instruments and consulting the DSM-IV and ICD-10 criteria for depression, we listed items corresponding to the main symptoms of depression. Based on the consensus of our group we selected 90 items corresponding to the following symptom clusters:

I.   Depressed mood
II.   Loss of interest and pleasure
III.   Psychovegetative changes
IV.   Psychomotor changes
V.   Tiredness and lack of energy
VI.   Feeling of worthlessness and suicidal tendencies
VII.   Decision and execution
VIII.   Somatic pains and complaints
IX.   Anxiety

The aim of our present study was to investigating the psychometric properties of Depression Profile on a large number of patients with major depression.

PSYCHOMETRIC TESTING OF DEPRESSION PROFILE

Subjects

339 patients consecutively admitted with DSM-IV major depressive episode at the Department of Clinical and Theoretical Mental Health of Kútvölgyi Clinical Center, Semmelweis University completed the Depression Profile. All patients completed the instruments within the first two weeks of admission. 81 DSM-IV major depression patients at the Adult Outpatient Care Unit of Markusovszky Hospital of Vas County patients also completed the Zung Self-Rating Depression Scale in addition to Depression Profile for external validation. The questionnaire was given to the patients by their treating clinician. The Institutional Review Board in charge of experimentation with patients approved the study. All patients provided informed consent before participating in our study.

Statistical analysis

We tested the reliability and internal consistency of Depression Profile scales with item-analysis. We then tested the external validity of Depression Profile against the Zung Self-Rating Depression Scale. The convergent validity of the scales was checked with Pearson correlation.

RESULTS

Descriptive statistics including means, standard errors and standard deviations of the 9 subscales of Depression Profile in our study population are shown in Table 1.

The internal consistency of Depression Profile proved to be excellent. The Cronbach alpha values of the scales met the expectable minimum level

Table 1. Means and standard deviations for Depression Profile subscale scores in our study population

	Items	Mean	Minimum	Maximum	Std.Dev.	Std. Error
I. Depressed Mood	5	8.52	0	15	3.8656	0.2099
II. Loss of Interest and Pleasure	14	22.38	0	42	11.1839	0.6083
III. Psychovegetative changes	13	15.00	0	39	7.5241	0.4087
IV. Psychomotor changes	5	7.58	0	15	3.5960	0.1953
V. Tiredness and lack of energy	13	20.78	0	39	10.2528	0.5569
VI. Feeling of worthlessness and suicidal tendencies	14	18.21	0	42	11.0612	0.6008
VII. Decision and Execution	5	7.58	0	15	4.2120	0.2288
VIII. Somatic pains and complaints	6	6.16	0	18	4.5643	0.2479
IX. Anxiety	15	21.32	0	45	9.7628	0.5302

Table 2. Internal consistency of Depression Profile Subscales

Subscales	Number of items	Cronbach alpha
I. Depressed Mood	5	0.8373
II. Loss of Interest and Pleasure	14	0.9426
III. Psychovegetative changes	13	0.7723
IV. Psychomotor changes	5	0.7299
V. Tiredness and lack of energy	13	0.9377
VI. Feeling of worthlessness and suicidal tendencies	14	0.9396
VII. Decision and Execution	5	0.8855
VIII. Somatic pains and complaints	6	0.8238
IX. Anxiety	15	0.8951

derived from the number of items in the scales. The "Psychomotor changes" subscale showed the lowest Cronbach-alpha (0.73), and the "Loss of interest and pleasure" subscale had the highest (0.94) value (Table 2).

The convergent validity of the new structure of Depression Profile was tested against the Zung Self-Rating Depression Scale on a smaller sample of 81 patients. We have tested Pearson correlations on subsamples of men and women separately as well. All correlation coefficients were significant. The correlation coefficients were moderate to high in all cases. The lowest correlation coefficient emerged in case of the "Somatic pains and complaints" scale (r=0.5), while the highest in case if the "Depressed mood" scale (Table 3).

Diagram 1. Graphical presentations of the scores on the Depression Profile subscales
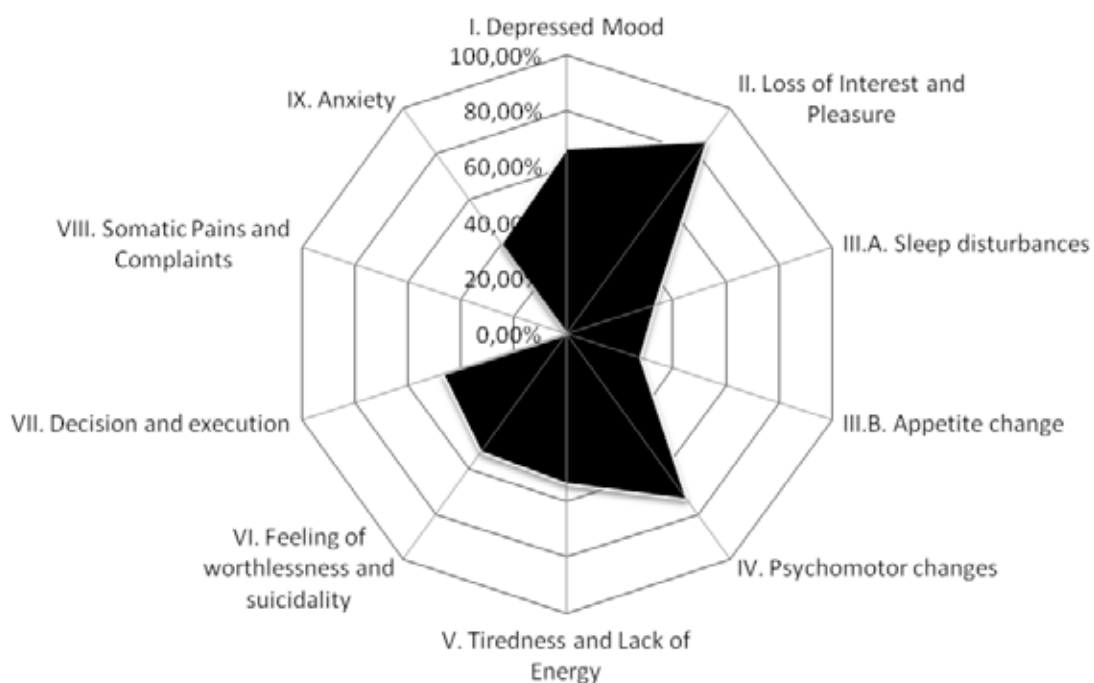
Table 3. Convergent validity of Depression Profile against the Zung Self-Rating Depression Scale
(Pearson's correlation coefficients)

	Whole sample (n=81)	Women (n=55)	Men (n=26)
Depressed mood	0.76	0.71	0.87
Loss of interest and pleasure	0.73	0.70	0.80
Psychovegetative symptoms	0.65	0.63	0.74
Psychomotor changes	0.64	0.64	0.70
Tiredness and lack of energy	0.70	0.69	0.75
Feeling of worthlessness and suicidality	0.69	0.69	0.74
Decision and execution	0.71	0.65	0.81
Somatic complaints	0.59	0.57	0.67
Anxiety	0.71	0.70	0.72

All correlation coefficients were significant.

DISCUSSION

The initial psychometric evaluation of Depression Profile indicates that our instrument has good reliability and internal and external validity. All the 9 subscales of our instrument showed excellent internal consistency, and all of the subscales had a moderate to high correlation with the Zung Self-rating Depression Scale score. We tested our instrument on a large number of patients, and we found it also clinically useful in establishing the constellation of depressive symptoms in case of each patient, determining the prominent symptoms and selecting medication to suit the individual constellation of symptoms.

We also developed a software to simplify data input and calculation of the subscales, as well as to store data for future research. The software also yields a cobweb diagram for the graphical presentation of the symptoms measured by the 9 subscales, also depicting the relative severity of each symptom cluster (Diagram 1). The graphical presentation of different symptoms is a helpful tool in evaluating the severity of a given depressive episode and in selecting the appropriate treatment.

The development of Depression Profile is a major step towards establishing tailor-made pharmacological therapies, adjusted to the leading symptoms of a given episode, maximising treatment of depressive symptoms and also minimizing side effects and adverse reactions. Thus our instrument fills a previous gap in the field of depression assessment instruments, where the majority of previously developed well-known and widely used self-report questionnaires yielded a single composite score without the possibility for investigating individual symptoms. Our new instrument, however, is useful not only for aiding clinical work and selecting the most adequate pharmacotherapy, but also for depression research in general, since it allows for subtyping of depression based on the relative prominence, presence and absence of individual symptoms. Furthermore, since our instrument was developed theoretically, based on the neruocircuity theory, it anchors these symptom clusters to neurotransmitter systems and neuroanatomical structures.

Our instrument has proved to be very useful in everyday clinical work. In the next step we will need to validate Depression Profile and the 9 main symptom clusters with respect to their neuroanatomical and neurochemical bases, as well as their pharmacological characteristics using neuroimaging techniques and neurochemical profiling.

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REFERENCES

1. APA, 2000. Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision. American Psychiatric Association.
2. Arango, V., Underwood, M. D. & Mann, J. J., 2002. Serotonin brain circuits involved in major depression and suicide. Prog Brain Res 136, 443-53.
3. Asnis, G. M., McGinn, L. K. & Sanderson, W. C., 1995. Atypical depression: clinical aspects and noradrenergic function. Am J Psychiatry 152, 31-6.
4. Carragher, N., Adamson, G., Bunting, B. & McCann, S., 2009. Subtypes of depression in a nationally representative sample. J Affect Disord 113, 88-99.
5. Charney, D. S., 2003. Neuroanatomical circuits modulating fear and anxiety behaviors. Acta Psychiatr Scand Suppl, 38-50.
6. Drevets, W. C., 2001. Neuroimaging and neuropathological studies of depression: implications for the cognitive-emotional features of mood disorders. Curr Opin Neurobiol 11, 240-9.
7. Ernst, M. & Paulus, M. P., 2005. Neurobiology of decision making: a selective review from a neurocognitive and clinical perspective. Biol Psychiatry 58, 597-604.
8. Fava, M., 2003. The role of the serotonergic and noradrenergic neurotransmitter systems in the treatment of psychological and physical symptoms of depression. J Clin Psychiatry 64 Suppl 13, 26-9.
9. Gelenberg, A. J., 2010. Depression symptomatology and neurobiology. J Clin Psychiatry 71, e02.
10. Gorwood, P., 2008. Neurobiological mechanisms of anhedonia. Dialogues Clin Neurosci 10, 291-9.
11. Hindmarch, I., 2001. Expanding the horizons of depression: beyond the monoamine hypothesis. Hum Psychopharmacol 16, 203-218.
12. Judd, L. L., 1997. Pleomorphic expressions of unipolar depressive disease: summary of the 1996 CINP President's Workshop. J Affect Disord 45, 109-16.
13. Kendler, K. S., Fiske, A., Gardner, C. O. & Gatz, M., 2009. Delineation of two genetic pathways to major depression. Biol Psychiatry 65, 808-11.
14. Lanni, C., Govoni, S., Lucchelli, A. & Boselli, C., 2009. Depression and antidepressants: molecular and cellular aspects. Cell Mol Life Sci 66, 2985-3008.
15. MacDonald, A. W., 3rd, Cohen, J. D., Stenger, V. A. & Carter, C. S., 2000. Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. Science 288, 1835-8.
16. Madaan, V. & Wilson, D. R., 2009. Neuropeptides: relevance in treatment of depression and anxiety disorders. Drug News Perspect 22, 319-24.
17. McClintock, S. M., Husain, M. M., Greer, T. L. & Cullum, C. M., 2010. Association between depression severity and neurocognitive function in major depressive disorder: a review and synthesis. Neuropsychology 24, 9-34.
18. Muller, N., Schennach, R., Riedel, M. & Moller, H. J., 2008. Duloxetine in the treatment of major psychiatric and neuropathic disorders. Expert Rev Neurother 8, 527-36.

19. Nofzinger, E. A., 2005. Functional neuroimaging of sleep. Semin Neurol 25, 9-18.

20. Nofzinger, E. A., 2008. Functional neuroimaging of sleep disorders. Curr Pharm Des 14, 3417-29.

21. Nutt, D. J., 2010. Rationale for, barriers to, and appropriate medication for the long-term treatment of depression. J Clin Psychiatry 71 Suppl E1, e02.

22. Oquendo, M. A., Placidi, G. P., Malone, K. M., Campbell, C., Keilp, J., Brodsky, B., Kegeles, L. S., Cooper, T. B., Parsey, R. V., van Heertum, R. L. & Mann, J. J., 2003. Positron emission tomography of regional brain metabolic responses to a serotonergic challenge and lethality of suicide attempts in major depression. Arch Gen Psychiatry 60, 14-22.

23. Overstreet, D. H., Friedman, E., Mathe, A. A. & Yadid, G., 2005. The Flinders Sensitive Line rat: a selectively bred putative animal model of depression. Neurosci Biobehav Rev 29, 739-59.

24. Owens, M. J., 2004. Selectivity of antidepressants: from the monoamine hypothesis of depression to the SSRI revolution and beyond. J Clin Psychiatry 65 Suppl 4, 5-10.

25. Ressler, K. J. & Nemeroff, C. B., 2000. Role of serotonergic and noradrenergic systems in the pathophysiology of depression and anxiety disorders. Depress Anxiety 12 Suppl 1, 2-19.

26. Schildkraut, J. J., 1965. The catecholamine hypothesis of affective disorders: a review of supporting evidence. Am J Psychiatry 122, 509-22.

27. Schildkraut, J. J., 1995. The catecholamine hypothesis of affective disorders: a review of supporting evidence. 1965. J Neuropsychiatry Clin Neurosci 7, 524-33; discussion 523-4.

28. Soares, J. C. & Mann, J. J., 1997. The functional neuroanatomy of mood disorders. J Psychiatr Res 31, 393-432.

29. Stahl, S. & Briley, M., 2004. Understanding pain in depression. Hum Psychopharmacol 19 Suppl 1, S9-S13.

30. Stahl, S. M., 2008. Stahl's Essential Psychopharmacology. Cambridge University Press, Cambridge.

31. Stahl, S. M., Zhang, L., Damatarca, C. & Grady, M., 2003. Brain circuits determine destiny in depression: a novel approach to the psychopharmacology of wakefulness, fatigue, and executive dysfunction in major depressive disorder. J Clin Psychiatry 64 Suppl 14, 6-17.

32. Steiger, A., 2007. Neurochemical regulation of sleep. J Psychiatr Res 41, 537-52.

33. Steketee, J. D., 2003. Neurotransmitter systems of the medial prefrontal cortex: potential role in sensitization to psychostimulants. Brain Res Brain Res Rev 41, 203-28.

34. Wise, T. N., Fishbain, D. A. & Holder-Perkins, V., 2007. Painful physical symptoms in depression: a clinical challenge. Pain Med 8 Suppl 2, S75-82.

Depresszió Profil: egy új mérőeszköz a különböző neuronális körökkel összefüggésbe hozható depressziós tünetek szelektív vizsgálatára

Bevezetés: Bár számos önbeszámoló kérdőívet kidolgoztak a depresszió mérésére, ezek általában egyetlen összetett pontszámot adnak, így nem teszik lehetővé a depresszióval összefüggő főbb tünetcsoportok külön-külön történő vizsgálatát. Egy ilyen mérőeszköz azonban hasznos lenne egyrészt a depressziós betegség altípusának megállapításában, másrészt az adott beteg számára legalkalmasabb gyógyszer kiválasztásában. Az idegi körökkel kapcsolatos elmélet kitér a depresszió fő tüneteinek hátterében feltételezett biokémiai és neuroanatómiai elemekre. Ezen elmélet alapján kutatócsoportunk egy új depressziókérdőívet alakított ki, a Depresszió Profilt, amely külön-külön méri a különböző neurotranszmitter rendszerekkel, illetve neuroanatómiai struktúrákkal összefüggésbe hozható depressziós tünetcsoportokat. Vizsgálatunk célja a Depresszió Profil pszichometriai jellemzőinek vizsgálata volt. **Módszer:** 339 egymás után felvett DSM-IV szerinti major depresszióban szenvedő beteg töltötte ki a Depresszió Profilt, míg 81 járóbeteg rendelésen megjelent páciens a Zung Önértékelő Depresszióskálát is kitöltötte. A Depresszió Profil belső konzisztenciáját itemanalízissel vizsgáltuk. A Depresszió Profil külső validitását a Zung Önértékelő Depresszióskálával szemben Pearson korreláció segítségével vizsgáltuk. **Eredmények:** A Depresszió Profil belső konzisztenciája kiválónak bizonyult. Valamennyi alskála esetében a Cronbach alfa mutatók értéke a várt minimum érték felett volt. A külső validitás vizsgálata esetében az alskálák Zung Önértékelő Depresszióskálával mutatott korrelációja minden esetben szignifikáns, és az együttható értéke közepesen magas - magas volt. **Megvitatás:** A Depresszió Profil elsődleges pszichometriai vizsgálatának eredménye arra utal, hogy mérőeszközünk belső konzisztenciája és külső validitása egyaránt kiváló. A kérdőív emellett hasznosnak bizonyult a klinikai munka során is a gyógyszerválasztásban és a depresszió altípusának meghatározásában. További, képalkotó és biokémiai módszereket alkalmazó kutatások szükségesek a Depresszió Profil által mért 9 tünetcsoport és a hátterükben álló feltételezett neuroanatómiai struktúrák és neurokémiai folyamatok összefüggéseinek meghatározására.

Kulcsszavak: depresszió, pszichometriai validálás, depressziós tünetek, idegi körök