

REVIEW ARTICLE

Personality and its Importance for Treatment in Psychiatry

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Compiled with the support of grant MSMT ME Kontakt 949.

Submitted: 2010-06-18 *Accepted:* 2010-07-06 *Published online:* 2010-09-28

Key words: **personality; temperament; character; treatment; psychiatry**

Act Nerv Super Rediviva 2010; 52(2): 114–118

ANSR52020R03

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Abstract

Personality is defined as an individual integration of mental and somatic properties, which is manifested in social interactions and its structure consists of temperament, character and intelligence. It is also responsible for the human's adaptation capability, behavior, emotional processing, feeling and experiencing well-being and illness. This is also the reason why personality type is an important factor in the pathogenesis of various mental disorders. The unique conceptual framework of personality based on a neurobiological ground is the Cloninger's theory of personality. According to the theory temperament is a „hereditary“ and „biological“ core of personality with relative resistance to the environmental stimuli. By contrast, character accrues especially from social learning. Both, temperament and character consist of dimensions. The theory describes the relationships between the biogenetic structure of personality and psychiatric disorders and mutual interactions of all temperament and character dimensions may influence vulnerability to psychiatric disorder. Several studies showed some dimensions of temperament are related to the development of depression and anxiety and have also possible predictive potential for antidepressant treatment outcome. On the other hand, character dimensions could react during the psychotherapy. Changes in character and temperament dimensions could reflect positive treatment effect and also support synergy of antidepressant treatment and psychotherapy in depressed patients. Future neuroscientific research of personality could lead to more complex and effective treatment in psychiatry.

INTRODUCTION

Personality is defined as an individual integration of mental and somatic properties, which is manifested in social interactions. It can be also characterized as dynamic organization of relatively stable properties, which are responsible for the human's adaptation capability. It is widely accepted that personality develops through the interaction of hereditary dispositions and environmental influences (Balon 2004).

From the structural standpoint, most authors agree that personality consists of temperament, character, and intelligence. In the simplified way distinguishing, temperament reflects biological and hereditary contri-

butions, and character reflects social and cultural contributions to personality. Intelligence influences both constitutional and social traits and modifies overall personality functions. It is known, temperament is more genetically determined, with relatively low or no effect of environmental factors on basic temperament traits. The environmental factors do influence other aspects of personality, having an important role in its development. Basic functions of personality are to feel, think, and perceive and to incorporate these into purposeful behaviors (Cloninger & Svrakic 2000).

It is unquestionable personality plays an important role not only in the maintenance of well-being, but also in the developing pathologic conditions. It

is known, personality traits could be potentially risk factors for serious disorders. For example, psychological studies showed, personality type A behavior is an important risk factor for developing cardiovascular diseases. Such individuals manifest high level of rivalry, ambitions and self-achievement. By contrast, personality type C behavior is an important risk factor for developing oncological diseases. These individuals manifest low level of aspirations with tendency to cooperate with and support others and to control negative emotions (Baštecká & Goldman 2001). Personality traits are of critical importance for developing and maintenance of stress coping strategies, which is also the reason why personality type is an important factor in the pathophysiology of various mental disorders. Despite the massive progress in neuroscience and various personality theories, complexity of interactions within personality and also between personality and environment remains not fully understood. From this point of view, Cloninger's personality theory could be of special importance.

CLONINGER'S THEORY OF PERSONALITY

The Cloninger theory of personality created the biopsychosocial model of temperament and character, which is derived from biological, neurophysiological, genetic and psychological studies (Cloninger et al 1993; Cloninger & Svrakic 2000). It describes the relationships between the biogenetic structure of personality and psychiatric disorders. Cloninger has suggested that mutual interactions of all dimensions may influence vulnerability to depression. According to this theory, personality is a complex stepped system which comprises different psychobiological dimensions of temperament and character.

Temperament is largely genetically determined and configures automatic behavior responses. It consists of four hereditary dimensions that are even observable from early childhood and include procedural and unconscious learning. They are designated as *novelty seeking* (NS), *harm avoidance* (HA), *reward dependence* (RD) and *persistence* (P). NS is the system of behavioral activation with dopamine as its neurotransmitter. NS is expressed as a tendency to be excitable, exploratory, enthusiastic and impulsive. HA is the system of behavioral inhibition with GABA and serotonin neurotransmitters. HA is a tendency to be cautious, tense, apprehensive and pessimistic. RD is the system for maintenance of ongoing behavior and noradrenalin and serotonin are its neurotransmitters. RD is a tendency to be warm, sensitive, dependent and sociable. P is the system of partial reinforcement and active behavior despite fatigue and frustration with glutamate and serotonin as its neurotransmitters. Individuals high in P tend to be tenacious, stable, hard-working and determined (Cloninger & Svrakic 2000).

Character develops in the course of ontogenesis and it is mostly affected by social learning. Character regulates the cognitive processes of sensory perception and emotion provoked by temperament. It consists of *self-directedness* (SD), *cooperativeness* (CO) and *self-transcendence* (ST) dimensions. These are determined more by environment than heredity. SD refers to identification with the autonomous self and ability to solve situations according to individual goals and values. CO indicates the extent to which individuals view other people as a part of the self. ST responds to identification with a unity of all things in the world (Cloninger & Svrakic 2000).

Dimensions of personality can be detected as well as measured with the help of a special questionnaire called the TCI (Temperament and Character Inventory). It was developed by Cloninger and associates as a self-rating personality questionnaire. This inventory has been shown to have sufficient validity in the general population (Cloninger et al 1993). Consequently, Cloninger et al have developed a new version of this questionnaire, The Temperament and Character Inventory-Revised (TCI-R).

Cloninger's model of personality has been revealed to be appropriate for clinical utility especially in depressive and anxiety disorders, but also in eating disorders, substance abuse and dependency and personality disorders.

PERSONALITY TRAITS AND TREATMENT STRATEGY

Cloninger's model of personality has been revealed to be appropriate for clinical utility especially in depressive disorders and to even have a probable predictive potential for developing future depression or treatment response (Cloninger et al 2006). Consequently, many studies continue to investigate its clinical application in patients with major depression. These studies have demonstrated that especially high HA is associated with depression and anxiety (Abrams et al 2004; Jylhä & Isometsä 2006; Hruby et al 2009). Some of these studies also suggest, that some dimensions may be related to the response to antidepressant treatment in patients with major depression.

There is evidence that depressive patients display higher HA scores, which decrease after successful treatment, but could remain elevated in comparison to healthy controls following clinical remission (Richter et al 2000; Abrams et al 2004; Joffe et al 1993). Individuals high in HA are at higher risk of developing depression and the familial vulnerability to major depression is predicted most strongly by high HA and also by low SD (Farmer et al 2003). These facts suggest HA could be a personality trait potentially the most appropriate to characterize the typical structure of personality in depressed patients. On the other hand, Farmer et al (2003) identified RD together with NS as a factor

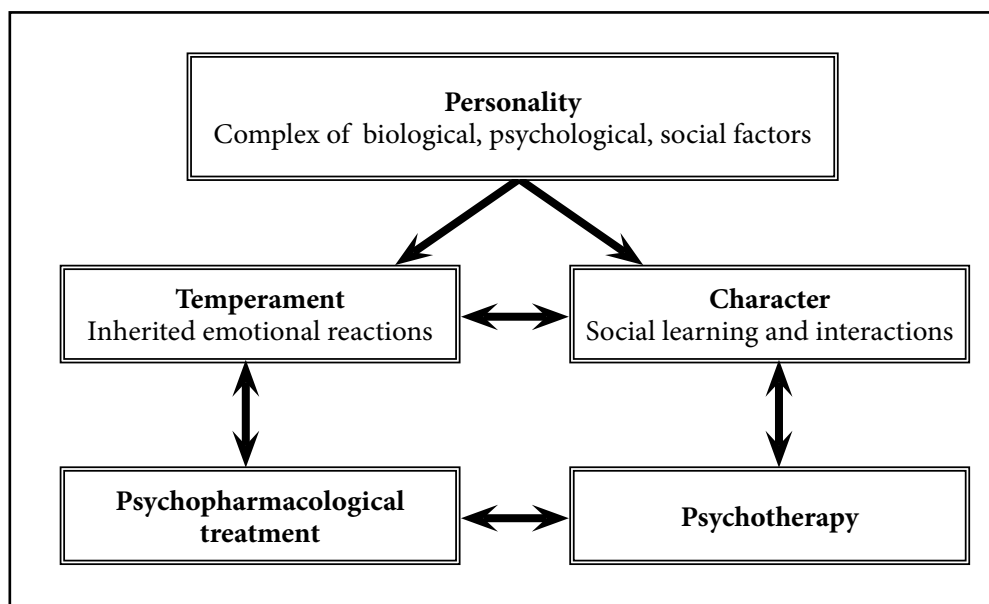


Figure 1. Relations between treatment modalities and personality

decreasing the risk of development of major depression. Such personality traits could probably be considered as a protective factor against the rise of depression or improving the chances of better recovery from developed depression. As the best predictors of antidepressant treatment outcome were recognized HA and RD dimensions (Joyce *et al* 2003; Nelson & Cloninger 1995; Nelson & Cloninger 1997; Hrubý *et al* 2010) and HA could be useful to distinguish between treatment responders and nonresponders (Hrubý *et al* 2010).

To summarize these findings, the majority of studies showed HA as the most markedly influenced by depression and anxiety and have also the strongest predictive potential for treatment outcome together with RD and ST. Some of the temperament dimensions are probably appropriate to predict treatment outcome and distinguish between treatment responders and nonresponders. The findings show that changes in personality dimensions and its predictive value for antidepressant treatment could point at an important role for personality traits in the pathogenesis, course and treatment of depressive disorder. The alterations in temperament dimensions may reflect that personality mechanisms involved in pathogenesis of depression were affected. The changes in character dimensions could reflect the way that personality reacts to depressive disorder, using psychological mechanisms and higher cognitive functions.

Gabbard (2000) highlights that distinguishing between temperament and character can be useful for effective treatment planning. Temperament has a tendency to remain with relatively stable characteristics and it is rather resistant to psychotherapeutic treatment. Character, on the other hand, develops also during the adulthood and it can distinctively react to psychotherapeutic interventions. Considering these

differences, for example the impulsivity conditioned mainly by temperament, we would rather affect by psychopharmacological treatment (e.g. antidepressants), whereas psychotherapy would be beneficial in affecting self-control and interactions with people which emerge mostly from the character. Such approach with combined but aimed usage of different therapeutic methods can bring high-quality and more effective treatment. *Figure 1* illustrates relations between treatment modalities and personality.

Interesting findings showing possible differences in mechanism of actions between psychopharmacological treatment and psychotherapy brought neuroimaging studies. The overview of studies with the use of neuroimaging techniques and functional neuroanatomy in psychotherapy was published by Roffman *et al* (2005). The authors focused mainly on the effect of psychotherapeutic interventions on brain functions in patients suffering from depression or anxiety disorders, and its confrontation with pharmacotherapy. From their findings arose that behavioral therapy of anxiety disorders was associated with moderation of abnormalities in areas connected with the pathophysiology of anxiety and activation of areas which related to positive reworking of anxiogenic stimuli. The changes of similar character in cortical-subcortical circuits occurred in studies related to cognitive-behavioral therapy in major depression. In confrontation of psychotherapy and psychopharmacological treatment in all mental disorders only partial similarity (correlation) of changes detected by neuroimaging methods was found out. This findings support the assumption that psychotherapy and antidepressant psychopharmacological treatment could act through different mechanisms.

For example, Furmark *et al* (2002) used positron emission tomography (PET) for examination of a group

of patients treated by cognitive behavioral therapy (CBT) and a group of patients treated with citalopram for social phobia. During the examination the subjects were instructed to read the text in front of 6–8 closely standing observers and at the same time they were observed by video camera. The patients with social phobia demonstrated an increased activity in limbic structures, including amygdala, hippocampus and adjacent temporal cortex before the treatment. The patients from the CBT group underwent 8 weeks of lasting group psychotherapy specifically focused on anxiety associated with verbal manifestation in public. In comparison with a control group, a significant decrease of activity in mentioned areas was observed in both groups of patients. In a group of patients treated with citalopram, the decrease in an area of ventral PFC (prefrontal cortex) was observed which was not observed in the CBT group. The decrease of cerebral blood flow (CBF) in an area of periaqueductal grey matter was found in the CBT group, reduction of CBF in an area of thalamus was found in the citalopram group. Goldapple *et al* (2004) presented as an example of CBT effects in patients with major depressive disorder the study of authors who realized FDG-PET examination before and after psychotherapeutical treatment and compared these results with depressive patients who were treated with paroxetine. The finding that in a CBT group the decrease of metabolism in numerous regions of frontal area including dorsolateral PFC occurred after the treatment was surprising. The authors assume that this finding is possible to explain by the treatment conditioned “active reassessment of emotionally relevant thoughts”. The finding in a group of patients treated with paroxetine was opposite – increasing of metabolism in PFC, whereas the effectiveness of both treatment methods was comparable. Moreover, the authors found out after the treatment in the CBT group a significant increase of activity in hippocampus, parahippocampal gyrus and dorsal gyrus cinguli. In a group treated with paroxetine the patients in comparison to the previous group demonstrated lower activity in an area of hippocampus and parahippocampus and a decrease of activity in posterior and ventral subgenual cingulum.

CONCLUSIONS

Various forms of gene expression and incomplete penetrance that are typical for serious mental disorders most probably indicate that in their manifestation it is the interaction between the genome on one side and the developmental and environmental factors on the other side (Gabbard 2000). The study of brain plasticity shows that the gene expression activated by cellular processes and its frequency are distinctively regulated by the external environmental signals during the whole life. These facts are extremely important for development of personality, but on the other hand personal-

ity traits directly regulate human behavior, emotional processing, adaptive copying strategies, feeling and experiencing well-being and illness, social interactions and so on. Moreover, increasing body of evidence points at important role of the personality traits in the pathogenesis, course and treatment of neuropsychiatric disorders. Some findings show, different personality components differ in its psychobiological background and could be target for different forms of therapy (psychopharmacology vs. psychotherapy) with different mechanisms of action. Despite such differences, both methods could lead to the similar result on the molecular level, which could be improving of neuroplasticity. For example, depression is treated by antidepressants together with distinct extend of psychotherapy. It is well known untreated depression leads to the impairment of neuroplasticity and morphological changes consequently, mainly in the hippocampus (Frodl *et al* 2002; Sheline *et al* 2003). Studies indicate that both methods have positive treatment effect, but differ in effects on the hippocampal metabolism. This enables the hypothesis to be put forward that these two types of therapy act in synergy. Clinical findings confirm that the combination of psychotherapy and an antidepressant drug is superior to treatment with drugs alone or psychotherapy alone (Olie *et al* 2005).

These positive effects of both psychopharmacological treatment and psychotherapy in increasing neuroplasticity could be explained by principles formulated by Kandel (1998; 2001). He postulated that genes and their protein products significantly affect both relations and interconnections among neurons and also their functions. Genes have an important influence on behavior and they represent one of significant components which participate in a development of mental disorder. The change of genetic information does not generally explain the variations of mental disorder which is affected also by social and developmental factors. Transcript of genetic information affects behavior, however, on the other side behavior and social influences can affect the brain activity and modify gene expression. Regarding these principles, antidepressant treatment targets mainly inherited temperament mediated and disturbed mechanisms, meanwhile psychotherapy affects character mediated changes arising from social and psychological interactions. So it could be possible explanation, why combination of antidepressant treatment together with psychotherapy has kind of synergistic treatment effect.

It is highly probable that intensive neuroscientific research of personality could lead to explicit improvement in planning a treatment strategy in psychiatry, which could reflect patient's individual personality traits in higher extend and be more effective consequently. Investigation of multilevel neuronal regulations and interactions is a very perspective area, which reflects principles and ideas of modern integrated neuroscience.

REFERENCES

- 1 Abrams KY, Yune SK, Kim SJ, Jeon HJ, Han SO, Hwang J et al (2004). Trait and state aspects of harm avoidance and its implication for treatment in major depressive disorder, dysthymic disorder, and depressive personality disorder. *Psychiatry Clin Neurosci.* **58**(3): 240–248.
- 2 Balon R (2004). Poruchy osobnosti. In: Höschl C, Libiger J, Švestka J, editors. *Psychiatrie. II. doplněné a opravené vydání.* Praha: Tigris, ISBN 80-900130-7-4, p. 540–555 (in Czech).
- 3 Baštecká B & Goldman P (2001). *Základy klinické psychologie. I. vydání,* Praha: Portál, ISBN 80-7178-550-4, p. 255–268 (in Czech).
- 4 Cloninger CR & Svrakic DM (2000). Personality disorders. In: Sadock BJ, Sadock VA, editors. *Kaplan and Sadock's Comprehensive Textbook of Psychiatry.* 7th edition. Philadelphia: Lippincott Williams & Wilkins, ISBN 0-683-30128-4, p. 1723–1764.
- 5 Cloninger CR, Svrakic DM, Przybek TR (1993). A psychobiological model of temperament and character. *Arch Gen Psychiatry.* **50**: 975–989.
- 6 Cloninger CR, Svrakic DM, Przybek TR (2006). Can personality assessment predict future depression? A twelve-month follow-up of 631 subjects. *J Affect Disord.* **92**: 35–44.
- 7 Farmer A, Mahmood A, Redman K, Harris T, Sadler S, McGuffin P (2003). A sib-pair study of the Temperament and Character Inventory scales in major depression. *Arch Gen Psychiatry.* **60**(5): 490–496.
- 8 Frodl T, Meisenzahl EM, Zetsche T (2002). Hippocampal changes in patients with a first episode of major depression. *Am J Psychiatry.* **159**: 837–841.
- 9 Furmark T, Tillfors M, Marteinsdottir I, Fischer H, Pissioti A, Langstrom B et al (2002). Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. *Arch Gen Psychiatry.* **59**: 425–433.
- 10 Gabbard GO (2000). A neurobiologically informed perspective on psychotherapy. *Brit J Psychiatry.* **177**: 117–122.
- 11 Goldapple K, Segal Z, Garson C, Lau M, Bieling P, Kennedy S et al (2004). Modulation of cortical-limbic pathways in major depression. *Arch Gen Psychiatry.* **61**: 34–41.
- 12 Hruby R, Nosalova G, Ondrejka I, Preiss M (2009). Personality changes during antidepressant treatment. *Psychiatr Danub.* **21**(1): 25–32.
- 13 Hruby R, Nosalova G, Hrubá S (2010). Predictive significance of TCI-R for antidepressant treatment. *Med Sci Monit.* **16**(8): CR 383–388.
- 14 Joffe RT, Bagby RM, Levitt AJ, Regan JJ, Parker JDA (1993). The Tridimensional Personality Questionnaire in major depression. *Am J Psychiatry.* **150**: 959–960.
- 15 Joyce PR, Mulder RT, Luty SE, McKenzie JM, Sullivan PF, Cloninger CR (2003). Borderline personality disorder in major depression: differential drug response, and 6-month outcome. *Compr Psychiatry.* **44**: 35–43.
- 16 Jylhä P & Isometsä E (2006). Temperament, character and symptoms of anxiety and depression in the general population. *Eur Psychiatry.* **21**: 389–395.
- 17 Kendler ER (1998). A new intellectual framework for psychiatry. *Am J Psychiatry.* **155**: 457–469.
- 18 Kendler ER (2001). Psychotherapy and the Single Synapse. *J Neuropsychiatry Clin Neurosci.* **13**: 290–300.
- 19 Nelson EC & Cloninger CR (1995). The tridimensional personality questionnaire as a predictor of response to nefazodone treatment of depression. *J Affect Disord.* **35**: 51–57.
- 20 Nelson EC & Cloninger CR (1997). Exploring the TPQ as a possible predictor of antidepressant response to nefazodone in a large multi-site study. *J Affect Disord.* **44**: 197–200.
- 21 Olie JP, Costa e Silva JA, Macher JP, editors (2005). *Neuroplasticity. A new approach to the pathophysiology of depression.* London: Current Medicine Group Ltd., ISBN 1-85873-829-6, 69 pp.
- 22 Richter J, Eisemann M, Richter G (2000). Temperament and character during the course of unipolar depression among patients. *Eur Arch Psychiatr Clin Neurosci.* **250**: 40–47.
- 23 Roffman JL, Marci CD, Glick DM, Dougherty DD, Rauch SL (2005). Neuroimaging and the functional neuroanatomy of psychotherapy. *Psychol Med.* **35**: 1385–1398.
- 24 Sheline YI, Gado MH, Kraemer HC (2003). Untreated depression and hippocampal volume loss. *Am J Psychiatry.* **160**(8): 1516–1518.