Univerzita Karlova v Praze

1. lékařská fakulta

Studijní program: Psychologie

Studijní obor: Lékařská psychologie a psychopatologie

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Štěpení a disociace u schizofrenie

Splitting and Dissociation in Schizophrenia

Typ závěrečné práce

Disertační

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Praha, 2014

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Podpis

Identifikační záznam:

PĚČ. Ondřej. *Štěpení a disociace u schizofrenie. [Splitting and Dissociation in Schizophrenia]*. Praha, 2014. 109 s., 4 příl. Disertační práce. Univerzita Karlova v Praze, 1. lékařská fakulta, Psychiatrická klinika. Vedoucí práce Raboch, Jiří.

Acknowledgements

I would like to express my gratitude to my mentors Prof. Jiří Raboch and Ass. Prof. Petr Bob for their supervision, guidance, support and helpful recommendations.

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1. THEORETICAL INTRODUCTION

Neuroscientic approach to psychodynamic description in its historical origin has been proposed by Freud (1895) in his "Project of scientific psychology". In principle this "Project" is focused on relationship between psychoanalysis and neuroscience with the aim to study relations of the nervous system with conscious and unconscious mental phenomena described within the framework of psychoanalytic theory. Basic postulate of this concept is that psychoanalytic explanations and interpretations of mental phenomena refer to processes that may be described using language of neuroscience, physics and other scientific disciplines as originally Freud emphasized and other authors proposed (Beutel et al., 2004; Edelson, 1986; Schore, 1997; Kandel, 1999; Solms, 1997; Westen and Gabbard, 2002a, 2002b; Yovell, 2000).

In this context, Solms proposed the concept of neuropsychoanalysis (Solms and Saling, 1986; Solms, 2004; Solms and Turnbull, 2011; Panksepp and Solms, 2012). The neuro-psychoanalytical concept develops substantial links between Freud's neurological and psychoanalytic ideas and suggests that both sciences may collaborate on issues of common interest (Solms and Saling, 1986). This neuropsychoanalytical concept is based on biological descriptions of the brain that in principle might be connected with Freud's psychological theories and in clinical

practice may provide integration of psychotherapy with psychoactive medications in agreement with current brain imaging data which show that "talk therapy" affects the brain in similar ways as psychoactive drugs (Solms, 2004). Based on modern concepts, neuropsychoanalysis is aimed to understand the human mind and especially its relationship to first-person perspective of subjective experience that in principle may be linked to processes in the human brain but its existence cannot be reduced only on information processing in neural networks and has specific dynamics that is called 'mind' (Panksepp and Solms, 2012). In this context, historical foundations of neuropsychoanalysis are closely linked to discussion of the basic philosophical concepts in this field that seem to be mainly focused on dual-aspect monism which in principle may present a new "school" of psychoanalysis (Solms and Turnbull, 2011).

In the current psychoanalytical thinking the "neuro-viewpoint" related to dual-aspect monism presents interesting and controversial issue and may be misunderstood as neural reductionism that has been repeatedly a subject of psychoanalytic criticism (Pulver, 2003; Vivona, 2009; Talvitie and Ihanus, 2011). In this context some authors pointed out on limits and clinical insignificance of neuroscience for psychoanalysis simply because "we cannot analyse human experience in neuroscientific terms" (Blass and Carmeli, 2007, p. 36).

On the other hand, there is a valid assumption that using neuroscience for description of psychoanalytic phenomena may help to study relationships between psychological and neuroscientific viewpoints without reductionism (Talvitie and Ihanus, 2011). Talvitie and Ihanus (2011) suggested that this purpose could be possible to achieve within the framework of "neuropsychoanalysis" based on dual-aspect monism that could be understood as an "interfield" theory providing unifying knowledge connecting both fields and viewpoints. Connections between both fields as related to objectively conceptualized scientific view may be based on "circular causality" principle that explains causal chain of phenomena in the field of mind and in the field of physiological events but also interfield causality which may describe a physiological change due to an event in the mind and vice versa. For example, conversion of a stressful event and intrapsychic conflict into somatic symptoms, behavioral disorders or seizures and on the contrary there is evidence that somatic disturbances influence the mind. In this context, it is possible to suppose that a neural event has its representation on conscious or unconscious mental level and vice versa.

With respect to the concept of causality between mind and brain, it is possible to suppose that both structures in principle reflect each other and are isomorphic with respect to general principles of mathematics, in which isomorphism is studied in order to extend insights from one phenomenon to another. In fact, if two objects are isomorphic, then any property described in scientific description of one

phenomenon is on a different "isomorphic" level valid for the objects in another descriptive domain (Marker, 2002; Mazur, 2008). Based on definition of isomorphism this problem is well defined in mathematics for scientific description of observed natural process in which the "same" object can be presented to us in different ways as for example "wave-particle dualism" in physics representing typical example of dual-aspect monism in scientific description of physical phenomena.

Applied to experimental work in science isomorphic description means that if an isomorphism can be found between some fields in which many theorems are already proved and is possible to describe analogies between those fields then they in principle may be linked to a kind of "superobject" or superstructure that underlies both of them and represents their conceptual unity in duality. In this context, even when the super-object is presented to us by two different ways and enables two different descriptions, there is a potential description that enables to consider both of them as equal and isomorphic. On this level of general abstraction we must separate a general quality connecting both descriptions and find how this general quality is specifically reduced to create the first one or the second descriptive level.

In the specific general description of the mind-brain relationship it means that mind cannot be fully understood using description of its brain, and on the contrary, mind theory may not provide description of brain phenomena even if in certain conditions, mind may be significantly determined by events in its brain. Both phenomenological levels of description, mind and the brain, have coupled internal dynamic structures that are mutually related and causally influenced. In fact it means that structure as a pattern of relations on various levels of organization creates a system organized by these characteristic patterns of its defined components and creates dynamic and metastable structural changes that define dynamics of the system during the time. Based on this principle, brain as well as mind, may be defined on a principle of structural patterns of relations that are created by its basic defined components. These components may be described by language of physics and chemistry describing creation of various chemical and biological aggregates in the brain and relations between them are essentially understood as information flows between various levels of the system components.

With respect to current findings there is a basic dilemma whether structural "disorder" in the biological system that specifically may emerge as mental or physical disease presents only increased randomness in the system as a response to injurious stimuli or presents specific and different level of organization of its components that is not random and present "meaningful response" of the self-organizing system to these stimuli. This problem seems to be critical for psychoanalytical viewpoint because when a mental disorder is only a transition increasing randomness in the mind then this pathological process is physically and bio-

logically determined. Historically this dilemma is contained in basic principles of psychoanalysis proposed by Freud (1895). Based on his scientific and therapeutic experience Freud proposed that even if mental disorder may be seen as an increased randomness of mental processing, in fact this randomness is caused by intrapsychic conflict, which determines that some mental aggregates that are integrated in healthy state of mind may be pathologically disintegrated.

This Freud's original viewpoint suggests that psychoanalysis as a theory is not reductionistic and in principle it has important theoretical elements that are also contained in modern theories of self-organizing systems which implicate that increased randomness and disorder in the system lead to different relations of system components. On the other hand, self-organizing rearrangement and repatterning of system components implicates increased disorder and random-like behavior in the system. The concept of self-organizing system is in principle in agreement with current neuroscientific evidence that mental disorder may be induced by injurious stimuli from the outside world like chemical substances or various biological or physical influences that increase randomness in the brain and mental system and may cause relational changes of its components. On the other hand mental disease may be induced by information overload leading to confusion and increased randomness in mental processing as well as painful and traumatic experiences affecting mental components and structural patterns of the human mind (Breakspear, 2006; Bob, 2008) that lead to psychological conflict and mental disintegration which may cause physiological and morphological brain abnormalities (Teicher et al., 2006; Bremner et al., 2007).

In this context, main purpose of this theoretical introduction is to show that current psychoanalytical and some neuroscientific theories of schizophrenia present isomorphic descriptions of the disease that in both descriptions in principle may be understood as a disorder of mental and neural unity leading to disintegration of consciousness and on the other hand as cognitive disorder due to increased randomness in mental and brain processing leading to disturbances in cognitive functions which in an extreme case may cause loss of differentiation between self and non-self.

1.1. SPLITTING IN SCHIZOPHRENIA AS A DISCONNECTION IN THE MIND

The term splitting can be defined as a forming of mental aggregates of incompatible experiences that cannot be synthesized (Freedman, 1980). Freud and Breuer (Breuer, 1893) defined splitting process as a division in mental apparatus and as a split of different psychic groupings or ego nuclei, for example due to post-hypnotic suggestion, hypnoid states, multiple personality disorder or somnambulism (Freedman, 1980; Breuer, 1893). The process of splitting creates "…nucleus and center of crystallization for the formation of a psychical group divorced from ego" (Breuer, 1893, p. 123).

1.1.1. Splitting of the Mind

Characteristic description of the splitting suggested Freud (1912), who proposed that as a consequence of splitting consciousness "…oscillates between the two different psychic complexes, which became conscious and unconscious in alternation" (Freud 1912, p.262) and found that the splitted mental representations related to emotional memories may be linked to a real trauma in infancy (Freedman, 1980).

In this context, may be defined "splitting of representations" and characterized by disaggregation of mental images and semantic descriptions of objects, events and the self, for example splitting of one object into two images – "good" one and "bad" one (Freud, 1915a; Brook, 1992). Following Freud's view (1915a, p. 135-136) later psychoanalytical concept proposed by Klein (1946; Lerner and Lerner, 1982) explained splitting as a consequence of a defense mechanism against destructive impulses within the self (Bychowski, 1956; Klein, 1946, p.101) through the process of projective identification that using projective mechanism transfers inacceptable parts of the mind into another person (Hinshelwood, 2008, p.510-516).

Developmental context in "splitting of representations" was elaborated by Kernberg (1982), who developed original Freud's postulates (1938, p.149) on ego

differentiation and its development from an undifferentiated ego-id structure. Kernberg developed a concept in which primarily undifferentiated self-object representation is structured through two contradictory sets of relatively independent memory traces related to pleasurable-good-rewarding and painful-bad-punishing experiences (Kernberg, 1982). Based on these tendencies, experiencing of object's contradictory qualities ["all good" or "all bad"] is linked to undifferentiated self-object representations. In later stages of development these contradictory tendencies may be distinguished and differentiated through interrelated processes of exteroception and interoception that enable to create inner and outer boundaries and differentiate between self and non-self (Kernberg, 1982; Lichtenberg and Slap, 1973).

In this context, differentiative processes related to splitting also may have adaptive functions, "…by which the ego organizes its participation in experience – essentially the early capacity to represent, distinguish between, and categorize extreme states" (Lustman, 1977, p.121). In that manner, splitting may be understood as a developmental precursor of repression and defensive mechanisms, reflecting undeveloped ego's synthetic functions such as primitive idealization, projective identification, denial and omnipotence/devaluation mechanisms (Kernberg, 1975; Lerner and Lerner, 1982; Lustman, 1977; Hinshelwood, 2008).

Using splitting processes, defense mechanisms lead to formation of relatively autonomous independent units within the self. For example, Fairbairn (1941) proposed that psyche is organized through relational structures coupled to the process of introjection based on a process of internal adoption of certain parts of others mind (Sharf and Birles, 1997). The "relational structures" were comprehensively described by Glover using his ontogenetic concept of primitive *ego nuclei*. Glover suggested that the infant psychic structure is multinuclear and consists of psychic ego-nuclei representing innate predispositions of relations between self and intentional objects (Glover, 1943, 1961). In this context, the ego-nucleus functions as a 'miniature ego' that during development of psychic apparatus and its synthetic functions creates relations to other nuclei. This process enables organization of the nuclei into coherent psychic structures. However, under stress the ego tends to regression and splits again, and distinct ego nuclei are expressed via previous early conflicts and fixations. In this context, Glover suggested the term "nucleation" for the process when a coherent structure breaks down into separated and relatively isolated nuclei forming dissociated mental structures (Glover, 1943).

Similarly, Bromberg later proposed existence of many *self-states* in the mind that all have a capability of subjective quality of "I" (Bromberg, 1995, 2003, 2009). According to Bromberg, transitions between individual self-states are subjectively experienced as continuous and create an illusion of one "I-ness". In this context, healthy mind has a certain ability to "stand in spaces" between the self-states

(Bromberg, 2009, p.643). On the contrary, Stern (2004, 2009) proposed that pathologically disintegrated self-states may be characterized by splitting between ordinary experiences and "unformulated" experience that are conflicting and "unacceptable".

Basic description of the splitting process has been proposed by Kohut (1971, p. 240), who suggested that in principle it is possible to define distinctions between horizontal splitting (repression) and vertical splitting (the splitting proper). In the horizontal splitting repressed material is different from unrepressed and content of the split exist on "vertical" axis (e.g. in terms of rawness of affect) while in vertical splitting the split of content exists on the same level on "horizontal" axis, for example represented as "ego nuclei" (Lustman, 1977; Garfield, 2005; Hinshelwood, 2008; Stern, 2004). Both processes define clear differences in which horizontal splitting (repression) allows substitutions by other representations, while vertical splitting results to ego fragmentation (Hinshelwood, 2008). The concept of splitting in general on its various levels leads to a separation of mental groupings that creates relatively autonomous psychic structures due to reduction of the synthetic and integrative capabilities in order to protect mind against conflicting processes within mental apparatus.

1.1.2. Splitting of the Mind in Schizophrenia

Systematic description of splitting— as a form of dissolution— based on psychodynamic approach was suggested by Bleuler (1911/1950) in his monograph *'Dementia praecox or the group of schizophrenias'*. In this groundbreaking work Bleuler described basic forms of splitting in schizophrenia as a consequence of disturbed associations. According to Bleuler, the first form of this process represents *associative splitting* or "loosening of associations", when normal pathways of associations have decreased their "cohesiveness", which leads to displacements, condensations, confusions, generalizations, clang-associations, illogical thinking and incoherence. The second form of splitting is related to *splitting of psychic functions,* when complex psychic structures disintegrate due to emotionally charged complexes that become isolated and independent.

Specific form of the splitting in psychosis may occur in cases, when a patient's mind is split off into *'many different persons'*. This splitting of the mind likely may occur due to a lack of inhibition of normal associative links (Bion, 1957; Katan, 1954) in which the ego changes it's links to one or another of the individual complexes (Bleuler 1911/1950, p.381). In this context, Bleuler in 1911 proposed the term 'schizophrenia', instead of 'dementia praecox' and thought that: "…the 'split-

ting' of the different psychic functions is one of its most important characteristics" (Bleuler 1911/1950, p.8). According to Bleuler (1911/1950) some psychic complexes may dominate personality structure, while other complexes are "split off" and operate as fragments connected with the others in an "illogical" way. Similarly described personality structure also Jung (1909), who proposed that associations are linked to complex mental aggregates that connect emotional, episodic and semantic memories.

According to Bleuler (1911/1950) associative forms of splitting are related to distorted relations between associations and affectivity due to weakened logical functions and abnormally facilitated affects. Bleuler's concept of splitting in schizophrenia was historically linked to psychoanalysis (Falzeder, 2007). In this context, Freud's thinking on schizophrenia (Freud, 1896; 1911) was conceptualized as continuity between schizophrenia and neurosis in which traumatic developmental factors of schizophrenia are quite similar to neurosis. Typically, an intrapsychic conflict and primitive defense (projection, disavowal) may play a role in development of ego weakness, regression of ego functions. Moreover, prevailing conflicts connected with aggressive impulses predetermine an increased severity of the disease and schizophrenic symptoms (delusions, hallucinations) (London, 1973; Grotstein, 1977b). In later Freud's work "On Narcissism: An Introduction" (Freud 1914, p.74-86) he suggested that psychosis is mainly characterized by 'intentional disappointment' that represents libido withdrawal from people and things in the external world which is not replaced in phantasy by other 'intentional expectations' (i.e. libido investment). This process is typically related to instinctual arousal (i.e. free energy) that cannot be 'bound' or 'released' and results to psychic dissolution (i.e. splitting), and 'innervates' ideational complexes that chaotically enter into consciousness. (Freud, 1914; 1915b, p.203; Novey, 1958; Freeman, 1997, p. 39). Based on this approach Freud (1923) postulated that basic aspect of schizophrenia represents a conflict between the ego and external reality that leads to defense mechanisms related to preservation of the ego identity against mental fragmentation.

The concept of schizophrenia as a psychical condition related to similar mechanisms like in neurosis was later elaborated by Klein (1946) and Rosenfeld (1947). According to this concept, schizophrenia represents fixation of paranoid schizoid position, in which primitive defense mechanisms as splitting [in a sense of splitting of representations], projective identification and introjective identification likely play a major role (Klein, 1946). Later Rosenfeld (1947) proposed that projective identification may be excessive, and may cause that parts of ego are split off and projected into other persons with resulting restriction of thinking and behavioral patterns. Similarly, Bion (1957) wrote about "splitting attacks", in which a person splits internal mental objects or functions and identifies them with real ob-

jects or persons that enables to sense and experience his/her own mental states as a part of the external world. For example, various bizarre objects or events, such as "listening chair", "sight" of unanimous objects, and others, based on projective mechanism in which a splitted [unconscious] seeing or other sensations may be transferred to other persons or things (Bion, 1957).

In this context, splitting has been also described as a disturbance or disconnection between two (or more) mental states or thoughts preventing conscious awareness against unbearable negative emotions. A consequence of these "attacks on the link" is an inability to bring together two disconnected thoughts and form a new mental object and related emotional experience (Bion, 1957, 1959).

1.1.3. Lack of differentiation between inner and outer

A limited capacity to differentiate between inner and outer world, and between subject and object is also closely linked to schizophrenic splitting. In this context, several studies compared splitting in schizophrenia with splitting in patients with borderline personality disorder (BPD) (Lustman, 1977; Blatt and Auerbach, 2001). The schizophrenic splitting in comparison to BPD has more "molecular" form of fragmentation (fractured actions, affects, raw drives, perceptions, memories and fantasy elaborations) as well as it lacks reality testing of subjective identification. On the contrary, the borderline splitting typically contains configurations of opposite representational aggregates that occurs in successive alternations and preserves reality testing and distinction between subjective and objective forms of self-awareness (Lustman, 1977; Freedman, 1980; Lerner and Lerner, 1982; Trimboli and Farr, 2000; Blatt and Auerbach, 2001).

The differentiation deficit likely may be linked to Freud´s concept of libido withdrawal from external reality (Freud, 1911) and mental representations of objects (decathexis) (Freud, 1915b). On the other hand schizophrenic fragmentation and loss of differentiation between internal and external world can have a common origin in effects of the very early traumatic factors that do not allow differentiation of primarily undifferentiated self-object representation through interrelated processes of exteroception and interoception as described by Kernberg (1982). Similarly, Grotstein (1977a; 1977b; 1990) described projective and introjective processes related to experiences of missing objects and emphasized a basic deficit in the development of proprioreceptive functions and perceptual awareness which disables narcistic representations of objects (internal objects) that cannot be used for projective identification and re-introjection, which may lead to confusion be-

tween internal and external reality. Caparros (1999) called this process as "uncompleted narcissistic splitting", which is characterized by a lack of borders between inner and outer world in which there is no separation, and any attachment is not possible. The core of this splitting is a denial of a primitive object (Burnham, 1969) as a *need-fear dilemma*, consisting in an extreme need for external structure of the object simultaneously with a fear of abandonment or destruction. Similarly description of schizophrenic mind suggested also Benedetti (Peciccia and Benedetti, 1996), who found that the basis of schizophrenic splitting is a blocked dynamic communication between 'disintegrated separate self' (in which autistic separation predominates) and 'disintegrated symbiotic self' (in which the nuclei of symbiotic fusion predominate). In a case of separateness between these states, the symbol of self cannot be created (Benedetti and Peciccia, 1998; Pestalozzi, 2003).

In this context Stern (1985) in his developmental theory described four basic self structures: 1. self-agency: sense of authorship of one's own actions with self-invariants of volition, proprioreceptive feedback, and predictability of consequences; 2. self-coherence: sense of being non-fragmented, and as a physical whole; 3. self-affectivity: inner qualities of feelings patterned by experience; 4. self-history: sense of continuity in time. The agencies are formed in the first months of life in mutual interactions between a child and a caregiver, which in the case of a disability to form '*sense of a core self*' is linked to inability to appropriately differentiate and distinguish between self and non-self as an agent of an internal experience (Stern, 1985).

Also other authors were focused on the process leading to a loss of differentiation and distinctions that causes undifferentiated state. For example, Robbins (1993, 2011) described the undifferentiated state of mind as a form of undifferentiated experience of the world - 'protosymbiosis'. According to Bleger (1974) this 'symbiotic' state disables to process projective identification as an ability to connect past mental experience with perceptual representations of actually experienced events. In this context, the 'schizophrenic autism' likely presents 'preconscious' undifferentiated 'symbiotic' formation. In this undifferentiated state 'agglutinated nuclei' [Latin agglutinare- "to glue"] are split-off and deposited in an external object with symbiotic link between self and external object (Sas, 1992).

Bleger also considered this mental state as an independent earlier position preceding developmental stages experienced as paranoid schizoid position and described them as deficient and undeveloped mental states [glyschro-caryc position, from Greek γλίσχροσ- sticky and καρυο- nucleus]: "schizophrenia results from a pathological splitting with inability to build up the schizoid content of the paranoid-schizoid position" (Bleger, 1974, p. 24).

Similar concept of primordial "undifferentiated state" can be found in Ogden's theory (1989), who called it as '*autistic contiguous position*'. Experiential mo-

dalities characterizing the undifferentiated state are body touches on the skin surface, rhythmicity and regularity of sucking (Ogden, 1989). Typical experiences characterizing the undifferentiated state were also described by Tustin (1988), who defined them as autistic shapes (e.g. gentle touches) or autistic objects (that cause hard pressure against the infant's skin). The cause of a deficient differentiation from mother-child symbiosis likely is linked to a negative attachment, connected with a fantasy of "…an area of skin common to both mother and infant" (Anzieu, 1993, p.46). Through this area they can communicate, they have the same feelings and sensations and may mutually "…read the thoughts". The self is fragmented into incoherent collection of pieces which create internal undifferentiated '*black holes*' of separateness in which actual representations of otherness are missing (Anzieu, 1974; 1993).

1.2. NEUROSCIENCE OF SPLITTING IN SCHIZOPHRENIA AND NEUROPSYCHONALYTIC VIEWPOINT

According to recent evidence, mental disintegration in schizophrenia is related to disturbed binding and integration of multiple and disparate neural activities underlying cognitive brain functions and consciousness (Singer, 1993, 2001; Varela et al., 2001; Fries, 2005; Tononi and Edelman, 2000; Peled, 1999; Lee et al., 2003; Ford et al., 2007). These findings indicate basic experimental paradigm for understanding of conscious integration, which implicates understanding of schizophrenia as a disorder of neural integration (Tononi and Edelman, 2000). The concept of brain and cognitive disintegration in schizophrenia is reminiscent of the concept of "schizophrenia" proposed by Bleuler in his "Dementia Praecox, or the group of the schizophrenias" emphasizing the splitting or disintegration of consciousness (Bleuler, 1911/1950, 1919/1906, 1924).

In this recent and historical context the "interfield postulate" would implicate that the process of mental splitting likely might be related to some form of "neural splitting" in coordinated brain activity and information processing. Particular consequence of the splitting is also disturbed process of self-reference based on an ability to distinguish between the self and an external world which according to the interfield principle also is related to specific disturbances and disunity of the brain information processing. Within this context, various neuroscientific data and theories seem to provide a basic support for certain neuropsychoanalytic viewpoints of splitting as a specific process on neural levels of information processing. Current findings indicate that psychological splitting in schizophrenia is likely specifically presented on a neural level as disrupted organization in neural communication underlying deficits in mental processing described by three basic neuroscientific concepts of schizophrenia, i.e. theory of disturbed connectivity, theory of corollary discharges and dynamic theory of neural complexity.

1.2.1. Disturbed connectivity and splitting in schizophrenia

The relationship between mental disintegration and disturbed functional integration of the brain has been significantly developed in the theory of disconnection (Friston, 1996) or dysconnectivity (Stephan et al., 2009) of neural activities across different brain regions which likely may significantly influence perceptual, emotional and cognitive processes (Park and Thakkar, 2010). In this context, disconnection means a deficit of functional integration among functionally specialized systems of various neural populations represented in different cortical areas (Friston, 1996; Park and Thakkar, 2010). The problem of functional integration of functionally specialized subsystems was also developed by Hoffman and McGlashan (1993), who proposed the parallel distributed processing (PDP) model of schizophrenia. The PDP model is based on experimental data and analysis of psychotic states induced by phencyclidine, or as a consequence of syndrome of metachromatic leukodystrophy and also neurometabolic studies suggesting that schizophrenia reflects a breakdown in communication between cortical areas. Using a computer simulation of the brain pathology they found that main neurocognitive consequences present functionally autonomous cortical circuits called "parasitic foci" representing disconnected states that create delusions of control, paranoid delusions "idee fixe" related to thought disturbances, hallucination, and cognitive deficits (Hoffman and McGlashan, 1993). These parasitic areas in memory, excluded from other parts of conscious and pre-conscious mental processing, disturb integrative functions and may lead to information interference and conflict (Piagnol et al., 2003).

The neural disconnections may also be linked to various dysplastic changes and related to abnormal modulation of plasticity through dopamine and acetylcholine neurotransmitter systems (Friston, 2002). The disconnection processes also significantly influence regionally specialized neural activities responsible for emotional learning and memory such as prefrontal and temporal cortex, mainly mediated by NMDA glutamate transmissions (Friston, 1996, 1998; Stephan et al., 2006, 2009).

The concept of disturbed connectivity in schizophrenia has been also developed within the concept of multiple constraint organization proposed by Peled (1999; 2008), who emphasized a specific role of multiple connections that enable multimodal representations in the brain integrative processes through various connections among widespread brain units represented by neural assemblies. In this context, Andreasen et al. (1998) proposed that *cognitive dysmetria* in schizophrenia is mainly based on disruptions of connectivity between several brain areas such as prefrontal regions, thalamic nuclei, and the cerebellum. In addition, large-scale simulations focused on dynamics of thalamo-cortical integrations sug-

gest that an altered dynamics of cortico-thalamic and cortico-cortical re-entrant circuits may be influenced by disruptions of the local connectivity within single cortical areas (Tononi and Edelman, 2000). Similarly, Friston (1998) also supposed that these local disconnections might be a consequence of a failure of the brain binding mechanisms that reflect processes of 'disconnection' or on the other hand splitting in the mind. In addition, several data suggest that the connectivity may be decreased in some 'symptomatic' subtypes of schizophrenia and increased in other subtypes (Lee et al., 2003).

Following these findings current neuroimaging studies indicate that the idea of "split mind" becomes relevant in an updated context, and these studies address the mechanism by which splitting of the mind potentially occurs (Park and Thakkar, 2010). Together these studies are mainly based on reflecting disturbed connectivity or dysconnectivity (i.e. increased or decreased connectivity) between several parts of the brain during information processing related to specific psychotic experiences or various experimental tasks (Park and Thakkar, 2010). For example, Satterthwaite et al. (2010) reported that recognition memory of faces is impaired in patients with schizophrenia and used an affective face recognition paradigm to examine possible interactions between cognitive and affective neural systems, namely cortical regions and amygdala, in schizophrenia using a functional magnetic resonance imaging (fMRI) during tasks with affective face recognition. They found that patients performed the task more slowly than healthy control participants. In comparison to control participants schizophrenic patients exhibited weakening of an interaction and correlation between activity in the amygdala and cortical regions involved in cognition, while the controls showed a negative correlation between these regions. This finding may have a relevance to psychical splitting between affect and cognition in schizophrenia as described by Bleuler (1911) and Ciompi (1982).

Other interesting example is a study reported by Diederen et al. (2010), who examined fMRI activation in a network of language-related regions during auditory verbal hallucinations and related signal changes preceding auditory hallucinations. During auditory verbal hallucinations they found heightened brain activation in bilateral (right more than left) language-related regions and bilateral motor regions. They also found prominent deactivation preceding these hallucinations in the left parahippocampal gyrus and significant deactivation preceding hallucinations has been also found in the left superior temporal, right inferior frontal, left middle frontal gyri, in the right insula and the left cerebellum. The authors concluded that a prominent finding of this study shows that the auditory verbal hallucinations were consistently preceded by deactivation of the parahippocampal gyrus that play a central role in memory recollection and sending information from the hippocampus to the association areas. Dysfunctions in

this region caused by disinhibition of the parahippocampal gyrus via dopaminergic innervations, trigger inadequate activity in language-related networks and auditory verbal hallucinations may result from re-experiencing old memories (Diederen et al., 2010).

In this context, it is possible to assume that hallucinations are essentially intrusions of unexpected or unintended information from long-term memory due to unstructured sensory input (Park and Thakkar, 2010). In agreement with recent cognitive models of schizophrenia these data suggest that contextual information exists within a frame of reference which influences access to conscious experience and within this context the positive symptoms of schizophrenia is possible to understand as "contextual disturbances" (Gray et al., 1991; Hemsley, 2005; Bion, 1957; 1959) that on neural level might be represented by deficits of information transfer within the brain and neural communication on synaptic levels.

1.2.2. Dysbalance of neural complexity and splitting in schizophrenia

According to current evidence neural basis of consciousness as an integrative experience likely represents synchronized neural processes that connect distributed brain activities related to various mental events into a coherent whole which is significantly disturbed in schizophrenia (Tononi and Edelman, 2000; Peled, 1999; Lee et al., 2003). Through these coherent links regulatory functions enable inhibition or enhancement of neural excitability to create representational maps that form a basis for integrative mental processing. This dynamic functional organization likely also enables to create simultaneously active groups of neurons involved in dynamic alterations of communicating neuronal assemblies which may be competitive or may generate synchrony and coherent networks. In this context, this ability to come into synchrony (in gamma band or other frequency bands) defines neural integration of the network or its complexity represented by simultaneously active groups of neurons (Tononi and Edelman, 2000; Lee et al., 2003).

Critical role in these integrative processes likely play cortical inhibitory systems that enable modulation of neural plasticity which is manifested by a functional reorganization of synaptic connections (Daskalakis et al., 2007). At the same time the inhibitory systems enable orchestrated activation of a parallel set of inhibitory interneurons that organize cortical processes to an intended action and prevent aberrant activation (Jones, 1993). Nevertheless, although disturbances of these regulatory functions are influenced by inhibitory deficits, also normal inhibitory functions may fail because of enhancement of certain signals related to increased neural excitability. In this context, the regulatory functions are vulnerable

to both inhibitory failure and signal enhancement which suggest that disruptions of temporal binding in distributed neural networks may be influenced by multiple mechanisms. Basic role in this regulatory functions likely play various neuromodulatory processes that likely have a profound influence on the cerebral functions through their effects on the neural excitability and synaptic functions that are significantly disturbed in schizophrenia and other psychiatric disorders (Gordon and Hen, 2004; Gray and Roth, 2007; Kruglikov and Rudy, 2008).

According to recent evidence, the process of disturbed neural integration leading to increased or decreased functional segregation among groups of neurons might also be quantified using concepts from statistical information theory and in particular by defining a measure of neural complexity that could provide a possible explanation for a failure of stability and self-regulatory processes related to disorganized cognition in schizophrenia (Bob, 2008; Breakspear, 2006; Sporns et al., 2000; Sporns et al., 2002; Bob et al., 2009; Balduzzi and Tononi, 2008; Tononi, 2004; Tononi and Koch, 2008). An increase in complexity is often associated with symmetry breaking and the ability of a system to have different states, which is also associated with a decrease in coherence in space over the long range (Weng et al., 1999).

The mathematical and physical concept of neural complexity (Tononi et al., 1994) is characterized by dynamic changes between integration, related to functional connectivity, and segregation, linked to functional specialization of distinct neural subsystems. In this context, neural complexity, measured by EEG and other psychophysiological measures, reflects processes during activity of independent areas that enable fast parallel information processing that runs in a distributed mode (Klonowski et al., 1999; Sammer, 1996; Elbert et al., 1992; Svetlak et al., 2010; Bob et al., 2009; Bob and Svetlak, 2011). This means that numerous processes from sensory and cognitive channels are executed simultaneously and this desynchronized neural state may be related to active information processing in the cortex (Tirsch et al., 2004).

Together these findings suggest that the process of neural or cognitive uncoupling may influence more irregular neural states with higher complexity and negatively affect connectivity patterns and integrative phenomena in the brain that are closely linked to an integrated conscious experience.

In this context, application of nonlinear methods of complexity analysis applied to EEG and behavioral data obtained from schizophrenia patients have shown abnormal patterns of complexity and irregularity (Breakspear, 2006) and altered sequential architecture of their choice-task behavior as a coexistence of highly predictable and highly unpredictable sequences (Paulus and Braff, 2003). Similarly, nonlinear and statistical analysis of data from measurement of

electrodermal activity (EDA) also indicated heightened neural complexity in schizophrenic patients (Bob et al., 2009).

In this context, psychological splitting and brain disunity is possible to study on levels of mental disintegration reflected by psychotic symptoms in schizophrenia that may be related to decreased or increased synchrony. For example relationship between wavelet coherence in some pairs of EEG signal and psychotic symptoms (Bob et al., 2008) or similar relationship between dissociative symptoms in schizophrenia and cross-correlations between EEG pairs likely as a consequence of pathological influences of traumatic 'splitting' or 'dissociation' in schizophrenia (Bob et al., 2010).

1.2.3. Splitting, lack of differentiation and corollary discharges

Cognitive and affective representations of one's identity or the subject of experience present a basis for self-recognition as a specific cognitive process typically involving conscious experience and interpretation of own activity as was previously mentioned in various psychoanalytical concepts (Capparos, 1999; Bleger, 1974; Ogden, 1989) and child observational studies (Stern, 1985). Disruptions of these self-interpretation processes likely represent a neurophysiological substrate for the process of fragmentation of consciousness because of misattribution of certain inner states that may be interpreted as external objects because they are "disowned" and dissociated from consciousness (Bob, 2008). In this context, a specific form of mental disintegration in schizophrenia linked to loss of differentiation of the internal and external world is likely related to deficits in communication between the frontal and temporal lobes (Ford et al., 2005). According to recent evidence, this disintegration of consciousness probably produces defective self-monitoring and self-experiencing (Feinberg, 1978; Ford et al., 2001, 2007) and this lack of interaction and disintegration may reflect the process of functional segregation of sets of neurons localized in different cortical areas.

According to recent findings this loss of distinctions between internally generated psychic activity and external input might be a neural substrate for hallucinations leading to defective self-monitoring and self-integrity originating in motor brain structures (Feinberg, 1978; Feinberg and Guazzelli, 1999). The motor commands from the brain structures are associated with neural discharges that alter activity in both sensory and motor pathways. These neural discharges called corollary discharges (or efference copy) have unique integrative functions that enable monitoring and modification of the commands themselves before an effector event is processed. In addition, they enable to inform sensory systems that the

stimulation produced by movement is self-generated or produced by an environment, which is crucial for the distinction between self and non-self (Feinberg, 1978; Ford et al., 2001; Ford et al., 2005; Ford et al., 2007; Poulet and Hedwig, 2007). In addition, this loss of distinctions may cause autonoetic agnosia as an inability to discriminate a self-generated mental activity from an externally generated one (Keefe et al., 2002). For example, patients with Schneiderian first rank symptoms of schizophrenia characterized by a loss of ego boundaries showed an aberrant relationships between a degree of discrepancy between seen (a virtual hand) and self-executed movements and brain activity as measured by regional cerebral blood flow by PET in the right angular gyrus and in the insular cortex (Farrer et al., 2004).

Other experimental evidence for the relationship between conscious disintegration and defective self-monitoring or self-experiencing has been reported in several studies during hallucinations (Feinberg, 1978; Ford et al., 2001, 2007; Poulet and Hedwig, 2007). For example, using the PET scan it has been found that applications of the same task to people with schizophrenia, and comparing hallucinators to nonhallucinators, show that the hallucinators have decreased blood flow in the speech monitoring areas, such as the left middle temporal gyrus and supplementary motor area (Andreasen, 1997).

In addition, there is also evidence that derangements of corollary discharges included in motor mechanisms of thinking produce many symptoms of schizophrenia in the visual or auditory system (Ford et al. 2001, 2007). For example, self-generated eye movements are related to a corollary discharge of the motor plan, informing the visual cortex that the changing of a visual input results from a self-generated action. A similar mechanism likely exists also in the auditory system, where corollary discharges from motor speech commands prepare the auditory cortex for self-generated speech, likely through a link between frontal lobes, where speech is generated, and temporal lobes, where it is heard, for example, inner speech is misidentified as external voices (Ford et al., 2007; Poulet and Hedwig, 2007).

In summary, recent findings on corollary discharges show that process of disintegration in schizophrenia is related to defective communication between structures of the frontal, temporal and occipital lobes which produces patterns of temporal disorganization and decreased functional connectivity. In addition, efference copy deficits in schizophrenia basically present lack of information processing that is related to specific decrease in brain connectivity that may reflect a level of independence between parts of the brain (Ford et al., 2008).

1.3. CONCLUSION

Current findings suggest that the psychoanalytical concept of splitting may present an essence that may be found in various current neuroscientific theories of schizophrenia focused on processes of dysconnection in information processing. Although this relationship between the neural and mental processes of splitting is evident, there is an epistemological limitation in understanding these processes. This limitation is necessary, because although there is evidence that mental phenomena are related to various physical, molecular, and neurophysiological processes, it is not simply possible to suppose that all brain information processes have mental representation strictly defined in time and space. Nevertheless it is evident and without doubt that large (indefinable) parts of the brain's information content and related neural and information processes are linked to the conscious and unconscious mind.

In addition, including the time dimension as a sequence of mental phenomena that produce states of consciousness enables the mind to be defined as a specific set of mental states represented by various states of consciousness and their connections, which provide associative chains. In this context, it is possible to connect physical and molecular processing linked to neural microstates with the level of information processes in the mind (Bob, 2011). Although the brain–mind interfield principle and dual aspect monism present basic epistemological principles, at the same time they suggest experimentally testable consequences for neuropsychoanalysis and other brain–mind sciences. Using those principles it is possible to connect psychophysiological and psychometric methods and experimentally assess various neurophysiological, molecular and physical processes as well as processes in the psychological domain with the purpose to study relationships between them.

As a consequence, application of the brain–mind interfield principle and dual aspect monism, although it is limited, may help to integrate various findings between the both fields in a meaningful way and using this principle in neuropsychoanalysis may be useful in making specific, experimentally testable predictions and defining methods for their performance. In this context, Freud (1895) in agreement with general thermodynamic and neuropsychological princi-

ples proposed that mind and brain transform the free energy into mental and behavioral activities and focus it on a target in the process of projection or transference which enables that the free psychic energy is ”bound”. Based on this principle mind and brain tend to prefer dynamic activity patterns related to feelings of balance with lowest possible level of free energy. Freud suggested that mental integration as a potentiality of the “balanced” state of the mind without a conflict might be linked to “neural unity”. On the other hand, within the framework of the dual aspect monism it is possible to suppose that splitting of the mind is related to a form of neural disunity and both processes could be consequences of processes of information disintegration that manifest in the brain as well as in the mind.

In this context, interfield theory presents theoretical postulate suggesting that connections between the field of mind and brain may be build on scientific and experimentally testable principles. These principles most likely are linked to isomorphism based on mutually corresponding levels of order or disorder in the brain as well as in the mind that enables measurable distinguishing between ordered or disordered states of the mind and the body.

In the brain ordered states are based on coordinated brain activities related to information processing based on series of timely and spatially organized microstates (i.e. neural binding) that provide a unique ability to combine neural bits of information into complex patterns of activities which manifest as mental functions and behavior. This ability of the brain self-organization to coordinate neural microstates and “bind” them into complex patterns of information enables to use and extrapolate basic terms used in thermodynamics and information theory into specific brain information processing.

This relationship between thermodynamics and information theory is based on physical principles that enable to connect Shannon’s concept of information entropy with Boltzmann’s statistical formulation of the entropy which indicate that the loss of information increases spatial disorder (Scott, 2005; Volkenstein, 2009). This relationship is based on the works published by Szilard (1929/1964), Rothstein (1951) and Brillouin (1956), who found an interesting connection between the Boltzmann entropy and Shannon information entropy (Frieden, 2004). The seminal article “On the Decrease of Entropy in a Thermodynamic System by the Intervention of Intelligent Beings” connecting statistical Boltzmann entropy with information theory was published by Szilard in 1929 who formulated physical connection between thermodynamic entropy and information (Szilard, 1929/1964; Zurek, 1984). Similarly, Brillouin (1956) found that information connected with certain specific physical system, which is bound in the system, is related to its entropy, i.e. bound information = decrease in entropy = increase in negentropy [i.e negative entropy] and on the other hand information loss = increase in entropy =

decrease in negentropy, which means that increase of entropy and loss of information proceed together (Brillouin, 1956).

Based on this relationship, it is possible to connect time and spatial coordination of neural microstates with information patterns in the brain and formulate the Brain-Mind information principle (Bob, 2011). This connection specifically means that disturbed neural coordination of neural microstates implicates increase in statistical disorder on a neural level related to various physical and molecular processes that consequently [on higher level of disarrangement] may appear as functional or structural deficits of the brain. There is evidence that mental phenomena are related to various physical, molecular and neurophysiological processes and it is evident that a part of the brain information content is linked to the human consciousness. In this context, it is possible to connect physical and molecular processing connected to neural microstates with the level of information processes in the human mind. Based on this relationship, it is possible to define brain entropy as a level of disorder reflecting deficits of time and spatial coordination of neural microstates which consequently defines "entropy" of mind as a specific level of disorder that is subjectively experienced and reflected in human mind behavior.

This physical-information relationship enables to define a basic principle of brain-mind information exchange that is based on the relationship between brain physical processes and information processing in the human mind. The relationship between statistical (material) entropy in the brain and information entropy related to information content in the human mind therefore presents and constitutes the basic brain-mind information principle, which specifically states that the loss of information during brain information processing implicates increased disorder (and entropy) of the brain and mind (Bob, 2011).

In this context, interfield view presents philosophical postulate that may be scientifically described using thermodynamics and information theory that represents basis for the relationship between psychological and neuroscience descriptions that also enable to explain mutual influences between mental and neural level of schizophrenia.

Typical example of application of the brain-mind information principle is possible to apply in research of schizophrenic patients. The *brain-mind information principle* states that the loss of information during brain information processing implicates increased disorder (and entropy) of the brain and mind. In the context of schizophrenia, the brain-mind information principle predicts that the loss of information typically implicates disorder and increased statistical randomness in the microscopic spatial domain of the brain. On the other hand the loss of information also predicts increased randomness in the temporal domain of mind, as for example pseudo-randomness in association flow of schizophrenic patients and other

temporal discontinuities of mental experience such as amnesia, depersonalization or derealization and other symptoms.

This consequence of brain-mind information principle is in agreement with findings documenting the disorder in the brain spatial domain of schizophrenic patients and there is evidence that spatio-temporal binding and synchronization mainly in gamma range related to brain information processing is significantly affected in schizophrenia (Tononi and Edelman, 2000; Peled, 1999; Lee et al., 2003; Ford et al., 2008; Uhlhaas et al., 2008; Uhlhaas and Singer, 2010).

On the other hand disturbed order related to information loss in the temporal domain is documented in studies of associative process and other studies focused on discontinuities in schizophrenic thinking and its pseudorandom behavior observed in word associations (Jung, 1909; Kent and Rosonoff, 1910; Moran et al., 1964; Shakow, 1980; Goldberg and Weinberger, 2000), impaired verbal fluency (Allen et al., 1993; Himelhoch et al., 1996; Vinogradov et al., 2002) and textual analyses of the semantic processing (Manschreck et al., 1979, 1981; Hoffman et al., 1982; Goldberg and Weinberger, 2000) indicating deficits in organization of semantic memory in schizophrenia (Davis et al., 1995; Paulsen et al., 1996; Vinogradov et al., 2002).

These findings are in agreement with long-term observational data in schizophrenia which show that the psychological influences on patients' mind likely may explain radical improvements (Harding, 2003; DeSisto et al., 1995; Harrison et al., 2001) and there is evidence that psychological therapies may significantly influence treatment outcome in schizophrenia (Mojtabaj et al., 1998; Gottdiener and Haslam, 2002; Sjostrom, 1985; Wunderlich et al., 1996) and on a parallel levels also brain functions (Andreasen, 1997; Bob, 2011). In recent time new psychotherapeutic approaches have been developed enhancing binding and synthetic capacity of thinking and fostering differentiation abilities between inner and outer world, based on psychoanalytical principles. There should be mentioned particularly mentalization based psychodynamic psychotherapy (Bateman and Fonagy, 2006; Brent, 2009) or approaches focused on intersubjective experience and metacognitive capacity (Lysaker et al., 2012a).

However, evaluation of the effect of psychoanalytically based therapies is complicated by the fact that the effect is likely to rise during follow-up after completion of treatment and the benefits of the therapeutic relationship can be expected no sooner than in five years of therapy (Mojtabaj et al., 1998; Robbins, 1993). Assuming validated hypothesis of dual aspect monism of splitting and dedifferentiation in schizophrenia we might have a chance to observe the effect of treatment using neurophysiological measurements earlier than by usual clinical assessments and also previously estimate which patients benefit more from the treatment.

In addition, the brain-mind information principle predicts process of splitting as a consequence of the information loss, related to inability to integrate some mental contents into consciousness that determines disorder in the temporal information domain of mind. Those typical events of the information loss it is also possible to observe in various experimental and clinical situations such as in hypnosis, during stressful tasks leading to conflict or information overload and in various clinical cases. In this context, some presentations of dissociation in its severe forms such as dissociative identity disorder or in some cases of hypnosis could present logical consequence of basic principles of nature that predict disorder and pseudorandomness in information integration within the temporal domain of mind as a consequence of specific psychological and neurobiological changes leading to information loss in brain information processing. Similarly, the information loss in schizophrenia most likely might be related to mental fragmentation and connected with primitive defense mechanisms, loss of differentiation between inner and outer world and loss of ability to symbolize. In this context, the word "information" (from Latin *informare*) means to form or to shape and create spatial and temporal relations.

Critical epistemological aspect of the brain-mind information principle (Bob, 2011) is that it enables to integrate various finding in the field in a meaningful way, it is compatible with substantial findings in the field and using this principle may help to establish a novel brain-mind information theory which can be useful to make specific experimentally testable predictions and define methods for their performance.

Application of the brain-mind information principle in psychology, cognitive neurosciences and psychoanalysis is capable to provide specific experimental predictions representing regular implications of the theory. In addition, application of the concept could in principle enable to integrate experimental psychology and neuropsychoanalysis into the domain of interdisciplinary physical sciences based on rigorous applications of the mathematical theory and experimental data acquisition that provides promising perspective to utilize theoretical and experimental principles of physical sciences in the field of neuroscientific, psychological and psychoanalytical research and connect both parts of the "Cartesian" universe using scientific methods as originally proposed Freud (1875) in his "Project for Scientific Psychology".

2. EMPIRICAL RESEARCH

2.1. SPLITTING IN SCHIZOPHRENIA AND BORDERLINE PERSONALITY DISORDER

2.1.1. Introduction

Splitting reflects shifts of mind related to a consciously experienced conflict of opposing mental forces. In principle it describes fragmentation of conscious experience that is typically related to long-term or acute stress that significantly disturbs selfconcept, identity, memory and perception of the external world (Breuer & Freud, 1895; Kohut, 1971; Stone, 1988; Bob, 2008; Ellenberger, 1970). Nevertheless, empirical studies of psychopathological processes related to splitting are very rare.

In schizophrenia the term splitting was developed by Bleuler (1911), who described process of mental fragmentation in schizophrenia as associative splitting or "loosening of associations" and considered it as a basic factor in pathogenesis of the disease. Later concept of splitting was described by Kernberg (1975), who used the process of splitting as a specific characteristic of cognitive and affective disturbances in borderline personality disorder (BPD) which typically manifest as shifts of emotional perception of objects, other persons and the self with typical fluctuations between idealization and devaluation.

These alterations on mental level consequently may be linked to great and abrupt changes in patterns of neural activity that may dissociate, or split off, certain external and internal stimuli and information out of awareness, which may lead to distinct states of divided consciousness (Hilgard, 1986; Crawford, 1994; Rainville et al., 2002; Vermetten and Douglas, 2004; Bob, 2008) and disorganization of semantic memory (Goldberg et al. 1998, Paulsen et al. 1996, Robert et al. 1998).

With respect to recent findings and theoretical concept a purpose of this study is to examine relationships between psychological process of splitting and disturbed cognitive and affective functions in schizophrenia and BPD.

2.1.2. Methods

Participants

The participants were recruited from regular daily treatment programs for schizophrenic and BPD patients at the Psychotherapeutic and Psychosomatic Clinic ESET in Prague. All participants signed informed consent and the study was approved by Charles University ethical committee. In the study were included only patients who had not compromised capacity and ability to consent. This ability was confirmed by clinical data about the patients and specific written statement regarding each participant by his/her psychiatrist. Each included participant was able to consider his/her participation and no one was included in the study based on agreement of legally authorized representative consented on the behalf of a participant.

The participants had diagnosis of schizophrenia or borderline personality disorder. Exclusion criteria were organic illnesses involving the central nervous system, substance, and/or alcohol abuse and mental retardation (IQ Raven lower than 90) (Raven, 1960). Clinical diagnoses were reassessed using the Mini-International Neuropsychiatric Interview (M.I.N.I.) (Sheehan et al., 1998) in schizophrenia patients and in BPD patients it was confirmed using semistructured interview for borderline personality disorder based on DSM-IV criteria. The sample included 30 patients with schizophrenia, i.e. 15 men and 15 women, mean age 35.7 (SD =9.2) with mean period of psychiatric treatment 12.89 (SD = 7.8) years and with average of 4.1 hospitalizations. The sample of BPD patients included 35 participants, i.e. 10 men and 25 women, mean age 32.0 (SD = 7.9) years with mean period of psychiatric treatment 6.2 (SD = 3.97) years and with average of 2.28 hospitalizations.

Psychometric measures

With respect to current theoretical concepts and empirical data we have tested relationship between splitting based on Splitting Index score (Gould et al., 1996) and verbal fluency as an indicator for semantic memory disorganization (Goldberg et al., 1998; Paulsen et al., 1996; Franceschi P., 2013) in patients with schizophrenia and BPD. To test how the splitting process is typically represented in schizophrenia and BPD we have compared occurrence of these psychopathological manifestations in schizophrenia and BPD and their relationships to other symptoms.

The symptoms of splitting were measured using self-reported Splitting Index (SI) (Gould et al., 1996) that enables to assess defense mechanisms related to splitting according to concept proposed by Kernberg (1976). Splitting Index is 24-items self-reported questionnaire assessed on 5-point Likert scale from 1 to 5 (Cronbach's alfa 0.92, test-retest reliability after one week 0.82). Using factor anal-

ysis three clusters of items have been identified that enable to describe the splitting process. These three factors represent: 1. the self factor (splitting of the self image), 2. the family factor (splitting of images of family members), and 3. the factor of others which describes splitting with respect to people outside the family.

Other psychopathological manifestations in both groups of patients were measured using Health of the Nation Outcome Scales (HoNOS) (Wing et al., 1996). The scale includes 12 items including overactive, aggressive, disruptive or agitated behavior; nonaccidental self-injury; problem drinking or drug-taking; cognitive problems; physical illness or disability problems; hallucinations or delusions; problems with depressed mood; other mental and behavioral problems; problems with relationships; problems with activities of daily living; problems with living conditions; problems with occupation and activities. This scale includes two versions, i.e. the version for external evaluators and the self-reported version (Cronbach's alfa 0.79, test-retest reliability after one week 0.85) (Pec et al., 2009).

As a measure of semantic memory disorganization, which is very close to Bleuler's concept of mental fragmentation, we have used verbal fluency test (Preiss et al., 2002; Goldberg et al., 1998, Paulsen et al., 1996). In this context, recent findings show that verbal fluency is severely disturbed in schizophrenia (Henry and Crawford, 2005) and it is closely related to disorganized dimension of psychopathology in schizophrenic patients (Robert et al., 1998).

Data analysis

Statistical evaluation of the results of SI and other psychometric measures included descriptive statistics, Mann-Whitney test for independent samples and Spearman correlation coefficients. The non-parametric analyses were preferred because SI data have not normal distribution. All the methods of statistical evaluation were performed using the software package Statistica version 6. To prevent Type II error which would disable to reject null hypothesis that the measure of splitting is not linked to verbal fluency and psychopathological symptoms we performed Power Analysis and assessed the effect sizes characterizing differences between means and correlation coefficients.

Results

Results show significant differences in scores of splitting, verbal fluency and psychopathological symptoms measured by HoNOS between BPD and schizophrenia groups that were compared using Man-Whitney test (Table 1). Mean score of the Splitting Index (SI) was significantly higher in BPD group than in schizophrenia.

On the other hand score of verbal fluency was significantly lower in schizophrenia group. In both assessments of HoNOS, for external evaluators and for self

evaluation, the BPD group scored significantly higher in means of total scores. In the power analysis we have tested significant differences which show that all differences between means have strong effect size (r =0.5 or higher; Table 1).

Results also show significant Spearman correlation coefficients characterizing relationships between splitting, verbal fluency and psychopathological symptoms measured by HoNOS in both samples (Table 2). Very significant relationship between verbal fluency and the SI ''factor of others'' in schizophrenia patients was found (Spearman r =-0.52, p, 0.01). Other significant correlations in schizophrenia patients were found between self-reported score of HoNOS(S) and total score of splitting (SI) (Spearman r = 0.42, p, 0.05) and between HoNOS(S) and SI(S) [representing splitting of the self] (Spearman r = 0.63, p, 0.01). On the other hand significant correlations in borderline personality disorder were found between HoNOS(S) and SI(S) [representing splitting of the self] (Spearman r = 0.45, p, 0.01) and between HoNOS self-reported score and verbal fluency (Spearman r =0.37, p, 0.01).

Table 1. Statistical comparison between schizophrenia and BPD patients using Mann-Whitney test.

	Schizophrenia N = 30	**BPD** N= 35	**Z**	**p**	**r**
SI	2.84	3.14	-2.2	0.0025	0.58
SI(S)	2.69	3.43	-2.8	0.0053	0.82
SI(F)	2.87	3.01	-1	0.3384	0.14
SI(O)	2.96	2.99	-0.2	0.8759	0.05
VF	34.5	41.43	-2.8	0.0052	0.79
HoNOS (E)	11.6	15	-2.6	0.0085	0.81
HoNOS (S)	7.8	14.25	-3.9	0.0000	0.97

Note: SI – Splitting Index, SI(S) – Splitting Index, factor of self, SI(F) – Splitting Index, factor of family, SI(O) – Splitting Index, factor of others, VF- verbal fluency, HoNOS(E)- version for external evaluation of HoNOS (mean), HoNOS(S) - self-rating version of HoNOS (mean), r - standardized effect size

Table 2. Spearman correlation coefficients between SI, verbal fluency, and HoNOS in schizophrenia and BPD.

	SI		**SI(S)**		**SI(F)**		**SI(O)**		**VF**		**HoNOS(E)**	
	Sch	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**
SI(S)	**0.76**	**0.68**	_	_								
SI(F)	**0,72**	**0,53**	**0,40**	0,05	_	_						
SI(O)	**0.73**	**0.47**	0.27	0.01	**0,50**	0,09	_	_				
VF	-0.31	0.15	0.02	0.19	-0,23	0,05	**-0.52**	-0.05	_	_		
HoNOS(E)	0.26	0.13	0.28	0.33	0,21	0,14	0.09	-0.07	-0.22	0.15	_	_
HoNOS(S)	**0.42**	0.28	**0.63**	**0.45**	0,14	0,10	0.10	-0.10	0.16	**0.37**	**0.48**	**0.70**

Note: Values at $p<0.05$ are in bold, Fisher Z higher than 0. 05, Sch – schizophrenia, BPD – borderline personality disorder, SI – Splitting Index, SI(S) – Splitting Index, factor of self, SI(F) – Splitting Index, factor of family, SI(O) – Splitting Index, factor of others, VF- verbal fluency, HoNOS(E)¨- version for external evaluation of HoNOS, HoNOS(S) - self-rating version of HoNOS.

2.1.3. Discussion

Main results of this study indicate significant differences in splitting, verbal fluency and psychopathological symptoms between schizophrenia and BPD patients. These findings show significantly higher level of splitting measured by SI in BPD patients compared to schizophrenia. On the other hand schizophrenia patients show significantly lower scores of verbal fluency most likely as a consequence of cognitive disorganization which in principle is in agreement with Bleuler's historical concept of splitting in schizophrenia (Bleuler, 1911). In this context, the correlation between verbal fluency and splitting (factor of others) in schizophrenia suggests that stronger levels of splitting into opposite aspects related to external objects and persons is related to disassociation of memory patterns that is manifested as disturbed verbal fluency.

Recent findings also show that impaired verbal fluency is associated with psychomotor slowness (Sumioshi et al., 2005; van Bailen et al., 2004) that might be related to disconnection between brain regions (van Beilen et al., 2004). The disconnection between brain regions also disables integrated response to emotional stimuli, which might be linked to specific differences in amygdala activity and prefrontal functions in schizophrenia and BPD (Barnow et al., 2010). Schizophrenia is typically characterized by reduced activation in amygdala and prefrontal cortex and on the other hand increased and excessive activation in amygdala and prefrontal cortex has been found during emotional tasks in BPD (Barnow et al., 2010; Williams et al., 2007; Reske et al., 2009; Schmahl et al., 2004; Scherpiet et al., 2013).

These typical neurophysiological changes might reflect typical differences related to splitting in schizophrenia and BPD based on psychological mechanisms of defense against unacceptable affective impulses (Klein, 1946). Responses to these impulses in BPD likely reflect disturbed levels of reality testing in response to various perceptual stimuli that typically result to increased emotional activation and irritability (Lustman, 1977; Blatt and Auerbach, 2001; Stone, 1988), which on neurophysiological level could be reflected in increased prefrontal and amygdala activation (Barnow et al., 2010). On the other hand disturbed verbal fluency in schizophrenia patients is likely related to decreased activity in amygdala and prefrontal activity in schizophrenia (Barnow et al., 2010; Williams et al., 2007; Reske et al., 2009), which might reflect inability to appropriately differentiate and reflect emotional stimuli (Anzieu, 1993; Ogden, 1989) likely due to disruption in attentional selection and decision related activation (Bob and Mashour, 2011).

In summary, the results show that the process of splitting has different forms in schizophrenia and BPD. In BPD patients splitting results to mental instability manifested as shifts in emotional perception of objects, other persons and the self, which are linked to increased mental tension and excessive prefrontal and amygdala activation. This specific form of splitting that occur in BPD is not typically present in schizophrenic patients, which is in agreement with the results indicating that SI score as a measure of borderline splitting is higher in BPD than in schizophrenia patients. On the other hand in schizophrenia the mental fragmentation leads to splitting of associations observed as lower scores of verbal fluency which in principle is in agreement with Bleuler's historical concept of splitting in schizophrenia (Bob and Mashour, 2011). This form of mental fragmentation in schizophrenia may represent a defense mechanism decreasing several psychopathological manifestations due to lowered mental tension and abnormally inhibited brain activities in amygdala and prefrontal cortices. Nevertheless it is also possible that mental fragmentation in schizophrenic patients is also related to deficits in contextual processing that may be primarily based on brain's ability to integrate information (Bob and Mashour, 2011; Rotenberg and Weinberg, 1999). This brain deficit to integrate information may be linked to various etiological conditions reflecting pathological processes on molecular, physiological and psychological levels. This brain potentiality to integrate information is on cognitive level specifically linked to ability to create integrated self-concept and synthetic capabilities related to various forms of metacognitive deficits that is typical impaired in schizophrenia (Mishara et al., 2014; Lysaker et al., 2013a; Kukla et al., 2013; Lysaker et al., 2013b; Tas et al., 2012; Dimaggio et al., 2008).

2.2. DISSOCIATION IN SCHIZOPHRENIA AND BORDERLINE PERSONALITY DISORDER

2.2.1. Introduction

Dissociation in principle describes fragmentation of conscious experience that is typically related to long-term or acute stress that significantly disturbs self-concept, identity, memory, and perception of the external world (Breuer and Freud, 1895; Stone, 1988; Bob, 2008; Ellenberger, 1970). Consequently, these alterations may be linked to marked and abrupt changes in patterns of neural activity that may dissociate, or split off, certain external and internal stimuli and information out of awareness, which may lead to distinct states of divided consciousness (Hilgard, 1986; Crawford, 1994; Rainville et al., 2002; Vermetten and Douglas, 2004; Bob, 2008).

Dissociation also reflects shifts of mind related to a consciously experienced conflict of opposing mental forces. In the similar context as Janet, also Bleuler coined the term splitting and described the process of mental fragmentation in schizophrenia as a basic step in the pathogenesis of the disease (Bob, 2012; Hilgard, 1986; Bleuler, 1911). The term fragmentation of consciousness in the sense of splitting was also defined in borderline personality disorder (BPD) as aspecific form of dissociation, and recent studies suggest that the relationship between dissociative symptoms and BPD per se is very close (Korzekwa et al., 2009a; Zanarini and Jager-Hyman, 2009; Zanarini et al., 2008).

With the aim of finding specific relationships between dissociative symptoms and other symptoms in BPD and schizophrenia, we assessed both groups of patients with the aim of comparing the occurrence of dissociation and also of assessing the possible influence of antipsychotic medication using chlorpromazine equivalents (EC).

2.2.2. Methods

Participants

The participants were recruited from regular daily treatment programs for outpatients with schizophrenia or BPD at the Psychotherapeutic and Psychosomatic Clinic ESET in Prague. The participants had a diagnosis of schizophrenia or BPD. Exclusion criteria were organic illnesses involving the central nervous system, substance and/or alcohol abuse, and mental retardation (Raven's IQ, 90) (Raven, 1960). Clinical diagnoses were based on DSM-IV (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*) criteria and were reassessed using The Mini-International Neuropsychiatric Interview (Sheehan et al., 1998) in patients with schizophrenia and confirmed by semistructured interview in patients with BPD. We calculated actual daily doses of antipsychotic medication in EC for all participants (Woods, 2003).

The schizophrenia sample comprised 31 patients (15 men and 16 women) of mean age 36.2±9.5 years. Their mean duration of psychiatric treatment was 13.3±8.2 years, and they had an average of 4.4 hospitalizations. The BPD sample comprised 36 patients (eleven men and 25 women) of mean age 31.0±8.7 years. Their mean duration of psychiatric treatment was 6.6±4.1 years, and they had an average of 2.39 hospitalizations. Because of their different durations of psychiatric treatment, the patients also had a different medication history and as measurable equivalent characterizing their current medication we have used EC.

Psychometric measures

We used the Dissociative Experiences Scale (DES) to screen for dissociative symptoms (Bernstein and Putnam, 1986). The DES is a 28-item self-report questionnaire that evaluates the frequencies of various experiences of dissociative phenomena in the patient's everyday life. Each item ranges from 0 to 100 and the mean of all item scores is calculated as the DES score. For more detailed assessment of the DES items, we analyzed the DES factors that have been used in previous research studies (Waller et al., 1996). In this analysis, we used three factors focused on absorption (items 2, 14, 15, 17, 18, and 20), amnesia related to dissociative states (items 3, 4, 5, 8, 25, and 26), and depersonalization/derealization (items 7, 11, 12, 13, 27, and 28). In the present study, we used the Czech version of the DES; like the original English version, it shows high reliability and internal consistency (Cronbach's alpha 0.92, test–retest reliability after one week 0.91)(Bob, 2000; Ptacek et al., 2006).

Symptoms related stress and traumatic experiences were measured using the Trauma Symptom Checklist-40 (TSC-40) (Elliot and Briere, 1992). The scale was designed for measurement of posttraumatic symptomatology associated with childhood trauma. TSC-40 is a self-reported scale that contains 40 items with 6 subscales, i.e., dissociation, anxiety, depression, a sexual abuse trauma index, sexual problems, and sleep disturbances. The Czech version of the TSC-40 has high reliability and internal consistency (Cronbach's alpha 0.91, test-retest reliability after one week 0.88)(Bob et al., 2003).

Psychotic manifestations in both groups of patients was measured with Health of the Nation Outcome Scales (HoNOS) (Wing, Curtis, & Beevor, 1996). The scale includes 12 items (overactive, aggressive, disruptive or agitated behavior; non-accidental self-injury; problem drinking or drug-taking; cognitive problems; physical illness or disability problems; problems with hallucinations or delusions; problems with depressed mood; other mental and behavioral problems; problems with relationships; problems with activities of daily living; problems with living conditions; problems with occupation and activities). There are two versions available: the version for external evaluators and the self-rating version for patients. The both versions were translated into the Czech language (Cronbach's alfa 0.797, test-retest reliability after one week 0.85) (Pec et al., 2009).

Statistical analysis

Statistical evaluation of the results for the DES and other psychometric measures included descriptive statistics, the Mann–Whitney U test for independent samples, and Spearman correlation coefficients. Nonparametric analyses were preferred because the DES data were not normally distributed. All the methods used for statistical evaluation were performed using Statistica version 6 software (StatSoft Inc., Tulsa, OK, USA). To prevent type II error, which would not be able to reject the null hypothesis that symptoms of dissociation are not linked to stress-related psychopathological symptoms, we performed a power analysis and assessed the effect sizes by characterizing differences between means or correlation coefficients of the samples.

2.2.3. Results

We compared scores from the psychometric measures using the Mann–Whitney U test to test for differences in dissociation, other psychopathological manifestations, and use of antipsychotic medication between the two disorders (see Table 1). Although the differences in DES scores between patients with BPD and those with

schizophrenia were not statistically significant, scores for symptoms of traumatic stress measured by the TSC-40 were significantly higher in the BPD group.

External evaluations as well as self-rating on the HoNOS showed that the BPD group had significantly higher scores than patients with schizophrenia. Nevertheless, in several subscales of the HoNOS, external evaluators reported that the schizophrenia group had higher scores for cognitive problems (1.55 in schizophrenia versus 0.66 in BPD, p=0.0001) and positive symptoms (1.26 in schizophrenia versus 0.54 in BPD, p=0.0273). Doses of antipsychotics measured by EC were significantly higher in the schizophrenia group. In the power analysis, we tested the differences between means, and found that all these had a strong effect size (r>0.5; Table 3).

We calculated Spearman correlation coefficients in both patient samples to assess the relationship between dissociation and other psychometric measures and the possible influence of antipsychotic medication on this relationship. The results show that scores on the DES, TSC-40, and HoNOS were significantly correlated (see Table 4). Interesting, statistically significant correlations were found in BPD patients between levels of EC and the DES score (Spearman's correlation r=0.37; refined Fisher's exact test Z=0.14) and between EC and depersonalization/derealization score on the DES (Spearman's correlation r=0.37; refined Fisher's exact test Z=0.38).

Table 3. Comparison schizophrenia to BPD (Mann-Whitney test).

	Schizophrenia N = 31	**BPD N= 36**	**MW – test Z**	**p-value**	**r Effect size**
DES	13.7	18.54	-1.8	0.0730	0.36
TSC-40	34.1	54.19	-3.7	0.0002	0.95
HoNOS (E)	11.6	15	-2.6	0.0085	0.81
HoNOS (S)	7.8	14.25	-3.9	0.0000	0.97
EC	518.4	98.6	5.5	0	0.97

Note: DES - Dissociative Experiences Scale; TSC-40 - Trauma Symptom Checklist-40; HoNOS(E) - version for external evaluation of HoNOS (mean), HoNOS(S) - self-rating version of HoNOS (mean), EC- day dosages of antipsychotic medication in equivalents of chlorpromazine (mean in mg); r - standardized effect size.

Table 4. Spearman correlation coefficients between use of antipsychotics, HoNOS, DES and TSC-40 in schizophrenia and BPD.

	EC		**HoNOS(E)**		**HoNOS(S)**		**DES**		**DES-AB**		**DES-AM**		**DES-DD**	
	Sch	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**	**Sch**	**BPD**
HoNOS (E)	0.2	0.04	—											
HoNOS (S)	-0.22	0.28	**0.48**	**0.7**	—									
DES	0.17	**0.37**	0.34	**0.37**	**0.53**	**0.52**	—							
DES-AB	0.08	0.29	0.25	0.25	**0.47**	**0.51**	**0.88**	**0.84**	—					
DES-AM	0.14	0.28	-0.02	0.25	0.17	0.26	**0.64**	**0.76**	**0.5**	**0.67**	—			
DES-DD	-0.03	**0.37**	**0.53**	**0.35**	**0.65**	**0.4**	**0.8**	**0.77**	**0.61**	**0.44**	**0.36**	**0.48**	—	
TSC-40	0.03	0.08	**0.41**	**0.56**	**0.7**	**0.7**	**0.6**	**0.53**	**0.49**	**0.46**	0.28	**0.38**	**0.75**	**0.43**

Note: Values at $p<0.05$ are in bold, Fisher Z higher than 0.05; EC-day dosages of antipsychotic medication in equivalents of chlorpromazine (mg), HoNOS(E) - version for external evaluation of HoNOS, HoNOS(S) - self-rating version of HoNOS, DES: Dissociative Experiences Scale, DES-AB - factor of absorption in DES, DES-AM - factor of amnesia in DES, DES-DD - factor for depersonalization/derealization of DES, TSC-40 - Trauma Symptom Checklist-40.

2.2.4. Discussion

Our results show that dissociative symptoms and symptoms of traumatic stress are significantly correlated in patients with BPD and in those with schizophrenia. The data also show that symptoms of traumatic stress are higher in BPD than in schizophrenia, which is in agreement with the findings of other (Kingdon et al., 2010; Putnam et al., 1996; Brunner et al., 2004).

On the other hand, DES and TSC-40 scores were significantly correlated with symptoms of psychosis in both disorders and, as in other studies of patients with schizophrenia, symptoms of traumatic stress were associated with psychotic symptoms (Read et al., 2005; Ross et al., 1994), higher levels of anxiety, and other psychopathological symptoms (Lysaker and Salyers, 2007; Lysaker et al., 2001; Lysaker et al., 2004; Janssen et al., 2004; Renard et al., 2012). In agreement with other studies, we also found that dissociation in schizophrenia is closely related to symptoms of trauma (Sar et al., 2010; Schafer et al., 2012; Moskowitz et al., 2009; Ross, 2009; Spitzer et al., 1997; Vogel et al., 2006; 2009a,b). Similar relationships between stress and dissociation have also been found in patients with BPD (Korzekwa et al., 2009a; Howel and Blizard, 2009).

An interesting finding of this study was the correlation between doses of antipsychotics measured in EC and dissociative symptoms in patients with BPD. This result suggests a specific psychotropic effect of antipsychotic medication in these patients. To the best of our knowledge, this has not been reported in the scientific literature before. Because our patients had different medication histories, it is necessary to investigate the possible relationship between medication and dissociative symptoms further in follow-up studies that could explain certain details and specific influences of medication on neurotransmitter systems. Nevertheless, the statistical finding of this relationship in the BPD group but not in schizophrenia group is of interest although further research is necessary. The possible influence of medication on dissociative symptoms might reflect the extremely important role of stress in BPD. According to current data, stress represents a more significant factor in BPD etiology than in schizophrenia (Weber et al., 2009; Barnow et al., 2009). Recent data show that antipsychotic treatment likely decreases activation of the anterior cingulate cortex (Yan et al., 2012; Yücel et al., 2007). In this context it is possible that conscious conflicting (Botvinick et al., 2004; Kerns et al., 2005) experiences due to antipsychotic medication in BPD may decrease conscious awareness of conflicting stressful experiences and cause their dissociation that may produce the dissociative symptoms measured by the DES. Although this interpretation is currently speculative, it might be useful for further research that could have significant consequences for the treatment of patients with BPD.

2.2.5. Conclusion and perspectives

These results support the conceptual but empirically rare findings concerning the important role of dissociative processes in schizophrenia and BPD and the specific relationship between them. A novel contribution of this study that needs further research is the significant finding that manifestations of dissociative symptoms might be specifically linked to antipsychotic medication in patients with BPD but not in those with schizophrenia.

2.3. CONCLUSIONS

In the presented experimental studies we aimed at finding relationships between psychological splitting or dissociation in schizophrenia on one side and other cognitive dysfunctions like semantic memory disorganization or symptoms on the other side. A way how to extend this type of research is to take into account also another perspectives how to study psychological splitting in schizophrenia. Bleuler's concept of splitting is also closely linked to the mind's metacognitive abilities (Lysaker et al., 2013a). As indicated by Moskowitz (2008), when Bleuler proposed disturbances of associations as the core feature of schizophrenia he did not refer merely to confusion due to the intrusion of unrelated ideas into thought but to the fundamental loss of the ability to "associational synthesis" (p. 44) which reduced the understanding of oneself as an embodied agent to a set of fragments which no longer served as a guide for goal directed activity. Bleuler assumed that this lack of synthesis had an organic origin but could also serve as a proximate cause of dysfunction. Bleuler presented detailed accounts of schizophrenia patients no longer able to function socially or vocationally and the loss of the ability to synthesize associations into larger images of oneself and others which then results in metacognitive dysfunction. In that way we can assess metacognition as a reciprocal measure to splitting.

Metacognition as a psychological process is defined as a spectrum of mental activities that involves thinking about thinking, ranging from discrete mental activities such as thinking about a specific isolated thought to more synthetic acts in which context of intentions, thoughts, feelings, and connections between events, are integrated into larger complex representations of self and others including also a reflection about that larger representation (Lysaker et al., 2013a). Metacognition in its synthetic form implies how basic elements of experience are recognized and then synthesized into complex wholes and in that way synthetic metacognitive acts also affect life in a different manner giving meaning to events, and thus, supplying reasons why to carry out a certain act and how to resolve dilemmas in the realm of the unique psychology of oneself and the others. Metacognition is related to the concept of mentalization (Bateman and Fonagy, 2006), though the latter construct considers that disruptions of the above mentioned psychological processes happen in the context of disturbed attachment. Synthetic metacognitive ac-

tivities allow persons to form evolving and flexible representations for themselves and others and thus form a basis for the ability to regulate affect and behavior.

Impaired metacogniction has been found in both earlier and later forms of schizophrenia (Lysaker et al., 2012b; Vohs et al., 2014). Disturbed metacognition is related with negative symptoms (Lysaker et al., 2005a; McLeod et al., 2014; Nicolo et al. 2012), intrinsic motivation (Tas et al. 2012; Vohs and Lysaker, 2014), functional competence (Lysaker et al., 2011a,b), subjective sense of recovery (Kukla et al., 2013), stigma resistance (Nabors et al., 2014), therapeutic alliance (Davis et al., 2011), vocational function (Lysaker et al., 2010a), and interpersonal relationships (Lysaker et al, 2010b; Lysaker et al, 2011).

Thus concept of deficit synthetic metacognition closely related to splitting or mental desorganization shows many links to symptoms and functioning in schizophrenia patients. For future research it would be advantageous to combine both approaches, splitting and deficit synthetic metacognition, and make use of methods of assessment of metacognition in studies concerning splitting and neural complexity.

Synthetic metacognition is assessed by analyzing discourse. The assessment is derived from a spontaneously generated speech sample in which persons discuss their lives and personal understanding of the situations they have faced. That speech sample is obtained through a semi-structured interview called the Indiana Psychiatry Illness Interview (IPII). The IPII asks the participant for their account or narrative of who they are as a person and also of their experience with psychiatric challenges. It thus allows for a life story to be told, in which there are opportunities for participants to spontaneously reveal how they think about themselves. To quantify synthetic metacognitive capacity within IPII narratives, the Metacognition Assessment Scale – Abbreviated [MAS-A] is used (Lysaker et al., 2005b). The MAS-A contains four scales. In each scale higher scores reflect abilities to perform increasingly complex synthetic acts.

The concept of synthetic metacognition links also splitting in schizophrenia in novel trends in psychoterapy of this disorder. Psychotherapeutic approaches directed to integration of splitted parts of mental content or to fostering of stabile mental representation in schizophrenia has been applied in the last decades, e.g. complex forms of cognitive remediation (Roder et al., 2011; McGurk et al., 2007), supportive psychodynamic approaches (Rosenbaum et al., 2012), psychoanalytic psychodrama (Corcos et al., 2012), self-complexity (Martens, 2009). Considering the possible close relationship between defective synthetic metacognition and splitting we can expect further progress from recent forms of therapy related to improvement of synthetic metacognition (Hasson-Ohayon, 2012; Lysaker et al., 2011c; Lysaker et al., 2013c; Brent, 2009). The aim of this novel treatments is to

help persons to form more complex and integrated representations about themselves and others and use this knowledge to respond to psychological problems.

Continuus research that would further clarify mutual relatedness between brain functions, splitting and metacognition might enable to follow a progress in psychotherapy not only by means of assessment on psychological level, but also to apply more measurements on neural level, e.g. EDA, EEG, to track neural binding processes interconnected with better integration on a psychological level.

3. APPENDIX - PSYCHOMETRIC MEASURES

3.1. SPLITTING INDEX - SI

SI

Jméno a příjmení..... Rodinný stav..... Věk .....

Zaměstnání..... Vzdělání.....

Odpověď znázorněte na škále od 1 (vůbec tomu tak není) do 5 (velmi dobře to odpovídá).

1. Cítím sám(a) sebe odlišně, když jsem s jinými lidmi.	1	2	3	4	5
2. Moje matka má své chyby, ale nikdy jsem nepochyboval(a) o její lásce ke mně.	1	2	3	4	5
3. Být schopen si udržet přátele, je pro mne jednou z nejdůležitějších věcí.	1	2	3	4	5
4. Moji rodiče vždy pečovali o mé potřeby.	1	2	3	4	5
5. Moje cítění sebe sama se dramaticky mění.	1	2	3	4	5
6. Je nemožné mé rodiče vždy milovat.	1	2	3	4	5
7. Odlišné části mé osobnosti je obtížné složit dohromady.	1	2	3	4	5
8. Moje pocity o mé matce se mění ze dne na den.	1	2	3	4	5
9. Moji rodiče pro mne udělali to nejlepší co mohli.	1	2	3	4	5
10. Mám pochybnosti o mých nejbližších přátelích.	1	2	3	4	5
11. Občas si nejsem jist kdo jsem.	1	2	3	4	5
12. Moje pocity o sobě jsou velmi silné, ale mohou se měnit od jednoho okamžiku k druhému.	1	2	3	4	5
13. Mé přátelské vztahy jsou téměř vždy uspokojivé.	1	2	3	4	5
14. Moje pocity o sobě se nemění snadno.	1	2	3	4	5
15. Měl jsem mnoho dlouhodobých přátelství.	1	2	3	4	5
16. Občas se cítím rozdělen(a) mými pocity o sobě.	1	2	3	4	5
17. Mé vztahy s rodinou jsou pevné.	1	2	3	4	5
18. Mé vztahy vůči mým blízkým zůstávají neměnné.	1	2	3	4	5
19. Byl(a) jsem si vždycky vědom(a), že moji blízcí přátelé se o mne opravdu starali.	1	2	3	4	5
20. Mé mínění o mých přátelích se zřídka mění.	1	2	3	4	5
21. Téměř vždy pociťuji jako dobré ty, kteří jsou mi blízcí.	1	2	3	4	5
22. Mám extrémně smíšené pocity o mojí matce.	1	2	3	4	5
23. Má rodina mne často zraňovala.	1	2	3	4	5
24. Kdo jsem záleží na tom jak se cítím.	1	2	3	4	5

3.2. DISSOCIATIVE EXPERIENCE SCALE - DES

DES

Jméno a příjmení..... Rodinný stav.....Věk.....

Zaměstnání..... Vzdělání.....

Pokyny:
Tento dotazník obsahuje 28 otázek, jež se týkají zkušeností, které se mohou vyskytovat ve vašem každodenním životě. Zajímá nás, jak často se Vám tyto události stávají. Je však důležité, aby Vaše odpovědi ukázaly, jak často tyto zkušenosti prožíváte, aniž jste pod vlivem alkoholu nebo drog. K tomu, abyste mohli odpovědět na otázku, je nutné, abyste vyjádřili odpovídající stupeň zkušenosti vyjádřené v otázce ve vztahu k sobě a vyznačili jej vertikální čarou na příslušném místě, jak je ukázáno na příkladu.
Příklad:
0% |——————————/——————————| 100%

1. Někteří lidé mají zkušenost, že si při řízení auta náhle uvědomí, že si nemohou vzpomenout na to, co se událo v průběhu celého výletu nebo jeho části. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————| 100%
2. Někteří lidé občas shledají, že si při poslechu něčí řeči náhle uvědomí, že neslyšeli část nebo vůbec nic z toho, co bylo řečeno. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————| 100%
3. Někteří lidé mají zkušenost v tom, že shledají sebe sama na nějakém místě a nevědí, jak se tam dostali . Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————| 100%
4. Někteří lidé mají zkušenost s tím, že naleznou sebe sama oblečené v oděvu a nevzpomínají si, že se oblékali. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————| 100%
5. Někteří lidé mají zkušenost, že naleznou nové věci mezi těmi jež vlastní a nemohou si vzpomenout, že je kupovali. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————| 100%
6. Někteří lidé občas shledají, že se setkají s lidmi, které neznají a kteří je nazývají jiným jménem a trvají na tom, že se spolu již setkali. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————| 100%
7. Někteří lidé mají občas zkušenost, že cítí, jakoby stáli vedle někoho, nebo hledíce na sebe sama něco dělají a vidí sebe sama, jakoby hleděli na jinou osobu. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————| 100%
8. Někteří lidé říkají, že občas nepoznávají přátele nebo členy rodiny. Vyznačte čarou, v jakém procentu času se to stává Vám.
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9. Někteří lidé někdy shledají, že si nevzpomínají na důležité události ve svém životě [například svatba, promoce, maturita a podobně].Vyznačte čarou, v jakém procentu času se to stává Vám.
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10.Někteří lidé mají zkušenost s tím, že jsou obviňováni ze lhaní, aniž by lhali. Vyznačte čarou, v jakém procentu času se to stává Vám.
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11. Někteří lidé mají zkušenost, že hledí do zrcadla a nepoznávají sami sebe. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————| 100%
12. Někteří lidé mají občas zkušenost s tím, že cítí, že jiní lidé, věci nebo svět kolem nich nejsou reálné. Vyznačte čarou, v jakém procentu času se to stává Vám.
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13. Někteří lidé mají občas zkušenost s tím, že cítí, jakoby jim jejich tělo nenáleželo. Vyznačte čarou, v jakém procentu času se to stává Vám.
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14. Někteří lidé mají zkušenost, že si občas vzpomenou na nějakou minulou událost, tak živě, že cítí, jakoby tuto událost znovu prožili.Vyznačte čarou, v jakém procentu času se to stává Vám.
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15. Někteří lidé mají zkušenost s tím, že si nejsou jisti, zda události, na něž si vzpomínají, se opravdu staly, nebo si je jen vysnili.Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%
16. Někteří lidé mají zkušenost s tím, že se octnou na známém místě, které jim připadá zvláštní a neznámé.
Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%
17. Některým lidem se stává, že když hledí na televizi nebo film, jsou tak pohlceni příběhem, že si nejsou vědomi ostatních událostí kolem nich.Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%
18. Některým lidem se občas stává, že jsou tak pohlceni fantazií nebo denním snem, že pociťují, jakoby se jim to opravdu stalo. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%
19. Některým lidem se stává, že jsou občas schopni ignorovat bolest. Vyznačte čarou, v jakém procentu času se to stává Vám.
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20. Některým lidem se stává, že občas sedí a upřeně hledí před sebe, o ničem nepřemýšlí a nejsou si vědomi uplynulého času. Vyznačte čarou, v jakém procentu času se to stává Vám.
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21. Některým lidem se občas stává, že když jsou sami, hovoří nahlas sami se sebou.
Vyznačte čarou, v jakém procentu času se to stává Vám.
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22. Někteří lidé shledávají, že v některé situaci jednají tak odlišně ve srovnání s jinou, že se cítí téměř tak, jakoby byli dvěma různými lidmi. Vyznačte čarou, v jakém procentu času se to stává Vám.
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23. Některým lidem se občas stává, že v některých situacích jsou schopni vykonávat věci, které jsou pro ně obvykle obtížné s úžasnou lehkostí a spontaneitou [například sport, práce, sociální situace]. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%

24. Někteří lidé si občas nemohou vzpomenout, zda-li něco udělali, neboť mají jen myšlenku o tom, že tu věc udělali [například nevědí, zda-li poslali dopis, nebo si jen myslí, že jej poslali]. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%
25. Někteří lidé někdy shledají, že udělali věci, na něž si nemohou vzpomenout, že je dělali. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%
26. Někteří lidé občas naleznou zápisky, kresby, nebo poznámky, mezi těmi jež jim náleží, které museli sami učinit, ale nemohou si vzpomenout kdy.Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%
27. Některým lidem se občas stává, že slyší hlasy uvnitř své hlavy, které jim říkají co mají dělat, nebo komentují to, co dělají. Vyznačte čarou, v jakém procentu času se to stává Vám
0% |——————————————————————————————| 100%
28. Někteří lidé občas pociťují, jako když hledí na svět skrze mlhu, takže lidé a objekty se jim jeví být vzdálenými a nejasnými. Vyznačte čarou, v jakém procentu času se to stává Vám.
0% |——————————————————————————————| 100%

3.3. HEALTH OF THE NATIONS - HONOS

HoNOS:
Hodnocení zdravotního stavu

Přehled pokynů k hodnocení

1. Ohodnoťte každou položku v pořadí od 1 do 12.
2. Neberte v úvahu informace hodnocené v předešlých položkách kromě položky 10, která představuje celkové hodnocení.
3. Hodnoťte NEJZÁVAŽNĚJŠÍ problém, který se vyskytl během hodnoceného období.
4. Všechny položky jsou hodnocené na škále, která má tuto podobu:
   0 = žádný problém
   1 = malý problém, který nevyžaduje žádné opatření
   2 = mírný problém, jehož existence je však zřejmá
   3 = středně vážný problém
   4 = vážný až velmi vážný problém

   Pokud není známo, hodnoťte jako 9.

   Položky zaškrtněte zvlášť na záznamovém archu.

Glosář - HoNOS

1. Nadměrně aktivní, agresivní, rušivé nebo agitované chování

- *Patří sem tento typ chování, ať už vznikl z jakékoliv příčiny (např. drogy, alkohol, demence, psychóza, deprese atd.).*
- *Nepatří sem bizarní chování hodnocené v položce 6.*

0 Žádný problém tohoto typu během hodnoceného období.
1 Podrážděnost, hádky, neklid apod., nejsou třeba žádná opatření.
2 Patří sem agresivní posunky, omezování a obtěžování ostatních, výhružky nebo verbální útok; drobné škody na majetku (např. rozbitý šálek nebo okno); zřetelná hyperaktivita nebo rozrušení.
3 Fyzická agresivita vůči lidem nebo zvířatům (v menší míře než vyjadřuje hodnocení 4); výhružné chování; závažnější hyperaktivita nebo poškozování majetku.
4 Nejméně jeden případ vážného fyzického napadení lidí nebo zvířat; ničení majetku (např. zakládání požárů); vážné hrozby nebo obscénní chování.

2. Úmyslné sebepoškozování

- *Nepatří sem neúmyslné sebepoškození (v důsledku např. demence nebo těžkého mentálního postižení); kognitivní potíže se hodnotí v položce 4 a poranění v položce 5.*
- *Nepatří sem nemoc nebo poranění jako přímý důsledek užívání drog nebo alkoholu, které se hodnotí na škále 3 (např. cirhóza jater nebo úraz jako následek řízení v opilosti se hodnotí v položce 5).*

0 Žádný problém tohoto typu během hodnoceného období.
1 Občasné myšlenky na skoncování se vším, avšak malé riziko během období hodnocení; žádné sebepoškození.
2 Mírné riziko během hodnoceného období; zahrnuje sebepoškozování, které není nebezpečné (např. povrchové pořezání zápěstí).
3 Střední až vážné riziko úmyslného sebepoškození během hodnoceného období; patří sem přípravná fáze (např. shromažďování tablet).
4 Vážný sebevražedný pokus nebo vážné úmyslné sebepoškození během hodnoceného období.

3. Problémy s alkoholem nebo užívání drog

- *Nepatří sem agresivní nebo destruktivní chování v důsledku užívání alkoholu nebo drog, které se hodnotí v položce 1.*
- *Nepatří sem tělesná nemoc nebo nezpůsobilost v důsledku užívání alkoholu nebo drog, která se hodnotí v položce 5.*

0 Žádný problém tohoto typu během hodnoceného období (případně občasná mírná konzumace alkoholu).
1 Občasná nadměrná konzumace, ale v rámci sociálních norem.
2 Ztráta sebekontroly v důsledku užívání alkoholu nebo drog, ale ne vážná závislost.
3 Zřetelná touha nebo závislost na alkoholu nebo drogách s častou ztrátou kontroly, rizikové chování pod vlivem alkoholu nebo drog.
4 Ztráta schopnosti normálně fungovat v důsledku problémů s alkoholem nebo drogami.

4. Kognitivní problémy

- *Patří sem problémy s pamětí, orientací a chápáním spojené s jakoukoli poruchou: mentální retardací, demencí, schizofrenií atd.*
- *Nepatří sem přechodné problémy (např. kocovina), které jsou důsledkem užívání drog nebo alkoholu a hodnotí se v položce 3.*

0 Žádný problém tohoto typu během hodnoceného období.
1 Malé problémy s pamětí nebo chápáním (např. občas zapomíná jména).
2 Mírný, ale zjevný problém (např. zabloudil na známém místě nebo nepoznal známou osobu); občas má potíže činit jednoduchá rozhodnutí.
3 Zřetelná dezorientace v čase, prostoru nebo osobě; zmatený z běžných událostí; řeč je občas nesouvislá; duševně zpomalený.
4 Vážná dezorientace (např. není schopen poznat příbuzné); nebezpečí úrazů; nesrozumitelná řeč; zastřené vědomí, stupor.

5. Tělesná nemoc nebo postižení

- *Patří sem nemoc nebo postižení z jakékoli příčiny, která omezuje nebo znemožňuje pohyb, nebo zhoršuje zrak nebo sluch, nebo jinak zasahuje do běžného života.*
- *Patří sem vedlejší účinky léků; účinky drog nebo alkoholu;tělesné postižení zapříčiněné úrazy nebo sebepoškozením souvisejícím s kognitivními problémy, řízení v opilosti atd.*
- *Nepatří sem duševní poruchy nebo poruchy chování hodnocené v položce 4.*

0 Žádný tělesný zdravotní problém během hodnoceného období.
1 Malé zdravotní problémy během hodnoceného období (např. nachlazení, pád bez závažných důsledků atd.).
2 Tělesný zdravotní problém způsobující mírné omezení pohyblivosti a aktivity.
3 Střední stupeň omezení aktivity v důsledku tělesného zdravotního problému.
4 Vážné nebo úplné zneschopnění (ztráta schopnosti normálně fungovat) v důsledku tělesného zdravotního problému.

6. Problémy spojené s halucinacemi a bludy

- *Patří sem halucinace a bludy bez ohledu na diagnózu.*
- *Patří sem zvláštní a bizarní chování spojené s halucinacemi a bludy.*
- *Nepatří sem agresivní, destruktivní nebo hyperaktivní chování, které lze přičíst halucinacím nebo bludům a které se hodnotí v položce 1.*

0 Žádné známky halucinací nebo bludů během hodnoceného období.
1 Určité zvláštní nebo výstřední představy, které nejsou v souladu s kulturními normami.
2 Bludy nebo halucinace (např. hlasy, vidiny) jsou přítomné,vedou však jen k mírnému znepokojení pacienta nebo mírným projevům netypického chování; tj. jsou klinicky přítomné, avšak mírné.
3 Zřetelná zaujatost bludy nebo halucinacemi, která způsobuje velké znepokojení nebo se projevuje zřejmým bizarním chováním, tj. středně vážný klinický problém.
4 Duševní stav a chování je vážně a nepříznivě ovlivněno bludy nebo halucinacemi, s vážným dopadem na pacienta.

7. Problémy s depresivní náladou

- *Nepatří sem nadměrná aktivita nebo neklid, které se hodnotí v položce 1.*
- *Nepatří sem sebevražedné myšlenky nebo pokusy, které se hodnotí v položce 2.*
- *Nepatří sem bludy nebo halucinace, které se hodnotí na škále 6.*

Žádný problém spojený s depresivní náladou během hodnoceného období.
1 Sklíčenost nebo malé změny nálady.
2 Mírná, avšak zjevná deprese a úzkost (např. pocity viny; ztráta sebedůvěry).
3 Deprese s nepřiměřeným sebeobviňováním; nadměrně se zabývá pocity viny.
4 Těžká nebo velmi těžká deprese s pocity viny nebo sebeobviňováním.

8. Jiné psychické problémy a poruchy chování

- *Hodnoťte pouze nejzávažnější klinický problém, který není zohledněn v položkách 6 a 7.*
- *Upřesněte typ problému vyplněním příslušného písmene: A fobický; B úzkostný; C obsedantně kompulzivní; D duševní napětí, tenze; E disociativní; F somatoformní; G příjem potravy; H spánek; I sexuální; J jiné – upřesněte.*

0 Žádné známky jakéhokoli z těchto problémů během hodnoceného období.
1 Pouze malé problémy.
2 Problém je klinicky přítomen v nízké míře (např. pacient má nad ním určitý stupeň kontroly)
3 Občasný prudký záchvat nebo zhoršení stavu se ztrátou kontroly (např. musí se vyhýbat situacím způsobujícím úzkost, zavolat souseda na pomoc atd.); tj. středně závažný problém.
4 Závažný problém, který výrazně ovlivňuje většinu činností.

9. Problémy se vztahy

- *Hodnoťte pacientův nejzávažnější problém spojený se stažením se ze sociálních vztahů nebo problém s nepřínosnými, destruktivními nebo sebepoškozujícími vztahy.*

0 Žádný významný problém během hodnoceného období.
1 Malý problém, nemá klinickou povahu.
2 Určitý problém v navazování nebo udržování podpůrných vztahů, pacient si stěžuje nebo jsou problémy zjevné pro okolí.
3 Přetrvávající závažné problémy kvůli aktivnímu nebo pasivnímu vyhýbání se sociálním vztahům nebo kvůli vztahům, které neposkytují dostatečné uspokojení nebo podporu.
4 Závažná a těžce snášená sociální izolace kvůli neschopnosti komunikovat s lidmi nebo kvůli vyhýbání se sociálním vztahům.

10. Problémy s každodenními činnostmi

- *Hodnoťte celkovou úroveň provádění každodenních činností (např. problémy se základními činnostmi sebeobsluhy jako je jídlo, umývání, oblékání, chození na toaletu; rovněž komplexní dovednosti, jako je vycházení s penězi, hledání bydlení, zaměstnání a rekreace, mobilita a používání dopravních prostředků, nakupování, vlastní rozvoj atd.).*

- *Patří sem jakýkoli nedostatek motivace využívat příležitostí pomoci si vlastními silami,což přispívá k celkově nižší úrovni.*
- *Nepatří sem nedostatek příležitostí k procvičování neporušených schopností a dovedností, což se hodnotí v položce 11 a 12.*

0 Žádný problém během hodnoceného období; dobrá schopnost fungovat ve všech oblastech.
1 Pouze malé problémy (např. nepořádnost, špatná organizace).
2 Přiměřená péče o sebe, ale vážný nedostatek ve vykonávání jedné nebo více komplexních dovedností *(viz výše)*.
3 Větší problém v jedné nebo více oblastech péče o sebe (jídlo, umývání, oblékání, používání toalety), stejně jako větší neschopnost vykonávat několik komplexních dovedností.
4 Závažná neschopnost nebo nezpůsobilost ve všech nebo téměř ve všech oblastech péče o sebe a komplexních dovednostech.

11. Problémy s podmínkami bydlení

- *Hodnoťte celkovou závažnost problémů s kvalitou podmínek bydlení a běžným každodenním životem.*
- *Jsou splněny základní životní potřeby (teplo, světlo, hygiena)? Pokud ano, je k dispozici pomoc, aby se pacient vyrovnal se svým postižením a má nabídku možností, jak využívat své schopnosti a rozvíjet nové?*
- *Nehodnoťte míru neschopnosti provádět činnosti, která se hodnotí v položce 10.*

Pozn.: Hodnoťte prostředí, ve kterém je pacient obvykle ubytován. Pokud je na akutním oddělení, hodnoťte jeho bydlení doma. Pokud nejsou informace k dispozici, hodnoťte jako 9.

0 Bydlení a životní podmínky jsou přijatelné; v těchto podmínkách mají problémy hodnocené v položce 10 minimální dopad, podmínky podporují svépomoc.
1 Bydlení je celkem přijatelné, přestože existují menší nebo přechodné problémy (např. umístění bytu není ideální, přál si něco jiného, nechutná mu strava atd.).
2 Výrazný problém s jedním nebo více aspekty ubytování nebo režimem (např. omezené rozhodování; personál nebo členové domácnosti neví, jak zlepšit soběstačnost.
3 Zatěžující četné problémy s bydlením (např. chybí základní životní nezbytnosti); domácí prostředí má minimální nebo žádné vybavení pro zvýšení pacientovy nezávislosti.
4 Ubytování je nepřijatelné (např. nedostatek základních životních nezbytností, pacientovi hrozí vystěhování nebo nemá „střechu nad hlavou" nebo jsou životní podmínky jiným způsobem nesnesitelné), což zhoršuje pacientovy problémy.

12. Problémy se zapojením se a dalšími činnostmi

- *Hodnoťte celkovou míru problémů s kvalitou denního prostředí. Existuje pomoc, aby se pacient vyrovnal se svým postižením a* příležitosti k udržení nebo zlepšení pracovních a rekreačních schopností a činností? *Zohledněte faktory, jako je stigma, nedostatek kvalifikovaného personálu, přístup k podpůrným zařízením (např. personální zajištění a vybavení denních center, dílny, společenské kluby atd.).*
- *Nehodnoťte míru neschopnosti provádět činnosti, která se hodnotí v položce 10.*

Pozn.: Hodnoťte pacientovu obvyklou situaci. Pokud je na akutním oddělení, hodnoťte jeho činnosti během období před přijetím. Pokud nejsou informace k dispozici, hodnoťte jako 9.

0 Pacientovo denní prostředí je přijatelné; v tomto prostředí mají problémy hodnocené v položce 10 minimální dopad, prostředí podporuje svépomoc.
1 Drobné nebo dočasné problémy (např. dostává pozdě dávky); k dispozici jsou přiměřená zařízení, ne však vždy v požadovanou dobu atd.
2 Omezený výběr aktivit; nedostatek přiměřené tolerance (např. nespravedlivě odmítnutý přístup do veřejné knihovny nebo bazénu atd.); znevýhodněn, protože nemá trvalé bydliště nebo nedostatečná pečovatelská služba nebo profesionální podpora, nebo je vhodné denní zařízení k dispozici, avšak pouze na velmi omezený čas.
3 Zřetelný nedostatek odborných služeb, které by pomohly minimalizovat úroveň stávající nezpůsobilosti; žádné příležitosti k využití neporušených schopností nebo získání nových; obtížný přístup k laické pomoci.
4 Nedostatek jakýchkoli příležitostí k denním aktivitám zhoršuje pacientovy problémy.

HoNOS – základní údaje

Identifikace pacienta

Datum narození pacienta

Pohlaví pacienta
1 muž
2 žena

Základní diagnóza pacienta

1 demence
2 abusus drog nebo alkoholu
3 psychóza
4 deprese, mánie nebo bipolární porucha
5 neurotická nebo úzkostná porucha
6 porucha příjmu potravy, porucha spánku nebo stresová porucha
7 porucha osobnosti
8 jiné

Zařízení, v němž bylo hodnocení provedeno:
1 akutní oddělení
2 dlouhodobý pobyt (včetně vězeňského oddělení)
3 denní sanatorium
4 denní stacionář
5 ambulantní oddělení
6 doma
7 jinde

Dnešní datum

Datum poslední kontroly pacienta

Délka hodnoceného období (v týdnech)

Profese osoby provádějící hodnocení

1 zdravotní sestra (v lůžkovém oddělení nebo v denním stacionáři)
2 komunitní psychiatrická sestra
3 lékař
4 pracovní terapeut
5 klinický psycholog
6 sociální pracovník
7 jiné

Iniciály osoby provádějící hodnocení

HoNOS – Záznam hodnocení

Identifikace pacienta ……

Dnešní datum ……

Pokud není známo, hodnoťte jako 9

1. Hyperaktivní, agresivní, rušivé nebo agitované chování	0 1 2 3 4
2. Úmyslné sebepoškozování	0 1 2 3 4
3. Problémy s alkoholem nebo užívání drog	0 1 2 3 4
4. Kognitivní problémy	0 1 2 3 4
5. Tělesná nemoc nebo postižení	0 1 2 3 4
6. Problémy spojené s halucinacemi a bludy	0 1 2 3 4
7. Problémy s depresivní náladou	0 1 2 3 4
8. Jiné psychické problémy a poruchy chování; určete poruchu A, B, C, D, E, F, G, H, I nebo J	0 1 2 3 4
9.Problémy se vztahy	0 1 2 3 4
10.Problémy s každodenními činnostmi	0 1 2 3 4
11.Problémy s podmínkami bydlení	0 1 2 3 4
12.Problémy se zaměstnáním a dalšími činnostmi	0 1 2 3 4

Zdravotní dotazník

Tento dotazník pomáhá v plánování a hodnocení Vaší léčby. Je důvěrný, takže na něj nepište jméno. Zodpovězte prosím všechny dotazy.

Pokyny
Přečtěte si postupně všechny části (od A do L) a uvažujte přitom o posledních čtrnácti dnech. Zakroužkujte pak číslo u výroku, který se na Vás nejlépe hodí. Pokud se Vás určitá část netýká zakroužkujte "0".(stejné jako, když není problém).

Část A. Agresivní, destruktivní chování nebo projevy rozrušení
V posledních čtrnácti dnech jsem...
0 nebyl/a agresivní, destruktivní ani přehnaně aktivní
1 se občas pohádal/a nebo byl/a podrážděný/á, ale většinou jsem byl/a klidný/á
2 byl/a občas agresivní nebo jsem vyhrožoval/a (slovně či gesty) nebo jsem byl/a velmi podrážděný/á, případně jsem způsobil/a drobnou škodu, např. jsem něčím praštil/a o zeď
3 byl/a agresivní k ostatním nebo jsem jim hodně vyhrožoval/a nebo jsem ničil/a věci
4 nejméně jednou závažným způsobem fyzicky napadl/a člověka nebo zvíře nebo způsobil/a vážnou škodu na majetku (např. jsem něco zapálil/a) nebo někoho vyděsil/a nebo pohoršil/a, např. nemravným chováním

Část B. Pokus o sebevraždu nebo sebepoškození: úmyslné poranění
V posledních čtrnácti dnech jsem...
0 nemyslel/a na sebevraždu nebo že bych si ublížil/a
1 pomyslel/a jednou nebo dvakrát na sebevraždu nebo že bych si ublížil/a, ale neměl/a jsem v úmyslu to udělat
2 vážně přemýšlel/a o sebevraždě nebo že bych si ublížil/a nebo jsem udělal/a gesto, např.jsem se trochu poranila na zápěstí
3 byl/a velmi blízko k tomu si vážně ublížit, např. jsem shromáždil/a prášky
4 učinil/a vážný pokus o sebevraždu nebo jsem se úmyslně zranil/a

Část C. Problémy spojené s alkoholem nebo užíváním nelegálních drog
V posledních čtrnácti dnech jsem...
0 nepil/a alkohol (případně občasná mírná konzumace alkoholu) ani nebral/a drogy
1 občas nadměrně pil/a alkohol nebo užíval/a drogu, ale nezpůsobilo to žádné problémy
2 ztratil/a sebekontrolu v důsledku pití alkoholu nebo užívání drog a způsobilo mi to problémy
3 zjistil/a, že nemohu žít bez alkoholu nebo drog, kvůli kterým jsem ztratil/a sebekontrolu a dělal/a podobné věci jako je např. řízení v opilosti
4 měl/a pocit, že požívání alkoholu nebo drog je hlavní příčinou mých tělesných nebo duševních problémů a nejsem schopný/á s tím něco udělat

Část D. Problémy s pamětí, orientací a porozuměním (chápáním)
V posledních čtrnácti dnech jsem...
0 neměl/a žádné takové problémy
1 měl/a menší problémy jako je zapomínání jmen nebo kam jsem si něco položil/a
2 měl/a závažné problémy s pamětí, např. jsem zapomněl/a kudy jít na známé místo nebo jsem nepoznal/a někoho, koho dobře znám nebo pro mě bylo velmi těžké učinit i velmi jednoduchá rozhodnutí
3 měl/a problémy pochopit, co se okolo mne děje, běžné události mne uváděly ve zmatek; v některých chvílích ostatní lidé jen stěží rozuměli mé řeči
4 byl/a zcela dezorientován/a, nepoznával/a jsem příbuzné nebo moje řeč byla tak zmatená, že mně nikdo nerozuměl

Část E. Tělesná nemoc nebo postižení
V posledních čtrnácti dnech jsem...
0 neměl/a žádné tělesné zdravotní problémy
1 měl/a přechodný zdravotní problém, např. chřipku nebo vyrážku
2 měl/a tělesný zdravotní problém, který mi ztěžoval běžný život
3 měl/a tělesný zdravotní problém, který mne výrazně omezoval
4 měl/a tělesný zdravotní problém, který mi znemožňoval normálně žít nebo se o sebe postarat

Část F. Halucinace a bludy
V posledních čtrnácti dnech jsem...
0 neměl/a žádné takové problémy
1 se choval/a způsobem, který ostatní považovali za výstřední
2 slyšel/a hlasy nebo měl/a vidiny, ale nerozrušovaly mě ani mě k ničemu nenutily
3 často slyšel/a hlasy nebo jsem měl/a vidiny, které mě rozrušovaly nebo mě nutily něco udělat
4 velmi trpěl/a kvůli svým hlasům nebo vidinám a ostatní to také velmi trápilo

Část G. Skleslá nálada (deprese)
V posledních čtrnácti dnech jsem...
0 neměl/a žádný problém se smutnou nebo skleslou náladou
1 byl/a velmi smutný/á kvůli něčemu, co se stalo nebo se má stát
2 byl/a depresivní, cítil/a se špatně (sklesle), byl/a plačtivý/á, měl/a pocity viny, nedostatek sebeúcty, málo radosti ze života
3 byl/a velmi depresivní, měl/a pocity viny, žádnou radost ze života, ztratil/a jsem sebeúctu a chtělo se mi plakat po většinu času
4 byl/a tak skleslý/á, že jsem celý den proseděl/a, ztratil/a veškerou radost ze života, měl/a pocity viny, cítil/a se zbytečný/á; nemohl/a spát nebo jíst a po celou dobu se mi chtělo plakat

Část H. Jiné problémy
Např.: panika, fobie, obsese, anorexie, bulimie, problémy se spánkem nebo příznaky, které lékař nedokáže vysvětlit. (Pokud jste měl/a více než jeden takový problém, hodnoťte pouze ten, který byl pro Vás nejhorší.)
Napište zde, o jaký problém se jedná:

V posledních čtrnácti dnech jsem...
0 neměl/a žádné takové problémy (kromě těch, které jsou uvedeny v předchozích částech A-G)
1 měl/a menší nebo krátkodobé potíže
2 byl/a po určitou dobu postižen/a tímto problémem, ale byla také doba, kdy se problém neprojevoval a měl/a jsem tedy věci pod kontrolou
3 nemohl/a se zbavit tohoto problému, někdy byl velmi výrazný a hodně mě trápil, takže jsem ztrácel/a sebekontrolu a potřeboval/a pomoc, abych se s tím vypořádal/a
4 zjistil/a, že tento problém výrazně ovlivnil všechno, co dělám a učinil můj život beznadějný a obtížný

Část I. Problémy ve vztazích s lidmi – včetně stranění se ostatním nebo neschopnosti s nimi hovořit
V posledních čtrnácti dnech jsem...
0 neměl/a žádné problémy s ostatními lidmi
1 měl/a menší problém(y), ale celkem přijatelné, případně se objeví a zase zmizí
2 byl/a v pořádku, když se mnou lidé mluvili, ale nejsem schopný/á navázat přátelství, vycházet s lidmi nebo s nimi přátelství udržovat; někteří lidé o tom vědí
3 se vyhýbal/a lidem co nejvíce, nikdo se nepokoušel o nějaký kontakt se mnou nebo jsem ostatní od kontaktu odrazoval/a
4 byl/a sám/sama, protože nejsem schopný/á s lidmi mluvit a nesnažím se s nimi být v kontaktu nebo vůbec neumím vycházet s lidmi

Část J. Problémy se zvládáním běžných činností
V posledních čtrnácti dnech jsem...
0 neměl/a žádné problémy s jídlem, mytím, oblékáním, chozením na toaletu, ani vycházet s penězi, organizovat si věci, dostat se, kam jsem potřeboval/a, nakoupit si nebo příjemně prožít čas
1 měl/a nějaké malé potíže, např. neschopnost si věci zorganizovat, což mi bránilo příjemně prožít čas
2 měl/a menší potíže, např. vycházet s penězi či s nakupováním nebo příjemným prožíváním času, ale neměl/a jsem problémy s jídlem, mytím, oblékáním ani chozením na toaletu
3 měl/a určité potíže s jídlem, mytím, oblékáním, používáním toalety, případně jsem nebyl/a schopný/á si nakoupit nebo si věci zorganizovat
4 nebyl/a schopný/á se o sebe postarat s jídlem, mytím, oblékáním, chozením na toaletu, natož pak hospodařit s penězi, nakupovat si apod.

Část K. Problémy s místem, kde žiji (pokud jste v nemocnici jenom krátkodobě, hodnoťte svůj domov)
V posledních čtrnácti dnech jsem...
0 neměl/a problémy s místem, kde žiji; chci tam bydlet a líbí se mi tam
1 mám nějaké problémy s místem, kde žiji, ale celkově je to v pořádku
2 si myslel/a, že místo, kde žiji, je pěkné, ale některé věci jako cena za bydlení nebo lidé, se kterými žiji, mi ztěžují život
3 měl/a problémy jak s lidmi, se kterými žiji, tak s tím, jak to tam vypadá, např. je tam vlhko nebo zima, nedostatek světla nebo tam není pořádná koupelna či kuchyně; nejsem vůbec rád/a, že tam bydlím
4 neměl/a vůbec žádné bydlení nebo jsem dostal/a výpověď nebo se tam už nemohu vrátit, protože se tam nedá bydlet – kvůli spolubydlícím nebo kvůli tomu, jak to tam vypadá

Část L. Problémy se zapojením se nebo s činnostmi během dne
V posledních čtrnácti dnech jsem...
0 měl/a vše, co jsem potřeboval/a, abych mohl/a plně využít své situace
1 měl/a určité problémy např. s tím, že mi přišly pozdě peníze nebo že tam, kam chci chodit, není otevřeno v době, kdy to potřebuji
2 měl/a nějaké problémy, protože nemám stálé bydliště nebo nemám někoho, kdo by mi pomohl, když to potřebuji apod.
3 to jakž tak zvládal/a, ale nemám pečovatele nebo terapeuta a potřeboval/a bych v mnoha ohledech pomoc
4 neměl/a žádnou šanci se dostat z problémů a nikdo mi s tím nepomohl

Problémy, které Vám nejvíce zasahují do života včetně těch, které nemusely být v dotazníku uvedeny.
Uveďte prosím pět nejzávažnějších problémů, se kterými jste se potýkal/a v posledních čtrnácti dnech:
1

2

3

4

5

Se kterými z těchto problémů by Vám podle Vašeho názoru měla pomoci zdravotní nebo sociální péče?

Zkontrolujte prosím, že jste ve všech částech A-L zakroužkovali jedno číslo.

Děkujeme za vyplnění dotazníku.

3.4. TRAUMA SYMPTOMS CHECKLIST - TSC-40

TSC-40

Jméno a příjmení..... Rodinný stav.....

Zaměstnání..... Vzdělání.....

Jak často jste zažil[a] každou z následujících položek v posledních dvou měsících

	Nikdy			Často
1. Bolesti hlavy.	0	1	2	3
2. Nespavost [problém s usnutím].	0	1	2	3
3. Ztráta váhy [bez diety].	0	1	2	3
4. Žaludeční problémy.	0	1	2	3
5. Sexuální problémy.	0	1	2	3
6. Pocit izolovanosti od ostatních.	0	1	2	3
7. "Retrospektivy" [náhlé, živé zneklidňující vzpomínky].	0	1	2	3
8. Neklidný spánek.	0	1	2	3
9. Snížený zájem o sex.	0	1	2	3
10. Záchvaty úzkosti.	0	1	2	3
11. Zvýšený sexuální zájem.	0	1	2	3
12. Pocit osamělosti.	0	1	2	3
13. Noční můry.	0	1	2	3
14. "Úlety" [úniky ve vaší mysli].	0	1	2	3
15. Smutek.	0	1	2	3
16. Závrať.	0	1	2	3
17. Nespokojenost se sexuálním životem.	0	1	2	3
18. Obtížná kontrola nálady.	0	1	2	3
19. Probouzení se brzy ráno a nemožnost opět usnout.	0	1	2	3
20. Nekontrolovatelný pláč.	0	1	2	3
21. Strach z mužů.	0	1	2	3
22. Rána bez pocitů odpočinku.	0	1	2	3
23. Máte sex, který Vás netěší.	0	1	2	3
24. Potíže ve vycházení s druhými.	0	1	2	3
25. Problémy s pamětí.	0	1	2	3
26. Zájem o sebepoškozování.	0	1	2	3
27. Strach ze žen.	0	1	2	3
28. Probouzení o půlnoci.	0	1	2	3
29. Špatné myšlenky nebo pocity v průběhu sexu.	0	1	2	3
30. Odchody někam.	0	1	2	3
31. Pocity, že věci jsou "nereálné".	0	1	2	3
32. Nadbytečné nebo příliš časté mytí.	0	1	2	3
33. Pocity ponížení.	0	1	2	3
34. Trvalé pocity napětí.	0	1	2	3
35. Zmatenost pokud jde o pocity související se sexualitou.	0	1	2	3
36. Přání fyzicky poškozovat druhé.	0	1	2	3
37. Pocity viny.	0	1	2	3
38. Pocity, že nejste vždy ve vašem těle.	0	1	2	3
39. Máte potíže s dýcháním.	0	1	2	3
40. Sexuální pocity tam, kde si je nepřejete mít.	0	1	2	3

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6. PUBLISHED ARTICLES

OPEN ACCESS Freely available online

PLOS ONE

Splitting in Schizophrenia and Borderline Personality Disorder

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Abstract

Background: Splitting describes fragmentation of conscious experience that may occur in various psychiatric disorders. A purpose of this study is to examine relationships between psychological process of splitting and disturbed cognitive and affective functions in schizophrenia and borderline personality disorder (BPD).

Methods: In the clinical study, we have assessed 30 patients with schizophrenia and 35 patients with BPD. The symptoms of splitting were measured using self-reported Splitting Index (SI). As a measure of semantic memory disorganization we have used verbal fluency test. Other psychopathological symptoms were assessed using Health of the Nation Outcome Scale (HoNOS).

Results: Main results show that SI is significantly higher in BPD group than in schizophrenia, and on the other hand, verbal fluency is significantly lower in schizophrenia group. Psychopathological symptoms measured by HoNOS are significantly higher in the BPD group than in schizophrenia. Significant relationship was found between verbal fluency and the SI "factor of others" (Spearman $r = -0.52$, $p < 0.01$) in schizophrenia patients.

Conclusions: Processes of splitting are different in schizophrenia and BPD. In BPD patients splitting results to mental instability, whereas in schizophrenia the mental fragmentation leads to splitting of associations observed as lower scores of verbal fluency, which in principle is in agreement with Bleuler's historical concept of splitting in schizophrenia.

Citation: Pec O, Bob P, Raboch J (2014) Splitting in Schizophrenia and Borderline Personality Disorder. PLoS ONE 9(3): e91228. doi:10.1371/journal.pone.0091228

Editor: Marianna Mazza, Catholic University of Sacred Heart of Rome, Italy

Received December 17, 2013; **Accepted** February 9, 2014; **Published** March 6, 2014

Funding: The study was supported by the Charles University grant (PRVOUK, www.cuni.cz), project provided by Czech Ministry of Education (LH11033, www.msmst.cz) and the project "CEITEC – Central European Institute of Technology" (CZ.1.05/1.1.00/02.0068) from European Regional Development Fund (http://ec.europa.eu/regional_policy/). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The authors have declared that no competing interests exist.

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Introduction

Splitting reflects shifts of mind related to a consciously experienced conflict of opposing mental forces. In principle it describes fragmentation of conscious experience that is typically related to long-term or acute stress that significantly disturbs self-concept, identity, memory and perception of the external world [1–5]. Nevertheless, empirical studies of psychopathological processes related to splitting are very rare.

In schizophrenia the term splitting was developed by Bleuler [6], who described process of mental fragmentation in schizophrenia as associative splitting or "loosening of associations" and considered it as a basic factor in pathogenesis of the disease. Later concept of splitting was described by Kernberg [7], who used the process of splitting as a specific characteristic of cognitive and affective disturbances in borderline personality disorder (BPD) which typically manifest as shifts of emotional perception of objects, other persons and the self with typical fluctuations between idealization and devaluation.

These alterations on mental level consequently may be linked to great and abrupt changes in patterns of neural activity that may dissociate, or split off, certain external and internal stimuli and information out of awareness, which may lead to distinct states of divided consciousness [4,8–11] and disorganization of semantic memory [12–14].

With respect to recent findings a purpose of this study is to examine relationships between psychological process of splitting and disturbed cognitive and affective functions in schizophrenia and BPD.

Materials and Methods

Participants

The participants were recruited from regular daily outpatients treatment programs for schizophrenic and BPD patients at the Psychotherapeutic and Psychosomatic Clinic ESET in Prague. All participants signed informed consent and the study was approved by Charles University ethical committee. In the study were included only patients who had not compromised capacity and ability to consent. This ability was confirmed by clinical data about the patients and specific written statement regarding each participant by his/her psychiatrist. Each included participant was able to consider his/her participation and no one was

Table 1. Statistical comparison between schizophrenia and BPD patients using Mann-Whitney test.

	Schizophrenia N = 30	BPD N = 35	Z	p	R
SI	2.84	3.14	−2.2	0.0025	0.58
SI(S)	2.69	3.43	−2.8	0.0053	0.82
SI(F)	2.87	3.01	−1.0	0.3384	0.14
SI(O)	2.96	2.99	−0.2	0.8759	0.05
VF	34.5	41.43	−2.8	0.0052	0.79
HoNOS (E)	11.6	15	−2.6	0.0085	0.81
HoNOS (S)	7.8	14.25	−3.9	0.0000	0.97

Note: SI – Splitting Index, SI(S) – Splitting Index, factor of self, SI(F) – Splitting Index, factor of family, SI(O) – Splitting Index, factor of others, VF- verbal fluency, HoNOS(E)-version for external evaluation of HoNOS (mean), HoNOS(S) - self-reported version of HoNOS (mean), BPD – borderline personality disorder, Z- Z value of Mann-Whitney test, r- standardized effect size.

doi:10.1371/journal.pone.0091228.t001

included in the study based on agreement of legally authorized representative consented on the behalf of a participant.

The participants had diagnosis of schizophrenia or borderline personality disorder. Exclusion criteria were organic illnesses involving the central nervous system, substance, and/or alcohol abuse and mental retardation (IQ Raven lower than 90) [15]. Clinical diagnosis was reassessed using the Mini-International Neuropsychiatric Interview (M.I.N.I.) [16] in schizophrenia patients and in BPD patients it was confirmed using semi-structured interview for borderline personality disorder based on DSM-IV criteria. The sample included 30 patients with schizophrenia, i.e. 15 men and 15 women, mean age 35.7 (SD = 9.2) with mean period of psychiatric treatment 12.89 (SD = 7.8 years) and with average of 4.1 hospitalizations. The sample of BPD patients included 35 participants, i.e. 10 men and 25 women, mean age 32.0 (SD = 7.9) years with mean period of psychiatric treatment 6.2 (SD = 3.97) years and with average of 2.28 hospitalizations.

Psychometric Measures

With respect to current theoretical concepts and empirical data we have tested relationship between splitting based on Splitting Index score [17] and verbal fluency as an indicator of semantic memory disorganization [12–13,18] in patients with schizophrenia and BPD. To test how the splitting process is typically represented in schizophrenia and BPD we have compared occurrence of these psychopathological manifestations in schizophrenia and BPD and their relationships to other symptoms.

The symptoms of splitting were measured using self-reported Splitting index (SI) [18] that enables to assess defense mechanisms related to splitting according to concept proposed by Kernberg [7]. Splitting Index is 24-items self-reported questionnaire rated on 5-point Likert scale from 1 to 5 (Cronbach's alfa 0.92, test-retest reliability after one week 0.82). Using factor analysis three clusters of items have been identified that enable to describe the splitting process. These three factors represent: 1. the self factor (splitting of the self image), 2. the family factor (splitting of images of family members), and 3. the factor of others which describes splitting with respect to people outside the family.

Other psychopathological manifestations in both groups of patients were measured using Health of the Nation Outcome Scales (HoNOS) [19]. The scale includes 12 items including overactive, aggressive, disruptive or agitated behavior; non-accidental self-injury; problem drinking or drug-taking; cognitive problems; physical illness or disability problems; hallucinations or delusions; problems with depressed mood; other mental and behavioral problems; problems with relationships; problems with activities of daily living; problems with living conditions; problems with occupation and activities. This scale includes two versions, i.e. the version for external evaluators and the self-reported version (Cronbach's alfa 0.79, test-retest reliability after one week 0.85) [20].

As a measure of semantic memory disorganization, which is very close to Bleuler's concept of mental fragmentation, we have used verbal fluency test [12–13,21]. In this context, recent findings show that verbal fluency is severely disturbed in schizophrenia [22] and it is closely related to disorganized dimension of psychopathology in schizophrenic patients [14].

Data Analysis

Statistical evaluation of the results of SI and other psychometric measures included descriptive statistics, Mann-Whitney test for independent samples and Spearman correlation coefficients. The non-parametric analyses were preferred because SI data have not normal distribution. All the methods of statistical evaluation were performed using the software package Statistica version 6. To prevent Type II error which would disable to reject null hypothesis that the measure of splitting is not linked to verbal fluency and psychopathological symptoms we performed Power Analysis and assessed the effect sizes characterizing differences between means and correlation coefficients.

Results

Results show significant differences in scores of splitting, verbal fluency and psychopathological symptoms measured by HoNOS between BPD and schizophrenia groups that were compared using Man-Whitney test (Table 1). Mean score of the Splitting Index (SI) was significantly higher in BPD group than in schizophrenia.

On the other hand score of verbal fluency was significantly lower in schizophrenia group. In both assessments of HoNOS, for external evaluators and for self evaluation, the BPD group scored significantly higher in means of total scores. In the power analysis we have tested significant differences which show that all differences between means have strong effect size ($r = 0.5$ or higher; Table 1).

Results also show significant Spearman correlation coefficients characterizing relationships between splitting, verbal fluency and psychopathological symptoms measured by HoNOS in both samples (Table 2). Very significant relationship between verbal fluency and the SI "factor of others" in schizophrenia patients was found (Spearman $r = -0.52$, $p < 0.01$). Other significant correla-

Table 2. Spearman correlation coefficients between SI, verbal fluency, and HoNOS in schizophrenia and BPD patients.

	SI		SI-S		SI-F		SI-O		VF		HoNOS(E)	
	Sch	BPD	Sch	BPD	Sch	BPD	Sch	BPD	Sch	BPD	Sch	BPD
SI-S	0.76	0.68	–	–								
SI-F	0.72	0.53	0.40	0,05	–	–						
SI-O	0.73	0.47	0.27	0.01	0.59	0.09	–	–				
VF	−0.31	0.15	0.02	0.19	−0,23	0.05	−0.52	−0.05	–	–		
HoNOS(E)	0.26	0.13	0.28	0.33	0.21	0.14	0.09	−0.07	−0.22	0.15	–	–
HoNOS(S)	0.42	0.26	0.63	0.45	0.14	0,10	0.10	−0.10	0.16	0.37	0.48	0.70

Note: Values of Spearman correlation coefficients significant at $p<0.05$ are in bold (0.45 or higher are significant at $p<0.01$); Fisher Z was higher than 0.05; Sch – schizophrenia; BPD – borderline personality disorder; SI – Splitting Index; SI(S) – Splitting Index, factor of self; SI(F) – Splitting Index, factor of family; SI(O) – Splitting Index, factor of others; VF: verbal fluency; HoNOS(E): version for external evaluation; HoNOS(S): self-reported version.

doi:10.1371/journal.pone.0091228.t002

tions in schizophrenia patients were found between self-reported score of HoNOS(S) and total score of splitting (SI) (Spearman $r=0.42$, $p<0.05$) and between HoNOS(S) and SI(S) [representing splitting of the self] (Spearman $r=0.63$, $p<0.01$). On the other hand significant correlations in borderline personality disorder were found between and between HoNOS(S) and SI(S) [representing splitting of the self] (Spearman $r=0.45$, $p<0.01$) and HoNOS self-reported score and verbal fluency in has been found (Spearman $r=0.37$, $p<0.01$).

Discussion

Main results of this study indicate significant differences in splitting, verbal fluency and psychopathological symptoms between schizophrenia and BPD patients. These findings show significantly higher level of splitting measured by SI in BPD patients compared to schizophrenia. On the other hand schizophrenia patients show significantly lower scores of verbal fluency most likely as a consequence of cognitive disorganization which in principle is in agreement with Bleuler's historical concept of splitting in schizophrenia [6]. In this context, the correlation between verbal fluency and splitting (factor of others) in schizophrenia suggests that stronger levels of splitting into opposite aspects related to external objects and persons is related to dissociation of memory patterns that is manifested as disturbed verbal fluency.

Recent findings also show that impaired verbal fluency is associated with psychomotor slowness [23–24] that might be related to disconnection between brain regions [24]. The disconnection between brain regions also disables integrated response to emotional stimuli, which might be linked to specific differences in amygdala activity and prefrontal functions in schizophrenia and BPD [25]. Schizophrenia is typically characterized by reduced activation in amygdala and prefrontal cortex and on the other hand increased and excessive activation in amygdala and prefrontal cortex has been found during emotional tasks in BPD [25–29].

These typical neurophysiological changes might reflect typical differences related to splitting in schizophrenia and BPD based on psychological mechanisms of defense against unacceptable affective impulses [30]. Responses to these impulses in BPD likely reflect disturbed levels of reality testing in response to various perceptual stimuli that typically result to increased and excessive emotional activation and irritability [3,31–32], which on neurophysiological level could be reflected in increased prefrontal and amygdala activation [25]. On the other hand disturbed verbal fluency in schizophrenia patients is likely related to decreased activity in amygdala and prefrontal activity in schizophrenia [25–27], which might reflect inability to appropriately differentiate and reflect emotional stimuli [33–34] likely due to disruption in attentional selection and decision related activation [35].

In summary, the results show that the process of splitting has different forms in schizophrenia and BPD. In BPD patients splitting results to mental instability manifested as shifts in emotional perception of objects, other persons and the self, which are linked to increased mental tension and excessive prefrontal and amygdala activation. This specific form of splitting that occur in BPD is not typically present in schizophrenic patients, which is in agreement with the results indicating that SI score as a measure of borderline splitting is higher in BPD than in schizophrenia patients. On the other hand in schizophrenia the mental fragmentation leads to splitting of associations observed as lower scores of verbal fluency which in principle is in agreement with Bleuler's historical concept of splitting in schizophrenia [35]. This

form of mental fragmentation in schizophrenia may represent a defense mechanism decreasing several psychopathological manifestations due to lowered mental tension and abnormally inhibited brain activities in amygdala and prefrontal cortices. Nevertheless it is also possible that mental fragmentation in schizophrenic patients is also related to deficits in contextual processing that may be primarily based on brain's ability to integrate information [35,36]. This brain deficit to integrate information may be linked to various etiological conditions reflecting pathological processes on molecular, physiological and psychological levels. This brain potentiality to integrate information is on cognitive level specifically linked to ability to create integrated self-concept and synthetic capabilities related to various forms of metacognitive deficits that is typical impaired in schizophrenia [37–43].

Author Contributions

Conceived and designed the experiments: OP PB JR. Performed the experiments: OP PB. Analyzed the data: OP PB JR. Contributed reagents/materials/analysis tools: OP PB JR. Wrote the paper: OP PB.

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Neuropsychiatric Disease and Treatment

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SHORT REPORT

Dissociation in schizophrenia and borderline personality disorder

This article was published in the following Dove Press journal:
Neuropsychiatric Disease and Treatment
17 March 2014
Number of times this article has been viewed

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Background: Dissociation likely plays a key role in schizophrenia and borderline personality disorder (BPD), although empirical studies that compare specific manifestations of these symptoms in schizophrenia and BPD are rare. In this context, the purpose of this study was to compare the occurrence of dissociative and other psychopathological symptoms in these disorders, and to assess the possible influence of antipsychotic medication on the dissociative symptoms.

Methods: We assessed 31 patients with schizophrenia and 36 patients with BPD. Dissociative symptoms were measured by the Dissociative Experiences Scale (DES), symptoms related to stress and traumatic experiences were assessed using the Trauma Symptom Checklist-40 (TSC-40), and other psychopathological symptoms were measured with the Health of the Nation Outcome Scales (HoNOS). We also assessed actual daily doses of antipsychotic medication in chlorpromazine equivalents in all participants.

Results: The results show that symptoms of traumatic stress measured by the TSC-40 had significantly higher scores in the BPD group. The data also show that dissociative symptoms (DES) were significantly correlated with symptoms of traumatic stress (TSC-40) and with symptoms assessed by the HoNOS. Remarkably significant correlations were found between levels of antipsychotic medication and the DES and between antipsychotic medication and the depersonalization/derealization component of the DES in BPD patients.

Conclusion: The results support an important role of dissociative processes in schizophrenia and BPD and suggest a significant relationship between manifestations of dissociative symptoms in BPD and antipsychotic medication.

Keywords: dissociation, stress-related symptoms, schizophrenia, borderline personality disorder, antipsychotic medication

Introduction

Dissociation in principle describes fragmentation of conscious experience that is typically related to long-term or acute stress that significantly disturbs self-concept, identity, memory, and perception of the external world.[1–4] Consequently, these alterations may be linked to marked and abrupt changes in patterns of neural activity that may dissociate, or split off, certain external and internal stimuli and information out of awareness, which may lead to distinct states of divided consciousness.[3,5–8]

Dissociation also reflects shifts of mind related to a consciously experienced conflict of opposing mental forces. In the similar context as Janet, also Bleuler coined the term splitting and described the process of mental fragmentation in schizophrenia as a basic step in the pathogenesis of the disease.[4,5,9] The term fragmentation of consciousness in the sense of splitting was also defined in borderline personality disorder (BPD) as a

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http://dx.doi.org/10.2147/NDT.S57617

specific form of dissociation, and recent studies suggest that the relationship between dissociative symptoms and BPD per se is very close.[10–12]

With the aim of finding specific relationships between dissociative symptoms and other symptoms in BPD and schizophrenia, we assessed both groups of patients with the aim of comparing the occurrence of dissociation and also of assessing the possible influence of antipsychotic medication using chlorpromazine equivalents (EC).

Materials and methods

Participants

The participants were recruited from regular daily treatment programs for outpatients with schizophrenia or BPD at the Psychotherapeutic and Psychosomatic Clinic ESET in Prague. The participants had a diagnosis of schizophrenia or BPD. Exclusion criteria were organic illnesses involving the central nervous system, substance and/or alcohol abuse, and mental retardation (Raven's IQ <90).[13] Clinical diagnoses were based on DSM-IV (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*) criteria and were reassessed using The Mini-International Neuropsychiatric Interview[14] in patients with schizophrenia and confirmed by semistructured interview in patients with BPD. We calculated actual daily doses of antipsychotic medication in EC for all participants.[15]

The schizophrenia sample comprised 31 patients (15 men and 16 women) of mean age 36.2±9.5 years. Their mean duration of psychiatric treatment was 13.3±8.2 years, and they had an average of 4.4 hospitalizations. The BPD sample comprised 36 patients (eleven men and 25 women) of mean age 31.0±8.7 years. Their mean duration of psychiatric treatment was 6.6±4.1 years, and they had an average of 2.39 hospitalizations. Because of their different durations of psychiatric treatment, the patients also had a different medication history and as measurable equivalent characterizing their current medication we have used EC.

Psychometric measures

We used the Dissociative Experiences Scale (DES) to screen for dissociative symptoms.[16] The DES is a 28-item self-report questionnaire that evaluates the frequencies of various experiences of dissociative phenomena in the patient's everyday life. Each item ranges from 0 to 100 and the mean of all item scores is calculated as the DES score. For more detailed assessment of the DES items, we analyzed the DES factors that have been used in previous research studies.[17] In this analysis, we used three factors focused on absorption (items 2, 14, 15, 17, 18, and 20), amnesia related to dissociative states (items 3, 4, 5, 8, 25, and 26), and depersonalization/derealization (items 7, 11, 12, 13, 27, and 28). In the present study, we used the Czech version of the DES; like the original English version, it shows high reliability and internal consistency (Cronbach's alpha 0.92, test–retest reliability after one week 0.91).[18,19]

Symptoms related to stress and traumatic experiences were measured using the Trauma Symptom Checklist-40 (TSC-40).[20] This scale was designed for measurement of post-traumatic symptomatology associated with childhood trauma. The TSC-40 is a self-reported scale containing 40 items with six subscales, ie, dissociation, anxiety, depression, a sexual abuse trauma index, sexual problems, and sleep disturbances. The Czech version of the TSC-40 has high reliability and internal consistency (Cronbach's alpha 0.91, test–retest reliability after one week 0.88).[21]

Psychotic manifestations were measured using the Health of the Nation Outcome Scales (HoNOS) in both groups of patients.[22] The scale includes twelve items (overactive, aggressive, disruptive or agitated behavior; nonaccidental self-injury; problem drinking or drug-taking; cognitive problems; physical illness or disability problems; problems with hallucinations or delusions; problems with depressed mood; other mental and behavioral problems; problems with relationships; problems with activities of daily living; problems with living conditions; problems with occupation and activities). There are two versions available, ie, the version for external evaluators and the self-rating version for patients. Both versions were translated into the Czech language (Cronbach's alpha 0.797, test–retest reliability after one week 0.85).[23]

Statistical analysis

Statistical evaluation of the results for the DES and other psychometric measures included descriptive statistics, the Mann–Whitney *U* test for independent samples, and Spearman correlation coefficients. Nonparametric analyses were preferred because the DES data were not normally distributed. All the methods used for statistical evaluation were performed using Statistica version 6 software (StatSoft Inc., Tulsa, OK, USA). To prevent type II error, which would not be able to reject the null hypothesis that symptoms of dissociation are not linked to stress-related psychopathological symptoms, we performed a power analysis and assessed the effect sizes by characterizing differences between means or correlation coefficients of the samples.

Results

We compared scores from the psychometric measures using the Mann–Whitney *U* test to test for differences in dissociation, other psychopathological manifestations, and use of antipsychotic medication between the two disorders (see Table 1). Although the differences in DES scores between patients with BPD and those with schizophrenia were not statistically significant, scores for symptoms of traumatic stress measured by the TSC-40 were significantly higher in the BPD group.

External evaluations as well as self-rating on the HoNOS showed that the BPD group had significantly higher scores than patients with schizophrenia. Nevertheless, in several subscales of the HoNOS, external evaluators reported that the schizophrenia group had higher scores for cognitive problems (1.55 in schizophrenia versus 0.66 in BPD, P=0.0001) and positive symptoms (1.26 in schizophrenia versus 0.54 in BPD, P=0.0273). Doses of antipsychotics measured by EC were significantly higher in the schizophrenia group. In the power analysis, we tested the differences between means, and found that all these had a strong effect size ($r \geq 0.5$; Table 1).

We calculated Spearman correlation coefficients in both patient samples to assess the relationship between dissociation and other psychometric measures and the possible influence of antipsychotic medication on this relationship. The results show that scores on the DES, TSC-40, and HoNOS were significantly correlated (see Table 2). Interesting, statistically significant correlations were found in BPD patients between levels of EC and the DES score (Spearman's correlation R=0.37; refined Fisher's exact test Z=0.14) and between EC and depersonalization/derealization score on the DES (Spearman's correlation R=0.37; refined Fisher's exact test Z=0.38).

Discussion

Our results show that dissociative symptoms and symptoms of traumatic stress are significantly correlated in patients with

Table 1 Comparison between schizophrenia and bipolar disorder using the Mann–Whitney *U* test

	Schizophrenia n=31	BPD n=36	MW Z	*P*-value	*r* Effect size
DES	13.7	18.54	−1.8	0.0730	0.36
TSC-40	34.1	54.19	−3.7	0.0002	0.95
HoNOS (E)	11.6	15	−2.6	0.0085	0.81
HoNOS (S)	7.8	14.25	−3.9	0.0000	0.97
EC	518.4	98.6	5.5	0	0.97

Abbreviations: BPD, bipolar disorder; DES, Dissociative Experiences Scale; TSC-40, Trauma Symptom Checklist-40; HoNOS (E), version for external evaluation of HoNOS (mean); HoNOS (S), self-rating version of HoNOS (mean); EC, daily doses of antipsychotic medication in equivalents of chlorpromazine (mean in ml); *r*, standardized effect size; MW, Mann–Whitney *U* test.

Table 2 Spearman correlation coefficients describing relationships of dissociation with other psychopathological symptoms and with doses of antipsychotic medication in patients with schizophrenia and borderline personality disorder

	EC		HoNOS (E)		HoNOS (S)		DES		DES-AB		DES-AM		DES-DD	
	Sch	BPD	Sch	BPD	Sch	BPD	Sch	BPD	Sch	BPD	Sch	BPD	Sch	BPD
HoNOS (E)	0.2	0.04	–											
HoNOS (S)	−0.22	0.28	**0.48**	0.7	–									
DES	0.17	**0.37**	0.34	**0.37**	**0.53**	**0.52**	–							
DES-AB	0.08	0.29	0.25	0.25	**0.47**	**0.51**	**0.88**	**0.84**	–					
DES-AM	0.14	0.28	−0.02	0.25	0.17	0.26	**0.64**	**0.76**	**0.5**	**0.67**	–			
DES-DD	−0.03	**0.37**	**0.53**	**0.35**	**0.65**	**0.4**	**0.8**	**0.77**	**0.61**	**0.44**	**0.36**	**0.48**	–	
TSC-40	0.03	0.08	**0.41**	**0.56**	**0.7**	**0.7**	**0.6**	**0.53**	**0.49**	**0.46**	**0.28**	**0.38**	**0.75**	**0.43**

Notes: values at P<0.05 are in bold; Fisher's exact test Z is higher than 0.05.

Abbreviations: EC, daily doses of antipsychotic medication in equivalents of chlorpromazine (mg); HoNOS (E), version for external evaluation of HoNOS; HoNOS (S), self-rating version of HoNOS; DES, Dissociative Experiences Scale; DES-AB, factor of absorption in DES; DES-AM, factor of amnesia in DES; DES-DD, factor for depersonalization/derealization of DES; TSC-40, Trauma Symptom Checklist-40; Sch, schizophrenia; BPD, borderline personality disorder.

BPD and in those with schizophrenia. The data also show that symptoms of traumatic stress are higher in BPD than in schizophrenia, which is in agreement with the findings of other studies.[24–26]

On the other hand, DES and TSC-40 scores were significantly correlated with symptoms of psychosis in both disorders and, as in other studies of patients with schizophrenia, symptoms of traumatic stress were associated with psychotic symptoms,[27,28] higher levels of anxiety, and other psychopathological symptoms.[29–33] In agreement with other studies, we also found that dissociation in schizophrenia is closely related to symptoms of trauma.[34–41] Similar relationships between stress and dissociation have also been found in patients with BPD.[10,42]

An interesting finding of this study was the correlation between doses of antipsychotics measured in EC and dissociative symptoms in patients with BPD. This result suggests a specific psychotropic effect of antipsychotic medication in these patients. To the best of our knowledge, this has not been reported in the scientific literature before. Because our patients had different medication histories, it is necessary to investigate the possible relationship between medication and dissociative symptoms further in follow-up studies that could explain certain details and specific influences of medication on neurotransmitter systems. Nevertheless, the statistical finding of this relationship in the BPD group but not in schizophrenia group is of interest although further research is necessary. The possible influence of medication on dissociative symptoms might reflect the extremely important role of stress in BPD. According to current data, stress represents a more significant factor in BPD etiology than in schizophrenia.[43,44] Recent data show that antipsychotic treatment likely decreases activation of the anterior cingulate cortex.[45,46] In this context it is possible that conscious conflicting[47,48] experiences due to antipsychotic medication in BPD may decrease conscious awareness of conflicting stressful experiences and cause their dissociation that may produce the dissociative symptoms measured by the DES. Although this interpretation is currently speculative, it might be useful for further research that could have significant consequences for the treatment of patients with BPD.

Conclusion and perspectives

These results support the conceptual but empirically rare findings concerning the important role of dissociative processes in schizophrenia and BPD and the specific relationship between them. A novel contribution of this study that needs further research is the significant finding that manifestations of dissociative symptoms might be specifically linked to antipsychotic medication in patients with BPD but not in those with schizophrenia.

Acknowledgment

The study was supported by a grant from Charles University (PRVOUK), project grants provided by the Czech Ministry of Education (LH11032) and the "CEITEC – Central European Institute of Technology" (CZ.1.05/1.1.00/02.0068) from the European Regional Development Fund.

Disclosure

The authors report that they have no conflicts of interest in this work.

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Review Article • DOI: 10.2478/s13380-013-0131-4 • Translational Neuroscience • 4(3) • 2013 • 368-377
Translational Neuroscience

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Received 06 May 2013
accepted 31 May 2013

SYNTHETIC METACOGNITION AS A LINK BETWEEN BRAIN AND BEHAVIOR IN SCHIZOPHRENIA

Abstract
Deficits in metacognitive capacity in schizophrenia can be conceptualized as existing along a spectrum from more discrete to more synthetic activities. These capacities may be of great importance in schizophrenia research given their potential to mediate and moderate the impact of illness-related factors on outcome. To explore this possibility this review summarizes research on synthetic metacognition using a paradigm in which metacognitive capacity is rated on the basis of spontaneously produced personal narratives. Evidence from a review of the literature shows that these deficits are detectable in patients with schizophrenia and are related to, but not reducible to, symptom severity and poorer neurocognitive function. Independent of symptoms and neurocognition, deficits in synthetic metacognition, which are likely linked to the brain's ability to integrate information, are related to a range of outcomes including functional competence, learning potential, and insight. These deficits may also play a role in long term psychosocial functioning via their impact on the ability to sustain social functions.

Keywords
• Schizophrenia • Neurocognition • Recovery • Narrative • Metacognition • Psychosis • Quality of life • Self

Bleuler suggested that the interruption of goal-directed behavior that characterizes schizophrenia is a result of complex, biologically based processes that lead to the unbinding of associations and the collapse of higher order understanding of oneself and others [1]. In the more than 100 years since, a more detailed picture has emerged of the sequence of events that result in dysfunction in schizophrenia. Genetic vulnerabilities for schizophrenia have, for instance, been linked to abnormalities in brain development that are reflected in neurocognitive deficits, such as impairments in information processing, verbal memory and executive function [2-7]. Further, these cognitive deficits may be linked to disruption in synchronized oscillatory activity of neural networks, which would allow for the effective integration of information and hence binding of associations [8-11].

Connecting these processes to behavior, neurocognitive dysfunction has been linked with poorer social, vocational, and community function. Bowie *et al.* for instance, found that processing speed and attention/working memory were uniquely linked with social competence and that social and functional competence mediated the effects of neurocognition on community and work functioning [12]. These neurocognitive deficits have been detected prior to the onset of illness [13,14] and observed to interact with symptom severity [15]. Neurocognitive compromise has also been implicated as a predictor of poorer response to psychosocial treatments [16].

Considering forces taking place outside of the mind of individuals, research has also suggested that social and community factors such as trauma, stigma, poverty, isolation, and attachment patterns exacerbate or are exacerbated by both brain function and neurocognition on their own, and in conjunction with more biologically based paths to dysfunction [17,18]. For example, Walker and Diforio proposed "a neural diathesis-stress model" of schizophrenia in which a neural mechanism for these phenomena results from the stimulating effect of the hypothalamus-pituitary-adrenal (HPA) axis on dopamine synthesis and receptors [19]. This gain of function may lead to abnormalities in dopamine receptors, which together with hippocampal damage, significantly influences hypersensitivity to stress in persons with schizophrenia. In the context of this model, Read *et al.* [20] developed "a traumagenic neurodevelopmental model" that proposes that genetic alterations lead to predisposing vulnerability in the form of hypersensitivity to stress related to adverse life events such as child abuse, which, according to recent evidence, contributes to the development of schizophrenia, likely through the influence of traumatic events on the developing brain and neurobiological abnormalities that frequently occur in patients with schizophrenia, such as over reactivation of the HPA axis or structural changes to the brain (e.g. hippocampal damage, cerebral atrophy, reversed cerebral asymmetry, ventricular enlargement), as well as dopamine, norepinephrine, and serotonin abnormalities.

As shown in Figure 1, this work, which initiated with Bleuler [1], offers a path from brain to behavior, linking genetics, brain

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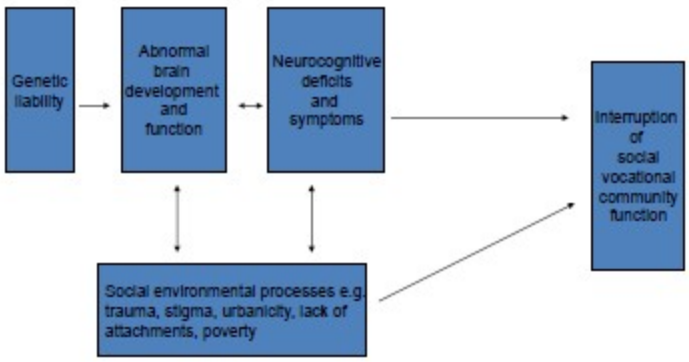

Figure 1. A path from gene to brain to dysfunctional behavior in schizophrenia.

development, cognition, symptoms, social and community experiences, and ultimately the interruption of goal directed behavior. This larger scheme provides an idea of different points for clinical intervention but also allows for a more nuanced inquiry into the proximate psychological factors affecting function in schizophrenia.

One specific area of interest concerns what is happening in the space in Figure 1 that lies in between manifestations of illness and psychosocial dysfunction. Certainly, it is not surprising that phenomena such as diminished neurocognitive capacity are related to impaired function because thought is needed for function. But exactly how does this occur? What psychological factors mediate the impact of symptoms and neurocognitive deficits on behavior? Are there phenomena specific to schizophrenia that play a role in dysfunction at this point in the model? Answers to these questions seem essential in order to develop treatments that intervene at the level most proximate to function. If we know how neurocognitive impairments and symptoms culminate in dysfunction, it may be possible to design treatments for persons with treatment-resistant cognitive deficits or symptoms, or for those whose impairments persist after cognitive deficits or symptoms have been resolved.

To address this issue, a program of research will be reviewed that has examined one set of processes which may mediate and/or moderate the impact of illness related variables such as symptoms and neurocognitive deficits on real world function. We propose that psychosocial dysfunction may result from a feedback loop in which symptoms and neurocognitive deficits influence and are influenced by deficits in synthetic forms of metacognition. We suggest that deficits in synthetic metacognition leave persons unable to make meaning of both illness related phenomenon and psychosocial challenges. Without possessing a sufficiently complex account of themselves and others, these individuals may be unable to make sense of what is happening. They may thus not know why they should take certain courses of action and hence social and vocational function may languish.

It has been proposed that phenomena such as neurocognitive deficits affect function because they leave persons less able to know how to perform basic functional and interpersonal tasks. Neurocognitive deficits may limit the rate at which persons can learn new skills, as well as accurately detect and respond to the demands of the environment [21]. In further iterations of this model, a lack of ability to perform tasks has been thought to lead to defeatism and withdrawal [22]. For instance, persons with deficits in executive function might incorrectly identify the motives or intentions of others and so not be able to know how to interact appropriately. They might similarly be unsure what actions are needed to achieve a certain end in a work setting and so struggle with an instrumental task and decide to give up. This work is certainly tied to an emerging set of interventions that try to teach people how to perform certain tasks, such as how to deliver a compliment or carry out a specific work task when under stress.

In this review we will not contradict that skill deficits are important to understanding outcome but add to it with the consideration of an additional possibility that deficits in metacognitive capacity interfere with the ability to know why to do certain things and not just how to do them. Harkening back to existential psychiatry, we will suggest that many with schizophrenia often fail to take certain action not only because they do not know how, or because they do not expect they will be successful, but often because they do not know why they should take those actions [23,24]. In daily life persons seek to repair rifts in relationships and endure difficult times at work because the hopes and dreams they possess make such repairs and persistence meaningful. Applied to schizophrenia then we wish to suggest, for instance, that metacognitive

deficits do not remove knowledge of how to make a compliment in a social encounter but may leave persons without an idea of why to deliver a compliment and so they may offer no compliment. Persons with schizophrenia might similarly not persist at work when under stress, not because they do not know how to persist but because of a lack of having any larger sense of themselves which would supply a reason for persisting. Figure 2 provides an illustration of how metacognitive deficits could be incorporated into standard models of the path from brain to behavior in schizophrenia.

To explore the possibilities offered by figure two, this review will first offer a definition of metacognition as a spectrum of mental activities which can involve synthesizing experience into integrated representations of self and other and can be a subject for reflection and subsequent revision. We suggest that disruptions in this form of metacognition closely parallel what Bleuler [1] described as the fundamental disturbance in schizophrenia. Second, we discuss recent efforts to operationalize and measure synthetic metacognitive deficits in schizophrenia and detail research offering preliminary confirmation of their potential links with both neurocognition as well as functional outcomes. Limitations and directions for future research and clinical implications are also explored. Of note, as portrayed in Figure 2, metacognition may potentially play a mediating role in the link between social phenomena and function, but this will not be discussed in the present review.

The concept of metacognition

Metacognition refers to a mental act in which persons forms a thought or idea about their own mental activities. The construct was first used in the education literature to capture learners' awareness of their own learning and what conditions best enabled learning, and then spread to different dimensions of experience such as self-regulation [25-27]. The psychological literature has similarly used the term to describe a broad range of phenomena. As we have summarized elsewhere [28], metacognition applied to human development and psychopathology has been used to describe a range of different mental activities. These activities may vary from one another according to the extent to which they involve focusing on discrete mental activities such as thinking about a specific isolated thought [e.g. 29] versus the integration or synthesis of a range of different experiences into a complex representation of self and others and then a reflection about that larger representation [30,31]. Intuitively, these discrete and synthetic metacognitive activities influence one another but as such are not reducible to one another. For instance, an awareness of discrete elements is needed to form larger ideas of oneself while awareness of oneself is needed to make meaning of discrete experience with the flow of daily life. Further, human beings' larger reflection about themselves and others has long been seen as more than a sum of psychological facts but rather emerging from a particular way of connecting and meaningfully situating those facts in relation to one another [32].

The distinction between discrete and synthetic forms of metacognition maps well onto a range of schizophrenia research [28,33]. Deficits have been found in the ability to detect specific mental activities such as behaviors, emotions and memories [34-36]. Looking at larger psychological phenomena, others have found difficulties reflecting on broader cognitive habits, such as reasoning style [37] as well as the ability to pull together autobiographical memories into a meaningful whole [38-40]. Importantly, the idea of a spectrum may also help clarify slightly different applications of the term metacognition with some authors concerned with the detection of errors [34] or reflections about specific beliefs, [29] and others, as will be explored in this review, with reflections about various integrations of thoughts and feelings into representations of self.

A rationale for why synthetic metacognition may be linked with psychosocial function in schizophrenia

In this review we focus on more synthetic forms of metacognition as a mediator of the effects of

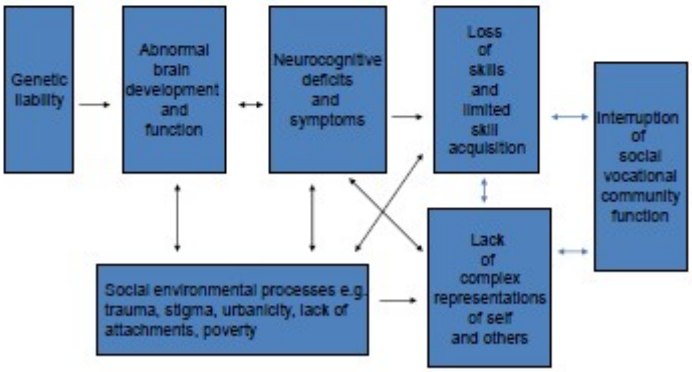

Figure 2. A path from gene to brain to dysfunctional behavior mediated by deficits in metacognitive capacity.

illness-related phenomena on function for three reasons, although we do not wish to suggest that more discrete forms of metacognitive activity would be less important or valid. First, synthetic metacognitive activity, in general, allows for meaning to be bestowed upon experience and activity. Synthetic forms of metacognition lend the potential for different meanings to be made, for instance, of conflicts at work or with others. For example, more pain is likely if one loses a job that is a cherished part of one's life vs. just a way to make money to support something else. More joy may come from one interaction vs. another because of the meaning one person has within one's life (e.g. a very promising junior colleague who might carry one's work forward vs. an acquaintance).

Second, the meaning bestowed on activity provides a context for reasoning about why one response makes more sense than another in the face of conflict or pain. As an illustration, if holding a certain job is an integral part of one's identity given longstanding career aspirations, one may find a reason why one should tolerate frustration with colleagues and supervisors rather than quit and find a new job. However, this would not make sense if the job is merely a way to make money. Similarly there would be far more reason to attempt to salvage a relationship with another person after a falling out if that relationship was seen as deeply connected with one's own life story. Thus, in the face of deficits in metacognitive capacity, a patient with schizophrenia might see less reason to persist or cope in the face of distressing developments if those events were without the personal meaning that is imbued by rich and complex understanding of oneself and others. Therefore, metacognitive deficits may result in there being no reason to undertake a course of action, regardless of whether or not a person knows how to undertake that action. In fact, without a reason to perform that action, there may be no search to figure out how to undertake that action.

Beyond the general connection between metacognition and responding to challenge, there is also a historic reason to think about schizophrenia as involving at its core a disturbance in the ability to synthesize material into complex wholes. As discussed also by Moskowitz [41], when Bleuler [1] proposed disturbances of associations as the core feature of schizophrenia, he did not refer merely to confusion due to the intrusion of unrelated ideas into thought but to the loss of a fundamental ability to form "associational synthesis" (p. 44), which reduced the understanding of oneself as an agent to a set of fragments which no longer served as a guide for goal directed activity. This lack of synthesis, as noted above, was assumed to have an organic origin but also to serve as a proximate cause of dysfunction as Bleuler [1] indeed detailed in accounts of schizophrenia patients no longer able to function socially or vocationally. Applied to the research presented above, it seems intuitively plausible that the feedback loop between brain development, social forces, symptoms, and neurocognition might well culminate in the loss of the ability to synthesize associations into larger images of oneself and others which, then, as exposed in Figure 2, may play into the larger feedback loop which results in dysfunction.

Assessing synthetic aspects of metacognition in schizophrenia using the Metacognition Assessment Scale Abbreviated (MAS-A)

To date one barrier to exploring the possibility that deficits in synthetic metacognitive activites play a role in the path from brain to behavior in schizophrenia concerns the issue of its measurement. Laboratory tasks have been developed to assess discrete aspects of metacognition, for instance to determine whether participants can detect they have made an error, made a certain action or recognize they do or do not know something [42-46]. These type of assessments, while useful for determining the effectiveness of error detection, are not useful for assessing more synthetic abilities which are a matter of complexity, flexibility, and adaptiveness.

In response, we have recently developed a method to assess synthetic metacognitive abilities from a spontaneously generated speech sample in which persons discuss their lives and personal understanding of the trials they have faced. That speech sample is obtained through a semi-structured interview called the Indiana Psychiatry Illness Interview (IPII). The IPII asks participants for their account or narrative of who they are as a person and also of their experience with psychiatric challenges. It thus allows for a life story to be told, in which there are opportunities for participants to spontaneously reveal how they think about themselves.

To quantify synthetic metacognitive capacity within IPII narratives, the Metacognition Assessment Scale – Abbreviated [MAS-A; 47] is used. The MAS-A contains four scales: "Self-reflectivity," or the comprehension of one's own mental states, "Understanding of others' minds," or the comprehension of other individuals' mental states, "Decentration," which is the ability to see the world as existing with others having independent motives, and "Mastery," which is the ability to use one's mental states to respond to social and psychological dilemmas. It is assumed that the metacognitive capacities assessed by each scale are semi-independent. Higher scores reflect abilities to perform increasingly complex synthetic acts within the domain captured by that scale. For instance, higher scores on Self-reflectivity would suggest a capacity to form more integrated representations of oneself, while higher scores on Mastery would suggest the capacity to use more complex forms of metacognition to respond to psychological and social challenges.

Acceptable levels of inter-rater reliability and internal consistency have been reported along with evidence of stability of MAS-A assessments across a 6-month interval [47-49]. Evidence that these procedures capture difficulties specific to psychosis includes findings that participants with schizophrenia have lower scores on all of the MAS-A subscales compared to others who also have significant medical and social adversity but not psychosis [50]. Concerning their validity, MAS-A scores have been linked with independent assessments of awareness of illness and cognitive insight [47,51].

Associations of metacognition with symptoms and neurocognition

To determine whether synthetic metacognitive activity may be linked with symptoms and

neurocognition, as portrayed in figure two, we have examined whether more severe symptom levels and neurocognitive deficits were linked with poorer MAS-A scores. In our first study, we correlated symptoms and neurocognitive functioning with MAS-A scores among men with schizophrenia in a non-acute phase of illness enrolled in rehabilitation [47]. The results revealed that a greater capacity for Self-reflectivity was linked with better verbal and visual memory, processing speed and premorbid intelligence. Greater capacities for Understanding the mind of the other, and Mastery were also related to better verbal memory. Concerning symptoms, higher levels of negative symptoms related to emotional withdrawal were linked with greater deficits in Self-reflectivity, Understanding the mind of the other, and Mastery.

Following up on the issue of neurocognitive capacity and metacognition, in a second sample of patients in a non-acute phase of illness we found that patients categorized as having achieved basic levels of Self-reflectivity on the MAS-A performed better on tests of executive function, working memory and social cognition [48]. In a third study [51] we turned to the issue specifically of executive function and correlated MAS-A scores with selected subtests of the Delis-Kaplan Executive Function System [D-KEFS; 52] including tests of inhibition, set shifting and mental flexibility. The sample consisted of 49 participants drawn from the two studies described above, who had completed the D-KEFS. Results revealed that the MAS-A Self-reflectivity subscale was more closely linked to D-KEFS subtests which tapped mental flexibility while D-KEFS subtests, which tested inhibition, were more closely linked to Decentration, Understanding of the other's mind, and Mastery, with correlations ranging from 0.30 to 0.47. We speculated that neurocognitive deficits contribute to deficits in synthetic metacognition. For instance, with a generalized reduction in the ability to flexibly think about daily events, persons may have difficulties forming an image of themselves as multifaceted. In contrast, without an ability to inhibit thoughts about events in the world, some may find it difficult to call to mind the perspectives of others and to detect a range of possible reactions others are having in rapidly evolving situations.

Exploring the links between metacognition and symptoms in a new sample, we compared MAS-A and symptom assessments over two time points six months apart in a group of 49 adults with schizophrenia in a stable phase of illness [49]. Correlational analyses found that the Total score of the MAS-A was correlated with both concurrent assessment of positive, negative, and disorganization symptoms as rated on the Positive and Negative Syndrome Scale [PANSS; 53]. In a multiple regression analysis, the MAS-A total score was found to predict prospective ratings of negative symptoms even after co-varying for baseline negative symptoms scores. We speculated that this may provide evidence as suggested in figure two of a bidirectional relationship between synthetic metacognition and at least some forms of symptoms. For instance, negative symptoms may dampen synthetic forms of metacognition while metacognitive deficits may be a risk factor for the emergence of negative symptoms. The findings linking negative symptoms and neurocognition with MAS-A scores in patients with prolonged psychosis have been replicated in separate Italian and Israeli samples [54,55]. McLeod *et al.* [56] have also reported metacognition rated using the MAS-A, on the basis of the Adult Attachment interview, prospectively predicted negative symptoms in 45 first episode patients in the United Kingdom above and beyond variance accounted for by premorbid function.

Metacognition and functional outcomes

Turning to the issue of synthetic metacognitive function and outcome, we first sought to see whether MAS-A scores could prospectively predict work function in a sample of 56 adults with schizophrenia enrolled in a vocational rehabilitation program. To accomplish this we divided participants who had completed a work placement program into three groups on the basis of Self-reflectivity score on the MAS-A which had been obtained prior to going to work: high (n=13), intermediate (n=21), and low Self-reflectivity (n=22) [57]. We then compared the biweekly rating of work performance completed during the six months following the initial MAS-A. Here we found that high Self-reflectivity group had significantly better work performance than either of the other two groups. That difference persisted after controlling for executive function as assessed prior to starting work. In a re-analysis of this data we also found that greater Self-reflectivity on the MAS-A was related to more accurate estimates of work ability as defined by the difference between self and supervisors assessments of work quality [58]. Links between metacognition and rehabilitation outcome were also found by Tas *et al.* [59] who reported that poorer metacognition predicted poorer response to cognitive remediation over time in a Turkish sample of 52 schizophrenia patients in a state of symptomatic remission.

In a second study we looked at the issue of social function, to assess whether we could show that metacognition, as assessed with the MAS-A, mediated the impact of neurocognitive deficits on rater assessment of the frequency of social interaction (quantity of social relationships) and on the basic building blocks which allow for social connection (quality of social relationships). Participants were 102 adults with a schizophrenia symptomatology in a post-acute phase of illness [60]. A Principal Components Analysis was used to reduce five different assessments of neurocognition, including executive function, verbal memory, visual memory, processing speed, and verbal ability into a single index and then structural equation modeling techniques were used to test the model that the capacity for metacognitive mastery mediates the impact of neurocognition upon the quality and quantity of social relationships after controlling for symptoms. Results revealed that an acceptable fit was observed between the model and data. Results persisted even after controlling for symptom and illness severity. In a third study, we followed this up by examining whether the links between metacognition and social function persisted over time [61]. Specifically, we examined the links between MAS-A scores and assessments of quality and quantity of social function conducted two times at an interval of five months apart for 72 of the original

102 participants. In a path analysis, acceptable levels of fit were found for a model in which Mastery predicted concurrent social function, and Mastery at baseline affected Mastery five months later, which similarly affected social function at that time. We interpreted the results of these studies along with the study of vocational function to provide evidence of the proximal link between synthetic metacognition and psychosocial function.

Support for these interpretations has been bolstered by another study of the relationship of metacognition with the perception of social interactions. Specifically, this study correlated MAS-A scores with assessments of social schema in a sample of 37 adults with schizophrenia in a non-acute phase of illness [62]. Social schema was measured using the Social Cognition Object Relations Scale [63], which assesses awareness of interpersonal relationships as a result of complex psychological forces, as well as the recognition that people in relationships have independent needs. Correlational analyses controlling for symptom severity and neurocognition revealed that higher levels of Mastery were linked to a greater understanding of the complex psychological forces that affect relationships and the existence of independent needs of individuals in relationships. The possible interpretation that the capacity for social connection is adversely affected by lower levels of metacognition has also been mirrored in another study, which found that greater levels of Mastery were related to self-reports of greater therapeutic alliance in cognitive behavior therapy [64].

In contrast to the studies noted above that used rater assessments of function, we also conducted studies examining the links of metacognition with self perception of illness and recovery. First, in a study of awareness of illness, we correlated assessment of awareness of symptoms, treatment needs and consequences of illness with assessments of social cognition, neurocognition and the MAS-A among 65 adults in a non-acute phase of illness [65]. After controlling for neurocognition, regressions revealed that Self-reflectivity was most closely linked to awareness of symptoms of psychosis while Mastery was most closely linked to awareness of treatment and consequence of illness. Mastery and social cognition were found to contribute independently to the prediction of awareness of consequences of illness. In terms of self-reported wellness as assessed by the Recovery Assessment Scale (RAS), with a new sample of 44 schizophrenia patients in a non-acute phase of illness, we found that those with greater levels of self-reflectivity felt less dominated by their symptoms while patients with more decentration were more able to reach out to others for help while feeling hopeful [66]. We have interpreted these findings as suggesting that synthetic metacognitive capacity may be linked to not only concrete social and vocational outcomes but also how persons appraise both illness and their achievement of health.

Finally, regarding functional competence or knowledge of how to perform tasks as indicated in Figure 2, a fourth study [67] examined the relationship between metacognition and performance on an assessment of functional skills, the UCSD Performance-Based Skills Assessment Battery (UPSA) [68]. Participants were 45 adults in a non-acute phase of schizophrenia that completed the IPII, assessments of neurocognition, symptoms and the UPSA. Correlational analyses revealed that Mastery was related to scores on the comprehension/ planning subscale of the UPSA even after controlling for symptoms and executive function. Results were interpreted as suggesting that decrements in Mastery may make some persons withdraw from more complex daily tasks such as organizing complex plans, resulting in the deficits in those functional abilities.

Discussion and limitations

In summary, decades of international research indicate that psychosocial dysfunction in schizophrenia is in part the result of the interaction of factors including symptoms and deficits in neurocognition. Less clear is how symptoms and neurocognitive deficits translate into the interruption of daily life. In this paper we sought to explore the possibility that synthetic metacognitive capacity may mediate the impact of these illness-related variables on outcome in schizophrenia. In other words, perhaps the path from brain to behavior in schizophrenia is influenced by the recursive process in which persons facing the illness reflect upon and interpret it in an ongoing inner experience. We have suggested a rationale for why this might be: metacognitive deficits seem likely linked in a bidirectional manner with symptoms and neurocognition and metacognitive deficits may leave persons less able to know why to pursue certain forms of action, resulting in the widespread interruption of goal directed activity. We have then detailed a method for assessing synthetic forms of metacognition and described findings from a number of studies that have linked synthetic metacognitive deficits with illness related phenomenon as well as multiple outcomes. These included at least one study that provided evidence that metacognitive deficits play a role as mediators.

Taken together the work reviewed provides evidence that deficits in synthetic metacognition are detectable in schizophrenia patients and that these deficits are related to outcome in a manner suggested in Figure 2. Deficits in synthetic metacognitive capacity appear to be distinct from a lack of functional skill and are not synonymous with defeatist beliefs linked with functional skill deficits [22]. Whereas a defeatist belief refers to a particular idea a person has about oneself, at issue here is the deeper ability to organize and integrate information into a larger whole, which would bestow meaning on daily activity. While this work is far from being able to offer proof that metacognitive deficits occupy the proposed role connecting brain to behavior, it appears to have offered a promising start, producing findings consistent with what one would expect if synthetic metacognition plays the role we have speculated.

As a whole this work can be seen to parallel recent converging anatomical and electrophysiological research on brain function which suggests that, as anticipated by Bleuler [1], the experience of "cognitive wholeness" is created through neural network synchronization of various association pathways [8,69]. Specifically, synchronization in the gamma and beta frequency bands has been linked to cognitive processes altered

in schizophrenia, such as perception and memory [9]. In the context of the subtitle of Crick's "Scientific search of the soul" [70] the research presented here may suggest that in schizophrenia, functional brain disintegration may unfold as psychological disintegration. Concerning treatment, this work is also consistent with other work suggesting that building metacognitive abilities through specific forms of learning during psychotherapy may also influence the brain based integrative processes as well [71,72].

There were important issues not discussed for reasons of space. This review has not dealt fully with the issue of etiology. It is unknown whether and if so how often, metacognitive deficits predate the illness and/or whether they can result from a number of different causal influences, including atrophy, loss of cognitive functioning, attachment style or exposure and response to trauma. It was also not discussed how the construct of metacognition converges and diverges with related constructs including social cognition, mentalization, mindfulness, and emotional intelligence. Future empirical and theoretical work is needed to tease apart how the phenomena studied here are related to these and other constructs. While we have explored capacity for forming and reflecting about integrated images of oneself, it should be noted that a range of work focused on autobiographical memory (e.g. [39-41]) should be considered in order to have a more complete view of the broader picture.

The research detailed here has also been most interested in links between illness related phenomenon and outcome and so the links between metacognition with social factors has been relatively neglected. We have also not fully explored the link between metacognitive disturbance and lack of functional competence. More nuanced theory is needed to develop a model of how skills deficits and metacognitive impairments impact and influence one another.

There are other limitations to the work presented here. Most of the studies discussed, though not all [54-56,59], were carried out in one laboratory. Participants also tended to be males in a later stage of illness enrolled in treatment. Replication is needed with broader samples including women, persons in an early stage of treatment and others who reject treatment. Long term longitudinal work is also needed to better understand the relationships suggested above as well as work comparing the metacognitive function of persons with schizophrenia to others with different forms of psychopathology. While we are not aware of other current methods for assessing synthetic forms of metacognition, much older work on psychotherapy and schizophrenia, such as that conducted by Carl Rogers [73], included quantitative scales for assessing personal distance from internal experience on the basis of clinical interviews. The constructs these scales assessed seem related to the central ideas of this review. Future work examining how the MAS-A may be related to these instruments could allow greater integration of the current findings into this much older and unduly neglected literature.

Clinical Implications

Finally, with replication there may be important clinical implications to consider. If neurocognition and symptoms affect function through an interaction with metacognitive processes, treatment may need to do more than provide general support, education and skill remediation. Persons could be taught how to perform various functional activities but not develop any conception of why they should do so. For example, they may learn as we noted earlier how to offer compliments in a social setting but have no idea about why to and hence mere skill enhancement may not meet on its own the goal of truly enhancing patient self-direction. Suggested by the work reviewed in this paper, it may be that some with schizophrenia need assistance, possibly through a form of psychotherapy, to integrate information and form their own personal and adaptive accounts of themselves and their challenges, ultimately allowing them to take charge of their lives and find a way to achieve a fully acceptable quality of life. Recent advances in integrative forms of psychotherapy have shown promise for assisting persons to form more complex and integrated representations about themselves and others and then reflect about those representations, evolving them with time and using that knowledge to respond to psychological problems [74-78]. These forms of interventions might then allow persons to know why to persist in certain difficult life circumstances and to find the kinds of meaning in pain that allow human beings to live a rich life despite adversity.

Acknowledgments

All the authors have contributed to the writing of the article. The authors declare no conflict of interest.

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7. LIST OF ABBREVIATION

BPD - borderline personality disorder (hraniční porucha osobnosti)
DES- Dissociative Experiences Scale (škála disociativních zkušeností)
DES-AB - factor of absorption in DES (faktor absorbce na škále DES)
DES-AM - factor of amnesia in DES (faktor amnézie na škále DES)
DES-DD - factor for depersonalization/derealization of DES (faktor depersonalizace / derealizace na škále DES)
EC - day dosages of antipsychotic medication in equivalents of chlorpromazine (denní dávky antipsychotické medikace v ekvivalentech chlorpromazinu)
EDA - electrodermal activity (elektrodermální aktivita)
EEG- electroencephalogram (elektroencefalogram)
fMRI- functional magnetic resonance imaging (funkční magnetická rezonance)
HoNOS - Health of the Nation Outcome Scales (škála Hodnocení zdravotního stavu)
HoNOS(E)- version for external evaluation of HoNOS (HoNOS, verze pro externí hodnotitele)
HoNOS(S) - self-rating version of HoNOS (sebehodnotící škála HoNOS)
M.I.N.I. - Mini-International Neuropsychiatric Interview
NMDA- N-metyl-D-aspartic acid (kyselina N-metyl-D-aspartátová)
PDP - parallel distributed processing (paralelní distribuované zpracování)
PET- positron emission tomography (pozitronová emisní tomografie)
SI - Splitting Index (index štěpení)
SI(F) – Splitting Index, factor of family (index štěpení, faktor rodiny)
SI(O) – Splitting Index, factor of others (index štěpení, factor ostatních)
SI(S) – Splitting Index, factor of self (index štěpení, faktor self)
TSC-40 - Trauma Symptom Checklist (dotazník pro zjišťování traumatické zkušenosti)
VF- verbal fluency (verbální fluence)

SOUHRN

Pojem štěpení je definován jako proces, při kterém se tvoří agregované soubory psychických elementů, které není možné vzájemně propojit pro neslučitelnou psychickou zkušenost. Soubory pak vytvářejí mnohočetná dělení uvnitř psychického aparátu. Současné poznatky ukazují, že psychické štěpení u schizofrenie se pravděpodobně specificky projevuje na neurální úrovni jako narušení organizace v neurální komunikaci. Narušená neurální komunikace je zřejmě podkladem deficitů v oblasti psychického zpracování, které popisuje řada neurovědních konceptů jako jsou například teorie narušené konektivity, souběžných výbojů a dynamické komplexity. V této souvislosti je cílem teoretické části disertační práce popsat hlavní neurovědní teorie, které komplementárně odrážejí vzájemně propojené procesy mezi psychickou a mozkovou činností, které jsou základem poruch mentální integrace a které pravděpodobně představují neurální reprezentace štěpení u schizofrenie.

Účelem první části empirického výzkumu bylo ozřejmit vztahy mezi psychologickým procesem štěpení a narušenými kognitivními a afektivními funkcemi u schizofrenie. Jako kontrolní skupina byl použit vzorek pacientů s hraniční poruchou osobnosti (HPO).

Metody: V rámci klinické studie jsme vyšetřovali 30 pacientů se schizofrenií a 35 pacientů s HPO. Symptomy štěpení byly měřeny za použití sebemonitorovací škály index štěpení (Splitting Index - SI). Pro ohodnocení stupně desorganizace sémantické paměti jsme použili test verbální fluence. Další psychopatologické symptomy byly vyšetřovány s použitím nástroje Hodnocení zdravotního stavu (Health of the Nation Outcome Scale - HoNOS).

Výsledky: Hlavní výsledky ukázaly, že index štěpení SI je signifikantně vyšší u HPO než u schizofrenie a na druhou stranu je verbální fluence signifikantně nižší ve skupině pacientů se schizofrenií. Psychopatologické symptomy měřené HoNOS byly signifikantně vyšší ve skupině s HPO než u schizofrenie. Signifikantní korelační vztahy byly nalezeny mezi verbální fluencí a „faktorem ostatních" na škále SI (Spearman r =-0.52, p, 0.01) u schizofrenních pacientů.

Závěr: Proces štěpení je odlišný u schizofrenie a HPO. U pacientů s HPO štěpení ústí do psychické nestability, zatímco u schizofrenie psychická fragmentace vede k asociačnímu štěpení, které lze sledovat v nízkých skórech verbální fluence. Tento závěr je v zásadě ve shodě s Bleulerovým historickým konceptem štěpení u schizofrenie.

Ve druhé části empirického výzkumu jsme zvažovali pravděpodobnou klíčovou roli disociace u schizofrenie. Současně jsme vycházeli také z toho, že podobnou roli může mít disociace také u hraniční poruchy osobnosti (HPO). Zároveň jsme zjistili, že studie, které by srovnávaly specifické projevy disociace u schizofrenie a

HPO jsou velmi řídké. V této souvislosti bylo účelem studie vyšetřit výskyt disociativních a dalších psychopatologických symptomů u schizofrenie a srovnat přítomnost těchto symptomů s jejich výskytem u HPO. Dalším cílem bylo zjistit možný vliv medikace antipsychotiky na symptomy disociace.

Metody: Vyšetřovali jsme 31 pacientů se schizofrenií a 36 pacientů s HPO. Symptomy disociace byly měřeny škálou disociativních zkušenosti (Dissociative Experiences Scale - DES). Symptomy vztažné ke stresu a traumatickým zkušenostem byly hodnoceny dotazníkem pro zjišťování traumatické zkušenosti (Trauma Symptom Checklist-40 - TSC-40) a další psychopatologické symptomy škálou Hodnocení zdravotního stavu (Health of the Nation Outcome Scales - HoNOS). U všech pacientů jsme také vyšetřovali aktuální denní dávky antipsychotické medikace v ekvivalentech chlorpromazinu.

Výsledky: Výsledky ukázaly, že symptomy traumatického stresu měřené TSC-40 mají signifikantně vyšší skóre ve skupině s HPO. Zjištěné údaje rovněž ukázaly, že disociativní symptomy (DES) byly signifikantně korelovány se symptomy traumatického stresu (TSC-40) a se symptomy hodnocenými na škále HoNOS. Výrazně signifikantní korelace jsme shledali mezi úrovní antipsychotické medikace a komponentou depersonalizace / derealizace na škále DES u pacientů s HPO.

Závěr: Výsledky podporují důležitou roli disociačních procesů u schizofrenie a HPO a naznačují signifikantní vztah mezi projevy disociace u HPO a antipsychotickou medikací.

V dalším výzkumu bychom chtěli využít těsné souvislosti mezi koncepcí štěpení u schizofrenie a deficitní syntetickou metakognicí. Syntetická metakognice jako psychický proces je schopností lidské mysli syntetizovat záměry, myšlenky, pocity a vztahy mezi událostmi a integrovat je do větších komplexních reprezentací včetně reflexivních funkcí (myšlení o myšlení). Syntetickou metakognici lze měřit analýzou diskurzu za použití standardizovaných postupů.

Klíčová slova: štěpení, disociace, metakognice, schizofrenie, hraniční porucha osobnosti, antipsychotika

SUMMARY

The term splitting is defined as a process of formation of mental aggregates linked incompatible experiences producing numerous divisions in mental apparatus. Current findings indicate that psychological splitting in schizophrenia is likely specifically presented on a neural level as disrupted organization in neural communication. This disrupted neural communication likely underlies deficits in mental processing described by various neuroscientific concepts such as theories of disturbed connectivity, corollary discharges and dynamic complexity. In this context, a purpose of the theoretical part of the dissertation is to describe basic neuroscience theories that complementarily reflect interrelated processes between mind and brain underlying disturbances of mental integration that likely present a neural representation of the splitting.

A purpose of the first part of the empirical research was to examine relationships between psychological process of splitting and disturbed cognitive and affective functions in schizophrenia. A sample of patients with borderline personality disorder (BPD) was used as a control group in this study.

Methods: In the clinical study, we have assessed 30 patients with schizophrenia and 35 patients with BPD. The symptoms of splitting were measured using self-reported Splitting Index (SI). As a measure of semantic memory disorganization we have used verbal fluency test. Other psychopathological symptoms were assessed using Health of the Nation Outcome Scale (HoNOS).

Results: Main results show that SI is significantly higher in BPD group than in schizophrenia, and on the other hand, verbal fluency is significantly lower in schizophrenia group. Psychopathological symptoms measured by HoNOS are significantly higher in the BPD group than in schizophrenia. Significant relationship was found between verbal fluency and the SI ''factor of others'' (Spearman r =-0.52, p,0.01) in schizophrenia patients.

Conclusions: Processes of splitting are different in schizophrenia and BPD. In BPD patients splitting results to mental instability, whereas in schizophrenia the mental fragmentation leads to splitting of associations observed as lower scores of verbal fluency, which in principle is in agreement with Bleuler's historical concept of splitting in schizophrenia.

In the second part of the empirical research we have considered that dissociation likely plays a key role in schizophrenia. Simultaneously we have found out that similar role dissociation could play also in borderline personality disorder (BPD), but empirical studies that compare specific manifestations of dissociation in schizophrenia and BPD are rare. In this context, the purpose of the study was to assess the occurrence of dissociative and other psychopathological symptoms

in schizophrenia and to compare the presence of these symptoms with BPD. A further aim was to assess the possible influence of antipsychotic medication on the dissociative symptoms.

Methods: We assessed 31 patients with schizophrenia and 36 patients with BPD. Dissociative symptoms were measured by the Dissociative Experiences Scale (DES), symptoms related to stress and traumatic experiences were assessed using the Trauma Symptom Checklist-40 (TSC-40), and other psychopathological symptoms were measured with the Health of the Nation Outcome Scales (HoNOS). We also assessed actual daily doses of antipsychotic medication in chlorpromazine equivalents in all participants.

Results: The results show that symptoms of traumatic stress measured by the TSC-40 had significantly higher scores in the BPD group. The data also show that dissociative symptoms (DES) were significantly correlated with symptoms of traumatic stress (TSC-40) and with symptoms assessed by the HoNOS. Remarkably significant correlations were found between levels of antipsychotic medication and the DES and between antipsychotic medication and the depersonalization /derealization component of the DES in BPD patients.

Conclusion: The results support an important role of dissociative processes in schizophrenia and BPD and suggest a significant relationship between manifestations of dissociative symptoms in BPD and antipsychotic medication.

In future research might be of interest to assess relationship between the concept of splitting in schizophrenia and deficit synthetic metacognition. The synthetic metacognition as a psychological process is capable to synthetisize intentions, thoughts, feelings, and connections between events, and to integrate them into larger complex representations of self and others including also a reflection about that larger representation that involves reflexive functions (thinking about thinking). Synthetic metacognition is measurable by analyzing discourse using standardized procedures.

Key words: splitting, dissociation, metacognition, schizophrenia, borderline personality disorder, antipsychotic medication