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BOREDOM, DOPAMINE, AND THE THRILL OF PSYCHOSIS: PSYCHIATRY IN A NEW KEY

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SUMMARY

Medication non-adherence is a great challenge in the treatment of psychotic disorders. Several factors leading to medication non-adherence in schizophrenia have been identified: drug side-effects, lack of illness insight, negative attitude of the patient and friends/relatives toward medication, stigma of mental illness and taking medication, poor therapeutic alliance, substance abuse, and role of the illness in maintaining the family system.

In this work I propose a new vista on the phenomenon of medication non-adherence in psychosis. Rather rule than exception, non-adherence is to be expected in psychosis, it can be considered as a symptom of psychosis similarly as substance craving and use are symptoms of the substance use disorders. Relying on the last refinements of the concepts of boredom, anticipatory anhedonia, intrinsic motivation, and thrill I assume that there is a lure of psychotic episode. In order to escape an extremely unpleasant and distressing experience of boredom and to experience the thrill of psychosis, the patients are prone to quit antipsychotic therapy.

The phenomena of boredom and the thrill of psychosis are evident but unexploited for strengthening the therapeutic adherence. Making the lure of psychosis an explicit reason for medication non-adherence would bring to the awareness a personal choice between short-term pleasure of the psychotic thrill and prevention of long-term losses due to a psychotic episode. Neurobiological and psychobiological underpinning of the psychotic thrill has been suggested. An explanation of the pleasure of psychosis and substance use, which overcomes the circular explanation of reward in which dopamine appears as the cause and consequence of reward, has been proposed.

The present synthesis can be regarded as a contribution to the field of theoretical psychiatry. It points to a chance for psychiatry to do more for patients' wellbeing and treatment adherence performing in a new key – dealing with boredom and pleasure in patients' everyday life.

Key words: *schizophrenia – psychotic disorders – boredom – dopamine – antipsychotics – compliance*

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INTRODUCTION

It is estimated that more than half of patients with schizophrenia or other psychotic disorder do not follow treatment recommendations (Gibson et al. 2013, Moritz et al. 2013, 2009, McCabe et al. 2012, Kahn et al. 2008, Byerly et al. 2007, Staring et al. 2006, Liberman et al. 2005). On the other hand, medication non-adherence in psychotic disorders raises the risk of psychotic relapse by a factor of three to five (Staring et al. 2006), the risk of suicide nearly by four (Hawton et al. 2005), and is associated with poor quality of life (McCabe et al. 2012). It has been also suggested that untreated psychosis may have neurotoxic effects (Wyatt 1991).

Specifically designed interventions aimed at improving adherence, referred to as 'adherence therapy' or 'compliance therapy' have shown lack of effect on medication adherence in schizophrenia (Gray et al. 2006). That is why the UK National Institute for Health and Clinical Excellence (NICE 2009) guideline for the treatment of schizophrenia advises against using 'adherence therapy'.

Several factors leading to medication non-adherence in schizophrenia have been identified: drug side-effects, lack of illness insight, negative attitude of the patient and friends/relatives toward medication, stigma of mental illness and taking medication, poor therapeutic alliance, substance abuse, and role of the illness in maintaining the family system (Moritz et al. 2013, 2009, Hill et al. 2010, Lacro et al. 2002).

Recently additional reasons for drug discontinuation in psychosis have been postulated. Moritz et al. (2013, 2009) have found that a subgroup of schizophrenia patients has positive attitudes towards positive psychotic symptoms and stops taking antipsychotic medication because the medicines make them free of: delusional feeling of importance and power, hearing voices, and experience of being another person. The finding supports an old notion that "some schizophrenics may prefer an ego-syntonic grandiose psychosis to a relative drug-induced reality" (Van Puten et al. 1976, p. 1443).

On the other hand, Gibson et al. (2013) argue that studies of adherence have not adequately explored consequences and meanings of non-adherence behaviors from patients' perspective. They have found that 'feeling well' day-to-day impacted on schizophrenia patients' treatment choices without taking into account longer-term risks of medication non-adherence.

The motivation for this work arose from an attempt to understand those numerous patients with schizophrenia, who are intelligent and well organized, sometimes academically successful, but whose life course

and achievements do not reach an objectively possible level for only one reason – medication non-adherence. The purpose of the present paper is to further elaborate the thesis about 'feeling well' of psychosis. In particular, the aims are: (1) to describe phenomenology of the pleasure of psychosis and the underlying neurobiology; (2) to understand non-adherence in the context of recent data about dopamine, motivation, anhedonia, and the state of boredom; and (3) to offer an explanation of the pleasure of psychosis and substance use which would overcome the circular explanation of reward in which dopamine appears as the cause and consequence of reward.

PHENOMENOLOGY OF THE THRILL OF PSYCHOSIS

Some descriptions of the pleasant experiences associated with prodromal phase of psychosis are shown in Table 1. They resemble the most pleasant subjective experiences induced by drugs (Earleywine 2005, Jay 2011). These pleasant experiences are neither pleasant dreams nor hallucinations. They are altered, strengthened receptions of the real world and enriched reflections on it; and they are not merely enjoyments of positive psychotic symptoms (cf. Moritz et al. 2013, 2009).

I was wondering if is it possible that these pleasant experiences could be so rewarding that a patient could get addicted to them. My internet search resulted in finding the confessions of two introspective patients who explain how they do use their psychoses to experience pleasure (Table 1). One of them designated his psychosis as 'addiction to the thrill, a metaphysical experience with enormous sense of excitement and immediacy' (Ados 2009). The description corresponds well to the Maslow's (1962) concept of the peak experience and the well documented phenomenon of chills, thrills or frisson (Grewe et al. 2010, Branković 2013).

Similarly, Nathan Foster (2012) compares his psychotic experiences with sexual pleasure and refuses to lose them through proper medical treatment. These two confessions do not prove that all patients reach that level of pleasure in psychosis but represent the extreme cases which illustrate the foundation for our thesis that the (pre)psychotic state could appear as a desirable mental state achievable through medication non-adherence.

NEUROLEPTIC DISPHORIA VS. BOREDOM: LURE OF PSYCHOSIS

The thesis on hedonic aspect of psychosis differs from the well developed concepts of neuroleptic dysphoria (Voruganti & Awad 2004), neuroleptic-induced anhedonia (Wise 2008), negative subjective experiences in patients taking antipsychotics (Mizrahi et al. 2007), and subjective tolerability of antipsychotic medication (Awad 2010). All these concepts imply that many patients treated with antipsychotic drugs experience dysphoric feelings, i.e. develop "a variety of unpleasant subjective changes in arousal, mood, thinking and motivation" (Voruganti & Awad 2004, p. 121). According to these concepts, patients are prone to treatment non-adherence and the use of illicit drugs as a way of relieving the unpleasant dysphoric feelings experienced while on antipsychotic medication (Voruganti & Awad 2004, Voruganti et al. 1997).

On the other hand, our thesis about pleasure of psychosis as a desirable mental state achievable through medication non-adherence holds that a patient in a non-psychotic state, presumably state of balanced dopamine function (see the next section) could like and want to experience pleasures of (pre-)psychotic phase described in Table 1.

Contrary to the differences from neuroleptic dysphoria and neuroleptic-induced anhedonia, our thesis on hedonic character of psychosis has much in common with some other recently developed concepts – (1) differentiated construct of anhedonia (Argyropoulos & Nutt 2013, Strauss & Gold 2012, Der-Avakian & Markou 2012, Treadway & Zald 2011, Choi et al. 2013, Buck & Lysaker 2013, Engel et al. 2013, Simpson et al. 2012, Gard et al. 2007); and (2) the phenomenon of boredom (Eastwood et al. 2012, Martin et al. 2006, Bench & Lench 2013).

Table 1. Pleasant experiences reported by patients with psychosis

"I developed a greater awareness of… My senses were sharpened. I became fascinated by the little insignificant things around me" (Bowers & Freedman 1966)
" Sights and sounds possessed a keenness that he (the patient) had never experienced before" (ibid.)
"It was as if parts of my brain awoke, which had been dormant" (McDonald 1960)
"My senses seemed alive… Things seemed clearcut, I noticed things I had never noticed before" (Bowers 1968)
"I felt that there was some overwhelming significance in this" (McDonald 1960)
"I felt like I was putting a piece of the puzzle together" (Bowers 1968)
"My capacities for aesthetic appreciation and heightened sensory receptiveness… were very keen at this time." (Anonymus 1950)
"Psychosis – addicted to the thrill: … it was rather a metaphysical experience… enormous sense of excitement and immediacy. While I'm normally a person that is not easily entertained or motivated, in psychosis I was plunged into a world full of mystery and discovery… my theory, that delusional disorder is an emotional compensation for needs that cannot be satisfied in reality… it is the addiction to the thrill of psychosis…" (Ados 2009)
"Psychosis as sexual pleasure: Psychosis can actually be one of the most pleasurable experiences you can have." (Foster 2012)

A view that anhedonia should be regarded as a heterogeneous symptom was suggested three decades ago (Peterson & Knudson 1983). Dealing with the concept, Klein (1987) and Argyropoulos & Nutt (1997) introduced the distinction between "appetitive" (anticipatory) and "consummatory" pleasure. The distinction showed useful during the last decade revealing a more differentiated view on motivational deficit in schizophrenia. It was found that anhedonia in patients with schizophrenia is not due to an inability to experience in-the-moment (consumatory) pleasure as hedonic reaction appears intact in patients. In other words, the patients do not manifest "consumatory anhedonia". Rather, the motivation deficit in schizophrenia reflects a reduced capacity for anticipating future pleasure resulting from goal-directed action, what is denoted as "anticipatory anhedonia" (Germans & Kring 2000, Gard et al. 2007, Strauss & Gold 2012, Simpson et al. 2012, Engel et al. 2013, Buck & Lysaker 2013).

Patients with anticipatory anhedonia could be prone to quit antipsychotic therapy in order to experience the thrill of psychosis (Table 1) as an equivalent behavior to the cannabis use in patients on antipsychotic medication who reported lower anticipatory pleasure (Cassidy et al. 2012).

Another concept, phenomenon, recently employed in theorizing on the subjective experience and course of schizophrenia and other psychotic disorders, which is congruent with our thesis about (pre)psychotic state as a preferable, pleasurable mental state, is the experience of boredom (Todman et al. 2008, Todman 2003). "Boredom is universally conceptualized as the aversive experience of wanting, but being unable, to engage in satisfying activity" (Eastwood et al. 2012, p. 482). Boredom is an extremely unpleasant and distressing experience (Martin et al. 2006). It can also be a chronic and pervasive stressor with significant psychosocial consequences such as death and a range of physical and mental health problems (Eastwood et al. 2012).

"Like anxiety, boredom has an important signal function: it informs us that the behavioral strategies currently in use have ceased to be effective in extracting novelty and positive reinforcement (pleasure) from a given environment" (Todman et al. 2008). Todman and colleagues (2008) suppose that "anhedonia (I would say 'anticipatory anhedonia' (see above)) and boredom exist on functional continuum of severity, with (anticipatory) anhedonia occurring when a persistently bored individual concludes that the source of their boredom is internal, uncontrollable, inescapable and permanent". In other words, Todman and collaborators have offered us a genetic definition of anticipatory anhedonia. The authors have also provided an empirical evidence for the thesis finding that in a sample of patients with schizophrenia, schizoaffective disorder, and bipolar disorder greater amounts of sustained boredom was associated with diminished expectations of future reward, with the notion that such an adaptation was more prevalent among the individuals with schizophrenia. They interpret this finding as "being consistent with a conception of (anticipatory) anhedonia as an extreme adaptation to sustained boredom in which there is an increasing conviction that monotony is permanent, ubiquitous, and largely uncontrollable". Another important finding of the study is that current substance abuse is associated with frequency of boredom over the last two weeks, demonstrating, according to the authors, the potential utility of the state boredom as a marker for a patient's current risk for substance abuse. On the other hand, the study also revealed the strong correlation between medication non-compliance and substance use.

A similar view on boredom as a discrete emotion with signal function has been recently elaborated by Bench & Lench (2013). The authors explain that as the intensity of an emotional experience of any kind diminishes, fades, the unpleasant emotional state of boredom arises and acts as an emotional signal that the current situation is no longer stimulating (i.e. is associated with less intense emotion) and encourages us to seek alternative, new goals and experiences. Further, "boredom does not discriminate the valence of a goal that should be switched to, it simply encourages changing to a new goal… Due to this, boredom could encourage changes that result in negative emotion… Boredom could even motivate goals that result in actual risk" (ibid. p. 462). The preferences for novel stimuli, including risky situations, are reflected also in choices of unfamiliar situations and objects, and challenging tasks. A challenging task and alternative goal for the patient in the state of boredom is certainly a discontinuation of medication and the pursuit of the pleasures and the thrill of the (pre-)psychotic phase. The option is likely "given that many patients (with schizophrenia) have limited resources and fewer opportunities for pleasure than healthy individuals" (Strauss & Gold 2012, p. 372). There is also a subjective factor for this, presumably the least creative solution, since "coping skills of individuals with severe and persistent mental illnesses such as schizophrenia and schizoaffective disorder are likely to be less effective, less efficient, less well developed and used less competently than those of healthy controls. It would therefore seem safe to assume that this would also apply to the coping strategies typically employed to avoid and manage boredom" (Todman et al. 2008).

For these reasons I assume that there is a lure of psychotic episode. In order to escape an extremely unpleasant and distressing experience of boredom and to experience the thrill of psychosis (Table 1), the patients are prone to quit antipsychotic therapy. This line of reasoning leads us to propose a new vista on the phenomenon of non-adherence in psychosis. Rather than being an exception or a reaction to adverse side-effects of antipsychotics, non-adherence is to be expected in psychosis. It can be considered as a feature of the course of psychosis in a similar way as substance craving and use characterize the substance use disorders.

NEUROBIOLOGY OF THE PSYCHOTIC THRILL: ELEVATED TONIC AND/OR PHASIC DOPAMINERGIC SIGNALING

What neurobiological mechanisms underpin the above described phenomena of psychotic pleasure (Table 1), escape from boredom, and lure of psychosis? There is abundant evidence that dopaminergic dysfunction underlies psychotic states and that the brain dopamine signaling is the primary common target for all antipsychotic drugs (Howes et al. 2009, Seeman et al. 2006, Seeman 2002). On the other hand, dopamine signaling is associated with different types of pleasure such as: sexual activity (Robinson et al. 2002), appetitive maternal behavior (Robinson et al. 2011), aesthetic pleasure (Salimpoor et al. 2011), intracranial self-stimulation (Owesson-White et al. 2008), and pleasures associated with substance use (Wanat et al. 2009, Volkow et al. 2007, Le Merrer et al. 2007, Grace 2000a, Polache & Granero 2013, Deehan et al. 2013). The association of dopamine activity with pleasure has brought many authors to the conclusion that "the crucial reward neurotransmitter in the brain is dopamine" (Gardner 2011, p. 30, see also Baik 2013, Berridge & Kringelbach 2011, Schultz 2010, Wightman & Robinson 2002).

Many theories have been developed to explain involvement of the dopamine system in psychosis (Seeman et al. 2006, Yang et al. 1999, Grace 1991, 2000b, Branković & Paunović 2002) and also many to clarify its role in the development of addiction (Wanat et al. 2009, Volkow et al. 2007, Kalivas 2002, Grace 2000a). Moreover, there is a conceptualization of dopamine signaling which has shared use in theorizing on psychosis, reward and addiction. It is the concept of dopamine as a mediator of motivational salience of environmental and internal stimuli. According to Kapur, a hyperdopaminergic state "leads to an aberrant assignment of salience to the elements of one's experience. Delusions are cognitive effort by the patient to make sense of these aberrantly salient experiences, whereas hallucinations reflect a direct experience of the aberrant salience of internal representations" (Kapur 2003, p. 13, see also Howes et al. 2009). Similarly, Robinson & Berridge (1993, 2008), Berridge & Robinson (1998), Berridge (2007, 2012), Robinson et al. (2013) proposed an explanation of reward and addiction employing the process of attribution of incentive salience to the drug mediated through sensitization of the mesolimbic dopamine system.

Moreover, the tonic-phasic balance of dopamine activity has been proposed to underlie normal and dysfunctional regulation as it relates to the pathophysiology both of schizophrenia and drug abuse (Grace 2002). Hamamura & Harada (2007) have suggested that due to the augmentation to a different extent of the phasic and tonic dopaminergic components, depending on the individual's pathophysiology, either "loose" antipsychotics (which dominantly suppress tonic activity) or "tight" ones (which equally diminish the tonic and phasic dopamine component) or aripiprazole (as "the phasic component buster") could be more effective than others. The proposed dopaminergic heterogeneity of schizophrenia parallels the dopaminergic effects of addictive substances (Table 2). For instance, alcohol, nicotine, opiates, and marijuana activate dopamine system primarily by increasing dopamine neuron spiking activity, i.e. augmenting the phasic dopamine function. Contrary, psychostimulants such as cocaine, amphetamine, methamphetamine, and ecstasy increase extracellular dopamine levels via a blockade of dopamine uptake system, i.e. they cause a much greater increase in the tonic dopamine function (Volkow & Li 2004, Grace 2000a, Bass et al. 2013).

Dopamine does not directly produce a motor output or reward signal, but instead acts on the gating of inputs, modulates inputs strength, and regulates information flow between compartments of neurons (Grace 2002, Yang et al. 1999). In the following section some specific roles of the tonic and phasic component of dopamine activity are further discussed.

BEYOND THE CIRCULAR EXPLANATION OF REWARD

The well established association between dopamine signaling and different types of pleasure (see the previous section) has caused some narrowing of the reward research focusing on the dopamine functioning. But, explanations such as "everything you want releases dopamine and you want it because it releases dopamine… Things are important and valuable only if they activate your dopamine neurons" (Nowell 2011) are circular. The circular logic is reflected also in one of

Table 2. Similarities between proposed dopaminergic profiles of psychoses and addictive substances

Dopaminergic heterogeneity of psychoses (Hamamura & Harada 2007)		Dopaminergic heterogeneity of addictive substances (Volkow & Li 2004, Grace 2000a, Bass et al. 2013)		
Tonic DA activity	Phasic DA activity	Tonic DA activity	Phasic DA activity	
↑	↑	↓	↑	alcohol, nicotine, opiates, marijuana
↑	↑	↑	↓	cocaine, amphetamine, ecstasy
↑	↑			

the criteria of identifying dopaminergic neurons – whether they respond to reward (Hong 2013). On the other hand, "it seems that increases in dopamine are not directly related to reward per se, as was previously believed, but rather to the prediction of reward and for salience. Salience refers to stimuli or environmental changes that are arousing or that elicit an attentional-behavioural switch" (Volkow & Li 2004, p. 964). The two currently most prominent hypotheses about what type of information is encoded by dopaminergic activity in the midbrain are: (1) the reward-prediction error hypothesis (Schultz et al. 1997) and (2) the incentive salience hypothesis of dopamine (Berridge 2007). But, "they (both) do not explain by themselves why or how people and other animals display certain types of phenomena related to learning, decision-making or motivation" (Colombo 2013, p. 9).

Beside the circularity of the dopamine thesis of reward, there is a need to understand reward in a larger neural framework since other monoamines and subcortical structures are involved in the brain's reward circuitry (Nakamura 2013, Mannella et al. 2013, Hikosaka et al. 2008, Vetulani 2001, Redgrave et al. 1999). Here I propose an explication of the separate roles of the tonic and phasic dopamine activity which: (1) overcomes the circular explanation of reward in which dopamine appears as the cause and consequence of reward and (2) frames the dopamine signaling in a broader scheme of the brainstem monoamines' mechanisms.

One notion is important for understanding the present model and its relevance for the psychotic thrill. It is the intense nature of the brain stimulation reward. "In awake humans, electrical stimulation can evoke intense subjective feelings of pleasure, in some instances similar to descriptions of intense medieval religious ecstasies. As the most addictive drugs (e.g. cocaine, methamphetamine) evoke comparable levels of subjective reward, it is easy to understand their intensely addictive nature" (Gardner 2011, p. 25). The question is how, through what mechanisms, dopamine contributes to experiencing these intense feelings of pleasure.

Firstly, tonic dopamine increases the gain of the input-output response of the pyramidal neurons (Thurley et al. 2008) and regulates information flow between compartments of neurons (Grace 2002, Yang et al. 1999). Through this "gain-amplification" mode tonic dopamine affects processing in target structures innervated by the mesolimbic, mesocortical, and mesostriatal pathways (Robbins & Everitt 2007), what explains why higher levels of dopamine are associated with more vigorous responding (Beierholm et al. 2013, Niv et al. 2006). For instance, both dopamine agonists and antagonists alter the strength of amygdala response to emotional stimuli (Salgado-Pineda et al. 2005, Takahashi et al. 2005, Exner et al. 2004). Considering these data in the context of the recently proposed scenario of neural computations during emotional responding to pleasant emotional stimuli I have suggested that the features (amplitude and duration) of the initial neural event in the process of emotional responding depend not only on the characteristics of the encountering stimulus but also on the tonic monoaminergic (including dopaminergic) innervation of the amygdalo-hippocampal circuit (Branković 2012, 2013) (Figure 1).

Dopamine is not the only neurotransmitter engaged in rewarding behavior and addiction. Noradrenergic and serotonergic systems also contribute to reward behavior and development of addiction (Nakamura 2013, Hikosaka et al. 2008, Vetulani 2001, Redgrave et al. 1999). Moreover, in his neurobiological model of emotion regulation Lewis (2005) emphasized multiple positive and negative feedback components originating from brainstem which back up to amygdala and other cortical and subcortical systems increasing the activation of some systems while decreasing it in others. According to the known neurobiology of emotional arousal the first candidates for the neurochemical substrate of the feedback loops could be bidirectional connections among amygdala, hypothalamus, and the brainstem (Pfaff et al. 2005), including brainstem originating monoaminergic inervation of amygdale, hypothalamus, and hippocampus.

The finding that interlinked positive and negative feedback loops design emotional response to pleasant stimuli (Branković 2011, 2012) puts more light to the joint contribution of the three brainstem monoamines to experiencing pleasant emotional arousal. According to the model, emotional response arises through a series of integrations with three feedback loops (Figure 1).

The foremost activated feedback loop in the model of emotional response to pleasant stimuli is positive and it presumably corresponds to the phasic dopamine activity. It rapidly induces the "on" state of the reward response (pleasant excitement) system. The third feedback loop in the model is the delayed positive feedback. It reflects the phasic serotonin activity and it robustly maintains the "on" state of the emotional component of the reward response. In between these two positive feedback loops is situated a negative feedback loop, which is proposed to reflect phasic noradrenergic function of the locus coeruleus. It reinstates the system in the original "off" state, prevents excessive response due to multiple positive feedback loops, and suppresses noise effects (Kim et al. 2006, Pfeuty & Kaneko 2009).

The dual-time switch, consisting of interconnected fast and slow positive feedback loops has been identified in many biological systems as the key regulatory scheme in the process of creation of output (Tsai et al. 2008, Brandman et al. 2005, Brandman & Meyer 2008). Mathematical simulations revealed that such coupled feedback circuits enable systems in noisy environment to produce perfect responses with respect to the response duration and amplitude (Pfeuty & Kaneko 2009, Mitrophanov & Groisman 2008, Kim et al. 2006). It is a mechanism which is able to assure both "quick onset" and "brief duration" as fundamental and adaptive characteristics of emotions (Ekman, 1992, Branković 2011).

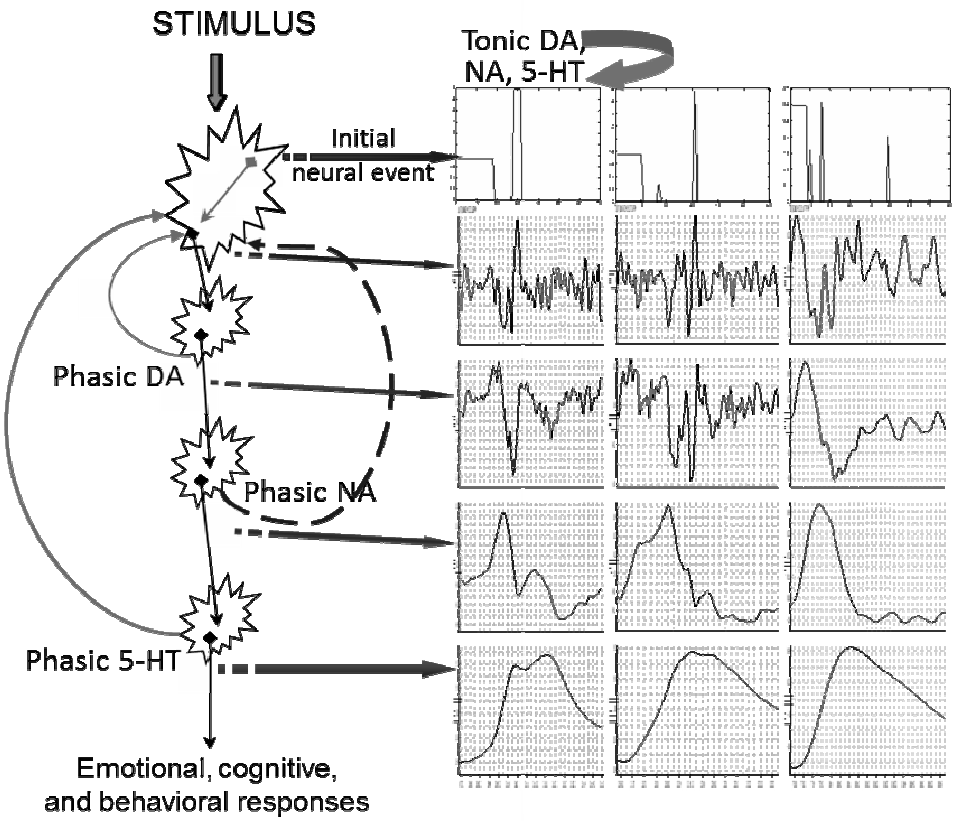

Figure 1. The response system to pleasant stimuli: a series of integrations with feedback (monoaminergic) loops which enable a transition from the time-scale of the brain operation (tens to hundreds of milliseconds) shown in the first row to the time-scale of emotional, cognitive, and behavioral responses (seconds or tens of seconds) shown in the last row. DA – dopamine, NA – noradrenaline, 5-HT – serotonin

The proposal that the phasic dopamine acts as a fast switch, which rapidly induces the "on" state of the reward (pleasant excitement) response, is in line with the thesis that "the initial burst of dopaminergic-neuron firing could represent an essential component in the process of switching attentional and behavioural selections to unexpected, behaviourally important stimuli" (Redgrave et al. 1999, p. 146). The suggestion that phasic serotonin acts as the delayed, second feedback loop in the reward response concurs with the evidence on differences in the temporal pattern of activity of dopamine and serotonin (Nakamura 2013).

An interpretation of the model of emotional response to pleasant stimuli (Figure 1, see also Branković 2011, 2012) could be that the series of integrations with feedback loops enables a transition from the time-scale of the brain operation, which is tens to hundreds of milliseconds (Varela et al. 2001, Koch et al. 1996, Koch 2005), to the time-scale of emotional, cognitive (e.g. focusing attention), and behavioral responses, which is seconds or tens of seconds (Ekman 1992). The transition is realized through the feedback enhancements from the brainstem, whereby dopamine has a special, the first position, in the regulatory chain providing a prompt turning on the emotional, cognitive, and cognitive response (cf. Redgrave et al. 1999). Probably, that position renders dopamine crucial for addictive behavior and prominent in the reward research.

The presented model of the aesthetic experience (elaborated in Branković 2011, 2012, 2013) fits well with the proposals: (1) that the brain reward system is closely related to the system of emotional arousal (Vetulani 2001) and "that the reward network and the arousal (or circadian) network have evolved by sharing the same mechanism (Hikosaka et al. 2008, p. 206), (2) that the dopamine system functions as "a manager of neural pathways" enabling through bidirectional modulation reward, alerting, vigor, etc. (Hong 2013), and (3) the model of a general catecholamine response to salient stimuli which enables behavioral switching initiated by significant events (Redgrave et al. 1999).

Also, this computational model of aesthetic arousal joined with "the two-factor model of hedonic value of stimuli" Branković (2001), which assumes unexpectedness (surprise) of stimuli as a necessary condition for eliciting pleasant excitement, is congruent with the proposals that both phasic dopamine (Schultz 2010, Kakade & Dayan 2002, Redgrave et al. 1999) and phasic noradrenaline (Dayan & Yu 2006) encode surprise (unexpected event). Moreover, the model explaines the mechanism (Figure 1) in which phasic dopamine and phasic noradrenaline (and also phasic serotonin) act as feedback signals at different levels of the neural integration over the cascade of the emotional arousal.

In the following section we continue with a framing of the intense subjective feelings of pleasure (arousal)

evoked by electrical stimulation, addictive drugs (Gardner 2011), or associated with hyperdopaminergic (pre)psychotic state into a broader context of human motivation.

EMBEDDING BOREDOM AND PSYCHOTIC THRILL INTO AN INTRINSIC MOTIVATIONAL FRAMEWORK

The thesis that the lure of psychotic episode consists in both providing an escape from boredom and experiencing pleasant excitements (thrills) in everyday life through elevated tonic and/or phasic dopaminergic signaling, similarly to the effects of drug abuse, can be viewed from a broader perspective, that of the intrinsic motivation. Already Bozarth (1990) has pointed to the relevance of motivational psychology for understanding the nature of drug addiction: "…the conceptual advances made in motivational theory can be used to guide the study of addictive behavior… fitting addiction into the framework provided by general motivational theory".

Relying on the Joseph Lichtenberg's (1989) conceptualization of motivation, where five mutually irreducible motivational systems have been delineated, we can notice that boredom emerges neither due to unsatisfying "the motivational system based on the psychic regulation of physiological requirements" (such as hunger, thirst, and sleep) nor "the aversive motivational system" (comprising fear, anger, disgust, etc.). On the other hand, boredom seems to be related to an inability to engage in pursuing in the other three Lichtenberg's (1989) motivational systems: "the attachment", "the exploratory/assertive", and "the sensual/sexual motivational system".

Pointing to an analogous nature of the sexual, the attachment, and the exploratory motivation I have exposed the theory of informational needs (TIN) and proposed a possible neurobiological substrate underlying the common, cyclic and informational, character of these three needs (Branković 2001, 2013). The analogy among the three informational needs (sexual, emotional, and exploratory) consists in the experience of excitement which accompanies satisfying of any of the three needs. Experiences of the sexual excitement, the emotional excitement (pleasure-in-intimacy), and the exploratory excitement are different feelings (Lichtenberg 1989). They sometimes reach the level of the respective kind of the peak experience – sexual orgasm, "emotional" orgasm ("emotional" peak experience), and "exploratory" orgasm ("exploratory" peak experience). The peak experiences are accompanied by somatic manifestations such as the phenomenon of chills, thrills or frisson (Branković 2013). The cycles of the informational needs include in an analogous fashion four phases: (1) the desire phase, (2) the excitement phase, (3) the peak experience phase, and (4) the resolution phase accompanied by refractory period to the kind of excitement in respected motivational system, i.e. the last one which brought to the peak experience (Figure 2). The phases correspond well to the description of the stages of the aesthetic expirience elaborated by Roman Ingarden (1968, 1969).

Affective character of the desire (appetitive) phase of the informational needs was described as "unpleasant psychic tension" in Branković (2001, p. 33). Here I argue that a blockade of the cycles of satisfying the informational needs at the desire phase, a prolongation of the desire phase and not a lack of desire (what could be the case in melancholy), leads to the experience of boredom. The inability to proceed to the next phase, to experience pleasant excitement (emotional, sexual or exploratory) could be caused by both objectively inadequate stimulation due to lack of stimulus uncertainty (unpredictability) and/or stimulus familiarity (see 'the two-factor model of hedonic value of stimuli' in Branković (2001, 2013)) and subjective factors such as poor, insuficient attention to become engrossed in satisfying activity (Eastwood et al. 2012). The question is how long is the time period of the subjective tolerance for this blockade, not moving on through the cycles of informational needs. I assume individual variation of the tolerance. I guess a range of its inter-individual varying from hours to days. The estimation is comparable with that suggested by Todman et al. (2008) in "The State Boredom Measure" where boredom is supposed to last and to be measured from less then few minutes up to two weeks. However, after the expiry of the personal time period of the tolerance extremely unpleasant and distressing experience of boredom eventually emerges and can be followed by the boredom escape behaviors including risky ones (Bench & Lench 2013, Draus & Carlson 2009, Martin et al. 2006, Anselme & Robinson 2013, Anselme 2013, Ashford 1996), unless a reentering into the cycles of the informational needs succeeds (Figure 2).

A similar cyclic model of pleasure, "a theoretical schematic depiction of the various facets of anhedonia and their relationship" has been recently proposed by Argyropoulos & Nutt (2013, p. 874). There are also four stages in the cyclic schema of Argyropoulos & Nutt: (1) the seeking stage (corresponding to the desire phase of the TIN), (2) anticipatory pleasure, (3) reward, enjoyment or consumatory pleasure, and (4) satiation or saturation (corresponding to the resolution phase of the TIN).

The present conceptualization of boredom through its embedding into the cycles of the informational needs enables a better understanding of the Martin Heidegger's (1995) reasoning on the subject: "If things evidently have their time in each specific case, and if we precisely come across things in their specific time, then perhaps boredom will fail to appear. Conversely: boredom is only possible at all because each thing, as we say, has its time. If each thing did not have its time, then there would be no boredom" (p. 105)… "Boredom springs from the temporality of Dasein. Boredom therefore, we may say in anticipation, arises from a quite determinate way and manner in which our own temporality temporalizes itself. This tallies with the thesis that we announced earlier, namely that boredom is possible only because every thing, and more fundamentally every Dasein as such, has its time" (p. 127).

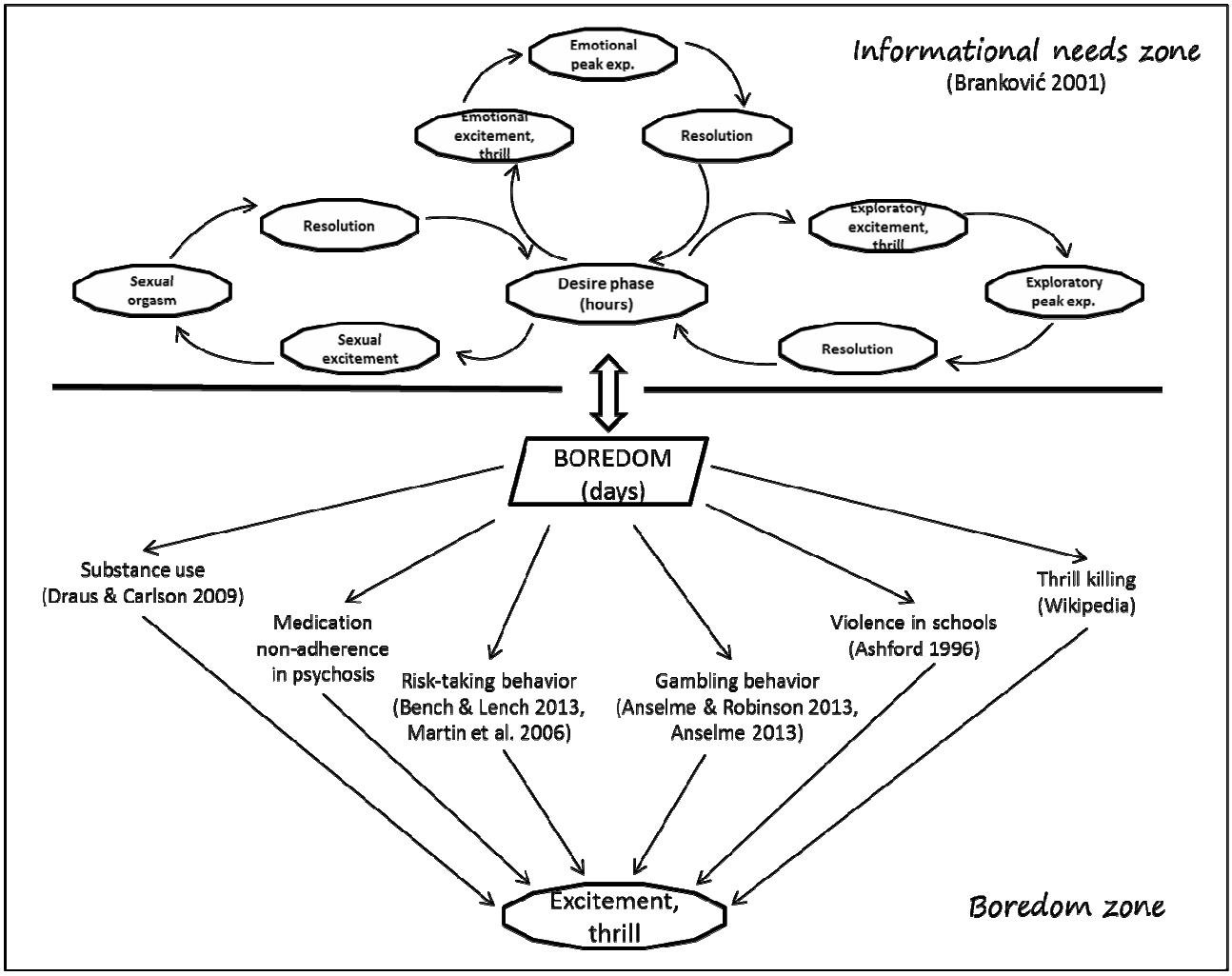

Figure 2. Cycles of informational needs, boredom, and boredom escape routines

Criticizing Heidegger's analysis of boredom, Lars Svendsen (2005) finds a weakness in the analysis when Heidegger contends that "boredom springs from the temporality of Dasein". Svendsen argues that since boredom can arise due to certain environment it is not necessary to turn to our temporality of Dasein. Realizing the correspondence between the concepts of the Heidegger's temporality of Dasein and the cyclic nature of the informational needs I argue that the presented view on boredom as a stoppage at the desire phase of the cycles of the informational needs justifies Heidegger's insistence on the temporality of Dasein for understanding boredom.

TREATMENT IMPLICATIONS

Our thesis on the lure of psychosis has its therapeutic implications. We can assume a benefit from informing a patient and his or her family that the (pre)psychotic phase could be experienced as a pleasurable mental state, biologically comparable to a kind of "inner substance abuse". Then, working with the patient and the family it could be useful to point to the risk of the occurrence of the state of boredom which could activate craving for the psychotic trill, which itself is easily achievable through quitting antipsychotic medication. Making this reason for medication non-adherence explicit would bring to the awareness the personal choice between short-term pleasure of the psychotic thrill and prevention of long-term losses due to a psychotic episode.

More specifically, in addition to "adherence/compliance therapy" (Gray et al. 2006) and "anticipatory pleasure skills training" (Favrod et al. 2010) I suggest that psychoeducation on the psychobiology of boredom and healthy mechanisms to overcome this extremely unpleasant and distressing experience (through reentering the informational needs zone, Figure 2) could be beneficial for both the strengthening one's motivation for mental health and rendering the goal feasible. The proposal is not a new one since already Todman (2003, p. 163) has suggested that "it might be possible to improve the boredom-coping skills of some boredom-prone individuals with adequate training… These (leisure-seeking skills as part of a more general social skills training) will probably not be engaged appropriately unless there is some awareness on the part of the patient of the significance of his or her sustained boredom… It seems clear that the challenge for treating clinician is to convince the patient to become interested in his or her boredom".

A novelty of the present work is the theoretical proposal which frames the state of boredom into a broader conceptualization of intrinsic motivation, i.e. the theory of informational needs (Branković 2001), which enables overcoming both negative (see Svendsen 2005) and circular (see Eastwood et al. 2012) defining of boredom. That is in line with the recently emerged research topic on the relationship among intrinsic motivation, neurocognition, and psychosocial functioning in schizophrenia (Vohs & Lysaker 2014, Tas et al. 2012, Choi & Medalia 2010, Medalia & Brekke 2010, Nakagami et al. 2010, Nakagami et al. 2008, Barch & Dowd 2010, Gard et al. 2009, Barch et al. 2008). The most prevalent conclusion of this research is that "intrinsic motivation is a critical mechanism for explaining the relationship between neurocognition and psychosocial functioning" (Nakagami et al. 2008, p. 95). Therefore, "treatments should be geared towards enhancing an individual's intrinsic motivation" (ibid. p. 101). I assume that the theory of informational needs (Branković 2001, 2013) and the presented psychobiological model of boredom (Figure 2) offer a sound conceptual ground for designing a treatment strategy which would revive one's intrinsic motivation yielding consequently a better psychosocial functioning and personal wellbeing.

CONCLUSION

In this work I have proposed a new vista on the phenomenon of medication non-adherence in psychosis: non-adherence is to be expected, rather rule than exception, it can be considered as a symptom of psychosis similarly as substance craving and use are symptoms of the substance use disorders. The phenomena of boredom and the thrill of psychosis are evident but unexploited for strengthening the therapeutic adherence. Making the lure of psychosis an explicit reason for medication non-adherence would bring to the awareness a personal choice between short-term pleasure of the psychotic thrill and prevention of long-term losses due to a psychotic episode.

Neurobiological and psychobiological underpinning of the psychotic thrill has been suggested. Through integration of the principles of dopamine signaling with the last refinements of the concepts of boredom, anticipatory anhedonia, intrinsic motivation, and thrill, the present synthesis can be regarded as a contribution to the field of theoretical psychiatry (Jakovljević 2013). It points to a chance for psychiatry to do more for patients' wellbeing and treatment adherence performing in a new key – dealing with boredom and cyclic and informational nature of pleasure (Branković 2001) in patients' everyday life.

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