are characterized by affective content; therefore, a cognitive framework of hallucinations does not come up to the mark. The tendency to link consciousness to cognition has led to definitions of consciousness that leave out any reference to meaning, emotion, and qualia. In these theoretical accounts, the neural correlate of consciousness (NCC) is suggested to involve widely distributed thalamo-cortico-cortical networks preferably resonating at gamma frequency rhythms. Gamma frequency oscillations and synchronization occur in the visual cortex after the detection of scenes, such as randomly moving dots. But is the detection of these meaningless scenes not a misadaptation supervening on the incapability of the visual-attentional system to shut down when nothing of interest is happening? Consciousness cannot be equated with such epiphenomenal forms of detection, but probably defensibly evolved to provide for more flexibility in social-emotional contexts, which are packed with meaning and stuffed with raw feelings – that is, qualia and emotions (Buck 1999; Ressler 2004; Schutter & Van Honk 2004a). This is exactly why hallucinations in schizophrenia carry their typical affective tone, as described by B&Y. The authors nevertheless discuss schizophrenia as a disorder of consciousness and cognition, and not primarily as a mood disorder. This is somewhat surprising, because schizophrenics have hallucinations with a strong negative affective content, and it is precisely this content which constitutes the essence of their suffering, because it makes them anxious, depressive, and suicidal (Meltzer & Fatemi 1995). B&Y, on the other hand, suggest that schizophrenia is often preceded by social anxiety and that schizophrenic hallucinations (not unexpectedly) often relate to social fear. Although the comorbidity between schizophrenia and social anxiety has been established (Pallanti et al. 2004), it does not fit with B&Y's reasoning that mood or emotion disturbances are only secondary to schizophrenia. Perhaps more on the right track, Lane (2003) recently argued that the core feature of schizophrenia is a deficit in affective function. In agreement, neuroimaging findings have provided evidence for the notion that the complex nature of affective abnormalities in schizophrenia is indeed associated with processing difficulties in subcortical emotion circuits (Paradiso et al. 2003).

In sum, the traditional cognitive-oriented explanations of consciousness and hallucinations emphasize thalamocortical architectures, whereas the affective-oriented interpretations stress the involvement of subcortical brain regions (Damasio 1999; Panksepp 1998). The subcortical structures generate the primary motivational and emotional drives and the cortical mantle is argued to internally represent and control the afferent subcortical information streams (Phillips et al. 2003; Schutter & Van Honk 2004b). Schizophrenia arguably finds its source on the subcortical level and, in particular, subcortical dysfunction that overrides cortical regulation might be the core brain deficit (Grossberg 2000). It is only on the cortical level that emotion and cognition interact; therefore it is not the affective, but the cognitive, deficit that is secondary to schizophrenia. The morphological brain abnormalities in schizophrenia that have been demonstrated in subcortical affective circuits (e.g., Sanfilipo et al. 2000) add further evidence to the notion that schizophrenia is primarily a disorder of emotion. In particular, positive symptoms in schizophrenia, which include hallucinations and delusions with negative affective content, can be explained in terms of defective cortical–subcortical interaction. The often affect-laden content of hallucinations and delusions arguably points at a cortical malfunction in the effective modulation of subcortical affective output. Moreover, this notion fits with findings of cortical hypoactivity and limbic hyperactivity in schizophrenia (Davis et al. 1991). Furthermore, recent findings by Epstein et al. (1999) suggest that positive symptoms are associated with increased mesotemporal and striatal activity in the context of decreased prefrontal activity. In particular, the amygdalar formation located in the mesotemporal region is argued to contribute to the affective nature of psychoses (Taylor et al. 2002).

According to B&Y, consciousness arguably involves constraining and fusing sensory input through prefrontal modulations. The lack of cortical control over the affective subcortical circuits may consequently manifest itself in a maelstrom of interoceptive and exteroceptive information left unbounded by prefrontal regulation. The inability to fit the perceptual input with the necessary internal schemata is suggested to lead to the positive symptomatology in schizophrenia (Kaprinis et al. 2002). From the functional neuroanatomical perspective, schizophrenia has also been described as a "misconnection" syndrome (Andreasen 1999), which refers to defective functional connectivity in the brain (Paradiso et al. 2003). Loss of executive frontal function might result in a derailment of cognitive processes, termed "cognitive dysmetria" (Andreasen 1999), but we mean to argue that perhaps it is better to use the term "affective dysmetria." Although consciousness and cognition are argued to stem from the higher cortical brain areas, they are both built on primordial motivational and emotional drives seated in the limbic system (Panksepp 1998). The hierarchical brain architecture implies an important role for affect in relation to consciousness, cognition, and psychopathology (Maclean 1990). Therefore, B&Y's cognitive explanation of consciousness and hallucinations in schizophrenia can in no way account for the emotional abnormalities observed in schizophrenia. Emotion is not merely the coloring of cognitive information processing or part of cognition, but rather is the essence of our processing system that controls consciousness and cognition (Damasio 1994).

We conclude that schizophrenia is a disorder of affective consciousness involving subcortically driven dysfunctional cortico–limbic interaction and accompanied by secondary cortical abnormalities in conscious aspects of cognition.

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Distinguishing schizophrenia from the mechanisms underlying hallucinations

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Abstract: This commentary challenges the argument that the diathesis for hallucinations is equivalent to that for schizophrenia. Evidence against this comes from data on the prevalence of hallucinations in schizophrenia, their nonspecificity, and their relationships with moderating variables. We also highlight, however, the manner in which the Behrendt & Young (B&Y) hypothesis extends recent neuroscientific theories of schizophrenia, and its potential treatment applications.

Behrendt & Young (B&Y) propose a theory of hallucinations that departs greatly from traditional views of this symptom, especially as applied to mental illness. On the other hand, some of its basic assumptions are consistent with current neuroscientific theories of schizophrenia. There are also several areas where the theory is either incomplete or unable to account for existing data. In this commentary, we will consider three issues: (1) the consistency of the authors' argument with our recently proposed view of cognitive coordination failures in schizophrenia; (2) general strengths of the article, especially in terms of applications to schizophrenia treatment; and (3) weaknesses of the theory for understanding schizophrenia.

B&Y's focus on abnormalities involving gamma-oscillations in schizophrenia is consistent with our recently proposed theory (Phillips & Silverstein 2003), which focuses on NMDA receptor hypofunction as the basis for reduced context-based cognitive coordination, and therefore as the basis of multiple forms of disorga-

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nization in schizophrenia (e.g., in perception, language, motor activity). The authors' concentration on hallucinations and consciousness goes beyond our focus on multiple manifestations of disorganization, and demonstrates the power of applying a model involving disturbed gamma-phase oscillations to other aspects of the illness. Similarly, their concentration on thalamic involvement in gamma-phase synchrony goes beyond our focus on local cortico-cortical circuitry found throughout the cortex. We suggested that other parts of the brain were likely to be involved and that the thalamus could provide the precise timing signals necessary for the binding of network activity, but this target article more clearly describes a role for subcortical structures. In addition, the authors' focus on the relationship between arousal and the onset of processing disturbances that could lead to hallucinations, and the roles of noradrenaline and acetylcholine in this process, demonstrates how disturbances in coordination can result from a cascade of events involving neurotransmitters other than dopamine and glutamate.

A general strength of the target article is that it emphasizes mechanisms hypothesized to be common to both "normal" perception and hallucinations, as well as specifying what factors lead to the emergence of the latter. This is similar to work being done regarding delusions, which views them as extreme forms of normal beliefs, as opposed to being discontinuous with normal experience, which has been the traditional view of positive symptoms for many years (Jaspers 1916/1963). The reconceptualization of the nature of delusions has formed the basis for successful cognitive-behavioral therapies, involving cognitive reframing techniques, for delusions, paranoid ideation, and distress secondary to hearing hallucinated voices (Chadwick et al. 1996; Kingdon & Turkington 1994). B&Y's thesis, if supported by future research, could provide a normalizing rationale for hallucinations that could greatly assist in psycho-educational and cognitive-behavioral treatment of this symptom. Another strength of the article is the recognition (in sect. 2) that hallucinations are unlikely to be simply thoughts or inner speech that are misattributed to an external origin. In suggesting that the key mechanism involves a deficient balance between the processing of external and internal stimuli rather than simply faulty self-monitoring, B&Y are laying the groundwork for a paradigm shift in how hallucinations are understood and studied. We believe this is important because it is unlikely that all phenomena in schizophrenia can be accounted for simply in terms of executive/frontal lobe dysfunction (Phillips & Silverstein 2003), which is thought to underlie self-monitoring (Turken et al. 2003).

Despite these strengths, B&Y's thesis suffers from at least three weaknesses. First, a central problem with this article is its reliance for its theory of schizophrenia on a theory of hallucinations. This is a problem because only approximately 60% of schizophrenia patients ever have auditory hallucinations, and only approximately 30% ever have visual hallucinations (Slade & Bentall 1988). Other types of hallucinations are rare in schizophrenia. If all schizophrenia patients had some form of hallucination at some point, but not at others (i.e., during the stable phase), then this theory could still hold because it could be argued that the diathesis for hallucinations is always there but that hallucinations only emerge under extreme stress or some other state factor. But, when up to one-third of patients do not have the symptom at all, ever, it seems strained to equate the diathesis for the symptom with the basis of the illness (as in sect. 2.1). Moreover, high rates of hallucinations can be found in other psychiatric disorders. In addition to the well-known findings of hallucinations in bipolar disorder and psychotic depression, high rates of hallucinations have been found in people with histories of child sexual abuse and post-traumatic stress disorder (Morrison et al. 2003; Read & Argyle 1999). Moreover, one study found a high rate (84%) of having had auditory hallucinations among mental health nurses (Milham & Easton 1998). These findings cast doubt on the hypothesis that the essence of schizophrenia is equivalent to the predisposition to develop hallucinations. Interestingly, they also support Bleuler's (1911/1950) seminal view that hallucinations should be considered an "accessory" (as opposed to "fundamental") symptom of schizophrenia.

Second, the argument that verbal hallucinations are more likely to occur in people with mental illness resulting from a combination of a predisposition to develop hallucinations and enduring social anxiety and interpersonal difficulties is problematic in several respects. First, positive symptoms, including hallucinations, are more likely to occur in schizophrenia patients with histories of good premorbid social functioning, whereas other features are characteristic of patients with poor premorbid social functioning (Ross et al. 1994). In addition, the idea that people with mental illness who develop hallucinations are more likely to develop auditory hallucinations is not supported by the evidence from substance abusers, where the prevalence of visual hallucinations is often greater than the prevalence of auditory hallucinations, even though the drugs used are thought to act on the same neurotransmitter systems that are involved in schizophrenia (e.g., amphetamines – dopamine, PCP/ketamine – glutamate; LSD – serotonin). Finally, the issue of moderating variables, exemplified by findings such as the greater incidence of hallucinations in female schizophrenia patients (Sharma et al. 1999), and the decrease in hallucinations as schizophrenia patients age (Schultz et al. 1997) are not addressed by B&Y's theory.

Third, the hypothesis (in sect. 2) that other symptoms of acute psychosis (e.g., thought disturbance, self-experience disturbance) are secondary to hallucinations is not supported by evidence. In contrast, disturbances of self-experience often precede the initial psychotic episode (Klosterkotter 1992), and can also be found in schizoid personality disorder, in which hallucinations are rare (Sass 1992).

In short, although B&Y have put forward an interesting theory of hallucinations, and one that may prove to be of value in understanding schizophrenia, this is not the same as proposing a theory of schizophrenia. In our view, it is unlikely that a single dysfunction can adequately account for all cases of schizophrenia. We also consider it unlikely that, if such a core deficit is found, it will be linked to only one symptom. We and others (e.g., Cohen & Servan-Schreiber 1992; Gray et al. 1991) have proposed views in which disturbances of circuitry can account for multiple phenomena in schizophrenia. However, even in these cases, both theory and data link these disturbances to specific symptom clusters (Barch et al. 2003; Phillips & Silverstein 2003) and do not try to account for all cases. Moreover, neuropsychological and imaging studies indicate that even the most robust deficits in schizophrenia research are rarely found in more than one-third of patients (Heinrichs 2001). Because hallucinations are found in a majority of patients, however, and because schizophrenia treatment involves reducing symptoms (as opposed to being curative), it is imperative that we move forward in our understanding of how these symptoms arise and how they can be reduced. B&Y have provided an important step in this direction.

Brainstem-thalamic neurons implicated in hallucinations

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Abstract: I propose that hyperactivity (or increased numbers) of neurons in the brainstem reticular core promote hallucinations by excessive excitation, coupled with disinhibition, of target thalamocortical neurons. This idea stems from animal experiments using kainate-induced overexcitation of midbrain reticular neurons leading to hallucinatory-type behavior during wakefulness, as well as from data in schizophrenic patients showing an increased number of neurons in mesopontine cholinergic neurons.

The article by Behrendt & Young (B&Y) has the merit of discussing neuronal operations in the circuitry of thalamocortical systems to explain the signs of a major psychiatric disturbance. Such

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