memories can be transformed into experiences with perceptual qualities just by virtue of their misattribution to an external origin.

Unfortunately, in their critique, the authors neglect a large body of evidence in favor of the external attribution bias hypothesis (for reviews, see Bentall 1990 and Seal et al. 2004). Any comprehensive account of the neurocognitive basis of hallucinations in schizophrenia should incorporate these findings. Nonetheless, we agree with B&Y that an external attribution bias cannot explain the sensory characteristics of hallucinations. However, attempts to address this issue have been extensively reported in the literature. Therefore, it is surprising that the authors seem to be unaware of the *imagery hypothesis* of hallucination. This is arguably the first scientific hypothesis of hallucinations: Sir Francis Galton proposed, more than a hundred years ago, that hallucinations result from an increased vividness of mental imagery (Galton 1883). He also devised introspective questionnaires to test his hypothesis and concluded that the evidence was in favor of his hypothesis. Indeed, an approach that focuses on mental imagery may have considerable explanatory power, as mental imagery has been shown to share important characteristics with bottom-up perception (Kosslyn 1994).

Most previous studies that investigated the imagery hypothesis of hallucinations (e.g., Mintz & Alpert 1972; Roman & Landis 1945; Starker & Jolin 1982) relied on subjective and introspective scales of imagery vividness, which may explain the inconsistent results reported in those studies. However, a number of recent studies have assessed the relationship between mental imagery and hallucination with objective, behavioral measures (Böcker et al. 2000; Evans et al. 2000;). Evans et al. (2000) used only auditory measures and failed to find differences between 12 hallucinating and 7 nonhallucinating patients. Böcker et al. (2000) assessed imagery and perception in the auditory and visual modalities in 13 hallucinating and 19 nonhallucinating patients. Consistent with Evans et al. (2000), no between-group differences were observed. However, regarding within-group comparisons, Böcker et al. observed a stronger influence of imagery on perception in the auditory compared to the visual modality in patients with auditory hallucinations. That is, imagining a stimulus enhanced subsequent detection of that stimulus to a larger extent in an auditory task than in an analog visual task. This significant difference was not evident for the nonhallucinating group. Moreover, in a recent cognitive neuropsychiatric case study (Aleman et al. 2002), we observed a modality-specific imbalance between imagery and perception in a patient with continuous auditory hallucinations, compared to five nonhallucinating patients. Specifically, whereas control subjects always performed better on a perception condition than on an imagery condition of the same task (the perception-superiority effect), this patient showed the reverse effect (imagery > perception) in the auditory but not the visual modality.

More recently, we investigated performance of 77 subjects on multiple behavioral measures of auditory and visual mental imagery and perception, and also on a measure of reality monitoring (Aleman et al. 2003). Our approach to the behavioral assessment of imagery-perception relations started from the assumption that, in normal conditions, mental images are less vivid; that is, they have fewer sensory and contextual characteristics than percepts. Kosslyn et al. (1999) and Aleman et al. (2000) recently provided evidence for this assumption in nonpsychiatric samples. According to the theory of Johnson and Raye (1981), increased vividness of images will make them less distinctive from percepts, which may lead to reality-monitoring errors. To test the imagery hypothesis, we assumed that a reduced distinctiveness of mental images with respect to bottom-up generated percepts would be observable differences in performance on tasks of imagery-perception relations. Therefore, we investigated whether patients with hallucinations would show smaller differences in performance on imagery and perception conditions of the same task, after controlling for nonspecific attentional variables. Comparisons were made between performance of schizophrenia patients with ($N = 22$) and without ($N = 35$) hallucinations and matched normal comparison subjects ($N = 20$), after controlling for attentional factors. No differences emerged on any of the mental imagery measures or on reality monitoring accuracy. This suggests that there is no stable disposition toward abnormal mental imagery associated with hallucinations. However, for patients with active hallucinations ($N = 12$), hallucination severity correlated positively with a measure of imagery-perception interaction in the auditory modality. Although preliminary, this finding is consistent with recent theoretical proposals in which hallucinations have been suggested to result from a larger influence of top-down sensory expectations (imagery) on conscious perception, and is therefore consistent with the approach taken by B&Y.

Top-down generated images can be confused with bottom-up percepts when there is a disconnection between prefrontal areas (involved in consciously generating an image) and temporal areas (involved in decoding the image, e.g., "image inspection" or "listening to the inner voice"). Evidence that such a disconnection is present in schizophrenia, and moreover, is associated with the occurrence of hallucinations, is accumulating at a rapid rate (Hubl et al. 2004; Lawrie et al. 2002; Shergill et al. 2003; Silbersweig & Stern 1996). In contrast, there is a lack of empirical data from patients with schizophrenia supporting the thalamocortical model proposed by B&Y. For example, a study by McKay et al. (2000) failed to find basic sensory impairment in patients with hallucinations as compared to patients without hallucinations, whereas deficits in perception of complex auditory stimuli were associated with hallucinatory status.

Nevertheless, we concur with B&Y that subcortical mechanisms may play an important role in the neurogenesis of hallucinations, as is apparent from studies reporting brain activation patterns during hallucinations (Shergill et al. 2000). Therefore, although the evidence remains tentative at best, the conception of hallucinations in schizophrenia as being underconstrained perception resulting from intrinsic thalamocortical resonance in sensory areas might complement current models of hallucination. However, in itself, the approach falls short of comprehensively explaining the neurogenesis of hallucinations in schizophrenia.

Underconstraint and overconstraint in psychiatry

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Abstract: Hallucination lies at an intriguing border between psychiatry and philosophy. Although Behrendt & Young (B&Y) tie their proposal to Kantian transcendental idealism, other philosophical positions are equally consistent. Cognition is underconstrained by reality not only in hallucination but also in autism and dreaming. Sensory underconstraint is insufficient to encompass schizophrenia. There is also a breakdown in integrative capacity on the cognitive side. From a wider clinical perspective than schizophrenia, there can be underconstraint or overconstraint in sensory and cognitive functionalities.

The problematic of hallucination brings psychiatry and philosophy up close. What adamantly *is* for the hallucinating patient, does not appear to the observer. This is a bona fide Being in itself, despite being hallucinated. What appears, the phenomena that onrushing experience discloses, is no basis for ontology, given the honest conviction of the hallucinator, who finds himself already thrown amidst a world in which, for example, voices may emanate from the shut-off television. And what if the non-schizophrenic world, too, is *maya*, illusion? Could non-schizophrenics in some deep ontological sense be hallucinators too? After all, Descartes meditating, seated by the fire, could find no certain truth that he was not dreaming it.

Behrendt & Young (B&Y) explicitly tie their theory of brain functioning to Kantian "transcendental idealism." "The world that we see around us is internally created and a fundamentally sub-

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jective experience that in the state of normal wakefulness is merely constrained by external physical reality" (target article, sect. 1, para. 2). Here, Being is a function of knowing. Our perceptions do not mirror reality but are shaped by it: "we do not *see, hear, feel, or smell* physical reality itself; instead physical reality *constrains* the internal and fundamentally subjective process of perception" (sect. 1, para. 3, emphasis in target article).

The difference between normal perception, dreaming imagery, and hallucination is only the degree to which they are constrained by sensory input. Hallucination is "*underconstrained* by sensory input" (sect. 1, para. 2, emphasis in original). The primacy of subjectivity, which is "merely constrained" by external physical reality, is clear. Kant's transcendental idealism is grounded in the subject. This is not Berkeley's solipsistic idealism. Kant's *ding an sich* is a reality that in itself is unknowable, but knowable via our subjective appropriation of it.

B&Y's theory is not necessarily tied to Kantian philosophy, however. In Neisser's *Cognition and Reality* (1976), perception is where cognition and reality meet. Subjectivity (in the form of schemata with conditions of satisfaction) and reality are at parity, but only in the normal case. In the psychopathology of schizophrenia, reality may underwhelm subjectivity, per B&Y. In Neisser, the balance shifts subjectivity from Kantian dominance over external reality to parity with external reality. When the particular schemata's conditions are satisfied, there is perception of a particular world. In schizophrenia, the schemata are underconstrained. So B&Y's theory is consistent with both Kant's transcendental idealism and Neisser's scientific realism.

Yet, another philosophical position quite consistent with B&Y's brain theory is monadological (Globus 1987; 2003; Globus & Bezzubova 2001). (See Nakagomi [2004] for a monadological interpretation of fundamental physics.) Here, world "thrownness" – to use Heidegger's term – rather than perception, is where cognition and reality meet. But the meeting is no longer one in which abstract conditions are satisfied. The meeting is instead a match – a belonging-together – some match between reality and cognitive attunement, which may be repeated across monadological brains. The mechanism is the same here – cognition and reality meet – but the result is no longer reflective perception *of* the world, but finding oneself already amidst it, in parallel with other world thrownnesses. In this view, reality is no longer the ground of the perceivable, but "is" instead a defaulting unknowable abground (more Heidegger; see Globus 2003). So there is no exclusive philosophical connection between Kantian transcendental idealism and B&Y's conception of brain functioning.

B&Y's theory can be clinically more broadly applied. For autistic mental activity, which is exaggerated in schizophrenia but is by no means confined to that condition, the inner life predominates over the external world. Autism, too, can be conceived of as underconstrained by reality. The Freudian unconscious mind is underconstrained by reality during dreaming. So underconstraint by input explains more than hallucination.

The key explanatory brain mechanism exploited by B&Y involves dysregulation in thalamocortical systems, so that reality underconstrains relative to cognition. The difference between normal perception and hallucination is only a matter of degree. In hallucination, "sensory constraints normally imposed on the process of perception" (sect. 2, para. 5) are disrupted. This explanation is restricted, however, leaving out primary pathology of the cognitive processes in schizophrenia. Bleulerian splitting (Bleuler 1968), so characteristic of schizophrenia, is a breakdown of integrative capacity, which disrupts experience, cognition, and action.

So a richer context for B&Y's account of brain functioning in hallucination is called for. Input may be underconstraining in schizophrenic hallucination and underconstrained in post-traumatic stress disorder. The subjective attunement may be overconstraining in obsessive-compulsive disorder and underconstraining in impulse control disorders. There is a variable imbalance in psychiatric disorders between constraints of the internal attunement and constraints from external reality. In the case of bipolar disorder, the shifts in attunement between depression and mania are striking.

We applaud B&Y for bringing in a philosophical framework, which is rare outside the phenomenological tradition in continental psychiatry (e.g., Jaspers 1962), and their approach to the relationship between symptoms and brain functioning is worth pursuing. We suggest that a broader clinical context would be beneficial to the development of their ideas.

Thalamocortical dysfunction and complex visual hallucinations in brain disease – Are the primary disturbances in the cerebral cortex?

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Abstract: Applying Behrendt & Young's (B&Y's) model of thalamocortical synchrony to complex visual hallucinations in neurodegenerative disorders, such as dementia with Lewy bodies and progressive supranuclear palsy, leads us to propose that the primary pathology may be cortical rather than thalamic. Additionally, the extinction of active hallucinations by eye closure challenges their conception of the role of reduced sensory input.

We are as dissatisfied as Behrendt & Young (B&Y) with previous models of the genesis of hallucinations. Applying them to complex visual hallucinations (CVH) in neurodegenerative disorders such as Alzheimer's disease (AD) and dementia with Lewy bodies (DLB), these models struggled to account for who hallucinated, what they saw, and when and where their hallucinations occurred.

We have devised a Perception and Attention Deficit model (PAD; Collerton et al., in press). CVH occur when combined impairments in visual attention and object perception, in the context of a relatively normal scene representation, disturb scene perception. We postulate that these functional impairments result from disturbances in a lateral frontal cortex–ventral stream system. Projections from basal forebrain cholinergic nuclei directly regulate this system, whereas cholinergic projections to the thalamus from the basal forebrain and the brainstem have an indirect influence.

B&Y's emphasis on the primacy of attentional processes in the phenomenology of CVH, and the role of cholinergic regulation, is consistent with PAD. Indeed, as a potential mechanism for synchronizing attention and visual perception, it fills a gap in PAD. Two differences, however, are the role given to the thalamus and the contribution of reduced sensory input to hallucinations.

If hallucinations reflect disruption of thalamocortical synchronized gamma oscillations, then, in principle, the primary locus of dysfunction could be either cortical or thalamic. In support of their thesis, B&Y point to single cases of thalamic lesions with CVH (e.g., Noda et al. 1993; Risser & Powell 1993). However, in group studies, CVH are infrequent in people with massive, but relatively restricted, thalamic damage resulting from infarcts (none reported, del Mar Saez de Ocariz et al. 1996) or fatal familial insomnia (none reported, Gallassi et al. 1996; Tabernero et al. 2000). They are more frequent in AD (average 23%, Ballard et al. 1995; 1999; 2001; Bathgate et al. 2001; Cummings et al. 1987; Holroyd & Sheldon-Keller 1995), Vascular dementia (VaD, average 33%, Ballard et al. 1995; Bathgate et al. 2001; Cummings et al. 1987), DLB (average 55%, Aarsland et al. 2001a; Ballard et al. 1995; 1999; 2001), and Parkinson's disease dementia (PDD, average 40%, Aarsland et al. 2001b; Neimark et al. 1996) – all disorders with more cortical than thalamic pathology. This suggests to us that the primary role that B&Y allocate to thalamic dysfunction cannot be generalized.

Cholinergic function is closely associated with CVH. Pharma-

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