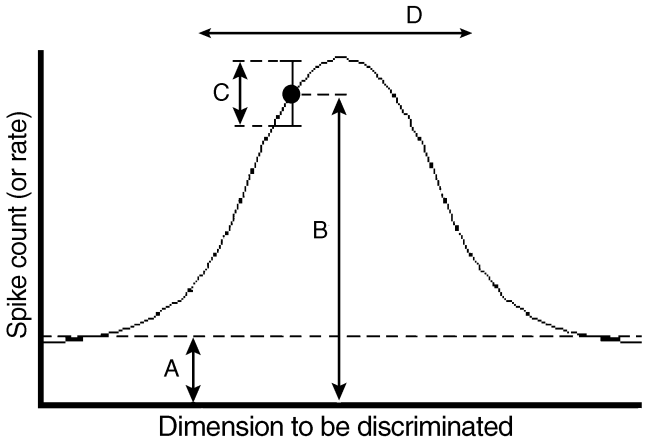


Figure 1 (Disney & Schultz). Two ways to measure signal-to-noise ratio. In this example, the method used in the cited physiological literature measures the SNR at the point indicated by the filled circle as B/A, the evoked divided by the spontaneous activity. SNR in the engineers' sense, however, would be measured as D/C – the variance across the distribution of signals to be discriminated (i.e., across a neuron's tuning curve), divided by the trial-to-trial variability (noise variance) around the point at C.

variance was calculated from only 10 trials per stimulus and the data were recorded from anaesthetized animals with basal forebrain lesions. Data from alert animals with an intact cholinergic afferent system are needed, ideally incorporating measurements of the effect of ACh on the Shannon information transmitted about sensory stimuli.

We can, however, look to other data that suggest what the effect of ACh on thalamocortical transmission may be. There is growing evidence that ACh enables action potentials arriving from the thalamus to elicit relatively larger excitatory postsynaptic potentials (EPSPs) in cortical neurons, compared with EPSPs arising from intracortical input. This effect has been shown in rat piriform (Hasselmo & Bower 1992), visual (Kimura et al. 1999), auditory (Hsieh et al. 2000), and somatosensory (Gil et al. 1997) cortices. The stronger thalamic drive is probably a result of increased glutamate release from thalamic terminals that express high-affinity nicotinic ACh receptors (Disney & Aoki 2003; Lavine et al. 1997; Parkinson et al. 1988; Prusky et al. 1987; Quik et al. 2000; Sahin et al. 1992). If summed EPSP amplitude at the cell soma is considered part of the "signal" to a cortical neuron, then increasing the gain on input from relay nuclei will allow the contribution of sensory input to somatic depolarization to exceed that arriving from intracortical sources.

The effect of thalamic input gain increases would be to give more weight in the cortical representation used by perceptual processes to the current sensory input, and less weight to recurrent, intracortical activity. This accords with both the framework put forward by B&Y and an earlier Bayesian model, which proposed that ACh controls the interaction between prior knowledge and sensory evidence (Yu & Dayan 2002). The chief differences in the two perspectives are: (1) The attractor states traversed by background activity: For B&Y, these are nonevoked gamma band oscillatory discharge cycles in a thalamocortical loop. In the Bayesian perspective, they are cortical recurrent activity representing prior knowledge of the world. (2) Where ACh exerts its critical influence: that is, in the interaction between the TRN and thalamic relay cells (B&Y, target article) or at thalamocortical synapses (Yu & Dayan). In both cases, the effect of sensory input is to constrain the states traversed by the network dynamics to those compatible with the current sensory evidence and, in both, ACh has considerable power to determine the strength of that constraint.

Recent optical imaging results may help us understand what background activity represents. Kenet et al. (2003) showed that activity in primary visual cortex, in the absence of visual stimulation, involves a dynamically switching set of cortical states whose spatial features correspond at above-chance levels to the coherent orientation maps observed with visual stimulation. Additionally, they observed that more time was spent visiting states such as the cardinal orientations, potentially reflecting the prior "knowledge" that arises from previous experience with the statistics of the natural environment (Ringach 2003). These statistics would presumably be represented in the connection strengths between cortical neurons, as a result of Hebbian-type processes. Activity in an individual neuron will then tend to excite neurons with which it is commonly coactive during natural stimulation. So hallucinations, viewed as spontaneous perception, will be like the sensory-evoked states that trained the network.

Good science, bad philosophy

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Abstract: Behrendt's & Young's (B&Y's) persuasive scientific theory explains hallucinations, and is supported by a wide variety of psychological evidence, both normal and abnormal – unlike their philosophical thesis, Kantian idealism. I argue that the evidence cited by the authors in support of idealism actually favors realism. Fortunately, their scientific theory is separable from their philosophy, and is methodologically consistent with realism.

Behrendt & Young's (B&Y's) elegant theory, simply put, is that hallucinations are otherwise normal perceptual processes that are not sufficiently constrained by sensory input. They present an impressively broad range of evidence for it, from microscopic to behavioral, and from normal to abnormal. Lest anyone be tempted to accept the dubious thesis of Kantian idealism on the coattails of this plausible scientific theory, I will argue that the evidence cited does not entail idealism, but instead favors realism. Thus, divorcing the scientific theory from philosophical idealism, I will argue that it be remarried with realistic materialism, its only true, methodologically compatible, partner.

The logical core of B&Y's philosophical thesis is that, "What we perceive as being around us is not the external physical world; instead, it is a part of our mind that is projected outside" (target article, sect. 1, Fig. 1 caption). This is said to be Kantian (transcendental) idealism, though this is in fact the central tenet of idealism of any species, and would be acceptable to Berkeley and Husserl, along with various other phenomenologists, existentialists, poststructuralists, and the like. B&Y adduce four arguments for it.

1. "Perceptual experience is an *active* achievement of the nervous system" (sect. 1, first para., emphasis in original). A parent might challenge a child: Did you really see that, or are you just making it up? Assuming this dichotomy between what we perceive and what we make up, the fact that perception is active (produced, at least in part, by us) seems to entail that we do not really see anything, but just make it up. However, this is not a genuine two-bin dichotomy. Perception need not be either entirely active or entirely passive. All perception requires *some* activity on the part of the perceiver. The only question is whether any external input is also required, and in what way. This is ontologically neutral. We may understand it realistically: Neural activity is necessary to perceive external physical realities. But it applies equally to the *idealist:* Some mental (or spiritual, or soul) activity is necessary to perceive ideas (or phantasms, or whatever). So the fact that perception is active does not favor idealism over realism.

2. "Only a minor part of thalamocortical connectivity is devoted

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to the transfer of sensory input" (sect. 1, para. 1). We know (by the previous point) that *some* activity is required by the perceiver, no matter what. Only if *all* perceptual activity were due to the perceiver would it follow that nothing external was being seen. So, the proportionality here tells us nothing. Most of the circuitry and activity in radar is used to generate the outgoing wave and an internal model of the range. The incoming signal makes up only the tiniest bit of the electrical information process, with the rest, including amplification, supplied by the system itself. It does not follow from this that radar does not detect and model external objects.

3. "Even normal perception in wakefulness is fundamentally a state of hallucinations" (sect. 1, para. 3). What is that word, "fundamentally," doing here? If idealism were true, then perception and hallucination would be the same. B&Y *must* distinguish between hallucination and perception – this is presupposed by their theory. And so they do: Perception is constrained by sensory input, hallucination is not. But that is a *fundamental* difference! We know (by point 2) that even a dynamically tiny external input makes a world of difference. Dynamically, there is scant difference between a radar image caused by an unshielded antenna and one caused by an aircraft – but they are fundamentally different in informational terms.

4. "Perception did not develop to copy the external world" (sect. 1, para. 3). True. But copying is not required for realism, and its absence does not entail idealism. Plato hypothesized the *eidos* to explain perception and cognition. *Eidos* means form or shape, and from it we get "video" and "idea." In seeing, thought Plato, the form of a material thing is transported into the immaterial mind: *same* form, different *substances* (material and mental). Cognition was the processing of mental forms. Thus, copying – identity of form – became a ubiquitous part of our theorizing about perception and cognition for the next 23 centuries. Within the last few generations, information theory has shown us how to escape the misdirection of copying theory. Now we can see that realism does not require that our perceptions copy reality, but instead that they provide us with information about it. The ghost of copying still haunts us everywhere, as indicated by B&Y's article, and the very word, in-*form*-ation, itself. But we can escape it, thanks largely to our familiarity with a sister concept: *trans*-formation. We have become conversant with the transformation of (the information in) DNA into an organism – and back again, the transformation of music (a computer program, movie, or whatever) into a CD (or DVD) disk – and back again, and so forth. The forms of external things are not copied in our brains, but rather transformed into neural activity and states. Perception is this transformation, and behavior transforms it back again. Perception is realistic in just those cases where the transformation informs us about its distal causes. Idealism would require *mis*information – that is, the internal creation of forms *as though* they carried information about distal causes, even though they do not. B&Y do not demonstrate, indeed do not even claim, any such misinformation – except for hallucinations.

So deeply ingrained is copying theory, that my admission that brains do not copy external objects – though surely true – might be grasped by some, perchance B&Y, as admitting to idealism itself. Nothing could be further from the heart of the matter, which is not copying itself, but whether our perceptions are causally connected to things in such a way as to carry (via transformation) information about them.

Finally, from a methodological point of view, all of B&Y's neuroscientific evidence would count for nothing if it were fundamentally equivalent to dreams and hallucinations. Idealism, like any philosophically decent metaphysics, can be logically sustained in the teeth of any evidence, if one isn't scared of wild ideas. But, as I have argued more fully elsewhere (Foss 2000), scientific methodology constrains metaphysics more decisively than the scientific evidence as such. Modern science is methodologically materialistic; hence, realism, not idealism, is the only philosophical mate compatible with B&Y's modern, and beautifully scientific, theory.

Perception and psychoses: The role of glutamatergic transmission within the nucleus accumbens septi

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Abstract: In agreement with Behrendt & Young (B&Y), we considered the role of perception disturbances in schizophrenia in our first clinical approaches, using the Bender test with schizophrenic patients. Following this, we reproduced nuclear symptoms of schizophrenia in animal models, showing that perceptual disturbances, acquisition disturbances, and decrease in affective levels can be induced by glutamatergic blockade within the nucleus accumbens septi. Our results link the proposed corticostriatal dysfunction with the thalamocortical disturbances underlying perceptual problems reviewed by B&Y.

The present article by Behrendt & Young (B&Y) is an interesting and strong effort to clarify a very important point: perception disturbances in psychoses. This topic has been recently taken as a central fact of this illness (Holden 2003a), as perceptual cognitive distortions are a core symptom of schizophrenic psychoses (see Costello 1993). One of the main goals of the article is to use biological explanations for illness in a strict sense (psychoses) in psychopathology, and, in this sense, the idea of causality is clearly present. One additional goal is the attempt to establish new relationships between objective study findings (electroencephalograms) and biological disorders underlying the psychoses. The description of and the large amount of evidence cited in support of the concept of a corticothalamic dysfunction in relation to perception, consciousness, neurotransmitters, and clinical entities is quite solid.

Not only hallucinations, but also other disturbances of cognition and perception, are symptoms of schizophrenia. The cognition involved in interpretation of perception is important, particularly in distinguishing facial expressions of emotion (Holden 2003a). Conrad (1966) argued that interpretation of interpersonal communication, because of its subtlety, was more affected than perceptions of external objects, facilitating delusional perceptions. All of these findings, taken together, strongly suggest that cognitive deficits may lie at the heart of schizophrenia (Holden 2003a).

We have studied these topics in the field of perception and psychoses, beginning with the ideas of Conrad about a gestaltic dysfunction underlying delusional perceptions (see Conrad 1966). We used the gestaltic Bender test (Del Vecchio & Gargiulo 1992; Gargiulo & Del Vecchio 1997), which showed that global scores and time employed were significantly different between healthy controls and acute and chronic patients, thereby allowing us to quantify the perceptual alterations resulting from the loss of objective structure of perception in schizophrenic patients (see Gargiulo 2003). Our results are in agreement with the failure of schizophrenic patients to perform well in visual backward masking tasks (Green et al. 1999, cited by B&Y), which are related to a failure to establish cortical oscillations in the gamma range in response to sensory stimulation (Green et al. 1999). Our next goal was to study perception in close relationship with cognition and anxiety in animal models, taking into account the current primary conceptions about the pathophysiology of schizophrenia.

Carlsson has proposed that psychotogenesis depends on an interplay between dopamine and glutamate pathways projecting to the striatum from the lower brainstem and cortex, respectively (see Carlsson et al. 2000). The action of these neurotransmitters on striatal GABAergic projection neurons (indirect striatothalamic pathways) exerts an inhibitory action on thalamocortical glutamatergic neurons, thereby protecting the cortex from a sensory overload and hyperarousal. This protection could be reduced by

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