that the dream had all the richness of normal perception, but I do not then mistake the clouds that I see out of the window for dreams or any other form of imagery. I see the rich, evanescent, and unpredictable forms that they appear to take, and know that I did not and could not imagine them. Why do I have such confidence, when I have never had any independent validation of the reality of perceived cloud forms? It is because, in cases where the things perceived are more accessible, the senses are constantly providing information about things that could not have been predicted and which (i.e., the information) is then confirmed by further action. Behrendt & Young (B&Y) say that normal perception ensues only if the pattern of sensory input matches the pattern of pre-activation provided by prefrontal attentional mechanisms. I do not see how they can look at the clouds and justify that view. Of course, my conscious experience is not of water droplets, and even less is it of the counterintuitive entities of subatomic physics, but that in no way implies that I can predict the images that clouds help form on my retina.

I am as sceptical of the richness of my own dream imagery as I am of the claims of others to have such richly detailed images. This is in part because for several years I studied visual imagery using a simple change detection paradigm that directly compared sensory storage (SS) with a basic form of imagery, that is, short-term visual memory (STVM; Phillips 1974; 1983). Many studies using many subjects showed unequivocally that the amount that can be voluntarily held in mind, even for only a few seconds, is many orders of magnitude less than that which vision delivers and which survives briefly in SS. Studies of change blindness (Rensink 2002; Simons & Levin 1997) show that these severe capacity limitations on internal representations apply also to natural scenes. A host of neurobiological and psychophysical studies confirms the everyday impression that our visual systems are designed to convey an enormous amount of information about the world. The huge channel capacity required is costly, both anatomically and energetically, and this cost is borne because it greatly enhances our adaptation to the real world. Imagination can do so too, but I do not see the need for it to operate at such a detailed level, nor do I know of any unequivocal evidence that it does. My working assumption is therefore that I can imagine something as having detail, without imagining the detail that it has.

What then of the descending pathways, which in channel capacity clearly rival the feed-forward pathways? Evidence that B&Y review, and plenty more, shows modulatory interactions to be widespread, but what kind of interaction are they and what do they do? One common view is that they increase the salience of relevant signals. This view can be made formally precise (e.g., Kay et al. 1998; Smyth et al. 1996), and can be related in detail to both neurophysiology (Phillips & Singer 1997) and psychopathology (Phillips & Silverstein 2003). This view of modulation requires the prior existence of signals to be modulated, however. B&Y seem to have a different conception in that they present modulation as something that can itself create images. Prima facie this seems highly counterproductive. For example, if attentional processes themselves generate images, then things searched for will be seen everywhere, and never found. It would therefore be helpful if the authors clarified their concept of modulation. Is it essentially the same as ours or not? If it is, then perhaps they emphasize noise because that might provide the prior signals on which modulatory processes operate. Such an argument does not require a belief in the primacy of fantasy, however. Indeed, it implies that the normal function of modulation is not to generate hallucinations but to increase the salience of signals that transmit information about the environment and which are relevant to the context within which they occur.

How then can I explain my richly detailed dream experiences and reports of such experiences by others? One possibility is that the reports are valid. We might then try to explain them as occurring when episodic and semantic memory are used to create vague, schematic scenes, which somehow are then filled-in with a rich array of details by selective enhancement of noise in lower-level corticothalamic pathways. Though this seems to me to be a defensible view, I do not see how it is in any way necessary. Another possibility seems to me more likely: that reports of highly detailed images are invalid. I thought that psychology learned long ago that although introspective reports may provide data worth explaining, they cannot be taken at face value as valid descriptions of internal mental processes (Dennett 1991; O'Regan & Noe 2001). Years of studying imagery using students have indicated to me that ratings of detail and vividness are highly unreliable, easily changed, and little related to objective performance measures. They are likely to be even less reliable in reports of hallucinations, which may differ in some fundamental ways from normal percepts, even though these differences are not a prominent part of the experience. Belief in the essential equivalence of hallucinations and valid perceptions may therefore be misleading, in that it could discourage therapies that aim to enhance their discrimination. Throughout their article B&Y seem to assume that reports of internally generated images can be taken at face value, and require no further validation or test. I do not see how this naive introspectionism can be justified.

It might be thought that no validation of internal imagery is possible, so we must either accept naive introspectionism or ignore the data altogether. Not so. We cannot argue that we must accept the truth of an introspective report simply because we have no way of testing it! Methods for objectively studying imagery are therefore needed. Showing that some aspect of reported content correlates with brain activity or behaviour will not do. I do not doubt that images have content. What I doubt is that they have such a richness of detail that it makes them essentially equivalent to normal percepts. Though they were not studies of hallucination, my studies of STVM are among many showing that the richness of detail in images can be tested. The paradigm I used could be applied to at least some of the hallucinatory experience to which B&Y refer. Included in the hallucinatory phenomenology described by Santhouse et al. (2000) is visual perseveration, where patients report continuing to see veridical objects or patterns after looking away. If even only one such patient is shown to have the ability to detect changes comparable to those reflecting the high capacity for sensory storage that we all have, but lasting much longer and after having looked at other things, I will recant in amazement.

Underconstrained thalamic activation + underconstrained top-down modulation of cortical input processing = underconstrained perceptions

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Abstract: Behrendt & Young's (B&Y's) theory offers a potentially important perspective on the neurobiology of schizophrenia, but it remains incomplete. In addition to bottom-up contributions, such as those associated with disturbances in sensory constraints on cognitive processes, a comprehensive model requires the integration of the consequences of abnormal top-down modulation of input processing for the evolution of "underconstrained" perceptions. Dysfunctional cholinergic modulation of input functions represents a necessary mechanism for the generation of false perceptions.

Behrendt & Young's (B&Y's) theory requires expansion to include integration of abnormal top-down effects on cortical input processing. The contribution of an abnormally reactive cortical cholinergic input system in schizophrenia to the defective augmentation and filtering of thalamic inputs is arguably a necessary component of a neurocognitive hypothesis of the positive symptoms of schizophrenia.

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The proposed loss of sensory-driven, bottom-up constraints on cortical/cognitive processing is intriguing, and is consistent with classical findings on the effects of severe sensory deprivation (Zubek 1969). The functional role of gamma frequency rhythms in cognition is far from settled, however, and the literature concerning gamma-frequency rhythms in schizophrenics is far more complex than the authors suggest (e.g., Lee et al. 2003). Nevertheless, the proposed defect in sensory constraints over cognitive processes is worthy of consideration. But a comprehensive model of schizophrenic symptomatology necessitates an additional class of top-down processes.

A central, and indeed signature, feature of schizophrenia is a fundamental disturbance in attentional processes. The available evidence, based on experiments assessing the effects of selective cortical cholinergic deafferentation and performance-associated cortical acetylcholine (ACh) release, conclusively indicates that the cortical cholinergic input system is necessary for the mediation of a wide range of attentional abilities (Arnold et al. 2002; Chiba et al. 1995; Everitt & Robbins 1997; McGaughy et al. 1996; Passetti et al. 2000; Sarter & Bruno 1997; 2000; Sarter et al. 2001; Turchi & Sarter 1997; 2000). In the cortex, ACh amplifies the processing of thalamic inputs and also suppresses cortico-cortical (or associational) throughput (Donoghue & Carroll 1987; Dykes 1997; Edeline 2003; Hars et al. 1993; Hasselmo & Bower 1992; Hasselmo & McGaughy 2004; Hsieh et al. 2000; Metherate & Ashe 1993; Metherate & Weinberger 1990; Murphy & Sillito 1991; Tremblay et al. 1990a; 1990b; Webster et al. 1991; Weinberger 2003). Presumably, these cholinergic mechanisms underlie behavioral findings such as the selectively disruptive effects of the loss of cortical cholinergic inputs on performance in signal trials in attention tasks (McGaughy et al. 1996).

The basal forebrain corticopetal cholinergic system (of which the nucleus basalis of Meynert represents only one of several regions that form this ascending projection system) mediates both bottom-up (Berntson et al. 1998; 2003a; 2003b; 2003c) and top-down (Sarter et al. 2001) modulation of input processing in attentional contexts. Top-down effects, reflecting the cognitive modulation of input processing, are mediated in part via cholinergic inputs to the prefrontal cortex, which in turn influence basal forebrain neurons (Sarter & Bruno 2002) and, via multisynaptic cortico-cortical innervation of cholinergic terminals, cortical cholinergic activity as well (Nelson et al., in press). Thus, abnormal regulation of the basal forebrain corticopetal cholinergic system has enormous consequences for the attentional modulation of input processing.

Although the current evidence indicating an abnormal regulation of cortical cholinergic inputs in schizophrenia is still limited, a reduction in muscarinic receptor densities has been reported in several post mortem studies (Crook et al. 2000; 2001; Mancama et al. 2003) and by single photon emission computed tomography (SPECT) in medication-free patients (Raedler et al. 2003). The latter study also reported a significant correlation between muscarinic receptor availability and positive symptoms. The interpretation of these data is not straightforward, but these findings correspond with other evidence and conceptualizations that collectively point to an abnormally reactive cortical cholinergic input system in schizophrenia (Hyde & Crook 2001; Sarter 1994; Sarter et al. 1999; 2005; Tandon et al. 1999). For example, chronic (accidental) cholinesterase inhibition yields psychotic symptoms (Bowers et al. 1964; Gershon & Shaw 1961). Furthermore, repeated exposure to amphetamine models the mesolimbic hyperdopaminergic transmission that is a hallmark of psychosis (Laruelle & Abi-Dargham 1999) and remains one of the more productive animal models of schizophrenia (Robinson & Becker 1986). Relevant to the current thesis, repeated amphetamine exposure has also been shown to sensitize cortical ACh release (Nelson et al. 2000). Abnormal increases in cortical ACh efflux are normalized by systemic or intra-accumbens administration of antipsychotic drugs (Moore et al. 1999). These and other data supported the general hypothesis that abnormal activity of cortical cholinergic inputs is a necessary correlate of abnormal mesolimbic dopaminergic transmission (see also Gerber et al. 2001) and that antipsychotic drug treatments act, at least in part, by normalizing the activity of cortical cholinergic transmission (Sarter 1994; Sarter et al. 1999; 2005).

An abnormal reactivity of basal forebrain cholinergic efferents (which may include the projections to the reticular thalamus) impacts the bottom-up and top-down modulation of stimulus processing in attention contexts. Theoretically, the exaggerated processing of normally filtered stimuli may constitute the primary effect of a dysregulated cortical cholinergic input system. Indeed, manipulations that disinhibit this neuronal system resulted in impairments in the performance of animals in non-signal trials of an operant sustained attention task (Deller & Sarter 1998; Holley et al. 1995; Turchi & Sarter 2001), likely reflecting an elaborated processing of non-signal information.

In interaction with an underconstrained thalamocortical input system, as proposed by B&Y, the contributions of an abnormally regulated cortical cholinergic input system to the formation of "false perceptions" could be even more fundamental. If cortical input processing is characterized by limited sensory information, top-down mechanisms would be expected to become more influential (see also Yu & Dayan 2002). Thus, abnormally strong, cholinergically mediated top-down effects may increasingly dominate the perceptual process. However, as the impairments in executive capacities escalate in schizophrenic patients, top-down mechanisms become increasingly dysregulated, eventually yielding a functional disconnection between prefrontal activity and the cholinergic modulation of input functions elsewhere in the cortex. Such a disconnection may be critical for the development of source monitoring problems (Frith & Dolan 1996; Johnson 1997) and thus for the emergence of false perceptions.

Forty years ago, Venables classified schizophrenia as an input dysfunction (Venables 1964). The disruption of thalamocortical information processing and the top-down modulation of cortical input processing appear to represent two necessary mechanisms yielding input dysfunctions. For insufficient sensory information to evolve into an underconstrained perception, abnormal augmentation and defective filtering are necessary mechanisms, and they are likely mediated, at least in part, via a dysregulated cortical cholinergic input system.

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Schizophrenia: A disorder of affective consciousness

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Abstract: Behrendt & Young (B&Y) propose an explanation for schizophrenia in terms of a *cortical* default in the interaction between consciousness and cognition. However, schizophrenia more likely involves miscommunication between subcortical and cortical affective circuits in the brain, a default in the interaction between consciousness and emotion. The typical "affective" nature of hallucinations in schizophrenia provides compelling evidence for *subcortical* involvement.

According to neurocognitive interpretations, hallucinations in schizophrenia result from cortical attentional mechanisms producing conscious experiences unconstrained by actual sensory input (B&Y). It has been argued that functional abnormalities in gating information streams in the brain result in a low signal-to-noise ratio that causes conscious experiences to be derailed (Taylor et al. 2002). Crucially, on the phenomenological level, hallucinations

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