Both models suggest a critical role of executive dysfunction. This is consistent with evidence of impairments of prefrontal cortex in disorders with high rates of visual (e.g., dementia with Lewy bodies) and auditory (e.g., schizophrenia) hallucinations. Whereas the PAD model suggests that this is an impairment of dynamic attentional binding, the HEAR model has focused on a deficit of intentional inhibition. Each hinges on the executive control of attention; that is, the selection of correct/incorrect proto-objects or relevant/irrelevant representations. According to Collerton and colleagues, hallucinatory experiences generally arise when there is impaired attentional binding together with a poor sensory response to the correct proto-object. Our model, however, emphasizes the heightened activation of an irrelevant, internal representation (incorrect proto-object); that is, hallucinations are related to a failure to inhibit currently irrelevant memory traces. This difference may simply be a matter of relative emphasis, given that dynamic attentional binding also involves resistance to irrelevant information. Indeed, we have suggested that the salience of currently relevant events depends critically on the ability to suppress memories of previous (now irrelevant) events (Badcock et al. 2005). Consequently, despite the difference in terminology, the mechanism of dynamic attentional binding appears to correspond closely to the process of intentional inhibition.

While the HEAR model links executive dysfunction to impaired memory, the PAD model combines attentional dysfunction with object perception impairments. Nevertheless, both are consistent with disturbed connectivity between frontal and temporal cortical circuits, and both attempt to describe sources of bias favoring the activation of the incorrect proto-object/irrelevant memory. For example, Collerton and colleagues suggest that current scene input/expectations are assumed to bias perception of an incorrect image. In contrast, in the HEAR model, the salience of irrelevant representations may derive from previous presentations and associated reward value. In sum, a perception/memory distinction appears to be a major difference between the two models. However, in studies of schizophrenia, a deficit emerges more consistently on higher-level object perception tasks closely related to memory (Gabrovska et al. 2002). Therefore, the overlap between these two models may be greater than it appears.

Collerton et al. stressed that an adequate model should account for the variation in frequency of hallucinations, yet support for the PAD model rests essentially on indirect observation of the overlap of cognitive and pathological impairments in disorders with high rates of RCVH. By contrast, our investigations have provided direct tests at the individual case level of the role of intentional inhibition and context memory in AH. For instance, we have shown that AH frequency in schizophrenia (but not the frequency of other symptoms) is correlated with degree of inhibitory dysfunction (Waters et al. 2003). We argued that this deficit underpins the intrusive nature of AH, a feature not directly addressed by the PAD model. In addition, we have also shown that intrusiveness is a key component of hallucinatory-like experiences in normal individuals (Paulik et al., submitted; Waters et al. 2003), raising the interesting possibility that inhibitory dysfunction may accompany other hallucinatory experiences in healthy individuals (e.g., across the sleep-wake cycle).

Because the HEAR model incorporates a context-binding deficit as well as an inhibitory control deficit, Waters et al. (in press) examined the percentage of patients with schizophrenia who were impaired on both cognitive processes. Almost 90% of schizophrenia patients currently experiencing AH showed the predicted combination of deficits, compared to only 33% of patients without hallucinations, representing approximately a sixfold increase in risk of having AH compared to patients without. Such findings provide compelling, direct support for the notion that these two deficits are significantly associated with the hallucinatory process.

Both models predict that isolated impairments would rarely produce hallucinations. Specifically, the HEAR model predicts that non-hallucinating individuals may exhibit deficits on either intentional inhibition or context memory, but not both. In direct confirmation of this prediction, patients with obsessive compulsive disorder (who, like schizophrenia patients with hallucinations, experience intrusive cognitions, but unlike hallucinators recognize them as self-generated) showed deficits in intentional inhibition but intact context memory (Badcock et al., submitted).

In sum, the possibility that deficient inhibitory control of attention, coupled with impaired memory (including context binding), could underpin both visual and auditory hallucinations merits its direct test, though the sufficiency of two deficits in accounting for these complex phenomena deserves scrutiny (see Waters et al., in press).

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Attentional deficit versus impaired reality testing: What is the role of executive dysfunction in complex visual hallucinations?

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Abstract: A "multifactorial" model should accommodate a psychological perspective, aiming to relate the phenomenology of complex visual hallucinations not only to neurobiological findings but also an understanding of the patient's psychological problems and situation in life. Greater attention needs to be paid to the role of the "lack of insight" patients may have into their hallucinations and its relationship to cognitive impairment.

> *We may . . . define an instinct as an innate disposition which determines the organism to perceive (to pay attention to) any object of a certain class, and to experience in its presence a certain emotional excitement and an impulse to action which find expression in a specific mode of action in relation to that object.*
> —William McDougall (1924, p. 110)

Perception is not a passive reflection of "things that are there" but an active process of unconsciously operating instinctive forces continuously creating a subjective, though usually adaptive, experience of seemingly external things and events (McDougall 1924; Schopenhauer 1844). Animals or people commonly feature in complex visual hallucinations because they meet unconscious affiliative impulses, and therefore it is not surprising that such hallucinations are associated with social isolation (Holroyd et al. 1992; Teunisse et al. 1994). For the same reason, and in response to unconscious social anxieties, patients with schizophrenia tend to hallucinate people's voices (see Behrendt & Young 2004).

Perceptual expectancies, which can be elicited by contextual or situational cues, or one's interests in certain kinds of objects, ultimately reflect, according to McDougall (1924), the working of instinctive impulses. Expectations or interests, which may not be explicitly conscious, are attentional mechanisms that crucially shape the content of subjective experience, although the possibilities are normally restricted by external sensory input. We hallucinate persons in their proper composition and place, rather than "floating on the ceiling" or with an "inverted face," because this is how we expect to see them. Insofar as hallucinations satisfy drives, the content of hallucinations should not surprise the hallucinator.

One is often struck in clinical practice to see how patients in

early stages of dementia are not puzzled by the impossibility of their observations of children or deceased relatives regularly visiting their home. Patients may repeatedly set the table for such hallucinated visitors without ever seriously questioning their actions. Less cognitively impaired patients who can show "insight" when describing their hallucinations may not necessarily be aware of the pathological nature of their experiences during the hallucinatory episode. We need to distinguish between insight on subsequent reflection and insight during actual experience. Despite demonstrating the former, patients with complex visual hallucinations may lack the latter, whereas patients with schizophrenia or paraphrenia, in whom hallucinatory experiences are entangled with delusions and persecutory fears, tend to lack both.

Of course, patients with Charles Bonnet syndrome, who have prominent peripheral visual impairment, see bizarre and apparently unexpected things, but in this condition, attentional mechanisms are much less restricted by peripheral sensory input in their effect on perception than is usually the case in dementia or schizophrenia. The question arises, how unexpected or bizarre do complex visual hallucinations really seem to patients with Charles Bonnet syndrome? How much of their insight is gained retrospectively, like the insight we gain into the implausible content of a dream only upon awakening? Indeed, during a dream we are not usually surprised to observe events that completely defy logic and past experience, as they would have been shaped at the time by attentional mechanisms reflecting unconscious desires or simply natural impulses of fear or curiosity.

By default, we accept externalised conscious experience as real, whether it occurs in wakefulness or as part of a dream. The dream intrusion hypothesis of complex visual hallucinations should not be discarded lightly on the basis of a lack of association with sleep disturbance. What should be of interest is that wakeful perception and dreaming are in a fundamental sense functionally equivalent states (Llinas & Pare 1991; Llinas & Ribary 1993) and, indeed, perception in wakefulness may be but an adaptive state of dreaming. Therefore, we could argue that reality testing is not something given to us by default; it relies on intact intellectual functioning accessible only in wakefulness.

It appears that inconsistencies in the perceived world prompt questioning of reality only if the perceiver has sufficient deductive or reflective cognitive capacity. One has to be able to note that an observation defies one's intuitive logic or does not conform to previous experience, while having at the same time access to the rather abstract notion that an experience one is having might not be real. What makes it even more difficult to perform this cognitive step is the fact that what we see in a hallucination or dream is usually expected unconsciously. Alternatively, it may be lack of capacity to interact with the environment in a coordinated and goal-directed fashion that prevents us from questioning the reality of our dream experiences. Cognitive executive impairment may similarly amount to a deficit in one's ability to translate instinctive impulses into sustained action in accordance with hierarchical behavioural strategies, while such impulses continue to manifest themselves in perception. Perception, whether in wakefulness or dreaming, primarily obeys the *pleasure principle*, in Freud's terms, whereas adherence to the *reality principle* can be regarded as higher cognitive performance involving the lateral prefrontal cortex.

It may be impaired reality testing, partly in combination with unconscious desires or fears (Asaad & Shapiro 1986), that converts a hallucinatory predisposition into recurrent complex hallucinations. Lack of insight as a result of cognitive impairment may be central in promoting the gradual development of simple visual hallucinations into recurrent complex visual hallucinations (which would explain the "double dissociation"), which is similar to how simple noises in patients with hearing impairment can develop over time into voices if there is concomitant psychological or cognitive impairment (Gordon 1987; 1995; 1996). For verbal hallucinations to become elaborate and personified in the course of mental illness (Nayani & David 1996), lack of insight can be maintained at the cost of relatively little cognitive impairment (voices can be heard from behind walls). In contrast, for visual hallucinations to acquire prominence in mental illness, the patient presumably will have to be more profoundly impaired in reality testing, which may partly explain the association between visual hallucinations and organic psychosis that is recognised clinically.

Lack of insight as a result of cognitive impairment and attentional pressures due to psychological problems play complementary roles in relation to the biological predisposition to hallucinate, as illustrated by Charles Bonnet syndrome, in which major psychopathology is absent and consciousness is unimpaired yet peripheral sensory impairment is prominent (Gold & Rabins 1989), or by bereavement states, in which yearning for the deceased can maintain complex visual hallucinations despite relatively intact sensory and cognitive functions.

No explanation is given by Collerton et al. in the target article as to precisely how executive dysfunction, frontal hyperactivity (as opposed to hypoactivity), impaired arousal, or cholinergic deficits that have been reported in clinical populations with complex visual hallucinations relate to their notion of "attentional impairment"; and it is not argued convincingly why "binding" of "incorrect proto-objects" into "scene representations" should be a common denominator of such impairments. Predictions regarding circumstances and content of complex visual hallucinations should be made using a model of attention and perception that is based – independently from what is to be predicted – on physiological and neuroanatomical insights, in order to prevent the impression that what is presented as an explanatory model does not go beyond an attempt to rephrase, in a hypothetical language, correlations between hallucinations and cognitive or visual impairments.

Furthermore, a distinction has to be made between sensory processing and perception. In our view, disruption of sensory constraints that are normally imposed on thalamocortical gamma synchronisation underlying conscious perception constitutes an essential biological predisposition to hallucinations – and it is here that we see the role of reticular thalamic nucleus dysfunction (not the "thalamus" as such) – but the extent to which this predisposition is turned into hallucinations and even psychosis crucially depends on personality problems, coping skills, and social stresses faced by the individual (Behrendt & Young 2004), as well as the individual's cognitive capacity for reality testing.

Catatonia is the Rosetta Stone of psychosis

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Abstract: Recurrent complex visual hallucinations (RCVH) represent a form of psychosis. It may be useful to compare RCVH to another form of psychosis, catatonia. Both include a long list of medical illnesses and have been examined using several different hypotheses. Catatonia has a variety of hypotheses, including neurocircuitry, neurochemistry, and an integrated neuropsychiatric hypothesis. This hypothesis for catatonia supports Collerton et al.'s Perception and Attention Deficit model (PAD) for RCVH.

There have been several reviews of catatonia and the ascribed causative illnesses. Two recent books on catatonia provide a variety of hypotheses for this form of psychosis (Caroff et al. 2004; Fink & Taylor 2003). Specifically, there are genetic, neuroanatomical, neurochemical, and neurophysiologic hypotheses to explain why it occurs. The hypothesis of top-down modulation as applied to catatonia (TDMC) shares some similarities with the Perception and Attention Deficit (PAD) model applied by Coller-

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