concerning the occurrence of visual hallucinations in both non-pathological conditions and a range of psychiatric and neurodegenerative disorders. By combining and developing previous models of visual alertness and its alterations, the PAD model gives an advantageous framework for understanding not only the nature of RCVH, but also the processes underpinning visual consciousness. However, in its attempt to provide a unique schema for RCVH in normal and pathological conditions, the PAD model may meet several limitations.

Most of these limitations come from research on the neurobiological mechanisms of the highly varying conscious states across the sleep-wake cycle. In particular, the transition from wake to sleep, when hypnagogic hallucinations normally occur, is basically characterized by a lowering of noradrenergic and serotonergic influences to the cortex rather than only by an acetylcholine underactivity, as Collerton et al. propose. Furthermore, during the transition from sleep to wake, when hypnopompic hallucinations are most frequent, there is a substantial enhancement of the activity of each of the noradrenaline, serotonin, and acetylcholine neurotransmitter systems (Gottesmann 1999; 2004a; Hobson et al. 1975; 2000; Pace-Schott & Hobson 2002). The occurrence of visual hallucinatory-like experiences across sleep stages is most frequently observed during rapid-eye-movement sleep (Fosse et al. 2001; 2004; Hobson et al. 2000), and this sleep stage is characterized by excessive acetylcholine overactivity (Gottesmann 1999; Hobson et al. 1975; Pace-Schott & Hobson 2002). Therefore, RCVH that are normally experienced at the borders of sleep may not be simply explained by acetylcholine underactivity only, as stated by Collerton et al. Rather, the role of either monoamines or monoamine-acetylcholine ratio in these types of RCVH is to be considered. Because the hypnagogic and hypnopompic are the most common visual hallucinations in non-pathological conditions, sleep research data and the neurochemical mechanisms of sleep-wake cycling may certainly be accounted for in explaining RCVH in psychiatric and neurodegenerative disorders.

Furthermore, Collerton et al. suggest that the attention deficit is an important contributing factor for RCVH, with the acetylcholine underactivity being the main neurochemical mechanism. However, many experimental (Aalto et al. 2005; Gao & Goldman-Rakic 2003; Nieoullon 2002; O'Donnell 2003) and clinical data concerning attention-deficit/hyperactivity disorder (Castellanos & Tannock 2002; Swanson et al. 1998) strongly point to the critical role of brain dopamine in the processes of attention. Also, Parkinson's disease (PD), where RCVH are frequently observed (Burn & Troster 2004; Poewe 2003), is caused by degeneration of dopaminergic neurons (Blandini et al. 2000; Eriksen et al. 2005; Fedorow et al. 2005; Montague et al. 2004; Nieoullon 2002). Moreover, there are clinical data documenting that the visual hallucinations in PD can be induced by the dopaminergic therapy (Burn & Troster 2004; Goetz et al. 2001b). Dopamine dysfunction is also generally recognized to underpin the phenomenology of schizophrenia (Hirvonen et al. 2005; Montague et al. 2004; Winterer & Weinberger 2004), which, as mentioned by Collerton et al., is one of the conditions associated with RCVH. Dopamine has an important role in controlling signal-to-noise ratio and top-down processes (Aalto et al. 2005; Gao & Goldman-Rakic 2003; Montague et al. 2004; O'Donnell 2003; Winterer & Weinberger 2004), both suggested in the PAD model to be impaired mainly as a result of acetylcholine underactivity. In addition, noradrenaline and serotonin, along with acetylcholine, are also shown to be significantly involved in modulating the signal-to-noise ratio (Gu 2002).

In the PAD model, the authors propose that hypo-functioning of the lateral frontal cortex resulting from a cholinergic deficit is another mechanism involved in RCVH. In this context, it is to be noted that animal-driven (Gao & Goldman-Rakic 2003; Seamans & Yang 2004; Zhou & Hablitz 1999) and human transcranial magnetic stimulation (Moll et al. 2000; 2003) data show that brain dopamine exerts a strong effect on cortical excitability.

In conclusion, the role of brain monoamines, and the role of dopamine in particular, appears very important for understanding the neurobiology of visual alertness and its alterations in normal and pathological conditions. Hence, the nature of RCVH could hardly be explained by acetylcholine underactivity only.

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Mental images: Always present, never there

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Abstract: Recent research on visual mental imagery plays an important role for the study of visual hallucinations. Not only are mental images involved in various cognitive processes, but they also share many processes with visual perception. However, we rarely confuse mental images with percepts, and recent neuroimaging studies shed light on the mechanisms that are differently activated in imagery and perception.

Visual mental images are generated from memory and therefore are of purely cognitive origin. Behavioral (e.g., Mast & Kosslyn 2002) and neuroimaging research (e.g., Ganis et al. 2004) suggests that the mechanisms associated with mental imagery are – at least to some extent – also involved in visual perception, and the functional value of this overlap has been widely discussed (e.g., perceptual anticipation theory; Kosslyn & Thompson 2003). Even though imagery and perception overlap, only rarely do we actually mistake images for percepts (an exception is the Perky-effect). Why is this the case? Despite the fact that images are essentially involved in a variety of cognitive processes, such as object recognition, spatial reasoning, and problem solving, we hardly ever experience mental images as perceptually real. Why are we able to reliably keep apart or separate when images are generated internally and when images are mediated via sensory stimulation? On the one hand, the fact that several mechanisms are shared by imagery and perception makes it even harder to address this question. On the other hand, research on mental imagery can provide helpful guidance on where to look when studying the mechanisms that account for the occurrence of recurrent complex visual hallucinations (RCVH).

Instead of mental imagery, Collerton et al. focus almost exclusively on attention. The question arises whether the mechanisms that underlie attention have enough explanatory value for a better understanding of RCVH. The major problem is that attention itself has no visual quality, even though it is often involved in visual cognition and visual perception. Mental images are not only phenomenologically related to RCVH, but they also share several common visual properties, which reflect the underlying mechanisms. The target article makes no reference to recent research on mental imagery, which renders Collerton et al.'s model of RCVH not only less compelling, but also incomplete. There are at least three separate points that are noteworthy in this context.

First, the spatial properties of RCVH resemble those of mental images. Collerton et al. point out that hallucinations are located in the central part of the visual field and – unlike afterimages – they do not move with eye movements. This description applies just as well to visual mental images. We often need to inspect images in order to retrieve more specific information from them. Neither images nor hallucinations disappear or move when attended to. Attention can be shifted over imagined or hallucinated objects. Thus, mental images and visual hallucinations share widely the same spatial properties.

Second, it has to be noted that the interplay between visual mental imagery and visual perception is an essential component of top-down processing. When objects are seen from a non-canon-

ical perspective or when they appear partially occluded, visual memories are used for the comparison between the input pattern and an already existing representation in memory. Therefore, the mechanisms engaged in object and scene recognition also rely on mental imagery and are partly identical with those mechanisms that enable us to voluntarily generate mental images (e.g., during daydreaming). Even though the approach proposed by Collerton et al. includes a top-down component, no reference is made to visual mental imagery.

Third, a growing amount of recent research revealed that the neural machinery engaged in visual perception is – to some extent – also drawn upon during visual mental imagery. In a recent study, the overlap was more pronounced in parietal and frontal regions, suggesting that at least some sensory processes are activated differently (Ganis et al. 2004). In other studies, however, differences between imagery and perception were found in parietal and prefrontal areas (Ishai et al. 2000). The discussion of these findings is absolutely crucial for a better understanding of RCVH. A more profound knowledge about the neural mechanisms that are engaged differently in mental imagery and perception, is likely to play a key role in the ability allowing for the continuous distinction between internally generated images and perceived images. The findings from recent research on mental imagery offer a more specific approach to investigate visual hallucinations than the failure of attentional binding, which is still a rather speculative explanation for the occurrence of RCVH.

In sum, Collerton et al. leave out major findings on mental imagery, which have a great potential to be useful for a general model of RCVH. There is no doubt that a model has the potential to reveal commonalities across diverse fields of enquiry, but it has to be based on solid grounds, integrating the most important issues relevant to the question.

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Now you see it, now you don't: More data at the cognitive level needed before the PAD model can be accepted

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Abstract: Before a general cognitive model for recurrent complex visual hallucinations (RCVH) is accepted, there must be more research into the neuropsychological and cognitive characteristics of the various disorders in which they occur. Currently available data are insufficient to distinguish whether the similar phenomenology of RCVH across different disorders is in fact produced by a single or by multiple cognitive mechanisms.

Collerton et al. have done a commendable job integrating a large array of clinical and experimental evidence to describe a plausible model for recurrent complex visual hallucinations (RCVH). Although many aspects of the model are congruent with the phenomenology one sees clinically, there are a few significant ways in which the model is incongruent.

The first difficulty is the claim that RCVH are generally appropriate to the scene in which they are observed. While the *category* of image might be considered appropriate (e.g., people and animals rather than, say, demons and cornfields), often the other features are not. For example, hallucinations are often reported as being in inappropriate positions (people/animals on the wall or ceiling, people floating outside the window, children under their bed; Gauntlett-Gilbert & Kuipers 2003; Howard & Levy 1994), inappropriate size (Lilliputian figures, "pixies" running along the window; Holroyd et al. 2001; Howard & Levy 1994), or inappropriate context (people being harmed, birds flying in a hospital; Lipowski 1990).

Even the two studies cited in Table 3 of the target article to support this claim, Asaad and Shapiro (1986) and Teunisse et al. (1996), in fact do not do so. Teunisse et al. (1996) screened elderly people with visual impairment and found that 63 of 505 had RCVH. The authors list some of the hallucinations described and, although the percentage of each type of hallucination is not listed, few could be described as appropriate to context (e.g., "miniature policemen guiding a midget villain"; "a dragon"; "an angel"; "an unfamiliar person"; p. 795). They also judged only 22% of hallucinations as "fitting in well" with the environment. The Asaad and Shapiro (1986) paper is a review of hallucinations in general and lists common features of visual hallucinations in psychosis as "people or animals or events taking place in front of them" without providing data or further detail (p. 1091). The unfamiliarity of hallucinated images in RCVH has also been found in Parkinson's disease (Barnes & David 2001; Holroyd et al. 2001), delirium (Lipowski 1990, pp. 86–87), and eye disease (ffytche & Howard 1999). Therefore, one can say that though the content of RCVH tends to be of people and animals, they are more often unfamiliar and just as often appear in inappropriate positions or contexts as they do in appropriate ones. If the PAD model's prediction is that scene representation bias is responsible for the content of hallucinations, one would expect the images to be at least more familiar, if not appropriate to location and context.

Another vulnerability in the model is the prediction that properly perceived external objects should displace the incorrect proto-object from attention and thus make the hallucination disappear. Clinical experience suggests this is not true. Although some patients may become absorbed in their hallucination and retain it as the focus of attention (as the PAD model predicts), often patients with RCVH actively hallucinate while they are being examined. For example, when looking at the examiner they will report seeing hallucinated images behind the examiner or in their peripheral vision. There is some indirect experimental evidence to suggest this as well. Teunisse et al. (1996) asked their subjects what acts would make the hallucinations stop. As one might expect, the most effective means was keeping eyes closed (38%). Interestingly, "looking/walking away," "putting on a light," and "concentrating on something else" were not effective (e.g., $< 15\%$ effective). Certainly this needs to be tested in more detail experimentally, but this finding would be a significant piece of evidence against the cognitive mechanism that Collerton et al. propose as generating RCVH.

The PAD model proposes a mechanistic *cognitive* theory to account for observations at the *phenomenological* level. Given the above-mentioned problems in accounting for the phenomenology, it would be important to have a more detailed look at how the various disorders with RCVH compare at the cognitive/neuropsychological level. As the authors cite, there are currently limited data in this area. Although the data in Figure 4 of the target article suggest that cognitive measures of "attentional/executive impairment" and "visual perceptual impairment" correspond to the predictions of the PAD model, these categories are quite vague and heavily biased to data from DLB (dementia with Lewy bodies) patients. For example, although Collerton et al. mention that "poor performance on tests of attention and visual perception are . . . the norm in delirium" (sect. 7.4.1, para. 5), the cited references actually evaluated only attention in any detail.

Greater precision at the cognitive level is important for the validity of the model because disorders that might seem similar at the phenomenological level may in fact have different mechanisms at the cognitive level. For example, consistent with findings in auditory and visual hallucinations in schizophrenia, Barnes et al. (2003) found that Parkinson's disease (PD) patients with visual hallucinations had intact visual imagery but poor object perception and deficits consistent with poor source and reality monitor-