RESEARCH ARTICLE

Effects of Schizophrenia on Attention

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Abstract

Attention is central to many functions such as vision, emotion, reward, memory, and consciousness. Attention is a neural signal that modulates main signals. Main signals are sensory signals in feed forward pathways (e.g., vision related ventral and dorsal pathways) and signals related to memory, emotion, reward, and so on. The attentional modulating signal could be in forward (bottom-up) or backward (top-down) direction from attentional sources such as thalamic reticular nucleus or fronto-parietal network, respectively. This attentional signal is disturbed in schizophrenia. Attention deficits in schizophrenia are critically reviewed. Schizophrenics appear to have deficit mostly in switching and selective (salience) attention. Attention modulates main signals via dopamine and glutamate neuromodulators in schizophrenia. An experimental design, based on switching attention deficit and functional magnetic resonance imaging, is proposed.

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Running head: Schizophrenia and Attention

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1. Introduction

Attention is central to many areas of both basic and clinical researches, and is essential to the construction of every subjective experience [1]. Attention plays important role in vision, perception, emotion, reward, memory, and action

selection [2]. Impaired mechanisms of attention are fundamental aspects of a variety of human illness states. These include schizophrenia, Alzheimer's disease, posttraumatic stress disorder (PTSD), dementia with Lewy Bodies, drug-abuse such as MDMA, cocaine, and marihuana use, Parkinson's disease, frontal lobe damage, attention deficit hyperactivity disorder, bipolar disorder, depression, autism, and pain (references in Table 1). Our **goal** is to (a) concisely review attention, schizophrenia, and attention deficit in schizophrenia, (b) propose an attention based schizophrenia model, and (c) present a functional magnetic resonance imaging (fMRI) based experimental design to study attention deficits in schizophrenia.

In Section 2, we concisely (a) define attention, (b) review the five forms of attention tasks and neurotransmitters involved, and (c) elucidate how attention affects emotion, face recognition, reward system, and working memory. In Section 3, we review schizophrenia and attention deficits, and models for schizophrenia. A future research project is suggested in Section 4. Finally, the conclusion synthesizes our main thesis.

2. Attention

2.1. Definition: Physiologically, visual attention can be defined as a neural signal that modulates main signal. Main signals are (a) sensory signals in feed forward pathways (e.g.: vision related ventral and dorsal pathways, (b) emotion, memory, and reward related signals. Attention modulates main signal by (a) facilitating or sharpening firings of already activated cells or (b) by suppressing or inhibiting neighboring deactivated cells [3]. Sensory signals includes signals related to all sensory systems such as vision, auditory, smell, taste, and somatosensory sensory; it could be unimodal, bimodal, or multimodal. Attentional neural signal could be re-entrant feedback signal from areas such as pre-frontal cortex [2] and could be feed forward signal from areas such as thalamic reticular nuclei [4].

Table 1. Attention deficits in patients: The references for attention deficits in various disorders related to the various forms of attention are listed. '?': Uncertain.

Disease ↓	Covert	Divided	Sustained	Selective	Switching	Attention deficit (non-specific)
Schizophrenia	[5]		[6]?, [7]	[8][9][10]	[11][12][13][14][15]	
AD		[16]	[17]	[18]		
PTSD		[19]	[19]	[20]		
DLB		[21]	[21]	[21]		[22]
MDMA use		[23]?, [24]	[25]	[23]?, [24]	[23]?	Chronic [26]
Cocaine use			[27][28]	[29]		[30]
Marihuana use		[31][32][33]	[34]	[35]		
PD				[36][37][38][39]	[40]	[41]
FLD			[42]	[36]?		
ADHD			[43]	[44]	[45][46]	[47][48]?
Bipolar disorder			[49]			[26]
Depression			[50][51]	[51][52]		[26]
Autism	[53]?		[53]?	[53]		

2.2. Visual attention tasks can be grouped into five forms[54]: **First**, in *foveal short-sustained, long-sustained, and vigilance attention tasks*, subjects attend the target foveally for short time-period (1-30 sec) in*short-sustained* attention, prolonged period of time (seconds to minutes) in *long-sustained* attention, and very long time-period (minutes to hours) in *vigilance* attention. **Second,** in *covert attention task,* subjects attend to peripheral stimulus without moving eyes while fixating foveally. **Third**, in *divided attention task*, subjects divide their attention between two (or more) different stimuli. **Fourth,** in *selective (or focused) attention task*, subjects attend the target and ignore distracters. Selective attention enhances activation in the cortical regions related to the attended stimulus and diminishes activation elsewhere. Since latent inhibition (LI) is defined as the normal ability to ignore nonrelevant (distracter) stimuli [11], LI may be helpful in explaining selective attention [8]. **Fifth**, in *switching attention task*, subjects switch attention to another task by disengaging attention from the first task and engaging to the second one. Switching tasks can be classified into 5 types [55]: (a) location switching: switching of attention between two spatial locations; (b) attribute switching: switching of attention from one feature of an object to another (e.g., from shape to color); (c) rule switching: switching of attention from one rule to reverse rule; (d) object switching: switching of attention between two stimuli or relevant objects; and (e) task switching: switching of attention between two responses or two operations that are applied to stimuli.

2.3. Neurotransmitters dopamine and glutamate, which are linked with attention and working memory, are disrupted by schizophrenia [56]. Excessive dopamine in some brain areas and reduced dopamine in other brain regions are reported in schizophrenia [57].

2.4. Effect of attention on emotion, face recognition, and reward system: Since attention modulates BOLD signal in the fusiform gyrus (GF) [58], which interacts with amygdala, attention may have effects on emotional and reward related facial stimuli. Selective attention is required to process the emotional content of faces [59] and can play an important role in dissociating facial emotion from facial identity (face recognition). Fronto-parietal attention network is involved in emotion perception and motivated attention [60]. Affectively arousing stimuli automatically draw attention, and emotion increases the facilitation effect of spatial attention and vice-versa [61]; these are consistent with *emotion-attention interaction* hypothesis. The presence of affectively arousing stimuli enhances the switching of attention to a given hemifield [62]. Attention signals from frontal cortex may potentiate the face recognition signal in GF; and attention signals from medial prefrontal cortex [63] may potentiate emotion discrimination signal in amygdale.

2.5. Attention and working memory (WM) are closely related cognitive processes. However, cognitive tasks measure performance. When performance is involved, then task characteristics also become important, which may influence the relationships between cognitive processes (such as attention and memory). The close relationship between WM and selective attention suggests that they may share common neural mechanisms [64]. Schizophrenics have selective attention deficit; therefore, their WM should also be degraded.

3. Attention Deficits

3.1. Schizophrenia: It is a chronic debilitating psychiatric disorder, which occurs in about 1% world population (lifetime prevalence is 0.5%-1%) [44]. Positive symptoms of schizophrenia [44] include (i) delusions, (ii) hallucinations, (iii) disorganized speech (e.g., frequent derailment or incoherence), (iv) grossly disorganized or catatonic behavior, and (v) positive formal thought disorder. Negative symptoms of schizophrenia are (i) affective flattening, (ii) alogia, (iii) avolition - apathy, (iv) anhedonia – asociality, and (v) disturbances in cognition (e.g., attentional impairment).

3.2. Attention deficits in schizophrenia: Schizophrenics could *split* their allocation of attention [65] and showed a peculiar mixture of over- and under-attention [66]. Chronic schizophrenics showed difficulty in maintaining a level of attention [67]. Selective attention were disturbed in schizophrenics [68].

More recent literature suggests that schizophrenics have impaired cognition, ranging from deficits in basic sensory gating to impairments in higher order functions such as attention [69][12], memory [70], vocational and psychosocial functions [71]. However, the pathophysiology of the cognitive deficits has not been fully elucidated. A hypothesis suggests that positive schizophrenic psychopathology (e.g., delusions and hallucinations) may result from exaggerated salience attribution or inability to switch attention from some inner psychic processes [72]. On the other hand, such salience ‘fixation’ may also underlie some of the negative symptomatology due to decreased salience of social activities and other natural reinforcers [73]. Schizophrenics attend to their own salient stimuli (which are abnormal) [72] and it is hard to switch their attention to normal stimuli [12][13]. Schizophrenic patients with negative symptoms (including apathy and social withdrawal) tend to ‘switch attention less’ and ‘perseverate more’ than that with the positive symptoms (including psychosis and mental disorganization) [14]. In addition, schizophrenics with positive symptoms ‘switch attention more’ and that with negative

symptoms ‘perseverate more’ than normal subjects [14]. Thus, deficits depend on the type of schizophrenia; this suggests that categorization of schizophrenics is essential during investigation. Furthermore, latent inhibition may be helpful in explaining selective attention [8] and over-switching [11] in schizophrenia. In over-switching, schizophrenics *‘tends to replace an adaptive response without being given a reason to do so*’ [11], i.e., switch attention without much reason. Positive schizophrenics who show high levels of over-switching, also exhibit impaired latent inhibition, whereas negative schizophrenics and normal controls show normal latent inhibition [11]. These findings suggest that over-switching is a specific attention deficit in positive schizophrenia. Sustained attention deficit contributes to deficit in communication in schizophrenics [7]. Schizophrenics have deficits in covert attention[5], switching attention (between color and form), and reloading of working memory; however, they appear normal on sustained attention or attention to multi-features [12][13]. Schizophrenics have deficit in selective attention to a single facial dimension (either identity or emotion) while ignoring the second [9].

In general, schizophrenics have deficit in various aspects of attention[69][12][13][5][6] as listed in Table 1, which also compares and contrasts various attention deficits in many mental diseases. It should be noted that schizophrenics do not have deficit in divided attention [66]. Many disorders have similar attention deficits. For example, patients with PD, ADHD, and schizophrenia have switching and selective attention deficits, but PD and ADHD patients do not show covert attention deficit whereas schizophrenics have this deficit according to Table 1. Thus, it is essential that all aspects of attention need to be robustly investigated for all diseases before we can identify the disease and attention deficit uniquely using comparison-and-contrast method from Table 1.

Furthermore, attention deficits of schizophrenics must be addressed in terms of their functional disability. For example, how the degraded attention influences their social life and interpersonal relationship, and how they ‘try and fail to negotiate socially mandated roles’ [74][75]. If they are unable to switch their attention to relevant traffic stimuli during driving from their task-unrelated salient stimuli, they may have fatal accident.

3.4. Attentional deficits related to emotion, face recognition, reward, and working memory in schizophrenics:
Schizophrenics show abnormal neural responses to emotionally salient stimuli in the amygdala, ventral striatum, and visual cortex [73][76]. Schizophrenics have deficits in (a) emotion recognition tasks such as differentiation of sad versus happy faces [77] and (b) facial emotion matching, facial identity matching (face recognition), and their interaction in a delayed matching task [9]. Subjects were unable to pay selective attention to emotion without interference from identity because facial identity tasks are faster than emotion tasks; emotion and identity are positively correlated; thus, emotion and identity perceptions interact and may reciprocally interfere [9][78]. Thus, an active dissociation process is needed to selectively attend and respond to one kind of facial information. In schizophrenic patients, problems in emotion correlated with problems in attention, verbal and spatial memory, and language abilities [14]. Schizophrenics have deficit in facial affect recognition and facial recognition tasks; this impaired facial processing can be explained (i) by non-specific attention deficit and selective attention deficit as performance on attention and facial tasks are significantly associated [79][80] and/or (ii) by impaired structural description of facial identity and emotion [81]. These explanations are supported by the eye movement recordings that showed fewer, shorter and abnormal fixations leading an abnormal pattern of face

exploration [82]. This suggests that schizophrenics do not attend to salient features of face resulting in an impaired facial structural description. Schizophrenics show functional deficits in (a) resolving interpersonal problems, (b) social perception, and (c) social functioning; these may be related to deficits in facial affect recognition [9][79][83][84]. In task-switching attention [55] , the decision to attend one task (or stimulus) may involve the integration of cognitive and affective information and reward related dopamine systems [85], which may be abnormal in schizophrenics[72]. In schizophrenics, attention and visual recall deficits in facial perceptual tasks may reveal unitary mechanism deficit; whereas in normal subjects, mechanisms for attention and visual recall may be distinct [9].

The severity of the negative symptoms (inattentiveness, affective flattening, and avolition–apathy) co-varied with deficits in facial processing [9]. This implies that expression of emotions and perception are closely linked. If the delusions are more severe, patients can recognize others' emotions better [9]. In general, schizophrenics have greater deficit in emotion perception than in other kinds of facial information (e.g. identity, gender or age) [9]. The dissociation processes are affected by such symptoms because of the association between negative symptoms and the difficulty in dissociating identity and emotion [9]. The patients impaired in recognizing others' expressions are not able to interact with other people in social activities [9]. In schizophrenic patients, since problems in emotion correlated with problems in attention[14] and attention-signal modulates emotion signal, *attention deficit could be the underlying mechanism in emotion deficit and in some functional disabilities*. This hypothesis needs further testing.

3.5. Neural correlates: Schizophrenics show abnormal functional MRI response to photic stimulation in visual area[86]. Schizophrenics have deficit in early-stage processing of faces [87]. The activity of the 'face' fusiform gyrus involved in the processing of facial information is more important for emotional faces [88]. The inability to process facial emotion can cause deficits in identity tasks that use expressive faces [89], and expressive faces increase the feeling of familiarity[90]. Therefore, the interference of identity in emotion processing is bi-directional; i.e. emotions may interfere in identity processing [91] and *vice-versa*. The fusiform gyrus (GF) is involved in face and object recognition; its activity is modulated by attention [58][92]; and it is abnormal (8-11% smaller gray matter volumes) in schizophrenia[93][94][95][96][97][98]. Chronic schizophrenics show gray matter volume reductions in temporal gyrus, which is related to hallucinations [99] and reduced right posterior fusiform gyrus volume, which is related to social disturbances (such as reduced sensitivity to human faces and low extraversion) [100]. Schizophrenics show abnormal processing of facial information, which may be related to reduced activity in the left fusiform gyrus (for facial working memory task) [101] and a significant increase in the volume of bilateral amygdala [102]. GF interacts with the emotion system (such as amygdala) for the evaluation of 'value'-category and with the attentional system for the evaluation of salience [58][92]. Schizophrenics with blunted affect (reduction in the intensity of emotional expression and response) show significantly reduced activation in fusiform gyrus (BA 20 and 21) compared to those with normal affect [103]. Schizophrenics show deficit in right lateral fusiform gyrus activity during the facial information (identity or affect) tasks [104]. Schizophrenics have emotion processing deficits (decreased BOLD activation) in the left amygdala and bilateral hippocampus on the emotional valence discrimination task [105]. Schizophrenics showed exaggerated amygdala activation in emotional intensity judgment task ('which face was more emotional'); this suggests that the gating of emotional input is impaired [106]. The neural correlates of facial emotion involve amygdala [9][89] which is an important area in (a) the cognitive dysfunction[107], (b) affect recognition deficit [105],

and (c) deficit in dissociating facial emotion and identity in schizophrenia. The reality distortion symptoms in schizophrenia may involve defects in parietal, frontal and hippocampal structures [108]. Schizophrenics do not show right hemisphere advantages on the facial task whereas normal controls do [109]. Frontal and prefrontal cortex and their interaction with the fusiform gyrus may also be involved in face recognition [110]. The frontal cortex modulates the activities of temporal area that are involved in facial analysis [9]. In schizophrenics, the frontal cortex is hypoactive and the coordination of activity between the frontal and temporal cortices is abnormal [111], which may lead to inability to dissociate facial emotion and identity [9]. Schizophrenics showed hypoactivity in bilateral frontal and right temporal area on CPT (Continuous Performance Test) task [112]. Perception and attention deficit (PAD) model for recurrent complex visual hallucinations (RCVH) in schizophrenia, delirium, dementing illnesses, eye disease, and on the borders of sleep suggests that RCVH may involve abnormal activations in a 'lateral frontal cortex-ventral visual stream system' with cholinergic dysfunction [113]. In the dorsolateral prefrontal cortex (DLPFC) of schizophrenics, GABA synthesis is reduced, which leads to reduction in the gamma-frequency synchronized neuronal activity; this in turn leads to a diminished capacity for working memory function [114]. Furthermore, the function of hippocampus may include contextual aspect of emotional processing in addition to usual processing of memory; it may be relevant for face identity as context and emotion discrimination as a function of amygdala [115][116]. Memory deficit is related to attention deficit, and working memory is a core deficit in schizophrenia; this implies lower capacity for object and face memory [117]. The disruption of memory-emotion-attention related left hippocampal–limbic–prefrontal neural net is critical in the loss of drive and initiative, perseveration, and irrelevant associations observed in schizophrenia.[118][119]

3.6. Models of schizophrenia and test hypotheses: Biochemical models suggest two models of schizophrenia, one based on excessive inhibitory neuromodulator dopamine (dopaminergic hyperactivity in the brain) and other based on lack of excitatory neurotransmitter glutamate, are reported [3][120][121], which reveal the interplay of the two signaling systems (dopamine and glutamate). Administration of dopamine agonist amphetamine or glutamate antagonist ketamine produces transient psychotic state, but the latter produces more "complete" schizophrenia [120]. The dopamine input in the indirect pathways is inhibitory and that in the direct pathways appears to be excitatory [122]. Imbalance of dopamine and glutamate leads to deficits in attention and working memory [3][120][121]. Serotonin, GABA, and norepinephrine may also be play role in schizophrenia. As attention degrades, memory also degrades. Schizophrenics have attention and memory deficits. Attention deficits in schizophrenia could be explained by (a) the dopaminergic dysregulation that disrupts the balance of specific attentional functions of facilitation and inhibition and (b) the failure of glutamatergic-mediated recurrent inhibition of discrete but widely distributed neural circuits [3][121]. Thus, *attention modulates main signals via dopamine and glutamate neuromodulators, which could be the underlying mechanisms of both (dopamine and glutamate) models of schizophrenia*. This hypothesis needs further testing.

From above review a following specific hypothesis can be generated:*The attention system (salience), visual system (face recognition), emotion and reward system (facial emotion), and their interactions are abnormal in schizophrenics. The cognitive processes involved in the dissociation of emotion and face recognition are altered in schizophrenia.*
Furthermore, a hypothesis can be formulated as follow: in schizophrenics, the selective attention deficit leads to deficit in (i) face discrimination (different faces with same or different emotion vs. same faces with same emotion), (ii) emotion

discrimination (different emotions with same or different faces vs. same emotions with same faces), (iii) facial identity (matching faces of same or different emotions), (iv) emotion identity (matching emotion of same or different faces), (v) emotion identification (identify emotion with same or different faces), (vi) face recognition (recognize face with same or different emotions), and (vii) disassociation of emotion (discrimination, identity, or identification) from face (discrimination, identity, or recognition).

4. Future directions

Psychophysical studies such as ours on visual mechanisms [123][124][125][126][127][128] and many others [129][130][131] were performed with high degree of attention. However, we need to collect data with a low level of attention or without attention to the test stimulus to separate attentional areas. For attention deficits in schizophrenics, we recommend investigating 'common' and 'specific' activated areas for at the least 2 aspects of attention using fMRI combined with psychophysical studies: switching attention and selective attention. The rationale is that schizophrenics have deficits mostly in these aspects [11][8][5][9]. However, other aspects of attention (covert, sustained, and divided attention) deficits in schizophrenics should also be investigated.

Switching attention deficit: Patients with schizophrenia were significantly impaired on visual attention tasks that required switching of attention (between color and form) [12][13]. Schizophrenics also have deficit in motion processing[132]. These separate reports of switching attention and motion deficits justify the investigation of both deficits in the same group of schizophrenics for the search of better marker. Therefore, we propose the following experimental design directly related to schizophrenia where subjects switch attention between color and motion attributes. In the reference task (R), target stimulus is not presented. Subjects are required to alternatively press the positive and negative buttons whenever a stimulus appeared as soon as possible after the presentation of each stimulus. This will allow for the control of non-cognitive visual processing and simple motor output by direct subtraction of fMR images. In the switching attention task (A), target stimulus is displayed and subjects are required to remember the color and motion of the stimulus. This is followed by 80 serially presented stimuli and subjects are required to respond a positive (stimulus matches with remembered stimulus) or negative response on a button press device. The reaction time is recorded. Incorrect responses to target or non-target stimuli are counted as errors. Subjects are required to alternate between responding (a) to stimuli matching the target for the color (ignoring motion), then (b) to stimuli matching the target for motion (ignoring color). Subjects are required to switch only when the appropriate match is found. This experiment may also involve working memory and active stimulus processing and hence we need to isolate them. For this purpose, the second experiment A is repeated without switching the attention (N), i.e., if a subject started matching color then the subject continues matching color (ignoring motion) for whole session. The outcome measures are reaction time (RT) and number of errors made in matching the targets. Functional MR images are obtained while performing the above psychophysical experiment. Using subtraction method, the results of experiments [A – R] are compared with that of [N – R] to estimate results for switching attention (mostly) alone.

Furthermore, the above group of experiments can be extended (a) to test the various forms of attention-deficits listed in

Table 1 for various disorders and (b) to find 'sources' of attention related to norephinephrine (locus coeruleus: LC), dopamine (substantia nigra: SN), and serotonin (raphe nucleus: RN) using fMRI and to apply structural equation modeling [133][134][135] to investigate how they modulate the visual processing for normal subjects and patients. Specifically, the test hypotheses for schizophrenia are given in Section 3.6. We proposed fMRI and psychophysical methods to separate attentional areas and to investigate the effects of attention on schizophrenia. This is one of many possible approaches (not discussed here) to address the same problem.

5. Conclusions

Attention is a neural signal, mostly reentrant signal[2], which modulates main signal. Main signals include (a) the feed forward sensory signals such as vision related signals in ventral and dorsal pathways and (b) signals related to memory, emotion, and reward systems. This modulating signal could be in forward (bottom-up) direction or mostly in backward (feedback, top-down) direction. The 'sources' of attention could be TRN, SN, LC, RN for bottom-up or frontal cortex (such as ACC, MFG) for top-down direction. The areas for the 'sources' of attention and the areas for the 'targets' of attentional signal (where attention signal modulates the main signal) need to carefully separated to make the attention research simple and to resolve controversies in both normal subjects and patients. Then basic attention mechanism may emerge in simpler form. Understanding the influence of attention on visual processing in healthy individuals can provide the foundation for studying the role of altered attention in various disease states characterized by impaired attention (Table 1). A simple working hypothesis is "*Attention is a neural signal that modulates main signals such as stimulus related feed forward signal. This modulation is disturbed in mental disorders. For example, schizophrenics may have deficit in switching and selective (salience) attention.*"

Statements and Declarations

Competing interests

The author declares that he has no competing financial interests.

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References

1. [^]*Kanwisher N, Wojciulik E: Visual attention: insights from brain imaging. Nat Rev Neurosci 2000, 1(2):91-100.*
2. [a, b, c]*Hamker FH: The Reentry Hypothesis: The Putative Interaction of the Frontal Eye Field, Ventrolateral Prefrontal*

Cortex, and Areas V4, IT for Attention and Eye Movement. Cereb Cortex 2004, 15(4):431-447.

3. [a, b, c, d] *Nestor PG, O'Donnell BF: The Mind Adrift: Attentional Dysregulation in Schizophrenia. In: The Attentive Brain. Edited by Parasuraman R. Cambridge, MA: The MIT Press; 1998: 527-546.*
4. [^] *Crick F: Function of the thalamic reticular complex: the searchlight hypothesis. Proc Natl Acad Sci U S A 1984, 81:4586– 4590.*
5. [a, b, c, d] *Posner MI, Early TS, Reiman E, Pardo EJ, Dhawan M: Asymmetries in hemispheric control of attention in schizophrenia. Archives of General Psychiatry 1988, 45:814-821.*
6. [a, b] *Salgado-Pineda P, Baeza I, Pe´rez-Go´mez M, Vendrell P, Junque´ C, Bargallo´ N, Bernardob M: Sustained attention impairment correlates to gray matter decreases in first episode neuroleptic-naive schizophrenic patients. NeuroImage 2003, 19:365–375.*
7. [a, b] *Docherty NM: Cognitive impairments and disordered speech in schizophrenia: thought disorder, disorganization, and communication failure perspectives. J Abnorm Psychol 2005, 114(2):269-278.*
8. [a, b, c, d] *Lubow RE: Construct validity of the animal latent inhibition model of selective attention deficits in schizophrenia. Schizophrenia Bulletin 2005, 31(1):139-153.*
9. [a, b, c, d, e, f, g, h, i, j, k, l, m, n, o] *Martin F, Baudouin JY, Tiberghien G, Franck N: Processing emotional expression and facial identity in schizophrenia. Psychiatry Res 2005, 134(1):43-53.*
10. [^] *Chudasama Y, Robbins TW: Psychopharmacological approaches to modulating attention in the five-choice serial reaction time task: implications for schizophrenia. Psychopharmacology (Berl) 2004, 174(1):86-98. Epub 2004 Apr 2008.*
11. [a, b, c, d, e, f] *Yogev H, Sirota P, Gutman Y, Hadar U: Latent inhibition and overswitching in schizophrenia. Schizophrenia Bulletin, 2004, 30(4):713-726.*
12. [a, b, c, d, e, f] *Smith GL, Large MM, Kavanagh DJ, Karayanidis F, Barrett NA, Michie PT, O'Sullivan BT: Further evidence for a deficit in switching attention in schizophrenia. J Abnorm Psychol 1998, 107:390–398.*
13. [a, b, c, d, e] *Barrett NA, Large MM, Smith GL, Karayanidis F, Michie PT, Kavanagh DJ, Fawdry R, Henderson D, O'Sullivan BT: Human brain regions required for the dividing and switching of attention between two features of a single object. Cognitive Brain Research 2003, 17:1–13.*
14. [a, b, c, d, e] *Yogev H, Hadar U, Gutman Y, Sirota P: Perseveration and over-switching in schizophrenia. Schizophr Res 2003, 61(2-3):315-321.*
15. [^] *Sharpe MH: Distractibility in early Parkinson's disease. Cortex 1990, 26:239-246.*
16. [^] *Baddeley AD, Baddeley HA, Bucks RS, Wilcock GK: Attentional control in Alzheimer's disease. Brain 2001, 124(Pt 8):1492-1508. Comment in: Brain. 2001 Aug;1124(Pt 1498):1479-1481.*
17. [^] *Estevez-Gonzalez A, Garcia-Sanchez C, Boltes A, Garcia-Nonell C, Rigau-Ratera E, Otermin P, Gironell A, Kulisevsky J: Sustained attention in the preclinical phase of Alzheimer's disease. Rev Neurol 2003, 36(9):829-832.*
18. [^] *Levinoff EJ, Li KZ, Murtha S, Chertkow H: Selective attention impairments in Alzheimer's disease: evidence for dissociable components. Neuropsychology 2004, 18(3):580-588.*
19. [a, b] *Jenkins MA, Langlais PJ, Delis DA, Cohen RA: Attentional dysfunction associated with posttraumatic stress disorder among rape survivors. Clin Neuropsychol 2000, 14(1):7-12.*

20. ^Attias J, Bleich A, Furman V, Zinger Y: Event-related potentials in post-traumatic stress disorder of combat origin. Biol Psychiatry 1996, 40(5):373-381.
21. a, b, cCalderon J, Perry RJ, Erzinclioglu SW, Berrios GE, Dening TR, Hodges JR: Perception, attention, and working memory are disproportionately impaired in dementia with Lewy bodies compared with Alzheimer's disease. J Neurol Neurosurg Psychiatry 2001, 70(2):157-164. Comment in: J Neurol Neurosurg Psychiatry. 2001 Feb;2070(2002):2148.
22. ^Ballard C, O'Brien J, Gray A, Cormack F, Ayre G, Rowan E, Thompson P, Bucks R, McKeith I, Walker M et al: Attention and fluctuating attention in patients with dementia with Lewy bodies and Alzheimer disease. Arch Neurol 2001, 58(6):977-982.
23. a, b, cZakzanis KK, Young DA, Radkhoshnoud NF: Attentional processes in abstinent Methylenedioxymethamphetamine (ecstasy) users. Appl Neuropsychol 2002, 9(2):84-91.
24. a, bGouzoulis-Mayfrank E, Daumann J, Tuchtenhagen F, Pelz S, Becker S, Kunert H-K, Fimm B, Sass H: Impaired cognitive performance in drug free users of recreational ecstasy (MDMA). J Neurol Neurosurg Psychiatry 2000, 68:719–725.
25. ^McCann UD, Mertl M, Eligulashvili V, Ricaurte GA: Cognitive performance in (+/-) 3,4-methylenedioxymethamphetamine (MDMA, "ecstasy") users: a controlled study. Psychopharmacology (Berl) 1999, 143(4):417-425.
26. a, b, cMontoya AG, Sorrentino R, Lukas SE, Price BH: Long-term neuropsychiatric consequences of "ecstasy" (MDMA): a review. Harv Rev Psychiatry 2002, 10(4):212-220.
27. ^Gendle MH, Strawderman M, Mactutus CF, Booze RM, Levitsky DA, Strupp BJ: Impaired sustained attention and altered reactivity to errors in an animal model of prenatal cocaine exposure. Brain Res Dev Brain Res 2003, 147(1-2):85-96.
28. ^Heffelfinger AK, Craft S, White DA, Shyken J: Visual attention in preschool children prenatally exposed to cocaine: implications for behavioral regulation. J Int Neuropsychol Soc 2002, 8(1):12-21.
29. ^Gendle MH, White TL, Strawderman M, Mactutus CF, Booze RM, Levitsky DA, Strupp BJ: Enduring effects of prenatal cocaine exposure on selective attention and reactivity to errors: evidence from an animal model. Behav Neurosci 2004, 118(2):290-297.
30. ^Rosselli M, Ardila A, Lubomski M, Murray S, King K: Personality profile and neuropsychological test performance in chronic cocaine-abusers. Int J Neurosci 2001, 110(1-2):55-72.
31. ^Verrico CD, Jentsch JD, Roth RH, Taylor JR: Repeated, intermittent delta (9)-tetrahydrocannabinol administration to rats impairs acquisition and performance of a test of visuospatial divided attention. Neuropsychopharmacology 2004, 29(3):522-529.
32. ^Macavoy MG, Marks DF: Divided attention performance of cannabis users and non-users following cannabis and alcohol. Psychopharmacologia 1975, 44(2):147-152.
33. ^Marks DF, MacAvoy MG: Divided attention performance in cannabis users and non-users following alcohol and cannabis separately and in combination. Psychopharmacology (Berl) 1989, 99(3):397-401.
34. ^Fried PA, Watkinson B, Gray R: A follow-up study of attentional behavior in 6-year-old children exposed prenatally to marihuana, cigarettes, and alcohol. Neurotoxicol Teratol 1992, 14(5):299-311.

35. ^Solowij N, Michie PT, Fox AM: Differential impairments of selective attention due to frequency and duration of cannabis use. Biol Psychiatry 1995, 37(10):731-739.
36. a, b Lee SS, Wild K, Hollnagel C, Grafman J: Selective visual attention in patients with frontal lobe lesions or Parkinson's disease. Neuropsychologia 1999, 37(5):595-604.
37. ^Filoteo JV, Maddox WT: Quantitative modeling of visual attention processes in patients with Parkinson's disease: effects of stimulus integrality on selective attention and dimensional integration. Neuropsychology 1999, 13(2):206-222.
38. ^Maddox WT, Filoteo JV, Delis DC, Salmon DP: Visual selective attention deficits in patients with Parkinson's disease: A quantitative model-based approach. Neuropsychology 1996, 10:197-218.
39. ^Filoteo JV, Williams BJ, Rilling LM, Roberts JV: Performance of Parkinson's disease patients on the Visual Search and Attention Test: impairment in single-feature but not dual-feature visual search. Arch Clin Neuropsychol 1997, 12(7):621-634.
40. ^Cools R, Barker RA, Sahakian BJ, Robbins TW: Mechanisms of cognitive set flexibility in Parkinson's disease. Brain 2001, 124(Pt 12):2503-2512.
41. ^Rowe J, Stephan KE, Friston K, Frackowiak R, Lees A, Passingham R: Attention to action in Parkinson's disease: impaired effective connectivity among frontal cortical regions. Brain 2002, 125(Pt 2):276-289.
42. ^Rueckert L, Grafman J: Sustained attention deficits in patients with right frontal lesions. Neuropsychologia 1996, 34(10):953-963.
43. ^Roth RM, Wishart. H.A., Flashman LA, Riordan HJ, Huey L, Saykin AJ: Contribution of organizational strategy to verbal learning and memory in adults with attention-deficit/hyperactivity disorder. Neuropsychology 2004, 18(1):78-84.
44. a, b, c DSM-IV: Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Fourth edn. Washington, DC: American Psychiatric Association; 1994.
45. ^Cepeda NJ, Kramer AF, Gonzalez de Sather JC: Changes in executive control across the life span: examination of task-switching performance. Dev Psychol 2001, 37(5):715-730.
46. ^Tamm L, Menon V, Ringel J, Reiss AL: Event-related FMRI evidence of frontotemporal involvement in aberrant response inhibition and task switching in attention-deficit/hyperactivity disorder. J Am Acad Child Adolesc Psychiatry 2004, 43(11):1430-1440.
47. ^Vaidya CJ, Austin G, Kirkorian G, Ridlehuber HW, Desmond JE, Glover GH, Gabrieli JD: Selective effects of methylphenidate in attention deficit hyperactivity disorder: a functional magnetic resonance study. Proc Natl Acad Sci USA 1998, 95:14494–14499.
48. ^Bunge SA, Dudukovic NM, Thomason ME, Vaidya CJ, Gabrieli JD: Immature frontal lobe contributions to cognitive control in children: evidence from fMRI. Neuron 2002, 33:301–311.
49. ^Clark L, Goodwin GM: State- and trait-related deficits in sustained attention in bipolar disorder. Eur Arch Psychiatry Clin Neurosci 2004, 254(2):61-68.
50. ^Sevigny MC, Everett J, Grondin S: Depression, attention, and time estimation. Brain and Cognition 2003, 53(2):351–353.
51. a, b Politis A, Lykouras L, Mourtzouchou P, Christodoulou GN: Attentional disturbances in patients with unipolar

psychotic depression: A selective and sustained attention study. Compr Psychiatry 2004, 45(6):452-459.

52. ^*Lang AJ, Sarmiento J: Relationship of attentional bias to anxiety sensitivity and panic. Depress Anxiety 2004:Sep 1; [Epub ahead of print].*
53. a, b, c *Belmonte MK, Yurgelun-Todd DA: Functional anatomy of impaired selective attention and compensatory processing in autism. Cognitive Brain Research 2003, 17:651–664.*
54. ^*Bushnell PJ, Levin ED, Marrocco RT, Sarter MF, Strupp BJ, Warburton DM: Attention as a target of intoxication: insights and methods from studies of drug abuse. Neurotoxicol Teratol 2000, 22(4):487-502.*
55. a, b *Wager TD, Jonides J, Reading S: Neuroimaging studies of shifting attention: a meta-analysis. Neuroimage 2004, 22(4):1679-1693.*
56. ^*Joseph MH, Frith CD, Waddington JL: Dopaminergic mechanisms and cognitive deficit in schizophrenia. A neurobiological model. Psychopharmacology (Berl) 1979, 63(3):273-280.*
57. ^*Davis KL, Fiori M, Davis BM: et al. Dopaminergic disregulation in schizophrenia: A target for new drugs. Drug Dev Res 1986, 9:71-83.*
58. a, b, c *Wojciulik E, Kanwisher N, Driver J: Covert visual attention modulates face-specific activity in the human fusiform gyrus: fMRI study. J Neurophysiol 1998, 79:1574–1578.*
59. ^*Pessoa L, Kastner S, Ungerleider LG: Attentional control of the processing of neutral and emotional stimuli. Cognitive Brain Research 2002, 15:31–45.*
60. ^*Moratti S, Keil A, Stolarova M: Motivated attention in emotional picture processing is reflected by activity modulation in cortical attention networks. Neuroimage 2004, 21(3):954-964.*
61. ^*Keil A, Moratti S, Sabatinelli D, Bradley MM, Lang PJ: Additive effects of emotional content and spatial selective attention on electrocortical facilitation. Cereb Cortex 2005, 15(8):1187-1197.*
62. ^*Stormark KM, Hugdahl K: Peripheral cuing of covert spatial attention before and after emotional conditioning of the cue. Int J Neurosci 1996, 86(3-4):225-240.*
63. ^*Geday J, Gjedde A, Boldsen AS, Kupers R: Emotional valence modulates activity in the posterior fusiform gyrus and inferior medial prefrontal cortex in social perception. Neuroimage 2003, 18(3):675-684.*
64. ^*Desimone R: Visual attention mediated by biased competition in extrastriate visual cortex. Philos Trans R Soc Lond B Biol Sci 1998, 353(1373):1245-1255.*
65. ^*Bleuler E: Dementia praecox or the group of schzophrenias. New York: International Universities Press (Original work published 1911); 1911/1950.*
66. a, b *Kraepelin E: Dementia Praecox and Paraphrenia. Edinburgh: E. & S. Livingstone (Originally published in 1913.); 1913/1919.*
67. ^*Huston PE, Senf R: Psychopathology of schizophrenia and depression. I. Effect of amytal and amphetamine sulfate on level and maintenance of attention. Am J Psychiatry 1952, 109(2):131-138.*
68. ^*McGhie A: Disturbances in Selective Attention in Schizophrenia. Proc R Soc Med 1964, 57:419-422.*
69. a, b *Hagh-Shenas H, Toobai S, Makaremi A: Selective, sustained, and shift in attention in patients with diagnoses of schizophrenia. Percept Mot Skills 2002, 95(3 Pt 2):1087-1095.*
70. ^*Heaton RK, Gladsjo JA, Palmer BW, Kuck J, Marcotte TD, Jeste DV: Stability and course of neuropsychological*

deficits in schizophrenia. Arch Gen Psychiatry 2001, 58(1):24-32.

71. ^*Meltzer HY: Cognitive factors in schizophrenia: causes, impact, and treatment. CNS Spectr 2004, 9((10 Suppl 11)):15-24.*
72. a, b, c*Kapur S: Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. Am J Psychiatry 2003, 160(1):13-23.*
73. a, b*Taylor SF, Phan KL, Britton JC, Liberzon I: Neural Response to Emotional Salience in Schizophrenia. Neuropsychopharmacology 2005:Feb 2; [Epub ahead of print].*
74. ^*Carter M, Flesher S: The neurosociology of schizophrenia: vulnerability and functional disability. Psychiatry 1995, 58(3):209-224.*
75. ^*Estroff SE: Self, identity, and subjective experiences of schizophrenia: in search of the subject. Schizophr Bull 1989, 15(2):189-196.*
76. ^*Phan KL, Taylor SF, Welsh RC, Ho SH, Britton JC, I. L: Neural correlates of individual ratings of emotional salience: a trial-related fMRI study. Neuroimage 2004, 21(2):768-780.*
77. ^*Sachs G, Steger-Wuchse D, Kryspin-Exner I, Gur R, Katschnig H: Facial recognition deficits and cognition in schizophrenia. Schizophr Res 2004, 68(1):27-35.*
78. ^*Schweinberger SR, Burton AM, Kelly SW: Asymmetric relationship between identity and emotion perception: experiments with morphed faces. Perception & Psychophysics 1999, 61:1102– 1115.*
79. a, b*Addington J, Addington D: Neurocognitive and social functioning in schizophrenia: a 2.5 year follow-up study. Schizophr Res 2000, 44(1):47-56.*
80. ^*Everett J, Laplante L, Thomas J: The selective attention deficit in schizophrenia. Limited resources or cognitive fatigue? J Nerv Ment Dis 1989, 177(12):735-738.*
81. ^*Archer J, Hay DC, Young AW: Movement, face processing and schizophrenia: Evidence of a differential deficit in expression analysis. British Journal of Clinical Psychology 1994, 33:517–528.*
82. ^*Williams LM, Loughland CM, Gordon E, Davidson D: Visual scanpaths in schizophrenia: Is there a deficit in face recognition? Schizophrenia Research 1999, 40:189–199.*
83. ^*Corrigan P, Toomey R: Interpersonal problem solving and information processing in schizophrenia. Schizophr Bull 1995, 21:395–403.*
84. ^*Treue S: Neural correlates of attention in primate visual cortex. Trends Neurosci 2001, 24(5):295-300.*
85. ^*O'Reilly RC, Noelle DC, Braver TS, Cohen JD: Prefrontal cortex and dynamic categorization tasks: representational organization and neuromodulatory control. Cereb Cortex 2002, 12(3):246-257.*
86. ^*Renshaw PF, Yurgelun-Todd DA, Cohen BM: Greater hemodynamic response to photic stimulation in schizophrenic patients: an echo planar MRI study. Am J Psychiatry 1994, 151:1493-1495.*
87. ^*Herrmann MJ, Ellgring H, Fallgatter AJ: Early-stage face processing dysfunction in patients with schizophrenia. Am J Psychiatry 2004, 161(5):915-917.*
88. ^*Dolan RJ, Fletcher P, Morris J, Kapur N, Deakin JFW, Frith CD: Neural activation during covert processing of positive emotional facial expressions. NeuroImage 1996, 4:194–200.*
89. a, b*Young AW, Hellawell DJ, Van De Wal C, Johnson M.: Facial expression processing after amygdalotomy.*

Neuropsychologia 1996, 34:31– 39.

90. ^*Baudouin J-Y, Gilibert D, Sansone S, Tiberghien G: When the smile is a cue to familiarity. Memory 2000, 8:285– 292.*
91. ^*Baudouin JY, Martin F, Tiberghien G, Verlut I, Franck N: Selective attention to facial emotion and identity in schizophrenia. Neuropsychologia 2002, 40(5):503-511.*
92. a, b*Aharon I, Etcoff N, Ariely D, Chabris CF, O'Connor E, Breiter HC: Beautiful Faces Have Variable Reward Value: fMRI and Behavioral Evidence. Neuron 2001, 32:537–551.*
93. ^*Dickey CC, McCarley RW, Voglmaier MM, Niznikiewicz MA, Seidman LJ, Frumin M, Toner S, Demeo S, Shenton ME: A MRI study of fusiform gyrus in schizotypal personality disorder. Schizophr Res 2003, 64(1):35-39.*
94. ^*Paillere-Martinot M, Caclin A, Artiges E, Poline JB, Joliot M, Mallet L, Recasens C, Attar-Levy D, Martinot JL: Cerebral gray and white matter reductions and clinical correlates in patients with early onset schizophrenia. Schizophr Res 2001, 50(1-2):19-26.*
95. ^*Pantelis C, Velakoulis D, McGorry PD, Wood SJ, Suckling J, Phillips LJ, Yung AR, Bullmore ET, Brewer W, Soulsby B et al: Neuroanatomical abnormalities before and after onset of psychosis: a cross-sectional and longitudinal MRI comparison. Lancet 2003, 361(9354):281-288; Comment in: Lancet. 2003 Jan 2025;2361(9354):2270-2001.*
96. ^*Lee CU, Shenton ME, Salisbury DF, Kasai K, Onitsuka T, Dickey CC, Yurgelun-Todd D, Kikinis R, Jolesz FA, McCarley RW: Fusiform gyrus volume reduction in first-episode schizophrenia: a magnetic resonance imaging study. Arch Gen Psychiatry 2002, 59(9):775-781.*
97. ^*Hooker C, Park S: Emotion processing and its relationship to social functioning in schizophrenia patients. Psychiatry Res 2002, 112(1):41-50.*
98. ^*Onitsuka T, Shenton ME, Kasai K, Nestor PG, Toner SK, Kikinis R, Jolesz FA, McCarley RW: Fusiform gyrus volume reduction and facial recognition in chronic schizophrenia. Arch Gen Psychiatry 2003, 60(4):349-355.*
99. ^*Onitsuka T, Shenton ME, Salisbury DF, Dickey CC, Kasai K, Toner SK, Frumin M, Kikinis R, Jolesz FA, McCarley RW: Middle and inferior temporal gyrus gray matter volume abnormalities in chronic schizophrenia: an MRI study. Am J Psychiatry 2004, 161(9):1603-1611.*
100. ^*Onitsuka T, Nestor PG, Gurrera RJ, Shenton M E, Kasai K, Frumin M, Niznikiewicz MA, McCarley RW: Association between reduced extraversion and right posterior fusiform gyrus gray matter reduction in chronic schizophrenia. Am J Psychiatry 2005, 162(3):599-601.*
101. ^*Yoo SS, Choi BG, Juh RH, Park JM, Pae CU, Kim JJ, Lee SJ, Lee C, Paik IH, Lee CU: Working memory processing of facial images in schizophrenia: fMRI investigation. Int J Neurosci 2005, 115(3): 351-366.*
102. ^*Morys JM, Dziewiatkowski J, Bobek-Billewicz B, Ratajczak I, Narkiewicz O, Morys J: Do the asymmetry and the size of the structures of the temporal lobe persist in early stages of schizophrenia? Folia Morphol (Warsz) 2004, 63(4):401-405.*
103. ^*Mancini-Marie A, Stip E, Fahim C, Mensour B, Leroux JM, Beaudoin G, Bentaleb LA, Bourgouin P, Beauregard M: Fusiform gyrus and possible impairment of the recognition of emotional expression in schizophrenia subjects with blunted affect: a fMRI preliminary report. Brain Cogn 2004, 54(2):153-155.*
104. ^*Quintana J, Wong T, Ortiz-Portillo E, Marder SR, Mazziotta JC: Right lateral fusiform gyrus dysfunction during facial information processing in schizophrenia. Biol Psychiatry 2003, 53(12):1099-1112.*

105. [a, b]*Gur RE, McGrath C, Chan RM, Schroeder L, Turner T, Turetsky BI, Kohler C, Alsop D, Maldjian J, Ragland JD et al: An fMRI Study of Facial Emotion Processing in Patients with Schizophrenia. Am J Psychiatry, 2002, 159:1992 - 1999.*

106. [^]*Kosaka H, Omori M, Murata T, Iidaka T, Yamada H, Okada T, Takahashi T, Sadato N, Itoh H, Yonekura Y et al: Differential amygdala response during facial recognition in patients with schizophrenia: an fMRI study. Schizophr Res 2002, 57(1):87-95.*

107. [^]*Evangeli M, Broks P: Face processing in schizophrenia: parallels with the effects of amygdala damage. Cognitive Neuropsychiatry 2000, 5: 81– 104.*

108. [^]*Guillem F, Bicu M, Pampoulova T, Hooper R, Bloom D, Wolf MA, Messier J, Desautels R, Todorov C, Lalonde P et al: The cognitive and anatomo-functional basis of reality distortion in schizophrenia: a view from memory event-related potentials. Psychiatry Res 2003, 117(2):137-158.*

109. [^]*White MS, Maher BA, Manschreck TC: Hemispheric specialization in schizophrenics with perceptual aberration. Schizophr Res 1998, 32(3):161-170.*

110. [^]*Kanwisher N, Moscovitch M: The cognitive neuroscience of face processing: an introduction. Cognitive Neuropsychology 2000, 17:1 – 11.*

111. [^]*Mitchell RCL, Elliott R, Woodruff PWR: fMRI and cognitive dysfunction in schizophrenia. Trends in Cognitive Science 2001, 5:71–81.*

112. [^]*Buchsbaum MS, Nuechterlein KH, Haier RJ, Wu J, Sicotte N, et-al.: Glucose metabolic rate in normalsand schizophrenics during the Continuous Performance Test assessed by positron emission tomography. Br J Psychiatry 1990, 156:216–227.*

113. [^]*Collerton D, Perry E, McKeith I: Why people see things that are not there: A novel Perception and Attention Deficit model for recurrent complex visual hallucinations. Behav Brain Sci 2005, 28(6):737-757.*

114. [^]*Lewis DA, Hashimoto T, Volk DW: Cortical inhibitory neurons and schizophrenia. Nature Reviews Neuroscience 2005, 6:312-324.*

115. [^]*LeDoux J, Phelps F: Emotional networks in the brain. In: Handbook of Emotions. Edited by Lewis M, Haviland-Jones JM, 2nd edn. New York: Guilford Press; 2000: 157-172.*

116. [^]*LeDoux JE: Emotion circuits in the brain. Annu Rev Neurosci 2000, 23:155-184.*

117. [^]*Silver H, Feldman P, Bilker W, Gur RC: Working memory deficit as a core neuropsychological dysfunction in schizophrenia. Am J Psychiatry 2003, 160(10):1809-1816.*

118. [^]*Melges FT: Disorders of time and the brain in severe mental illness. In: The study of time VI: Time and mind. Edited by Fraser JT. Madison, CT: International Universities Press; 1989.*

119. [^]*Dawson KA: Temporal organization of the brain: Neurocognitive mechanisms and clinical implications. Brain and Cognition 2004, 54(1):75–94.*

120. [a, b, c]*Krystal JH, Perry EB, Jr., Gueorguieva R, Belger A, Madonick SH, Abi-Dargham A, Cooper TB, Macdougall L, Abi-Saab W, D'Souza DC: Comparative and interactive human psychopharmacologic effects of ketamine and amphetamine: implications for glutamatergic and dopaminergic model psychoses and cognitive function. Arch Gen Psychiatry 2005, 62(9):985-994.*

121. [a, b, c]*Nestor PG, Han SD, Niznikiewicz M, Salisbury D, Spencer K, Shenton ME, McCarley RW: Semantic disturbance in schizophrenia and its relationship to the cognitive neuroscience of attention. Biol Psychol 2001, 57(1-3):23-46.*
122. ^*Carlsson A: The dopamine theory revisited. In: Schizophrenia. Edited by Hirsch SR, Weinberger DR: Blackwell Science; 1995: 379-400.*
123. ^*Vimal RLP: Spatial-frequency tuning of sustained nonoriented units of the Red-Green channel. J Opt Soc Am A Opt Image Sci Vis 1998, 15(1):1-15.*
124. ^*Vimal RLP: Spatial frequency tuned mechanisms of the Red-Green channel estimated by oblique masking. J Opt Soc Am A Opt Image Sci Vis 2002, 19(2):276-288.*
125. ^*Vimal RLP: Spatial frequency discrimination: a comparison of achromatic and chromatic conditions. Vision Research 2002, 42(5):599-611.*
126. ^*Vimal RLP: Spatial color contrast matching: broad-bandpass functions and the flattening effect. Vision Research 2000, 40(23):3231-3243.*
127. ^*Vimal RLP: Orientation tuning of the spatial-frequency-tuned mechanisms of the Red-Green channel. Journal of the Optical Society of America A 1997, 14:12622-12632; Errata, J. Opt. Soc. Am. A 12615, 12758.*
128. ^*Vimal RLP: Color-luminance interaction: data produced by oblique cross masking. J Opt Soc Am A Opt Image Sci Vis 1998, 15(7):1756-1766; Errata, J. Opt. Soc. Am. A 1715, 2931.*
129. ^*Wilson HR, McFarlane DK, Phillips GC: Spatial frequency tuning of orientation selective units estimated by oblique masking. Vision Research 1983, 23(9):873-882.*
130. ^*Kaiser PK, Boynton RM: Human Color Vision, 2nd edn. Washington, D.C.: Optical Society of America; 1996.*
131. ^*Phillips GC, Wilson HR: Orientation bandwidths of spatial mechanisms measured by masking. J Opt Soc Am A 1984, 1:226-232.*
132. ^*Chen Y, Nakayama K, Levy DL, Matthysse S, Holzman PS: Psychophysical isolation of a motion-processing deficit in schizophrenics and their relatives and its association with impaired smooth pursuit. Proc Natl Acad Sci USA 1999, 96(8):4724-4729.*
133. ^*McIntosh AR, Gonzalez-Lima F: Structural equation modelling and its application to network analysis in functional brain imaging. Hum Brain Mapping 1994, 2:2–22.*
134. ^*Buchel C, Friston KJ: Modulation of connectivity in visual pathways by attention: cortical interactions evaluated with structural equation modelling and fMRI. Cereb Cortex 1997, 7(8):768-778.*
135. ^*Goncalves MS, Hall DA: Connectivity analysis with structural equation modelling: an example of the effects of voxel selection. Neuroimage 2003, 20(3):1455-1467.*