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C. R. Biologies 328 (2005) 169–175



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Neurosciences

# The neural basis of hallucinations and delusions

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Received 10 September 2004; accepted after revision 19 October 2004

Available online 19 December 2004

Presented by Nicole Le Douarin & Henri Korn

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## Abstract

Schizophrenia is a biologically based disorder characterised by false perceptions (hallucinations) and false beliefs (delusions). The underlying physiological cause of these mental abnormalities remains unknown. There is increasing evidence that one class of symptom, the ‘made experiences’ including delusions of alien control and thought insertion, is associated with abnormalities in the mechanism that predicts the outcome of intended actions (the forward model). For these patients active movements feel like passive movements. As a result these patients do not feel in control of their actions. However, comparison with various neurological disorders, such as those associated with parietal lobe lesions, suggest that this abnormal experience is not sufficient to explain the feeling that some other agent is controlling is one’s actions. Preliminary evidence suggests that patients with schizophrenia have an exaggerated sense of agency. In combination with the feeling of not being in control, this exaggerated sense of agency could explain delusions of alien control in which the patient attributes his own actions to another agent. Little is yet known about the neural basis of the predictive mechanisms that create the feeling that we are in control of our movements. Such prediction requires integration of information about intended movements generated in frontal cortex with sensory processing in posterior regions of the brain. Measures of functional connectivity suggest that long-range interactions between frontal and posterior regions are abnormally reduced in patients with schizophrenia. Further research is needed to explore the precise involvement of long-range connections in the mechanisms of forward modelling. **To cite this article:** C. Frith, C. R. Biologies 328 (2005).

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## Résumé

**Bases neurales des délires et des hallucinations.** La schizophrénie est un désordre d’origine biologique, caractérisé par des perceptions fausses (hallucinations) et des croyances erronées (délires). La cause physiologique sous-jacente à ces anomalies mentales demeure inconnue. Il semble de plus en plus établi qu’une classe de symptômes, les « expériences vécues », incluant les délires de contrôle extérieur et d’insertion de pensée, est associée à des anomalies du mécanisme de prédiction de la survenue d’actions intentionnelles (modèle *forward*). Pour ces patients, les mouvements actifs sont comme les mouvements passifs. Il en résulte qu’ils ne se sentent pas en mesure de contrôler leurs actions. Toutefois, la comparaison avec divers désordres

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neurologiques, tels ceux associés à des lésions du lobe pariétal, suggère que cette expérience anormale ne suffit pas à expliquer le sentiment que quelque autre agent contrôle ses propres actions. Des données préliminaires indiquent que les patients souffrant de schizophrénie ont un sens exagéré de l'action. Combiné au sentiment de ne pouvoir se contrôler, ce sens exagéré de l'action pourrait expliquer les illusions de contrôle extérieur qui font que le patient attribue ses propres actions à un autre agent. On ne sait toujours que peu de choses quant à la base neurale des mécanismes prédictifs qui créent le sentiment que nous contrôlons nos mouvements. Une telle prédiction exige que les informations sur les mouvements intentionnels engendrées dans le cortex frontal soient intégrées au traitement sensoriel dans les régions postérieures du cerveau. Des mesures de connectivité fonctionnelle suggèrent que des interactions à longue distance entre les régions frontale et postérieure sont anormalement réduites chez les patients souffrant de schizophrénie. Des recherches plus approfondies seront nécessaires pour explorer l'implication précise de ces connexions longues dans les mécanismes de modélisation *forward*. **Pour citer cet article : C. Frith, C. R. Biologies 328 (2005).**

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*Keywords:* Schizophrenia; Delusions; Hallucinations; Connectivity; Forward models; Prediction

*Mots-clés:* Schizophrénie ; Délires ; Hallucinations ; Connectivité ; Modèles *forward* ; Prédiction

## 1. How can we understand schizophrenia?

Schizophrenia is a common disorder (lifetime risk 1%) found all over the world that causes great suffering to patients and their relatives [1]. The onset of the disorder is usually in early adulthood and many patients never fully recover. Since the 1950s the vast majority of patients have been treated with drugs. This treatment can reduce the severity of the symptoms, but often has unfortunate side effects. The diagnosis is usually made on the basis of hallucinations (false perceptions) and delusions (false beliefs) reported by the patient. But, as can be seen in the examples below, the terms hallucinations and delusions do not really do justice to the bizarre nature of the experiences reported.

Example 1: *“If I breathe without other people then they get stuck to me. I get stuck to people and the thoughts come through people. There are things I’ve learned just before I came in. It was so bad I could hear everybody in my mind. It is like being stuck on the same wavelength as people.”*

Example 2: *“I felt myself touched in such a way as if I were hypnotised, electrified, or generally controlled by some sort of medium or some other will.”*

Example 1 contains a report of an auditory hallucination, *“I could hear everybody in my mind”*, while Example 2 contains the report of a delusion, *“I felt... as if I were controlled by... some other will.”*

Because these reports are so bizarre, there is a temptation to conclude that there is no point in try-

ing to understand these symptoms. They are merely the products of a disordered brain. For example, for Jaspers [2] there was an ‘*abyss of difference*’ between psychosis and normal consciousness. *“The profoundest difference... seems to exist between that type of psychic life which we can intuit and understand, and that type which, in its own way, is not understandable and which is truly distorted and schizophrenic.”* In contrast, I believe that it is possible to understand the symptoms of schizophrenia in terms of specific defects in normal psychological processes. However, although the symptoms reflect mental disorders involving complex cognitions, there is abundant evidence that schizophrenia has a biological basis.

The illness tends to run in families although the precise genetic basis is not yet understood [3]. Significant structural abnormalities have been observed in the brains of patients with schizophrenia, but these are not specific to the disorder [4]. The clinical effectiveness of drugs is strongly related to their ability to block dopamine receptors [5]. In order to understand schizophrenia we need to link the psychological abnormalities with underlying neurophysiological disorders. This is best achieved using the language of cognitive neuroscience, since this can be applied at both levels. In addition our hope is that, by understanding symptoms in terms of cognitive processes, we can also get a better understanding of what it is like to experience such symptoms.

## 2. Are the symptoms of schizophrenia disorders of self-recognition?

Hallucinations and delusions can be observed in other psychiatric and neurological disorders, but in these cases the symptoms have a rather different form. Schneider [6] listed particular kinds of hallucinations and delusions that he believed were specific to schizophrenia (first rank symptoms). Although this list was constructed on an empirical rather than a theoretical basis, the majority of the symptoms have the common feature that they involve an abnormal perception of the patient's own acts. These include the 'made' symptoms in which the patient's own actions and experiences feel if they were made by external forces.

Example 3: *"It is not me who is unhappy, but they are injecting unhappiness into my brain."*

Example 4: *"It is my hand and arm that move, and my fingers pick up the pen, but I don't control them."*

Example 5: *"He treats my mind like a screen and flashes his thoughts onto it like you flash a picture."*

Auditory hallucinations can also be characterised in this way. There are a number of anecdotal reports showing that the 'voices' a patient reports hearing are associated with the patient's own sub-vocal speech (e.g., [7]).

Example 6: *'The patient whispered, "The only voice I hear is hers. She knows everything. She knows all about aviation." At this point she stated audibly, "I heard them say I have a knowledge of aviation."'*

From these examples, we see that a common feature of the symptoms of schizophrenia may be mis-attribution of one's own actions to some external agent.

## 3. We recognise the consequences of our actions through prediction

It has long been recognised that the acting self creates problems for the perceptual system [8]. Actions, such as moving the eyes, cause changes in sensation; the image moves across the retina. But there is nothing in the signal detected by the retina that can indicate whether this visual sensation is caused by the acting self or an independent event in the outside world. The problem can be solved by prediction. The brain can predict what changes will occur as a consequence of

the intended movement. In this way the effects of actions can be labelled as self-generated and distinguished from external events. Recent work on motor control [e.g. [9]] has shown that a mechanism for predicting the consequences of action is an essential component of any system, such as the brain, in which there is a delay between the initiation of an action and the detection of consequences of that action. Prediction in the motor system is called a forward model and is derived from the commands sent to the musculo-skeletal system that controls movements of the limbs. Two aspects of action can be predicted via forward modelling. First, we can predict the trajectory of the limb movement in space and time (the forward dynamic model). Second, we can predict the tactile and kinesthetic sensations that the movement will cause (the forward output model). There are now several studies showing that forward modelling is abnormal in patients with schizophrenia, particularly those currently reporting delusions of control.

## 4. Problems with the forward output model

Because we can predict the sensory consequences of our own actions, our response to these sensations is attenuated. A touch we apply to ourselves feels far less intense than the same touch applied by someone else [10,11]. This effect depends upon precise prediction since the subjective intensity of self-touching increases if the relationship between the movement and its sensory consequences is systematically distorted [12]. This attenuation of self-generated sensation can also be observed at the physiological level. Activity in somatosensory cortex is much reduced when tactile stimulation is self-applied [13]. This effect is not specific to self-touching, but applies in general to sensations caused by movements. Activity in parietal cortex is greater for passive movements than for the same movement performed actively [14].

Patients currently experiencing delusions of control do not show this attenuation of self-generated sensations. They rate the intensity of self-touch as high as the same touch applied by someone else [15]. Activity in parietal cortex is abnormally high during active movements [16]. Similar observations have been made in relation to the sensory consequences of speech. Responses to sound in auditory cortex are reduced when

the subject is speaking, but this attenuation is not seen in patients with schizophrenia, especially those who report auditory hallucinations [17].

### 5. Problems with the forward dynamic model

The forward dynamic model allows us to predict how long a movement will take and where the limb will be at the end of the movement. Through this prediction we can know that we have initiated the wrong movement before the sensory feedback associated with that movement arrives. As a result we are able to make very rapid error corrections [18]. Several studies have shown that patients with schizophrenia, especially those reporting delusions of control, make very few such rapid error corrections (e.g. [19,20]).

We can also use the forward dynamic model to detect differences between expected and observed visual feedback during the movement we are making. Mark Jeannerod's group in Lyon has carried out a series of ingenious experiments in which visual feedback relating to arm movements is distorted or replaced by feedback about someone else's movements. Normal subjects can detect distortions of  $> 15^\circ$  in space and  $> 150$  ms in time [21]. They can also detect that feedback is of someone else making the movement on about 70% of occasions [22]. Patients with schizophrenia are less able to detect distorted visual feedback, particularly when they report delusions of control [23].

If people are asked to imagine making a movement, then the time taken in the imagination closely mirrors that taken in reality. Difficult movements take longer to perform in imagination just as they do in reality. Presumably this effect is mediated by the forward dynamic model that allows us to predict how long any particular movement will take. Patients with delusions of control do not show this pattern. Imagined movements tend to take the same time whatever the level of difficulty [24,25].

However, not all aspects of forward modelling are impaired in schizophrenia. For example, patients show normal anticipatory adjustments of grip force when picking up objects or holding objects in anticipation of a collision [26]. Forward modelling is required in order to make these anticipatory adjustments. However, when patients initiate collisions adjustment of

grip force is abnormally delayed. These results suggest that implicit, automatic use of forward modelling is unimpaired in schizophrenia and that the problem is only seen when patients have to consciously attend to their actions. Support for this distinction comes from a recent study by Knoblich et al. [27]. Subjects drew circles on a writing pad connected to a computer and were asked to detect discrepancies between their hand movements and the visual consequences of these movements displayed on the computer screen. Patients with positive symptoms were impaired in their awareness of the discrepancies, but were not impaired in their ability to automatically adjust their hand movements to compensate for the discrepancies.

### 6. Does prediction depend upon long-range cortico-cortical connections?

At the physiological level our ability to make use of the predictions derived from forward modelling must depend, in broad terms, upon communication between brain areas concerned with initiating actions (frontal cortex) and sensory areas concerned with processing the consequences of actions. There is also evidence of a special role for anterior cingulate cortex (posterior rostral region) in the detection of discrepancies (e.g., [28]). In the case of schizophrenia, therefore, we might expect to find abnormal connectivity between these cortical regions. There is some preliminary evidence in favour of this hypothesis [29]. For example, Fletcher et al. [30] observed abnormally low connectivity between frontal and temporal regions in patients with schizophrenia. They also showed that the strength of this connectivity was modulated by activity in anterior cingulate cortex [31].

Ford et al. [32] used EEG to measure connectivity (i.e. coherence) between frontal and temporal areas involved in speech production and perception. In normal volunteers they found that this connectivity was greater when subjects were speaking rather than listening to speech, consistent with the idea that, during speech, forward modelling is being used to modulate responses in regions processing auditory signals. The increase in connectivity was not observed in patients with schizophrenia, especially those prone to experience hallucinations. An open question is whether different symptoms relate to different kinds of disconnection.

For example, delusions of control might arise from disconnections between frontal regions and parietal areas concerned with the perception of limb positions. Further work is needed to elucidate the brain mechanism underlying forward modelling in both normal and abnormal cases.

## 7. What is it like to have delusions of control?

The account of delusions of control as a failure of forward modelling should give us some understanding of what it is like to have these symptoms. Normally when we perform an action we are hardly aware of sensory consequences of that action. In contrast, if our arm is moved passively the sensory consequences are much more vivid. Because they are abnormally aware of the associated sensations, for patients with delusions of control an active movement feels like a passive movement. Given this experience, it is not surprising that they do not feel on control of their actions. But is this experience sufficient to explain why they feel that their actions are under the control of some external agent?

Studies of neurological patients suggest that a feeling of not being in control of one's actions is not sufficient to explain delusions of control. Lesions of supplementary motor area (SMA) or anterior corpus callosum can sometimes lead to a condition in which the contralesional hand performs actions not intended by the patient (the anarchic hand, [33]). The hand may grab doorknobs or scribble with a pencil. The patient recognises that these actions are not intended and tries to stop them by restraining the hand. But typically the patient reports that there is something wrong with his hand, not that his hand is being controlled by alien forces. This phenomenology is very different from that seen in patients with delusions of control. These patients perform the actions they intend and do not attempt to prevent them. Yet they still report that these actions are being controlled by alien forces [16].

Studies of patients with parietal lesions also show that failure to predict the consequences of one's actions is not sufficient to create delusions of control. There is much evidence for a failure of the forward dynamic model after parietal lesions. These patients resemble patients with schizophrenia in that their performance of movements in the imagination is abnor-

mal [34]. Also they have problems in using visual feedback to recognise their own actions [35]. But these patients do not report delusions of control.

## 8. The experience of agency

There seems to be a critical distinction between the feeling that one is not in control of one's actions and the feeling that one's actions are being controlled by an external agent. So where does this sense of agency come from? The idea of agency is intimately concerned with cause and effect; my desire causes my action, my action (e.g., pressing a button) causes an event (e.g., a tone). The model of motor control I have discussed so far does not address the question of how we are aware of agency in this sense of cause and effect. However, recent studies by Patrick Haggard and his colleagues have identified an empirical marker of the sense of agency. The experimental technique is based on that of Libet et al. [36] in which subjects are asked to indicate the time at which they initiated an action. This perceived time is not systematically related to actual time at which the movement begins, since it is altered by the context in which the movement occurs. When a button press causes an event (a tone) then the times of the action and the event are perceived as being closer together than was actually the case. In contrast, when an involuntary movement (caused by TMS) is followed by a tone, then the action and the event are perceived as being further apart in time [37]. When our action causes an event there is an 'intentional binding' whereby the cause and its effect are drawn together in perceived time.

## 9. Who is the agent?

Of particular relevance to our understanding of delusions of control is the demonstration that intentional binding does not depend upon the proprioceptive and efferent signals associated with movement initiation. When we watch another person pressing a button to cause a tone these signals are not available, but the intentional binding effects are just as strong. However, binding does not occur when the button is pressed by a mechanical device [38]. Thus intentional binding is a marker of agency, but does not specify

whether the agent is the self or another. Preliminary results suggest that patients with schizophrenia show exaggerated intentional binding [39]. If it is shown that this applies when watching other people also, then this would imply that these patients have an exaggerated sense of agency which is not specific to the self. It has been recently demonstrated that patients with delusions of persecution perceive intentional behaviour in the movements of abstract shapes where normal controls perceive none [40].

On the basis of these new observations we can put forward a more complete account of delusions of control. There are two problems. First, the patient does not feel properly in control of his actions. Second, he has a strong sense of agency associated with these actions. The lack of control, however, implies that the agency must belong to another.

## 10. Future research

This account of delusions of control suggests a number of fruitful lines for future research on the cognitive neuropsychology of schizophrenia. In the short term, we should explore the neural basis of the sense of agency though the use of brain imaging with normal volunteers and the behavioural study of patients with circumscribed lesions. In particular we need to know whether lesions to parietal cortex affect the sense of agency as well as the ability to predict the consequences of action. There is also a surprising lack in our knowledge of the phenomenology of delusions of control. For example, the account I have presented here would predict that these delusions should be associated with actions having a consequence (e.g., pressing a button to cause a tone), but not with actions that have no intended effect [41]. I am not aware of any account in the literature showing that only certain kinds of actions are associated with delusions of control.

In the longer term, we need to establish the physiological mechanisms underlying forward modelling and the attenuation of self-generated stimulation. These mechanisms are reasonably well understood in computational terms. Brain imaging studies are needed which examine interactions between brain regions rather than patterns of activation. For example, Ford et al. [32] showed that long-range connectivity between frontal and temporal regions altered depend-

ing upon whether subjects were listening or speaking. Recent methodological advances should enable a more precise identification of the circuitry involved in the modulation of brain connectivity in the context of action [29].

Finally we need to relate these mechanisms to neurotransmitter function. There is a clear role for dopamine in schizophrenia (e.g., [42]), but it is not yet clear whether there is a specific role for this neurotransmitter in the forward modelling mechanisms that are crucial to the normal experience of the self in action. One possibility is that the role of dopamine in broadcasting prediction errors [43] and in novelty detection [44] may also apply to the processing of mismatches between the output of the forward and subsequent sensory feedback.

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