# Functional Magnetic Resonance Imaging of Inner Speech in Schizophrenia

Claudia J.P. Simons, Derek K. Tracy, Kirandeep K. Sanghera, Owen O'Daly, James Gilleen, Maria-de-Gracia Dominguez, Lydia Krabbendam, and Sukhwinder S. Shergill

**Background:** Auditory verbal hallucinations in schizophrenia have been linked to defective monitoring of one's own verbal thoughts. Previous studies have shown that patients with auditory verbal hallucinations show attenuated activation of brain regions involved with auditory processing during the monitoring of inner speech. However, there are no functional magnetic resonance imaging studies explicitly comparing the perception of external speech with internal speech in the same patients with schizophrenia. The present study investigated the functional neuroanatomy of inner and external speech in both patients with schizophrenia and healthy control subjects.

**Methods:** Fifteen patients with schizophrenia and 12 healthy control subjects were studied using functional magnetic resonance imaging while listening to sentences or imagining sentences.

**Results:** Significant interactions between group (control subjects vs. patients) and task (listening vs. inner speech) were seen for the left superior temporal gyrus, as well as regions within the cingulate gyrus.

**Conclusions:** Attenuated deactivation of the left superior temporal gyrus in schizophrenia patients during the processing of inner speech may reflect deficits in the forward models subserving self-monitoring.

**Key Words:** Auditory verbal hallucinations, fMRI, inner speech, schizophrenia, temporal lobe, verbal self-monitoring

uditory verbal hallucinations (AVHs) are one of the most common symptoms in schizophrenia. Current cognitive models suggest that auditory hallucinations are the result of defective self-monitoring (1–4). A feed-forward mechanism (5) has been proposed to explain self-monitoring of motor actions. In this model, motor commands that are needed to achieve a specific goal are identified and subsequently issued. Simultaneously, an efference copy is generated and transmitted through a corollary discharge mechanism to the sensory brain regions that are relevant to the planned act. The efference copy of the motor command serves to predict the sensory effects of the motor act. If the actual and predicted sensory feedback match, the actual sensory feedback will be attenuated. If a movement is externally controlled, there will be no efference copy that can attenuate the sensory information. Hence, the monitoring of motor acts can help to recognize whether a movement is self-initiated or externally generated. If generation of language and thought can be considered as a type of motor act (6-8), then monitoring of the verbal act may contribute to the distinction between self as source versus others as source. If this monitoring system is defective, verbal thoughts will not be recognized as being self-generated, leading to the experience of AVHs (8).

From the Department of Psychiatry and Neuropsychology (CJPS, M-de-GD, LK), Maastricht University, European Graduate School of Neuroscience, Maastricht; and GGz Eindhoven (CJPS), Eindhoven, The Netherlands; Cognition Schizophrenia and Imaging Laboratory (DKT, KKS, OO, JG, SSS), Division of Psychological Medicine; and Department of Clinical Neuroscience (OO), Institute of Psychiatry, London; and University of Leicester School of Medicine (KKS) Leicester, United Kingdom; and Center Brain and Learning (LK), Department of Psychology and Education, VU University Amsterdam, Amsterdam, The Netherlands.

Address correspondence to Claudia J.P. Simons, M.Sc., Department of Psychiatry and Neuropsychology, Maastricht University, PO Box 616, 6200 MD Maastricht, The Netherlands; E-mail: c.simons@sp.unimaas.nl.

Received Jun 3, 2009; revised Sep 2, 2009; accepted Sep 3, 2009.

Neuroimaging studies investigating the generation of speech in healthy control subjects indicate that the monitoring process is associated with activation of the left prefrontal cortex and deactivation of the left and right superior temporal cortices (9), suggesting that corollary discharge from the areas involved in the generation of verbal speech results in attenuation of the activity in areas involved in speech perception. Initial studies of verbal fluency in schizophrenia indicated that this temporal deactivation was absent (9). However, more recent studies have failed to replicate this finding (10,11). Neuroimaging studies trying to "capture" auditory verbal hallucinations when they are naturally occurring have reported activation of temporal lobe structures during AVHs (12,13). In healthy control subjects, the generation of inner speech, or the silent articulation of words, is associated with activation in the left inferior frontal cortex/insula, the supplementary motor area, the left superior temporal/inferior parietal region, and the right posterior cerebellar region (14). The left inferior frontal cortex has also been shown to be activated during AVHs (12,15). A more recent study suggests the right inferior frontal gyrus may also be involved (16), indicating involvement of inner speech in AVHs. An early positron emission tomography (PET) study investigating auditory verbal imagery imaging another person's speech—revealed that schizophrenia patients who were prone to auditory hallucinations displayed normal activation of the left inferior frontal gyrus but abnormal activation of the left temporal cortex compared with both schizophrenia patients without a history of AVHs and healthy control subjects (17). More recently, we have used functional magnetic resonance imaging (fMRI) to show normal activation of the left inferior frontal gyrus and attenuation of right temporal cortex activation in hallucination-prone schizophrenia patients, relative to an inner speech task, during verbal imagery (18). To summarize, previous studies indicate that when generating verbal material, schizophrenia patients display normal activation of the inferior frontal cortex but abnormal activity in the temporal cortex, with the changes varying with the condition used as the baseline. However, to demonstrate the specificity of this hypothesis, ideally one would compare simple self-generated speech with comparable external speech to exclude any nonspecific

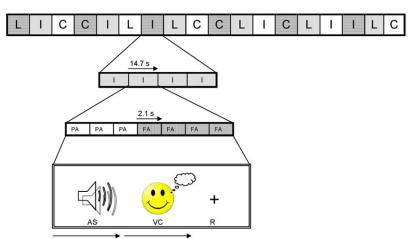


Figure 1. Stimulation protocol. Illustrated are the experimental blocks, the trials within a single block, and how stimulus acquisition, stimulus presentation, and response take place during a trial. AS, auditory stimulus; C, baseline; FA, full acquisition; I, inner speech; L, listening; PA, partial acquisition; R, response; VC, visual cue.

impairments in the auditory cortical processing and to exclude the increased demands associated with generating more complex stimuli such as imagery. To our knowledge, there are no fMRI studies explicitly comparing this in patients with schizophrenia.

1124-2862 ms

In the present study, we used fMRI to investigate brain activation during the perception of internally generated speech and external speech in both patients with schizophrenia and matched healthy control subjects. Activation during the selfmonitoring of inner speech was compared directly with activation during listening to externally generated speech, a task requiring no self-monitoring. We predicted that inner speech would lead to attenuation of temporal cortex activation in healthy control subjects as a consequence of corollary discharge, while the patients with schizophrenia would show less attenuated activation of the temporal cortex during inner speech trials, indicating faulty verbal self-monitoring. Since external sensory stimulation does not lead to an efference copy, we anticipated an interaction between task (listening vs. inner speech) and group (control subjects vs. patients) in the temporal cortex. As silent articulation is associated with activation of the left inferior frontal cortex, we also predicted that inner speech trials would be accompanied by an increased activation of this area compared with listening trials in both patients and the control subjects. The present study minimized the effects of acoustic scanner noise during the stimulus presentation through the use of a partially silent acquisition in which the auditory stimulus preceding the inner speech and listening prompts was presented in a partially silent gap followed by a period of continuous image acquisition (19).

#### **Methods and Materials**

# Subjects

Fifteen male patients with a DSM-IV diagnosis of schizophrenia and right-handed, as assessed with the Annett Handedness Inventory (20), participated in the study. Patients were recruited through consultant and key worker recommendations and had all experienced prominent auditory hallucinations during exacerbations of their illness. Fourteen subjects were outpatients and one subject was an inpatient. All patients were receiving regular doses of antipsychotic medication. Mean age of the patients was 34.7 years (SD = 8.7). Mean duration of illness was 11.2 years(range 3-27). Mean score on the Positive and Negative Syndrome Scale (21) was 48.5 (SD = 16.5, range 30-83). Patients were matched for age, sex, and handedness to a control group. Twelve

healthy right-handed, male control subjects were recruited through an advertisement in a city-wide newspaper. They did not suffer from psychiatric disorders and had no family history of psychiatric disorder. Mean age of the control subjects was 34.4 years (SD = 7.9). English was a first language for all subjects and all subjects had a minimum of 11 years of education. Exclusion criteria were any illicit drug use within the previous 6 months or any contraindications to magnetic resonance imaging (MRI) scanning. Potential subjects were assessed on their ability to perform the tasks (detailed below) outside the scanner, approximately 1 week before scanning. Subjects provided written informed consent, and ethical approval was provided by the Institute of Psychiatry and Maudsley National Health Services Trust.

## Tasks Performed During fMRI

Subjects performed two active tasks, listening and inner speech, and there was one additional null baseline condition. Each of these tasks was administered over six counterbalanced blocks, each block comprising four listening trials, four inner speech trials, and four baseline trials, with the baseline trials consisting of a silent period equal in length to four paired stimuli

Auditory stimuli used for the listening and inner speech conditions were 24 neutral sentences, spoken by an adult female native English speaker. During the listening trials, the auditory stimulus was followed by a visual cue prompting the subjects to listen to a second auditory stimulus identical to the first; during the inner speech trials, the auditory stimulus was followed by a visual cue prompting the subjects to covertly imagine repeating the sentence to themselves in their own voice and press a button with their right index finger once this was completed.

Each auditory stimulus was presented once during a listening trial and once during an inner speech trial. Sentences were presented via pneumatically driven earphones, incorporated within ear defenders, specifically designed for functional MRI (Quiet Muff 29 Earmuffs, Avotec, Jensen Beach, Florida). These reduced unattenuated noise from the scanner. As some sentences were longer than others, the duration of the stimuli varied from 1124 msec to 2862 msec, with an average length of 1971 msec. The auditory stimulus was followed by a 1000-msec period in which the visual prompt was presented, followed by the actual task (listening/inner speech). There was a gap before the onset of the next trial (intertrial interval: 14,728 msec). Image acquisi-

**Figure 2.** Brain activation map for the effects of **(A)** task (blue: inner speech > listening, yellow: listening > inner speech) and **(B)** group (p = .009, <1 false-positive cluster).

tion was performed during this nontrial interval. Total length of the task was 17 minutes and 39 seconds.

## **Image Acquisition**

Gradient-echo echo-planar imaging data were acquired on a neuro-optimized GE Signa 1.5 Telsa system (General Electric, Milwaukee, Wisconsin) at the Maudsley Hospital, London. For complete details on methodologies, please see Section 1 in Supplement 1.

#### **Image Analyses**

Data were realigned (22) to minimize motion-related artefacts, smoothed using a Gaussian filter (full-width at half maximum 7.2 mm) and transformed into Talairach space (23). Task (listening vs. inner speech) and group (control subjects vs. patients) specific comparisons were carried. Second, the interaction between task and group was tested by subtracting the inner speech trials from the listening trials. Cluster-level statistics (22) corrected for multiple comparisons were thresholded at p < .009 to ensure less than one false-positive cluster per image. For complete details on methodologies, please see Section 2 in Supplement 1.

# Results

#### **Behavioral Data**

The mean response time to complete the inner speech task was 2.62 seconds (SD = 1.59) for control subjects and 2.28 seconds (SD = .85) for patients. This difference was not statistically significant [t(22) = .64, p = .53]. On postscan debriefing, all subjects reported that they had been able to perform the tasks and thus data from all subjects were included in the analyses.

# **Imaging Data: Inner Speech Versus Listening**

The main effect of inner speech was associated with greater activation in the left inferior frontal gyrus and anterior cingulate gyrus compared with listening (Figure 2A, Table 1). Listening was associated with greater activation in the right superior temporal gyrus, left transverse temporal gyrus, and right inferior parietal lobule across both groups.

# **Between-Group Differences in Activation**

The main effect of group showed significant differences in the left and right occipital gyrus (Figure 2B, Table 1). Patients demonstrated greater bilateral activation of the occipital gyri

during the active tasks compared with the control subjects, who showed greater activation during the baseline condition.

# **Interaction Between Task and Group**

Significant interactions between group (control subjects vs. patients) and task (listening vs. inner speech) were seen for the left superior temporal gyrus, bilateral anterior cingulate gyrus, right hippocampus, and the left posterior cingulate gyrus (Figure 3A, Table 1). The plots from the peak sum of squares response in the left superior temporal gyrus of both patients and controls subjects demonstrated activation during the listening task compared with baseline. However, during the generation of inner speech, the control subjects showed markedly attenuated activation, while this difference between inner speech and listening was less pronounced in the patients (Figure 3B).

Within the other regions with significant interactions, including the right hippocampus and regions within the cingulate gyrus, control subjects showed activation during inner speech compared with listening, while the patients showed a decrease in activation during the inner speech.

**Table 1.** Regions of Differential Activation

	Coordinates			Cluster
Region of Activation (BA)	Χ	Υ	Z	Size
Effect of Task Inner Speech > Listening				
Left inferior frontal gyrus (BA 44)	-54	4	15	33
Anterior cingulate gyrus (BA 32)	4	15	42	45
Inner Speech < Listening				
Right superior temporal gyrus (BA 22)	58	-7	-2	168
Left transverse temporal gyrus (BA 41)	-58	-19	9	161
Right inferior parietal lobule (BA 40)	33	-37	53	42
Effect of Group (Control Subjects > Patients)				
Left middle occipital gyrus (BA 18)	-25	-81	4	59
Right middle occipital gyrus (BA 19)	33	-74	15	33
Interaction				
Left superior temporal gyrus (BA 22)	-54	-11	-2	20
Left cingulate gyrus (BA 24)	-11	-19	42	81
Right cingulate gyrus (BA 24)	18	-15	39	221
Right hippocampus	29	-37	-2	69
Left posterior cingulate (BA 29)	-11	-41	9	86

Thresholded at p < .009. Cluster size indicates total number of activated voxels in region.

BA, Brodmann area.

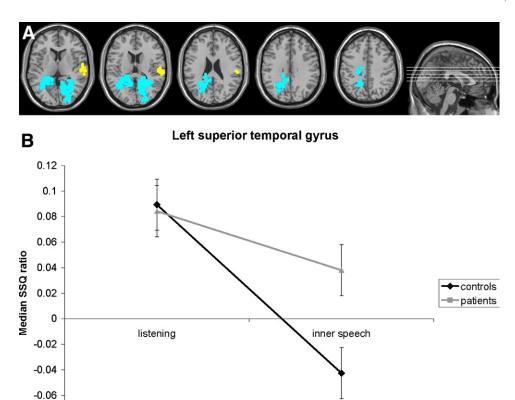


Figure 3. (A) Brain activation map for the interaction between the effects of task and group (p = .009, < 1 false-positive cluster; blue: listening minus inner speech has higher peak SSQ in patients than in control subjects; yellow: listening minus inner speech has higher peak SSQ in control subjects than in patients). (B) SSQ plots for the interaction between task and group in the left superior temporal cortex. SSQ, sum of squares.

#### Discussion

The main finding was a significant interaction between group and task for the left superior temporal cortex. During the listening trials, the anticipated activation of the left superior temporal cortex was evident in control subjects and in schizophrenia patients, suggesting that listening to spoken sentences is not impaired in schizophrenia patients. Control subjects showed greater decrease in activation during inner speech compared with listening than patients. This provides evidence for defective self-monitoring of inner speech in schizophrenia patients. Failure to attenuate the activity in the temporal cortex may lead to the attribution of the verbal material as being of external origin, ultimately leading to auditory hallucinatory experiences.

-0.08

The attenuation of temporal cortex activity is thought to be associated with corollary discharge from the inferior frontal cortex (24,25). Activation of the left inferior frontal cortex is thought to correspond to silent articulation (26–29). In line with previous studies (18,26), we found increased activation of the left inferior frontal gyrus during inner speech compared with the listening condition across groups.

Previous studies did not find clear differences between healthy control subjects and schizophrenia patients on an inner speech task (17,18). These studies used an inner speech task that required subjects to silently articulate a sentence in the form, "I like . . ." or "You are . . .," appended with a single word that was presented to them in the auditory or visual modality. The task used in the present study differs from these early studies in requiring the subjects to repeat longer sentences that were presented to them aurally, placing higher demands on verbal self-monitoring. The same previous studies (17,18) did, however, find abnormal activation in the inferior frontal and temporal cortex during auditory verbal imagery, a task presumably requiring more self-monitoring than simple silent repetition. Thus, the findings of the present and previous studies suggest that when self-monitoring demands are absent or minimal, patients show no language network related abnormality, and it is only when the verbal material gets more complex and requires greater selfmonitoring that the reduced attenuation of superior temporal cortex activation in patients with schizophrenia becomes appar-

Studies on repetition priming in healthy control subjects show a decrease in activity in the left inferior prefrontal cortex and temporal cortex in response to repeated words in comparison with novel words (30). It has been suggested that repetition priming for semantic material may be reduced in patients (31). Since the auditory stimuli were repeated after presentation of a visual cue, it could be argued that the reduced attenuation in the patient group may be the result of reduced repetition priming. However, there were no significant group differences in activation of the left superior temporal gyrus during listening and there were no group differences in activation of the left inferior frontal gyrus. Furthermore, reaction times during the inner speech condition were not significantly shorter in control subjects, which may be expected in case of differential priming effects. The present study also differs considerably from the typical repetition priming designs in that the stimuli are more complex and the stimulus onset asynchrony is comparatively long (2124– 3862 msec).

The present study found a main effect for the anterior cingulate gyrus, with inner speech trials eliciting more activation in this area than listening trials. There were significant interactions between group and task within the left and right anterior cingulate gyrus. Control subjects showed activation of these regions during inner speech and decreased activation when listening to external speech. Patients did not show this activation of the cingulate cortex during inner speech. The anterior cingulate has been implicated in divided attention (32) and competition monitoring (33), and patients with schizophrenia have been shown to display attenuated anterior cingulate activation during a cognitive inhibition task compared with control subjects (34). It has been suggested that the anterior cingulate may act as a top-down modulator of activity in the left superior temporal gyrus (35,36). This impaired modulation may be associated with external misattributions of inner speech (36).

The only between-group activations were in the occipital cortices; control subjects showed greater decreases in activation of the occipital cortex during the listening and inner speech tasks than the patients. Studies using fMRI have demonstrated that stimulation of one sensory modality can deactivate activation in other sensory regions (37,38). Functional MRI deactivation probably reflects inhibition of neural processes in task-irrelevant regions or regions that potentially interfere with the task at hand (39). This may indicate that schizophrenia patients show less cross-modal inhibition and may even engage some aspect of visual imagery upon hearing or generating the sentences. There were no group differences in the superior temporal gyrus during listening. Previous studies have found attenuated activation in response to listening to sentences (40,41); one reason for not replicating this finding may lie in the fact that the patients were not actively hallucinating during the imaging session. Earlier studies of inner speech per se have reported increased activation of the temporal cortex (42), but these differ significantly in including an active listening baseline rather than a null baseline as used in this study.

## Limitations

Inner speech is a subjective phenomenon and as such cannot be easily objectively monitored. Training subjects on the task before scanning was done to minimize the influence of variation in performance. We also included an external measure of completion of the inner speech with an explicit button press, and there was no difference from control subjects in mean response time, suggesting that both groups performed the inner speech task in a similar manner. One further issue is related to the level of intrinsic, implicit, inner speech activity, which may be occurring during the baseline task; however, this is difficult to remove completely, as it occurs implicitly. The use of the identical baseline during the listening condition should go some way to eliminating any systematic effects due to this.

We selected patients on the basis of a prior history of auditory hallucinations, and the inclusion of a nonhallucinating group would have permitted the examination of the specificity of this finding with regard to hallucinations versus schizophrenia. Ford and Mathalon (43) conducted several studies using event-related potentials and electroencephalogram (EEG) gamma coherence as proxies of corollary discharge. These studies reported abnormalities in the corollary discharge system in patients with schizophrenia, without specificity to hallucinations. Other studies (44–46) using similar EEG paradigms have shown that the corollary discharge system is disrupted in patients, especially in those prone to AVHs. Even if inner speech deficits can be linked to

AVHs, it remains to be seen whether these deficits can serve as the basis for all AVHs (47,48). Jones (48) argues that AVHs that involve voices attempting to regulate ongoing actions of the voice hearer may be consistent with inner speech based models, but other AVHs, e.g., those with a content clearly linked to previous traumatic experience, may be better explained by other cognitive models.

Intrinsic scanner noise poses a problem in all fMRI studies, especially in studies investigating auditory processing. The present study attempted to minimize scanner noise during the tasks by using a partially silent acquisition during the stimuli presentation. Nevertheless, scanner noise remains perceptible. Although scanner noise can significantly influence the blood oxygenation level-dependent response in the temporal cortex (19,49), the confounding effects of background noise should be considered as constant across conditions.

To summarize, our data indicate that when generating inner speech, schizophrenia patients show an attenuated deactivation in the left superior temporal gyrus, an area that has been implicated in verbal self-monitoring processes (18). This is consistent with the notion that auditory verbal hallucinations in schizophrenia may arise as a consequence of faulty predictive models underlying the monitoring of behaviors including inner speech (50,51).

Sukhwinder S. Shergill was supported by a Higher Education Funding Council for England Clinical Senior Lecturer Fellowship. Lydia Krabbendam was supported by the Netherlands Organisation for Scientific Research (VIDI grant).

Claudia J.P. Simons, Derek K. Tracy, Kirandeep K. Sanghera, Owen O'Daly, James Gilleen, and Maria-de-Gracia Dominguez report no biomedical financial interests or potential conflicts of interest.

Supplementary material cited in this article is available online.

- Ford JM, Mathalon DH, Kalba S, Whitfield S, Faustman WO, Roth WT (2001): Cortical responsiveness during inner speech in schizophrenia: An event-related potential study. Am J Psychiatry 158:1914–1916.
- Shergill SS, Samson G, Bays PM, Frith CD, Wolpert DM (2005): Evidence for sensory prediction deficits in schizophrenia. Am J Psychiatry 162: 2384–2238.
- 3. Kumari V, Fannon D, Ffytche DH, Raveendran V, Antonova E, Premkumar P, et al. (2008): Functional MRI of verbal self-monitoring in schizophrenia: Performance and illness-specific effects [published online ahead of print November 7]. Schizophr Bull.
- Allen P, Aleman A, McGuire PK (2007): Inner speech models of auditory verbal hallucinations: Evidence from behavioural and neuroimaging studies. Int Rev Psychiatry 19:407–415.
- Wolpert DM, Ghahramani Z, Jordan MI (1995): An internal model for sensorimotor integration. Science 269:1880–1882.
- Seal M, Aleman A, McGuire PK (2004): Compelling imagery, unanticipated speech and deceptive memory: Neurocognitive models of auditory verbal hallucinations in schizophrenia. Cogn Neuropsychiatry 9:43–72.
- Jones SR, Fernyhough C (2007): Thought as action: Inner speech, selfmonitoring, and auditory verbal hallucinations. Conscious Cogn 16:391– 399.
- 8. Feinberg I (1978): Efference copy and corollary discharge: Implications for thinking and its disorders. *Schizophr Bull* 4:636 640.
- Frith C, Friston KJ, Herold S, Silbersweig D, Fletcher P, Cahill C, et al. (1995): Regional brain activity in schizophrenic patients during the performance of a verbal fluency task. Br J Psychiatry 167:343–349.
- Spence SA, Liddle PF, Stefan MD, Hellewell JS, Sharma T, Friston KJ, et al. (2000): Functional anatomy of verbal fluency in people with schizophrenia and those at genetic risk. Focal dysfunction and distributed disconnectivity reappraised. Br J Psychiatry 176:52–60.

- 11. Dye SM, Spence SA, Bench CJ, Hirsch SR, Stefan MD, Sharma T, et al. (1999): No evidence for left superior temporal dysfunction in asymptomatic schizophrenia and bipolar disorder. PET study of verbal fluency. Br J Psychiatry 175:367-374.
- 12. Shergill SS, Brammer MJ, Williams SCR, Murray RM, McGuire PK (2000): Mapping auditory hallucinations in schizophrenia using functional magnetic resonance imaging. Arch Gen Psychiatry 57:1033-1038.
- 13. Dierks T, Linden DEJ, Jandl M, Formisano E, Goebel R, Lanfermann H, et al. (1999): Activation of Heschl's gyrus during auditory hallucinations. Neuron 22:615-621.
- 14. Sherqill SS, Brammer MJ, Fukuda R, Williams SCR, Murray RM, McGuire PK (2003): Engagement of brain areas implicated in processing inner speech in people with auditory hallucinations. Br J Psychiatry 182:525-531.
- 15. McGuire PK, Shah GMS, Murray RM (1993): Increased blood flow in Broca's area during auditory hallucinations in schizophrenia. Lancet
- 16. Sommer IEC, Diederen KMJ, Blom JD, Willems A, Kushan L, Slotema K, et al. (2008): Auditory verbal hallucinations predominantly activate the right inferior frontal area. Brain 131:3169-3177.
- 17. McGuire PK, Silbersweig DA, Wright I, Murray RM, Frackowiak RSJ, Frith CD (1996): The neural correlates of inner speech and auditory verbal imagery in schizophrenia: Relationship to auditory verbal hallucinations. Br J Psychiatry 169:148-159.
- 18. Shergill SS, Bullmore E, Simmons A, Murray R, McGuire P (2000): Functional anatomy of auditory verbal imagery in schizophrenic patients with auditory hallucinations. Am J Psychiatry 157:1691–1693.
- 19. Amaro E, Williams SCR, Shergill SS, Fu CHY, MacSweeney M, Picchioni MM, et al. (2002): Acoustic noise and functional magnetic resonance imaging: Current strategies and future prospects. J Magn Reson Imaging 16:497-510.
- 20. Annet MA (1970): A classification of hand preference by association analysis. Br J Psychol 61:303-321.
- 21. Kay SR, Fiszbein A, Opler LA (1987): The Positive and Negative Syndrome Scale (PANSS) for schizophrenia. Schizophr Bull 13:261-276.
- 22. Bullmore ET, Brammer MJ, Rabe-Hesketh S, Curtis VA, Morris RG, Williams SCR, et al. (1999): Methods for diagnosis and treatment of stimulus-correlated motion in generic brain activation studies using fMRI. Hum Brain Mapp 7:38 - 48.
- 23. Brammer MJ, Bullmore ET, Simmons A, Williams SCR, Grasby PM, Howard RJ, et al. (1997): Generic brain activation mapping in fMRI: A nonparametric approach. Magn Reson Imaging 15:763-770.
- 24. Ford JM, Mathalon DH (2004): Electrophysiological evidence of corollary discharge dysfunction in schizophrenia during talking and thinking. Int J Psychophysiol 58:179-189.
- 25. Frith CD (1992): The Cognitive Neuropsychology of Schizophrenia. Hove, UK: Psychology Press.
- 26. McGuire PK, Silbersweig DA, Frith CD (1996): Functional anatomy of inner speech and auditory verbal imagery. Psychol Med 26:177–189.
- 27. Smith JD, Reisberg D, Wilson M (1992): Subvocalisation and auditory imagery: Interactions between the inner ear and inner voice. In: Reisberg D, editor. Auditory Imagery. Mahwah, NJ: Lawrence Erlbaum Associates, 95-120.
- 28. Paulesu E, Frith CD, Frackowiak RSJ (1993): The neural correlates of the verbal component of working memory. Nature 362:342-345.
- 29. Price CJ, Wise RJS, Warburton E, Moore CJ, Howard D, Patterson K, et al. (1996): Hearing and saying: The functional neuroanatomy of auditory word processing. Brain Lang 119:919-931.
- 30. Buckner RL, Koutstaal W, Schacter DL, Rosen BR (2000): Functional MRI evidence for a role of frontal and inferior temporal cortex in amodal components of priming. Brain 123:620 - 640.
- 31. Kubicki M, McCarley RW, Nestor PG, Huh T, Kikinis R, Shenton ME, et al. (2003): An fMRI study of semantic processing in men with schizophrenia. Neuroimage 20:1923-1933.

- 32. Corbetta M, Miezin FM, Dobmeyer S, Shulman GL, Petersen SE (1991): Selective and divided attention during visual discriminations of shape, color, and speed: Functional anatomy by positron emission tomography. J Neurosci 11:2383-2402.
- 33. Carter CS, Braver TS, Barch DM, Botvinick MM, Nol D, Cohen JD (1998): Anterior cingulate cortex, error detection, and the online monitoring of performance. Science 280:747-749.
- 34. Krabbendam L, O'Daly O, Morley LA, Van Os J, Murray RM, Shergill SS (2009): Using the Stroop task to investigate the neural correlates of symptom change in schizophrenia. Br J Psychiatry 194:373–374.
- 35. Fletcher P, McKenna PJ, Friston KJ, Frith CD, Dolan RJ (1999): Abnormal cingulate modulation of fronto-temporal connectivity in schizophrenia. Neuroimage 9:337-342.
- 36. Allen P, Amaro E, Fu CHY, Williams SCR, Brammer MJ, Johns LC, et al. (2007): Neural correlates of the misattribution of speech in schizophrenia. Br J Psychiatry 190:162-169.
- 37. Laurienti PJ, Burdette JH, Wallace MT, Yen YF, Field AS, Stein BE (2002): Deactivation of sensory-specific cortex by cross-modal stimuli. J Cogn Neurosci 14:420-429.
- 38. Lewis JW, Beauchamp MS, DeYoe EA (2000): A comparison of visual and auditory motion processing in human cerebral cortex. Cereb Cortex 10:873-888.
- 39. Tomasi D, Ernst T, Caparelli EC, Chang L (2006): Common deactivation patterns during working memory and visual attention tasks: An intrasubject fMRI study at 4 Tesla. Hum Brain Mapp 27:694-705
- 40. Woodruff PWR, Wright IC, Bullmore ET, Brammer M, Howard RJ, Williams SCR, et al. (1997): Auditory hallucinations and the temporal cortical response to speech in schizophrenia: A functional magnetic resonance imaging study. Am J Psychiatry 154:1676-1682.
- 41. David AS, Woodruff PWR, Howard R, Mellers JDC, Brammer M, Bullmore E, et al. (1996): Auditory hallucinations inhibit exogenous activation of auditory association cortex. Neuroreport 7:932-936.
- 42. Shergill SS, Bullmore ET, Brammer MJ, Williams SCR, Murray RM, McGuire PK (2001): A functional study of auditory verbal imagery. Psychol Med 31:241-253.
- 43. Ford JM, Mathalon DH (2005): Corollary discharge dysfunction in schizophrenia: Can it explain auditory hallucinations? Int J Psychophysiol 58: 179 - 189
- 44. Ford JM, Mathalon DH, Whitfield S, Faustman WO, Roth WT (2002): Reduced communication between frontal and temporal lobes during talking in schizophrenia. Biol Psychiatry 51:485-492.
- 45. Heinks-Maldonado TH, Mathalon DH, Houde JF, Gray M, Faustman WO, Ford JM (2007): Relationship of imprecise corollary discharge in schizophrenia to auditory hallucinations. Arch Gen Psychiatry 64:286 – 296.
- 46. Ford JM, Roach BJ, Faustman WO, Mathalon DH (2007): Synch before you speak: Auditory hallucinations in schizophrenia. Am J Psychiatry 164:458-466.
- 47. Langdon R, Jones SR, Connaughton E, Fernyhough C (2009): The phenomenology of inner speech: Comparison of schizophrenia patients with auditory verbal hallucinations and healthy controls. Psychol Med
- 48. Jones SR (2008): Do we need multiple models of auditory verbal hallucinations? Examining the phenomenological fit of cognitive and neurological models [published online ahead of print September 26]. Schizo-
- 49. Bandettini PA, Jesmanowicz A, Van KJ, Birn RM, Hyde JS (1998): Functional MRI of brain activation induced by scanner acoustic noise. Magn Reson Med 39:410-416.
- 50. Frith CD, Done DJ (1989): Towards a neuropsychology of schizophrenia. Br J Psychiatry 153:437–443.
- 51. McGuire PK, Silbersweig DA, Wright I, Murray RM, David AS, Frackowiak RS, et al. (1995): Abnormal monitoring of inner speech: A physiological basis for auditory hallucinations. Lancet 346:596-600.