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Contributions of emotional prosody comprehension deficits to the formation of auditory verbal hallucinations in schizophrenia

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ABSTRACT

Deficits in emotional processing have been widely described in schizophrenia. Associations of positive symptoms with poor emotional prosody comprehension (EPC) have been reported at the phenomenological, behavioral, and neural levels. This review focuses on the relation between emotional processing deficits and auditory verbal hallucinations (AVH). We explore the possibility that the relation between AVH and EPC in schizophrenia might be mediated by the disruption of a common mechanism intrinsic to auditory processing, and that, moreover, prosodic feature processing deficits play a pivotal role in the formation of AVH. The review concludes with proposing a mechanism by which AVH are constituted and showing how different aspects of our neuropsychological model can explain the constellation of subjective experiences which occur in relation to AVH.

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1. Introduction: phenomenology of AVH and EPC

Emotional impairment in schizophrenia was first observed by Kraepelin (1919). Disturbances range from flat affect to intense bursts of inappropriate emotions such as anger and fear (Kraepelin, 1971 [1919]). The combination of these emotional disturbances points to one of the paradoxes of schizophrenia: whilst there can be a flattening of affect, as in negative symptoms, there can be also an increase in emotional arousal and reactivity, as in the positive symptoms of psychosis (Aleman & Kahn, 2005). Traditionally, deficits in emotion processing have been linked to negative symptoms such as inefficient social interaction, and apathy and avolition towards social stimuli (Hoekert, Kahn, Pijnenborg, & Aleman, 2007). However, a possible link between emotional processing difficulties, particularly for vocal stimuli, and the presence of hallucinations or delusions (Rossell & Boundy, 2005) has also been reported. In fact, dysfunctional emotion processing may also be associated with the positive symptoms of schizophrenia (Aleman & Kahn, 2005). In line with this idea, it has been reported that maladapted forms of emotion regulation, such as expressive suppression, have been associated with severity of hallucinatory experience (Badcock, Paulik, & Maybery, 2011).

Although emotional disturbances are of central importance in psychosis in general (van 't Wout, Aleman, Kessels, Laroi, & Kahn, 2004), this review will focus on the impact of such disturbance in relation to hallucinations. Deficits in the comprehension of vocal emotion are

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thought to be specific for schizophrenic patients suffering from hallucinations (Rossell & Boundy, 2005; Shea et al., 2007). Additionally hallucinations in schizophrenia tend to be more often auditory than visual (Mueser, Bellack, & Brady, 1990). The congruency in modality between affective processing deficits and abnormal perception might suggest an underlying common mechanism. Auditory modality emotion recognition abilities are likely to be of central importance in the formation of AVH, and the study of this connection promises to reveal a mechanism linking both phenomena.

In everyday interactions, humans are persistently exposed to verbal communication, meaning that large amounts of social information are carried by the voice (Belin, Fecteau, & Bedard, 2004). Speech encodes semantic information, but also carries non-linguistic information collectively known as prosody. These prosodic elements comprise acoustic features such as pitch, amplitude, and segment and pause duration, Prosody can be used to disambiguate the meaning of an utterance (i.e. statement vs. question, known as linguistic/stress prosody) as well as to encode affective information which is known as emotional prosody (Belin et al., 2004). Thus, prosody allows for the encoding and decoding of feelings in speech.

One of the central characteristics of AVH is that the voices patients hear are often spoken in emotional tones (Copolov, Mackinnon, & Trauer, 2004), most often with a negative emotional valence (Nayani & David, 1996), e.g., angry voices shouting abusive language. One interpretation of the link between AVH and EPC deficits lies in the coincidence in emotional valence between both phenomena. Patients with AVH seem to have difficulties correctly identifying happy prosody, possibly because of a tendency to attribute fear or sadness to any stimulus (Tsoi et al., 2008) and research in schizophrenia suggests that perception and labeling of emotions such as anger and fear may also be impaired (Allen et al., 2004). Regarding perceived intensity of prosodic emotions, it has also been discovered that patients with AVH generally tend to rate frightening stimuli as more intense than controls and patients without AVH (Rossell & Boundy, 2005). Given that patients who hear "malevolent" voices tend to rate them as "very powerful" more often than patients who hear benign or benevolent voices (Birchwood & Chadwick, 1997), these AVH patients might also rate fearful stimuli in EPC tasks as more intense. It should be noted that this review focuses on AVH in schizophrenia and not in non-clinical population, as the emotional valences of hallucinations between both populations seem to differ.

Delusions and hallucinations also seem to be linked. It is reasonable to assume that individuals experiencing hallucinations may need to explain this anomalous perceptual experience, and such attempts may rise to delusions. From this point of view, hallucinations are understood as a primary phenomenon, and delusion a consequence of the former (Maher, 2006).

The relation between EPC deficits and AVH might also be mediated by attentional mechanisms. In the early literature, it was proposed that a breakdown in selective attention may overload working memory with irrelevant sensory data. Such an overwhelming sensorial influx putatively makes it difficult to integrate current perceptions with past experience, giving rise to abnormal perception and resulting in hallucinations (Chapman & McGhie, 1964). This proposal, however, does not explain why the perceptions reaching consciousness very often have a negative affective tone. Instead, this review proposes that an attentional bias towards negative affects in EPC tasks may increase the likelihood of experiencing AVH or even act as a trigger for them. Therefore, it is essential to determine the direction of causation in any such relation. On the one hand, emotionally negative AVH might lead to a negative mood state, which is reflected in a bias toward negative affects in EPC tasks. This has been shown in the visual modality, in that depressed patients show a bias to perceive neutral emotional facial expressions as negative (Hale, Jansen, Bouhuys, & van den Hoofdakker, 1998). Thus, it is plausible that patients may live in an emotional negative state because of the intrusive hallucinations they experience, and this negative emotional state might bias judgements regarding the emotional expressions of others, such as in the evaluation of emotional prosody. On this interpretation, the bias in EPC may be based in the distress that results from the experience of emotionally negative AVH. In this causal model, AVH cause distress, and distress may lead to EPC deficits.

That interpretation notwithstanding, we intend to discuss how AVH might be associated with underlying EPC disturbances, with the direction of causation leading from emotion processing deficits to the psychotic symptom. We will consider how EPC deficits may contribute to the formation of AVH and to what extent aberrant auditory processing might be underlying both phenomena. In order to demonstrate this link we will consider behavioral and brain functional and structural findings connecting AVH and EPC, and we will integrate this evidence to suggest a new neuropsychological model of AVH with the aim of explaining the phenomenology of the abnormal experience.

2. The link between AVH and EPC at the behavioral level

Until recently, the idea of an affective prosody impairment as a modular deficit in schizophrenia was controversial. It was assumed that impaired prosodic processing in schizophrenia patients merely reflects basic sensory deficits such as misperception of pitch and amplitude (Leitman et al., 2005). For example, Leitman et al. (Leitman et al., 2005) suggested that prosody processing deficits may be due, in part, to low-level pitch discrimination defects (Leitman et al., 2005). However, there is evidence to suggest that prosody processing deficits in schizophrenia cannot be solely explained by pitch perception defects. In fact, pitch is a feature of emotional as well as linguistic prosody. If pitch perception impairment were the only cause of prosody processing deficits, linguistic as well as emotional prosody should be equally affected in schizophrenia, but contrary to this assumption, stress prosody comprehension seems to be preserved in patients with schizophrenia (Murphy & Cutting, 1990). In line with this observation, it is plausible that the dysfunction of low-level auditory processing, even if partially contributing to EPC impairment, is not sufficient to explain the complexity of the emotional prosody deficits in schizophrenia and that these may rather relate to a specific emotion processing deficit.

In addition to the specificity of EPC impairment as an emotion processing deficit, EPC difficulties are particularly prominent in the subgroup of patients with schizophrenia who have a tendency towards AVH. In a study comparing patients with and without AVH in an EPC discrimination task (Rossell & Boundy, 2005), it was observed that when EPC performance was tested with non-lexical speech sounds spoken in different tones of voice, only the AVH patient group were impaired relative to controls. When EPC stimuli contained prosodic as well as semantic elements, both patient groups showed significantly worse performance than controls. The authors interpret these findings as suggesting a dissociation between EPC for auditory stimuli with prosodic and semantic content, which further supports the idea of a specific connection between AVH and EPC. The additional processing of semantic content may mask the relation between EPC and AVH. Deficits in semantic processing are found in schizophrenia in general, without being linked to any symptomatic manifestations of the disorder in particular (Rossell & David, 2006) while emotional prosodic deficits seem to be specific to the AVH subgroup (Rossell & Boundy, 2005).

Interestingly, some research did not find an association between EPC deficits and hallucinations. On the contrary, an association between negative symptoms and EPC deficits was found (Leitman et al., 2005). However, it should be noted that the cited study collapsed patients with different diagnoses in the same group (i.e. schizophrenia and schizoaffective disorder) and it applied a tool for the measurement of positive symptoms which did not distinguished between different modalities of hallucinations. Thus, it might be the case that prosody deficits might

not be linked to hallucinations in other modalities and other diagnosis categories besides auditory hallucinations in schizophrenia.

A further factor mediating the formation of hallucinations is voice identity recognition. Prosodic features such as pitch intensity and pitch duration are important cues in differentiating voices (Belin et al., 2004). When prosodic features processing is impaired, recognition of voice identity becomes more difficult, and thus a voice may sound like someone else's (Cutting, 1990). In other words, voice identity recognition as well as vocal emotion recognition partly relies on processing of the same paralinguistic acoustical features (Belin et al., 2004), of which the most salient are fundamental frequency, intensity and interval (Perrot, Aversano, & Chollet, 2007). In fact, professional imitators use variations in pitch, tone and rhythm, to impersonate someone else (Orchard & Yarmey, 1995; Perrot et al., 2007). Variations in these same parameters can affect the emotion conveyed by the voice. Even so, it is important to note that some dissociations between voice identity recognition and emotional prosody comprehension have been found (Garrido et al., 2009), suggesting that both processes share a common paralinguistic ground. Following Cutting's approach (Cutting, 1990), Shea and colleagues suggested that vocal emotion may serve as one of the cues to determine the identity of the voice (Shea et al., 2007). Thus, prosody could be one of the contributing factors in identifying both others' as well as one's own voice, such as heard in inner speech. From a cognitive perspective, prosodic features impairment may lead to misattribution of inner speech via an incapacity to use the prosodic cues to estimate the origin of the stimuli. If the patients cannot recognize the origin and identity of their own inner voice, they might attribute the voice to someone else. It is important to note that many voices encountered in AVH have clear identities (Stephane, Thuras, Nasrallah, & Georgopoulos, 2003). Additionally, it has been claimed that misidentification of one's own inner speech relates to abnormal self-monitoring (Johns et al., 2001). Our interpretation explores the possibility that abnormal prosody processing may be a mechanism mediating misidentification of inner speech, which also contains prosodic cues helping the individual to recognize the identity of the speaker (Belin et al., 2004). Following this line of argument, inaccurate processing of the prosodic information encoded in inner speech might contribute to a misidentification of the identity of one's own inner voice.

Inaccurate decoding of prosodic features might also lay a foundation for the formation of certain AVH triggered by external sounds. In fact, a direct relationship has been reported between the timbre and pitch of real environmental sounds and how a degraded perception of them can be mistaken as an AVH (Hunter & Woodruff, 2004). A similar mechanism of degraded auditory perception might not only operate in the case of external environmental sounds but also for voices as features such as timbre are also involved in voice identification. It has been proposed that speaker identification is based on the extraction of paralinguistic features such as frequency and pitch (Formisano, De Martino, Bonte, & Goebel, 2008) (prosodic aspects of speech). EPC deficits, may account for the misidentification of self-generated auditory objects which lead to the formation of AVH. If this conclusion is correct, it might also explain why many AVH contain specific prosodic features and voice identities.

The idea that disrupted integration of external and propioceptive perceptions are the basis of hallucinations has already been formulated by Chapman (1967), who proposed that distorted sensory information from memory and proprioceptive information might be perceived abnormally. Such perceptions may have diffuse qualities and yet they might feel like a real perception from external stimuli (Chapman, 1967).

3. Neural underpinnings of auditory hallucinations and their overlap with EPC

Studying gray matter abnormalities in patients with a history of AVH offers additional evidence about the neural mechanisms underlying the abnormal perception phenomenon. A recent study in a large sample of 99 schizophrenia patients found reduced gray matter density bilaterally in the superior temporal gyri which was correlated with hallucination severity (Nenadic, Smesny, Schlosser, Sauer, & Gaser, 2010). Another recent study investigating the link between AVH and gray matter volume reported significant structural differences between AVH patients and controls particularly in the left and right superior temporal gyrus (STG), left inferior frontal gyrus, left amygdala, and insula bilaterally (Garcia-Marti et al., 2008). The authors, basing their interpretation on a previous model of emotional dysfunction in schizophrenia (Aleman & Kahn, 2005), concluded that the amygdala and insula as key regions of the emotional brain may be involved in the emotional load of AVH (Garcia-Marti et al., 2008), particularly in the emotional prosody conveyed by AVH.

One of the pioneering neuroimaging studies applied positron emission tomography (PET) to assess the pattern of regional cerebral blood flow in schizophrenia patients when they were actively hallucinating. Activation was found in left temporal lobe and Broca's area, suggesting that certain language processes, such as speech production, inner speech, and AVH share similar neural mechanisms (McGuire, Shah, & Murray, 1993). A related functional magnetic resonance (fMRI) study assessed patients with schizophrenia while hallucinating and during auditory stimulation (Dierks et al., 1999) and found an activation of the superior and medial temporal gyrus and primary auditory cortex (predominantly in the left hemisphere for two patients and in the right hemisphere for the third patient) for both conditions. Interestingly, a recent study demonstrated that hallucinators activate the left primary auditory cortex to a lesser extent that controls when listening to tunes, indicating that this region is tonically turned on to process irrelevant auditory objects (Ford et al., 2009).

Dierks and colleagues study also showed that during hallucinations, the fronto-parietal operculum, the hippocampus, and the amygdala showed an increase of the BOLD signal. The authors suggested that the activation of the primary auditory cortex during the hallucination may explain why hallucinations are perceived as real and often external sounds (Dierks et al., 1999), whereas the amygdala activation was considered to reflect an emotional response relating to the emotional content of the voices. Moreover, the authors interpreted the activation of the left temporal lobe as an indication of semantic processing in hallucinations, even though the relative contributions of semantic and prosodic aspects of language were not compared directly. Unfortunately, the lateralization of semantic (normally left-lateralized) and prosodic processes (normally right-lateralized) was not directly assessed in this study. Thus, these results cannot be related to evidence of a reduced lateralization of linguistic processing in schizophrenia particularly in those patients suffering from severe hallucinations (Sommer, Ramsey, & Kahn, 2001; Weiss et al., 2006).

Unlike the lateralization of linguistic processing, findings about the lateralization of prosody processing in schizophrenia are equivocal. While some evidence found a left lateralization of the normally right-lateralized response to prosody (Mitchell, Elliott, Barry, Cruttenden, & Woodruff, 2004), a more recent study found increased right lateralization of emotional prosody in schizophrenic patients (Bach et al., 2009). The findings from the cited studies are limited by small sample sizes.

Only a very few studies have directly assessed the neural link between EPC and AVH. Evidence comes from an fMRI study of patients with schizophrenia, in which neural activation in patients with and without history of AVH was compared while listening to external speech (Woodruff et al., 1997). This study found an increased activation particularly in the right middle temporal gyrus (MTG) for the patient group as a whole, relative to healthy controls during external speech stimulation. The authors suggested that this might indicate an inherent natural hyperresponsivity to emotional prosody of speech in the patient group. In fact, right hemisphere hyperresponsivity has

been found in other clinical populations such as violence offenders, who also show deficits in the perception of emotional cues (Lee, Chan, & Raine, 2009). Woodruff at al. also scanned schizophrenic patients during the hallucinating state when they were simultaneously listening to external speech, finding that during AVH the response to external speech in the right MTG was decreased. This finding suggests that AVH compete with external speech for auditory processing resources within the temporal cortex, including those regions which in healthy population are known to be necessary for EPC (Woodruff et al., 1997). However, as it has been previously said, the lateralization of EPC in patients with schizophrenia remains controversial (Bach et al., 2009; Mitchell et al., 2004).

Several studies addressed the overlap of the neural correlates of AVH and perception of prosodic and semantic aspects of speech. For listening to emotional speech, one study (Sanjuan et al., 2007) found enhanced activation of the left middle and right superior temporal lobe, insula, thalamus, and middle and superior frontal lobes in a group of patients with AVH in comparison to controls. The stimuli used in this study were based on patients' individual reports of the content of their AVH, where words were selected as stimuli based on their frequency in the reported AVH. The selected words contained an emotional semantic meaning and were recorded in an imperative prosodic tone. This study found an increase in brain activity in a fronto-temporal network involving the orbitofrontal cortex, the left medial temporal gyrus and right STG in the patient's group in comparison to controls, and this activation pattern was interpreted as related to AVH (Sanjuan et al., 2007). As this study did not test schizophrenia patients without AVH, however, the results should be interpreted with caution. Differences in the neural networks underlying semantic and prosodic aspects of speech processing between schizophrenia patients and healthy controls may be related to other aspects of the disorder and not specifically to AVH. In opposition to Sanjuan and colleagues, a recent study found decrease activity in superior temporal and inferior parietal areas, and in bilateral insula in AVH patients compared to patients without AVH when performing a memory recollection task (Wible et al., 2009). The authors interpret that as tonically elevated activity within these regions. The differences between studies might be due to the diverse paradigms they employed. Since the stimuli used by Sanjuan and colleagues may have triggered AVH, the task used in Wible et al. (2009) was meant to compete for neural resources with AVH.

Along with the STG, the amygdala is one of the structures that seem to be involved in AVH as well as in EPC, specifically for anger and fear. Amygdala involvement in processing of angry voices may relate to the fact that angry voices "threaten" the listener, which is especially pertinent in schizophrenia given the negative focus of AVH outlined above. Further, it has been demonstrated that, at a resting state, the amygdala shows higher metabolism in patients with schizophrenia than in healthy controls. This might indicate a tonic hyperactivation of the amygdala in schizophrenia, as opposed to a phasic activation in the presence of salient emotional stimuli in healthy controls. Interestingly, hypometabolism of the amygdala during emotion judgment tasks has also been found in schizophrenia (for a review, see Aleman & Kahn, 2005). In fact, a study assessing patients' neural response to nonverbal emotional auditory stimuli (crying and laughing) found that patients with a history of AVH showed reduced activity in the left amygdala and bilateral hippocampus during crying sounds in comparison with patients without such a history (Kang et al., 2008). A different study found increased amygdala activation in AVH patients during passive listening to emotional words in comparison to NAVH and controls (Escarti et al., 2010). However, this study did not control for occurrence of hallucinations during the scanning, which was a likely eventuality given that AVH patients obtained high scores in a scale measuring hallucinatory experience in the previous 24 h to the testing session. Thus, the fMRI results might rather have been related to the experience of hallucinations itself than to the experimental stimuli. Possibly, the constant hypermetabolism of the amygdala in schizophrenic patients prevents the detection of differences in BOLD response between baseline and threatening stimuli conditions. Furthermore, tonic pattern of amygdala activation might explain the ubiquitous perception of threats some patients suffer from, resulting in constant (implicit) processing of emotional stimuli leading to lead to feelings of being emotionally overwhelmed that precedes development of AVH.

4. Structural and functional connectivity within the EPC and AVH networks

Studies of structural connectivity with diffusion tensor imaging (DTI) have revealed that AVH schizophrenia patients, compared with non-AVH schizophrenia patients, showed an increased connectivity in the temporo-parietal section of the arcuate fasciculus (Hubl et al., 2004; Shergill et al., 2007), which connects the STG and medial temporal gyrus (MTG) with the inferior frontal lobe (Glasser & Rilling, 2008). It should be noted that a right temporo-parietal network has been identified as underlying emotional prosody processing (Alba-Ferrara, Hausmann, Mitchell, & Weis, 2011; Bach et al., 2008; Mitchell, Elliott, Barry, Cruttenden, & Woodruff, 2003). Moreover, it has been proposed that perturbations in a left posterior temporal and inferior parietal regions network comprising the dorsal "where" auditory pathway results in the AVH to be perceived in external space making them sound more "real"(Badcock, 2010). AVH patients also showed increased structural connectivity in the left cingulated bundle, which is part of the limbic system (Hubl et al., 2004; Shergill et al., 2007). The increased connectivity between language-related areas may lead to an abnormal hyperactivation of the circuit which processes external speech, increasing noise in the speech processing system and lowering the threshold for the intrusion of aberrant auditory perceptual input (Rotarska-Jagiela et al., 2009).

Additionally, a functional connectivity study showed that schizophrenic patients with AVH had poor functional integration between anterior cingulate cortex (ACC) and STG (Mechelli et al., 2007). The authors claim that this network underlies the evaluation of speech source (Mechelli et al., 2007). The aberrant activation of the neural correlates of speech perception without the regulation of the ACC might partially contribute to the formation of hallucinations via deficient source monitoring of inner speech. Moreover, increased structural connectivity within the temporo-frontal network might result in increased spreading of activation in the speech network, introducing noise and increasing excitability of the system (Rotarska-Jagiela et al., 2009), which, in addition to the poor functional integration in this network, might further contribute to abnormal perception. It is important to note that abnormal fluctuations in the BOLD signal can also be detected at resting state in patients with schizophrenia (Garrity et al., 2007), and it has been proposed that there is spontaneous activity in auditory sensory areas which might form the basis of auditory perception in the absence of external stimuli (Hunter et al., 2006).

In conclusion, AVH and EPC in schizophrenia seem to be based on overlapping neural networks, in which the STG seems to play a pivotal role. Interestingly, targeting the STG with TMS has been shown to reduce the severity of AVH (Aleman, Sommer, & Kahn, 2007), suggesting a causal involvement of this structure in hallucinations. Additionally, a combined fMRI and TMS study found that even when the BOLD response during hallucinations appear to be distributed in a network comprising the left and right inferior frontal gyri and the left superior temporal gyrus, only TMS applied on the left superior temporal gyrus ameliorated the AVH (Hoffman et al., 2007). The authors concluded that this area plays a critical role in the genesis of AVH (Hoffman et al., 2007). Additional evidence demonstrated that patients with a history of AVH have reduced gray matter density in the STG (Garcia-Marti et al., 2008; Nenadic et al., 2010) suggesting

that this structure is constitutively involved in the generation of hallucinations. Additionally, there is evidence of abnormal structural and functional connectivity of STG with frontal areas in AVH patients which might come on top of the gray matter abnormalities of this area. It is important to note that numerous findings have associated this structure not just with AVH and prosodic processing but also with a variety of cognitive domains, including audiovisual integration, theory of mind and speech processing. Within the variety of neural networks the STG is temporally coupled to (Hein & Knight, 2008), for our model we will focus on the cases in which it interacts with the EPC and AVH networks.

5. Towards a new neuropsychological model of AVH

Some AVH seem to be triggered by external sounds and there is some supplementary evidence indicating that inaccurate perception of prosodic features of external sounds, such as timbre, amplitude, and timing, might contribute to the formation of AVH. Along these lines, a case study has shown that false perceptions may derive from misinterpretation of real sounds as exemplify in the case of a patient who heard a real engine sound and parallelly perceived a voice with "mechanical timbre" (the voice sounded "like a machine") (Hunter & Woodruff, 2004). In this case, acoustical features of the AVH were already present in an original environmental sound (Hunter & Woodruff, 2004) and features corresponding to the timbre of sounds seem to be misprocessed in a way that triggers false perceptions. We suggest that this primary sensory processing impairment plays a necessary role in false perception, but it is not sufficient to form hallucinations *per se*.

While some AVH appear to be triggered by an external sound, other AVH appear to be generated intrinsically and in the absence of external stimuli. At the neurophysiological level, it has been shown at resting state there is spontaneous activity in the primary auditory cortex, superior temporal gyrus (STG), and anterior cingulate cortex (ACC) in healthy populations (Hunter et al., 2006). Such spontaneous activity establishes the possibility of this circuitry being involved in perception when external stimuli are absent. It has been proposed that disruption in the physiological modulation of this network at resting state, or some defect in the intrinsic functional connectivity of this network, could contribute to an enhanced sensitivity of the primary auditory cortex, which might lead to an increased sensitivity to irrelevant (auditory) signals, and thus to hallucinatory processes (Hunter et al., 2006). Considering that the primary auditory cortex has been shown to be prone to spontaneous activity, the existence of external stimulation might not be necessary for AVH and thus AVH might additionally be purely intrinsically generated. Thus, we hypothesize that such spontaneous fluctuation in the network might trigger the perception of AVH in absence of external stimuli in some cases, while in other cases it might lowers the excitability threshold facilitating neural firing in the presence of external stimuli.

Regardless whether auditory stimuli are internally or externally generated, they are integrated into a common percept after sensory evaluation. Evidence suggests that the STG is the neural substrate of this integration process in the healthy population (Leitman et al., 2008) and it has been shown that STG, a pivotal structure underlying EPC (Hoekert, Bais, Kahn, & Aleman, 2008; Mitchell et al., 2004), is also active during AVH (Stephane, Barton, & Boutros, 2001; Woodruff et al., 1997). The posterior section of STG in the left hemisphere is considered to be the location of Wernicke's area, the neural substrate of speech comprehension. Indeed, it may be the multifarious associations of the STG with numerous brain regions that explain why this structure is the focus of overlap between EPC and AVH. Functional connectivity analyses and DTI techniques have shown that STG is connected with auditory primary cortex, medial ventral frontal cortex, prefrontal cortex, ACC, and amygdala (Hunter et al., 2006; Mechelli et al., 2007).

Furthermore, the STG also forms part of an anatomically connected triadic network which also includes the amygdala and orbitofrontal cortices (Ghashghaei & Barbas, 2002). The amygdala may contribute to EPC specifically for the valences of fear and anger, and these two emotional valences are probably the most frequent affective tone of AVH. The amygdala receives inputs from the prefrontal cortex which regulates its activity via afferents suppressing amygdala output (Rosenkranz & Grace, 2001), but this amygdala regulation by prefrontal cortex is disrupted in schizophrenia (Leitman et al., 2008). Therefore, in schizophrenia, prefrontal circuitry may fail to down-regulate amygdala activity, which might therefore drive attention towards certain features of prosody, particularly negative tones, coincidently with the emotional valence of most of the AVH that patients suffer from. The amygdala thus lacks prefrontal inhibitory control and becomes hyperresponsive, i.e., it fails to suppress this stimulus-driven (bottom-up) process. In combination, the inefficient top-down control of the amygdala, and dysfunction of bottom-up processes (sensorial misperception) may account for the mechanism underlying the relationship between AVH and EPC in schizophrenia. Amygdala disinhibition increases emotional reactivity (Siever, 2008) and consequently biases attention towards threatening stimuli, which ultimately causes abnormal emotion processing.

Finally, we suggest that a final element of the network mediating the contribution of EPC to AVH is the abnormal connectivity between ACC and STG. Evidence suggests that this abnormal connectivity causes difficulty judging whether a stimulus was internally or externally generated (Erkwoh et al., 2006), thus leading to misattribution of inner auditory objects such as inner speech.

It should be noted that the proposed model also has some limitations and some aspects require further exploration and research. Moreover, the present model does not claim that the impaired mechanisms found in AVH patients and specifically EPC deficits are sufficient to produce hallucinations, nor can it explain the full phenomenology of AVH. Instead, our aim has been to draw attention to the association between EPC deficits and hallucinations which have so far been neglected in the literature. The present model does not invalidate previous cognitive models of AVH which have been useful to at explain certain phenomenological aspects of AVH. For example, Badcock (2010) model can elegantly explain why AVH are perceived in external auditory space. Additionally, memory frameworks can explain the content and derogatory tone of auditory hallucinations in those patients with traumatic experiences, although such experiences do not appear in all patients suffering from AVH. Our model should thus be seen as complementing previous models, and filling some gaps in previous theoretical frameworks. Pointed out by Jones (2008) different models may be needed to explain particular types of hallucinations because the phenomenological diversity of hallucinatory experiences is vast. The present model seeks to account for the specific type of AVH that is most characteristic of schizophrenia (Fig. 1).

6. Conclusion

The neuropsychological model proposed here accounts for the diverse phenomenology of AVH, considering that different aberrant processes may result in similar abnormal perceptions. We contribute to previous literature by integrating phenomenological and behavioral aspects of AVH in an empirically verifiable neural model. Regardless of whether AVH are triggered by the misperception of external sounds, by spontaneously generated intrinsic fluctuations in the auditory system, or by a combination of both, hallucinations in schizophrenia may occur at the stage of transition from primary sensory processing regions to integrative regions such as STG. Deficits in perception and integration of stimulus features may lead to EPC deficits which contribute to the formation of AVH, for example, via voice identity misidentification. Given that the right STG is involved in

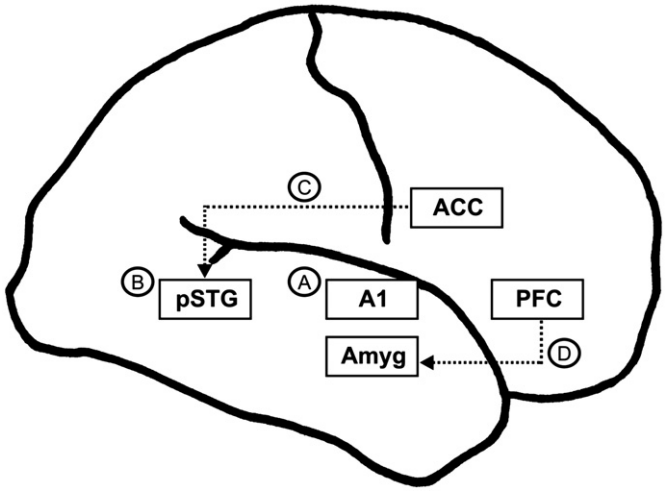

Fig. 1. Hypothetical model of the neural formation of AVH in schizophrenia. Precursors of AVH originate at the primary auditory cortex (A1). Spontaneously generated intrinsic fluctuations in the activation of A1 can result in a misperception of external low-level sound features and a perceptual experience of sounds even in the absence of external stimuli (A). A1 projects to the superior temporal gyrus. The posterior part of the superior temporal gyrus (pSTG) integrates prosodic features of external and intrinsically generated sounds. Structural and functional abnormalities in the pSTG in schizophrenia result in a degraded and aberrant integration of prosodic features (B). Additionally, a disruption of the functional connectivity between pSTG and anterior cingulate cortex (ACC) can lead to a misidentification of the source of both external and self-generated auditory objects (C). Finally, the amygdala is hyperactive due to aberrant top-down regulation by the prefrontal cortex (PFC), which results in increased emotionality and attention bias towards threatening auditory stimuli (D). These processes might independently or jointly contribute to the formation of AVH.

voice identity recognition and EPC, which are in turn associated with AVH, future empirical research should focus on how the STG is functionally and structurally altered in AVH patients and how these alterations are linked to deficits that patients might present at the behavioral level (such as prosody decoding and voice identity recognition deficits). Moreover, this model opens the possibility to intervene by training patients to withdraw attention from threatening auditory stimuli, as it has been shown that such stimuli capture attention triggering the formation of AVH. Lastly, we propose to perform an fMRI study in patients at the time they are actively hallucinating. From such study, three different strands of information can be extracted. a) Recent fMRI analyses show that it is possible to establish correlation in the activity of two or more brain regions and to demonstrate how the activity of one region is affected but the activity in another characterizing not only co-activated brain regions but also causal effects acting over time. Thus, such analysis could be applied to identify the coupling of neuronal activity along the brain structures pinpointed in the present AVH model. Such research would allow to test how adequate is the model given the empirical data. b) The application of this paradigm to non-clinical population of voice hearers could demonstrate that hallucinations between this group and schizophrenia patients not only differ in phenomenological aspects but also in its neural underpinnings. c) A seed region could be created based on the peak voxel during the AVH in schizophrenia patients. This region could become the target of clinical interventions such as the application of neurodisruptive techniques (i.e. TMS) which may hopefully diminish AVH precursor processes in patients resistant to the medication and thus reduce the human cost of the symptom.

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