Voice identity discrimination and hallucination-proneness in healthy young adults: A further challenge to the continuum model of psychosis?

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Abstract

*Introduction.* Auditory hallucinations occur in schizophrenia and also in the general population. However, evidence points to differences in the nature and mechanisms of clinical and non-clinical hallucinations, challenging the dominant assumption that they represent the same phenomenon. The current study extended this evidence by examining voice identity perception in hallucination-prone individuals. In schizophrenia, deficiencies discriminating between real (external) voices have been linked to basic acoustic cues, but voice discrimination has not yet been investigated in non-clinical hallucinations.

*Methods.* Using a task identical to that employed in patients, multidimensional scaling of voice dissimilarity judgements was used to examine how healthy individuals differing in hallucination-proneness (30 high and 30 low hallucination-prone individuals) distinguish pairs of unfamiliar voices. The resulting dimensions were interpreted with reference to acoustic measures relevant to voice identity.

*Results.* A two-dimensional “voice space”, defined by fundamental frequency ($F_0$) and formant dispersion ($D_f$), was derived for high and low hallucination-prone groups. There were no significant differences in speaker discrimination for high versus low hallucination-prone individuals on the basis of either $F_0$ or $D_f$.

*Conclusions.* These findings suggest voice identity perception is not impaired in healthy individuals predisposed to hallucinations, adding a further challenge to the continuum model of psychotic symptoms.
1. Introduction

Although auditory hallucinations (AH) are typically associated with schizophrenia (American Psychiatric Association, 2000; Blashki, Rudd, & Piterman, 2007; Sartorius et al., 1986; Sartorius, Shapiro, & Jablensky, 1974), they have also been found to be relatively frequent in the general population, with prevalence rates averaging 3-4% and rising to 14-71% in student populations (see Sommer et al., 2010; Stip & Letourneau, 2009; van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009 for reviews). While many healthy individuals will experience hallucinations with no other consequences, for some, hallucinations will progress to full psychosis (De Loore et al., 2011; Dominguez, Wichers, Lieb, Wittchen, & van Os, 2011; Johns & Van Os, 2001). These observations fit with the dominant “continuum hypothesis” of psychosis (Choong, Hunter, & Woodruff, 2007; Laroi, 2012; Laroi et al., 2012; Linscott & Van Os, 2010; Shevlin, Murphy, Dorahy, & Adamson, 2007; van Os et al., 2009) which rests on the idea that psychotic symptoms occur on a dimension between normality and psychosis and involve similar phenomenological characteristics and underlying mechanisms. However, several recent empirical reports and theoretical reviews of the literature have challenged this view, showing both similarities and differences in the nature (Daalman et al., 2011; Linscott & Van Os, 2010) and mechanisms (Badcock & Hugdahl, 2012; Diederan et al., 2011; Linden et al., 2011) of clinical and non-clinical hallucinations. As a result, there is a growing call for more debate and research on the continuum model (David, 2010; Kaymaz & van Os, 2010; Sommer et al., 2010). Uncovering the processes that are/are not involved in the predisposition to AH in non-clinical groups would be theoretically useful as well as providing valuable information for identifying those at risk of transitioning to schizophrenia.

AH typically involve voices (Beck & Rector, 2003; Wible, Preus, & Hashimoto, 2009), and both hallucinated and real (external) voices often convey information about the
identity (age, gender, and size) and emotional state of the speaker (Belin, Zatorre, Lafaille, Ahad, & Pike, 2000). For example, a person with AH may identify the hallucinated voice as belonging to a famous newsreader, who is angry at them (Chadwick & Birchwood, 1994; Hayward, 2003; Sorrell, Hayward, & Meddings, 2010). Consistent with the continuum model, there is growing evidence for emotional prosody deficits in both clinical (Rossell & Boundy, 2005; Shea et al., 2007) and non-clinical AH (Phillips & Seidman, 2008). A recent review suggests that emotional prosodic feature processing deficits play a pivotal role in the formation of AH (Alba-Ferrara, Fernyhough, Weis, Mitchell, & Hausmann, 2012), and these deficits are also thought to be important contributors to some of the psychosocial functioning difficulties in individuals with schizophrenia (Hoekert, Kahn, Pijnenborg, & Aleman, 2007). Affective prosody dysfunction has been shown to relate to basic auditory processing abnormalities (Jahshan, Wynn, & Green, 2013), including arising (at least in part) from difficulties processing pitch-related cues in voice (Kantrowitz et al., 2011; Leitman et al., 2005; Leitman et al., 2007; Leitman et al., 2010).

On the other hand, much less is known about the perception of voice identity. Models of human voice processing suggest that affect and identity information conveyed in speech are processed in partially segregated pathways (Belin, Bestelmeyer, Latinus, & Watson, 2011; Belin, Fecteau, & Bedard, 2004; Relander & Rama, 2009), hence it is possible that, independent of prosody deficits, individuals with clinical and non-clinical AH could have difficulties processing voice identity. Preliminary evidence in schizophrenia points to impairments in voice recognition (Zhang et al., 2008) and recall (Waters & Badcock, 2009) associated with AH. For example, Alba-Ferrara et al. (2012) have recently showed impaired recognition of famous voices, especially in patients with AH. These difficulties processing vocal identity information may be grounded in the mechanisms of human (i.e., real, external) voice perception (Badcock & Chhabra, 2013). Building on this proposal we recently reported
Voice identity & hallucination-proneness

A difference in discriminating speaker identity – linked to reduced use of resonance-based voice cues (formant dispersion; $D_f$) – in schizophrenia patients compared to healthy controls, using multidimensional scaling (MDS) (Chhabra, Badcock, Maybery, & Leung, 2012b). This difference was present both in patients with, and without AH, which raises the possibility that problems processing voice identity contribute to AH but may also play a role in other symptoms of psychosis such as delusions. Additionally, since patients currently without AH in this study had hallucinated in the past, it is possible that problems with voice identity perception may be relevant to the predisposition to hallucinate. The aim of the current study, therefore, was to examine voice identity discrimination in healthy (non-clinical) voice-hearers.

The adoption of identical tasks in studies of AH in different groups is rare (Amminger et al., 2012), resulting in inconsistencies in the literature which could simply reflect differences in stimuli and other aspects of methodology. In order to avoid this problem, the task used in the current study to measure perception of voice identity in relation to hallucination-proneness, was identical to that previously employed in patients with schizophrenia (Chhabra et al., 2012b). If individuals high in the predisposition to hallucinate show distorted differentiation of speaker identity compared to individuals low in the predisposition to hallucinate, this would support the continuity of vocal identity discrimination limitations in clinical and non-clinical hallucinations. More specifically, based on the results of Chhabra et al (2012b), support for the continuum model would take the form of individuals highly predisposed to hallucinations showing reduced use of $D_f$ to discriminate voices. Alternatively, if the high and low hallucination-prone individuals demonstrate similar speaker discrimination, this would be in keeping with a discontinuity of mechanisms underlying clinical and non-clinical hallucinations.
Finally, given evidence suggesting voice perception deficits may be a general vulnerability factor for psychosis (Chhabra et al., 2012), as well as recent evidence that hallucinatory and delusional tendency tend to co-occur in healthy individuals (Sommer et al., 2010), we measured delusional experiences to explore any potential relationship between voice identity discrimination and this symptom.

2. Method

2.1 Participants

Five hundred and twenty-two undergraduate psychology students completed the Launay-Slade Hallucination Scale-Revised (LSHS-R) questionnaire ($M = 14.08$; range = 0–40). Individuals scoring in the upper (scores of 28 and above) and lower (scores of 6 and below) quartiles were invited to participate in the study as one of several ways to meet a course requirement. Thirty high scorers (23 female) and 30 low scorers (22 female) responded to this invitation (see Table 1 for demographic information). Participants were free from current/previous history of psychosis (assessed using the Mini International Neuropsychiatric Interview for Schizophrenia and Psychotic Disorders; Sheehan et al., 1998) and all had normal hearing acuity (assessed with standard audiometry). Each participant provided informed consent using procedures approved by the Human Research Ethics Committee of the University of Western Australia.

2.2 Questionnaires and intelligence test

The LSHS-R questionnaire (Bentall & Slade, 1985) assesses a range of visual and auditory experiences. The 12 LSHS-R items are scored on a 5-point scale (ranging from 0 = *certainly does not apply to me*, to 4 = *certainly applies to me*). Items on the questionnaire describe both clinical hallucinatory experiences (e.g., item 8: *In the past, I have had the experience of*...
hearing a person’s voice and then found that no one was there) and sub-clinical intrusive mental events (e.g., item 5: The sounds I hear in my daydreams are usually clear and distinct). The LSHS-R has good reliability and validity (Laroi et al., 2012), with previous research showing that item endorsement patterns are stable across time (Aleman, Nieuwenstein, Bocker, & de Haan, 1999), and that the factor structure observed in non-clinical samples is comparable with the one obtained in clinical samples (Serper, Dill, Chang, Kot, & Elliot, 2005).

IQ was estimated using the vocabulary and matrix reasoning subtests of the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). The yes/no version of the Peter’s Delusion Inventory (PDI), which has been shown to have satisfactory reliability and validity (Peters, Joseph, Day, & Garety, 2004), was used to assess delusional thinking.

2.3 Similarity Ratings Task (Chhabra et al., 2012b)

Stimuli consisted of eight three-syllable words, matched in amplitude and spoken in 12 different voices (half male), presented via Sennheiser HD 205 headphones at 69 dB. On each trial, participants heard two speakers saying the same word in sequence and were instructed to make a judgment as to how similar/dissimilar the two voices sounded, by focusing on the qualities of the speakers’ voices. One second after the onset of the second word, a seven-point rating scale appeared on a touch screen, ranging from very similar (1) to very dissimilar (7), which participants used to record their responses. Participants were encouraged to use the full-range of the scale when making their voice ratings throughout the task. Testing was conducted in two blocks, each of which included presentation of all possible voice pairings, including pairings within-gender (i.e., male-male and female-female voice pairings) and between-gender (i.e., male-female and female-male voice pairings), in random order. The word used on each trial was randomly selected. Participants were
provided with four rest breaks throughout the task. Four practice trials were administered prior to commencing each block of testing, with 132 test trials in total. Stimuli were selected anew for each participant of each group. Total task duration was approximately 15 minutes (see Chhabra et al., 2012b for further details of the stimuli and procedure).

2.4 Analysis of acoustic characteristics

In order to explore the acoustic correlates of the dimensions found, we selected identical acoustic measures as in Chhabra et al. (2012b) since these have been highlighted as specifically relevant for voice identity (rather than voice emotion perception) recognition (Bachorowski & Owren, 1999; Baumann & Belin, 2010; Ko, Judd, & Blair, 2006; Leitman et al., 2010). Acoustic measurements were based on stimuli comprising a consistent order of all eight words spoken by each of the 12 speakers. PRAAT 5.0.32 software (Boersma, 2001) was used to compute the following acoustic characteristics:

**Pitch.** The pitch of each voice was measured by its average fundamental frequency ($F_0$) in Hertz (Hz).

**Pitch variability.** As an indicator of intonation, the standard deviation of pitch ($F_0$-SD) for each voice was calculated to assess the momentary variability in pitch during speech.

**First formant.** The peak frequency of the first formant ($F_1$), in Hz was obtained.

**Formant dispersion.** The resonance of each voice was represented by its formant dispersion ($D_f$, the average frequency difference between formants; Fitch, 1997), which is related to the speaker’s vocal tract size. $D_f$ was calculated as the average distance between adjacent pairs of the first five formant frequencies, in Hz (derived as in Baumann & Belin, 2010). The maximum formant frequency for female voices was set to 6500 Hz (Baumann & Belin, 2010). All other parameters were default values recommended by the authors of PRAAT.
2.5 Data analysis: multidimensional scaling (MDS) of similarity judgements

The average listener dissimilarity rating (out of 7, where 1 = *very similar* and 7 = *very dissimilar*) for each pair of the 12 voices was calculated for each participant, and matrices of these mean dissimilarity ratings were entered into MDS analyses which were conducted via an Individual-Squares Scaling (INDSCAL) model, with Euclidean distances and interval scaling. There were two phases of analysis; first, dissimilarity matrices from the two groups (i.e., one high LSHS-R group matrix and one low LSHS-R group matrix – provided in Supplementary material) were submitted to MDS to verify whether the data map onto a two-dimensional space as described in our recent study with schizophrenia patients (Chhabra et al., 2012b) and in previous studies of both healthy (non-clinical) and schizophrenia populations (Baumann & Belin, 2010; Leitman et al., 2010). Interpretation of these dimensions was then determined by examining correlations of the values for the 12 voices on each dimension with the acoustic measures computed for those voices. Based on our previous findings (Chhabra et al., 2012b), we expected a directional relationship between Dimension 1 and F0 and between Dimension 2 and Df; hence one-tailed tests were used.

Next, we included matrices from individual participants (i.e., 30 high LSHS-R and 30 low LSHS-R dissimilarity matrices) within the same INDSCAL model in order to assess whether there were any differences in the processes used to differentiate voices between high and low LSHS-R groups. INDSCAL produces a critical parameter in the form of a weight for each individual on each dimension, which accounts for individual variation in the perceptual processes when performing the rating task. The higher the weight, the greater the importance that individual gave to that dimension (Kring, Barrett, & Gard, 2003). The weights for each dimension were then compared for the high and low LSHS-R groups using independent-samples t tests. Prior to these comparisons, the dimension weights were screened for outliers.
(scores three or more standard deviation units away from their respective group means). No outliers were identified for these INDSCAL weights for the participants in either group.

To check for possible influences of delusional experiences and intelligence, the high and low LSHS-R groups were compared on IQ and PDI scores. If either variable yielded a significant group difference, the variable was correlated with the INDSCAL dimension weights for each of the two groups. No further action was taken if these correlations were not significant.

3. Results

3.1 Descriptive statistics

Participants’ scores on single measures were excluded if they were 3 standard deviations or more away from their respective group means. Two extreme data points were identified and excluded for WASI IQ scores. No other outliers were identified. A summary of cognitive and schizotypy measures for the high and low LSHS-R groups is provided in Table 1. Substantial group separation was obtained on the LSHS-R, as expected. No significant group differences were observed in age or WASI IQ scores, however high LSHS-R scorers obtained significantly higher scores than low LSHS-R scorers on the PDI.
Table 1

**LSHS-R group means, standard errors (SE), and t-tests for the age, PDI and WASI data.**

<table>
<thead>
<tr>
<th></th>
<th>Low LSHS-R</th>
<th>Mean</th>
<th>SE</th>
<th>High LSHS-R</th>
<th>Mean</th>
<th>SE</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>LSHS-R</td>
<td></td>
<td>3.53</td>
<td>.32</td>
<td></td>
<td>31.37</td>
<td>.74</td>
<td>34.56**</td>
</tr>
<tr>
<td>AGE (years)</td>
<td></td>
<td>17.93</td>
<td>.18</td>
<td></td>
<td>17.80</td>
<td>.16</td>
<td>.55</td>
</tr>
<tr>
<td>PDI</td>
<td></td>
<td>3.97</td>
<td>.44</td>
<td></td>
<td>8.43</td>
<td>.51</td>
<td>6.64**</td>
</tr>
<tr>
<td>WASI</td>
<td></td>
<td>109.23</td>
<td>1.37</td>
<td></td>
<td>109.87</td>
<td>1.39</td>
<td>.33</td>
</tr>
</tbody>
</table>

**p < .001

3.2 MDS of group dissimilarity matrices

A two-dimensional solution was found to be most appropriate for the MDS analysis conducted using the group dissimilarity matrices for the high and low LSHS-R samples *(proportion of variance accounted for (R²) = 0.78; stress = 0.19)* based on the interpretability, uniqueness, and percentage of accounted-for variance (Baumann & Belin, 2010), as well as on other recommendations from the literature (Borg & Groenen, 1997; Kruskal & Wish, 1978). This two-dimensional solution for the 12 voices for the high and low LSHS-R scorers combined is shown in Figure 1. In this voice space, the greater the distance between voices, the lower their perceived similarity.

*(insert Figure 1)*

Table 2 provides correlations between values on the two dimensions derived from this analysis and the acoustic measures for the 12 voices. Values for Dimension 1 correlated...
strongest with $F_0$, but also correlated with $F_0$-SD. Dimension 2 correlated significantly with $D_f$. Overall, it seems that participants mainly used the speaker attributes of $F_0$ and $D_f$ in making the similarity/dissimilarity judgements. This is consistent with MDS dimensions identified using the identical task in patients with schizophrenia (Chhabra et al., 2012b), as well as with those found in other studies that have examined voice dissimilarity using MDS (Baumann & Belin, 2010; Goh, 2005; Ko et al., 2006; Leitman et al., 2010).

Table 2

*Correlations between scores on MDS Dimensions 1 and 2, and acoustic measures, for high and low LSHS-R groups.*

<table>
<thead>
<tr>
<th>Acoustic measures</th>
<th>Dimension 1</th>
<th>Dimension 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pitch ($F_0$)</td>
<td>.93**</td>
<td>.43</td>
</tr>
<tr>
<td>Pitch variability ($F_0$-SD)</td>
<td>.60*</td>
<td>.17</td>
</tr>
<tr>
<td>First formant ($F_1$)</td>
<td>.05</td>
<td>.04</td>
</tr>
<tr>
<td>Formant dispersion ($D_f$)</td>
<td>.24</td>
<td>.55*</td>
</tr>
</tbody>
</table>

*p < .05, **p < .01

3.3 MDS using dissimilarity matrices for individual participants

The distribution of weightings within each subgroup was normal. To provide a visual comparison of the distributions of voices in two-dimensional space for the high and low LSHS-R groups, dimension values were averaged for the participants in each group. Figure 2 illustrates the similar distribution of voices for the high and low LSHS-R groups.

*insert Figure 2*
The subject weights for each dimension (1 and 2) were then compared for high and low LSHS-R scorers to assess whether the two groups differed in the extent to which their judgments relied on either dimension. Although the high LSHS-R group appeared to assign lower weightings (i.e., be less sensitive to variability between voices) compared to the low LSHS-R group (see Figure 2, Table 3), independent-samples t tests did not reveal any significant differences between groups in terms of their weightings for either Dimension 1, \( t(58) = 1.70, \) partial-\( \eta^2 = .05, \) \( p > .05, \) or Dimension 2, \( t(58) = 1.43, \) partial-\( \eta^2 = .03, \) \( p > .05. \)

It is unlikely that delusional tendency influenced the performance of high and low LSHS-R scorers since PDI scores did not correlate with individual subjects weights on either Dimension 1, \( r(30) = -.19, \) \( p > .05, \) or Dimension 2, \( r(30) = -.14, \) \( p > .05. \) As such, no further action was taken to account for PDI scores\(^1\).

Table 3

*Descriptive statistics for subject weights as a function of dimension in high and low LSHS-R groups.*

<table>
<thead>
<tr>
<th></th>
<th>Low LSHS-R</th>
<th>High LSHS-R</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Dimension 1</td>
<td>.37</td>
<td>.18</td>
</tr>
<tr>
<td>Dimension 2</td>
<td>.28</td>
<td>.10</td>
</tr>
</tbody>
</table>

4. Discussion

To our knowledge, this is the first study to use MDS to examine the perception/discrimination of voice identity in young adults highly predisposed to

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\(^1\) In additional analyses, no evidence of any influence of delusions was obtained in comparing the performance of high and low PDI scorers. Also, no additional effects were identified when sex was added to analyses as a between-subjects factor and IQ and age were added as covariates.
The main finding of this study was that individuals with high and low levels of hallucination-proneness showed a similar pattern of discrimination between voices, using both pitch ($F_0$) and formant dispersion ($D_f$) cues. These results suggest that the discrimination of voice identity is unimpaired in healthy (non-clinical) individuals who are prone to hallucinate, and contrast with those recently documented for patients with schizophrenia, which showed significant differences in some aspects of speaker discrimination. It is unlikely that this difference in outcome can be explained simply as a result of differences in stimuli or method since an identical task was used in the two studies (Chhabra et al., 2012b). It is possible that the difference in outcome between studies reflects general illness-related factors (medication, previous hospitalization, etc.) in patients rather than representing specific difficulties in voice-identity discrimination. However, such non-specific illness effects would have been expected to lead to diminished performance across all indices of voice-identity perception, which was not the case: individuals with schizophrenia demonstrated differences in processing resonance-based cues but not pitch-based cues to voice (Chhabra et al., 2012b). Similarly it seems unlikely that poor performance in patients could be explained as an effect of medication since the association between antipsychotic dosage and voice processing ability was not significant. Consequently our findings present a further challenge to the continuum model of psychosis.

The pattern of voice dissimilarity judgements obtained for both high and low hallucination-prone groups was well captured by representing individual voices in a two-dimensional space, defined by the average $F_0$ and $D_f$. This characterization of a "perceptual voice space" (see Latinus & Belin, 2011) is consistent with the MDS space previously described in healthy (i.e. non-clinical) individuals listening to brief vowel sounds (Baumann & Belin, 2010), and in patients with schizophrenia and their controls, using three-syllable words (Chhabra et al., 2012b). Statistical testing revealed no significant difference between
high and low LSHS-R groups in the weightings assigned to either Dimension 1 ($F_0$) or Dimension 2 ($D_f$), pointing to similar sensitivities to differences in speaker identity arising from lower level acoustic cues (pitch and formant dispersion) in healthy, non-clinical hallucinators and non voice hearers. This latter result in particular contrasts with that of our recent study comparing schizophrenia patient and healthy control samples (Chhabra et al., 2012b), whereby patients appeared to be less sensitive to differences in voices based on formant dispersion, and therefore, less able to differentiate between them on this basis. Of note, this difference in voice identity discrimination in schizophrenia appeared to be relevant to other psychotic symptoms as well as to AH (i.e., to be a general vulnerability factor for psychosis). Thus, one possible interpretation of the apparent discontinuity in the perception of voice identity in clinical and non-clinical AH is that differences in voice identity discrimination may only emerge further along the continuum of psychosis when early hallucinatory experiences become complicated with other symptoms such as delusional ideation (Smeets et al., 2010). However, in the present study, PDI scores – although relatively low – did not correlate with individual subject weights on either dimension, suggesting that there does not seem to be a relationship between delusional tendency and discrimination of voice identity in the current task. Future studies need to explore the potential link between voice perception and delusions. Alternatively, voice identity discrimination deficits may only arise as psychosis fully develops (Badcock, Chhabra, Maybery, & Paulik, 2008; Chhabra, Badcock, & Maybery, 2012a; Chhabra, Badcock, Maybery, & Leung, 2011; Waters & Badcock, 2009; Zhang et al., 2008). Importantly, AH in psychosis are more frequently experienced, are more intrusive and distressing, and have a different average age of onset than AH in the general population (Badcock et al., 2008; Choong et al., 2007). Thus a different explanation is that clinical and non-clinical hallucinations represent different phenomenological subtypes – rather than different points on a continuum – stemming from
different aetiological mechanisms. Alternatively, it may be necessary (and appropriate) to reconceptualise the continuum hypothesis of psychosis as *multiple continua* (see, for example, Kaymaz & van Os, 2010). If correct, an apparent surface-level continuum may nonetheless be underpinned by two discretely different groups (i.e., phenomena such as AH can be both continuous and discontinuous with normality at the same time; though not in the same individual).

Overall, the current results appear to indicate a discontinuity in the perception of voice identity in individuals experiencing clinical versus non-clinical hallucinations. In contrast, recent evidence indicates that the perception of vocal emotion (affective prosody) is impaired across the continuum of psychosis, that is, is present in patients with schizophrenia and AH as well as in individuals at risk of developing schizophrenia (Hoekert, Kahn, Pijnenborg, & Aleman, 2007; Phillips & Seidman, 2008). If correct, this difference may reflect the underlying separability of neural pathways specialized for processing vocal affect and vocal identity information in human voices (Belin et al., 2004; Garrido et al., 2009; Hailstone, Crutch, Vestergaard, Patterson, & Warren, 2010). Specific abnormalities within these pathways may result in differential contributions to the symptoms of schizophrenia: consequently continuity of deficits for healthy individuals prone to AH and individuals with psychosis may arise in one pathway, but not necessarily in the other. As such, hallucination-prone individuals may be impaired in the ability to process vocal emotions (van't Wout et al., 2004), but unimpaired in their ability to process vocal identity. Alternatively, it is possible that emotion prosody tasks may simply be more demanding, and that impairments may be present in both vocal affective and identity pathways in healthy individuals predisposed to hallucinations, but not revealed within the requirements of the current task (i.e., to judge whether voice pairs are similar or different in identity) (Chhabra et al., 2012b). Additional investigation of this proposal is warranted.
4.1 Limitations

It is necessary to note that Dimensions 1 and 2 are unlikely to be completely independent of each other. In particular, the sizeable - though non-significant - correlation also observed between Dimension 2 and $F0$, suggests this dimension is unlikely to be a pure reflection of $D_f$. Increased understanding of how these two dimensions contribute to voice identity perception could be attained by testing for the capacity to discriminate voices in one dimension while holding the other dimension constant (using, for instance, a same/different recognition task), and by testing for more basic impairments in auditory perception.

The current study was also limited in that the hallucination-predisposed individuals were all undergraduate university students, and hence, not necessarily representative of the general population, although young adults are the peak group in which hallucinations are reported (Stip & Letourneau, 2009). Future research should test a larger, more varied sample. Furthermore, Laroi (2012) distinguishes between two types of non-patient (healthy) AH: type i, in which AH are infrequent, and not very similar to patient AH; and type ii, in which frequent AH are experienced, which are very similar on a number of levels to those in patients with psychosis. As is the tendency in research into non-patient AH, the hallucination-predisposed sample (i.e., high LSHS-R scorers) in this study are likely to have comprised non-patient individuals with type i AH (though this was not formally assessed). Hence one approach would be to design a study employing the same task in comparisons of a clinical group of individuals experiencing AH to a group of healthy individuals who are selected because they experience phenomenologically similar AH (i.e., similar in frequency, form, and severity) to those experienced by patients (i.e., type ii AH). This approach would help to make firm conclusions about whether voice identity processing is or is not impaired in non-patient compared to patient AH.
Acknowledgement

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Dominguez, M. D., Wichers, M., Lieb, R., Wittchen, H. U., & van Os, J. (2011). Evidence that onset of clinical psychosis is an outcome of progressively more persisten


Jahshan, C., Wynn, K., & Green, M. F. (2013). Relationship between auditory processing and affective prosody in schizophrenia. *Schizophrenia Research, 143,* 348-353.


Sartorius, N., Jablensky, A., Korten, A., Ernberg, G., Anker, M., Cooper, J. E., et al. (1986). Early manifestations and first-contact incidence of schizophrenia in different cultures. A preliminary report on the initial evaluation phase of the WHO Collaborative Study
on determinants of outcome of severe mental disorders. *Psychological Medicine, 16*, 909-928.


Figure captions

Figure 1. The two-dimensional INSCAL voice space for high and low LSHS-R scorers combined, derived from dissimilarity ratings for the 12 voices. Dimensional interpretations ($F_0$ & $D$) are derived from correlational evidence (see text for details).

Figure 2. Super-imposition of the two-dimensional INDSCAL solutions obtained for individual high and low LSHS-R scorers, derived from dissimilarity ratings for the 12 voices.