VOICE-PITCH PERTURBATION IN NON-CLINICAL PSYCHOSIS

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Abstract

Patients with psychosis experience deficits in multisensory integration (MSI), or the communication between different sensory modalities, such as sound and sight. One way to assess multisensory integration is by utilizing voice-pitch tasks in which participants hear the pitch of their own voice artificially altered by a computer. When healthy individuals hear this computerized pitch-shift, they respond behaviorally by changing their own pitch. This is a reflexive attempt to "correct" a perceived error, demonstrating communication between the sensory system and the motor system. While voice-pitch tasks have been used to assess MSI in patients with Parkinson's Disease (another population with emergent MSI abnormalities), it is unclear what responses on voice-pitch tasks look like in psychosis populations. One way to address this unknown question is to assess voice-pitch task performance in individuals with non-clinical psychosis (NCP) who are otherwise healthy but experience occasional psychotic-like experiences (PLEs). In the present study, a total of 36 participants (11 NCP and 25 controls) were asked to complete a voice-pitch task in which they held a constant and steady "ahh" sound. While vocalizing, they heard the pitch of their voice artificially shifted by a computer, and the task determined if they consequently shifted pitch in response to the stimuli. Variables of interest on the task were 1) magnitude (the extent to which the response-pitch deviated from baseline) and 2) latency (the amount of time elapsed between the end of the pitch-shift stimulus and the beginning of the response). I hypothesized that NCP participants would show exaggerated responses (greater magnitudes) and slower reaction times (longer latencies), consistent with findings among patients with Parkinson's Disease. Although results from the study were not statistically significant, the effect size and pattern of trends both suggest that with increased power, the NCP group may show deficits in sensorimotor integration, reflected by abnormal performance on the voice-pitch task when compared to controls. Studying integration patterns among at-risk samples and psychosis populations may identify a potential vulnerability marker relevant for understanding the pathogenesis of psychosis.

Keywords: schizophrenia, NCP, sensorimotor integration, biomarker, vulnerability

Voice-Pitch Perturbation in Non-Clinical Psychosis

Introduction

Schizophrenia is a debilitating mental illness, affecting about 1% of the population (Barlow & Durand, 2011). The disorder has previously been viewed categorically (diagnosis or no diagnosis); however, more recent research suggests that the severity of psychosis symptoms actually exists along a continuum (Kelleher & Cannon, 2011). One group that falls on the psychosis continuum includes individuals reporting non-clinical psychosis (NCP). Representing 5-8% of the general population, these individuals are otherwise healthy but endorse occasional psychotic-like experiences (PLEs), such as fleeting auditory or visual hallucinations. For example, these individuals may think they see something that disappears or think they hear their name being called when nobody is around. Occurring only once or twice a year, these experiences are infrequent and nondistressing (Kelleher & Cannon, 2011). The present study explores multisensory integration (MSI), a neurological function that is known to be impaired among psychosis populations, within the context of NCP. MSI refers to the integration of information from multiple senses and is crucial for everyday functioning, social interactions, and behavior (Tseng et al., 2015). Particularly, the study explores a specific form of MSI called sensorimotor integration. As the name suggests, this form of MSI requires the sensory system and the motor system to communicate and coordinate with one another. The current study is the first to investigate this form of integration among an NCP group.

Non-Clinical Psychosis (NCP) and the Psychosis Continuum

There is extensive literature suggesting NCP groups share vulnerability factors with psychosis populations (Kelleher & Cannon, 2011; Polanczyk et al., 2010). Although there is a slight increased risk for developing psychosis among this group, the majority of these individuals

still do not (Jacobson et al., 2010). Rather, progression from PLEs to a clinical diagnosis depends heavily on exposure to environmental factors that interact with the inherited susceptibility (Polanczyk et al., 2010). Thus the goal of studying NCP is not to predict the onset of psychosis, but instead to add to the greater understanding of how certain biological and neurological variation may relate to the emergence of psychotic symptoms. In other words, the aim is to explore shared vulnerabilities existing on the continuum that, when met with certain environmental triggers (such as stress or trauma), may relate to the onset of psychosis.

Based on many shared vulnerability factors, both the criterion and construct validity of NCP suggest that this group is a valid population for studying the etiology of psychosis (Kelleher & Cannon, 2011). Within the literature, there has been research investigating NCP groups in order to understand the psychosis continuum more broadly. For example, in a study involving 205 NCP participants, both dermatoglyphic asymmetries and procedural learning were examined (Mittal et al., 2012). Dermatoglyphics, or skin patterns on the palms and fingers, are known to be asymmetrical among patients with schizophrenia (Reilly et al., 2001). Likewise, procedural learning (learning by doing) is also known to be impaired among patients with schizophrenia (Huston & Shakow, 1949; Eysenck & Frith, 1977). Mittal et al. (2012) found that NCP participants showed both elevated dermatoglyphic asymmetries and slower rates of learning on a procedural learning task when compared to controls. Further, these findings were consistent with previous studies among patients with formal psychosis diagnoses (Granholm et al., 1993; Gomar et al., 2011; Clare et al., 1993). Taken together, studying NCP groups provides important insights into understanding psychosis by allowing researchers to explore the broader psychosis spectrum and the trajectory of symptomatology. Additionally, studying individuals with NCP can aid in

teasing apart factors contributing to psychosis onset in the absence of clinical confounds that otherwise exist when studying psychosis groups (such as medication or substance use).

Multi-Sensory Integration

An area of particular interest to study among at-risk and psychosis populations is multisensory integration (MSI). MSI is a critical function of the brain that allows individuals to interpret information by communicating between multiple systems, such as the eyes and the ears working together to make sense of a scene (Stekelenburg, Maes, Gool, Sitskoorn, & Vroomen, 2013). For example, perhaps there is an ambiguous animal that is difficult to identify. People may be able to make sense of this animal by integrating what they *see* with what they *hear*. If the animal barks, they may conclude that it is a dog; if the animal purrs, they may conclude that it is a cat. The automatic MSI process allows individuals to generate holistic experiences by bringing together various pieces of information, as neurological systems coordinate and communicate with one another. In other words, brain activity does not exist in isolation, but rather within a larger network of neurological connectivity (Stekelenburg et al., 2013).

Patients with schizophrenia exhibit deficits in multisensory integration (Williams, Light, Braff, & Ramachandran, 2010), and this disruption in communication is referred to as the "disconnection syndrome" (Jacobson et al., 2010). In a study comparing 20 patients with schizophrenia to 20 healthy controls, participants were asked to complete a target-detection task involving unimodal targets (prompting only one sensory modality, either auditory or visual) and bimodal targets (prompting two sensory modalities, both auditory and visual). Results indicated that healthy individuals were quicker to detect bimodal targets than the patients with schizophrenia (Williams et al., 2010). As such— demonstrated by slower reaction times detecting targets that require the integration of two sensory modalities— those with

schizophrenia showed deficits in multisensory integration. These results held even when the researchers matched the participants for unimodal reaction times (Williams et al., 2010).

In another study (Stekelenburg et al., 2013), 18 healthy individuals and 18 patients with schizophrenia were presented with incongruent stimuli in which they heard a person say one syllable ("ba") but saw that person enunciate a different syllable ("fu"). Electroencephalography (EEG) results indicated that the patients with schizophrenia did not experience the same neurological spike that controls did when perceiving the incongruent stimuli. In other words, they were not able to simultaneously take in both audio information as well as visual information to understand and detect that the two did not match up. These results indicate deficits in multisensory integration at a neural level among patients with schizophrenia (Stekelenburg et al., 2013).

Auditory Feedback and Vocal Output

Auditory feedback plays a critical role in communication and is essential for normal, daily conversations and interactions. Speakers ultimately monitor auditory feedback to ensure it aligns with what they intend to say. For example, if what they hear themselves say (i.e., auditory feedback) does not match with what they expect, they will modify accordingly (Burnett, Freedland, Larson, & Hain, 1998). Simply, speakers control details of voice and speech, including loudness, pitch, or even actual words. To illustrate, if speakers accidentally use an incorrect word, they hear themselves use the wrong word and most likely repeat the sentence using the correct word. This modification is a result of an auditory feedback loop functioning properly: sensory information is perceived (i.e., hearing the incorrect word), speakers hear and recognize this word as an error, and neurological systems send signals to the motor cortex to correct for the error (Kiran & Larson, 2001).

When listening to themselves sustain the sound "ahhh," ten healthy participants (Flagmeier et al., 2014) briefly heard the pitch of their own voice electronically altered (either raised or lowered). These changes sounded to the participants as if they made a mistake because of a discrepancy between what they expected to hear and what they did hear. They compensated for these computer-generated pitch-shifts by either lowering or raising their pitch in response. This reflects a successful feedback loop: pitch-shift "information" comes in that the brain perceives as an error needing to be fixed (Flagmeier et al., 2014). This triggers corrective behavior, again sending a signal to the motor cortex to react verbally (Parkinson et al., 2012). Thus the sensory system and the motor system communicate during this detection and correction process by perceiving, identifying, and compensating for perceived errors. These findings reflect voice production to be a highly complex motor skill that integrates important sensory input into motor output.

Reflexive Deficits

Because this complex, neural control of the voice requires coordination among multiple systems, patients with psychiatric disorders who experience deficits in MSI may not possess behavioral control over vocalization or respond with the same reflexive modification that controls do (Flagmeier et al., 2014). For example, the effects of pitch-shifts were studied among ten patients with Parkinson's Disease, a population known to experience abnormal processing of auditory, visual, and somesthetic information (Kiran & Larson, 2001) and ten healthy controls. Patients with Parkinson's Disease showed more variability, demonstrating overall exaggerated responses. When sustaining an initial "ahh," participants were vocalizing at baseline. After a pitch-shift stimulus was presented and participants responded accordingly, the magnitude (change in pitch) was measured in cents and recorded. The Parkinson's Disease group

demonstrated responses of greater magnitude, meaning their reflexive responses to the pitch-shift deviated from baseline more than the controls' responses did. Additionally, latency refers to the amount of time in between the end of the pitch-shift stimulus and the beginning of the response. The Parkinson's Disease group demonstrated longer latencies, meaning it took longer for them to react than it did for controls. Taken together, Kiran and Larson (2001) concluded that the clinical sample demonstrated less control over their vocalization than the control sample.

In Kiran and Larson's (2001) study, the duration of the pitch-shifts varied between 100 milliseconds, 500 milliseconds, and 1000 milliseconds. As the duration of the shift became longer, responses became more "voluntary" and less "reflexive," because as duration increases, participants began to predict what came next. This allowed them to incorporate more of their own mental cognition (Kiran & Larson, 2001). While all participants (both healthy and those with Parkinson's Disease) exhibited pitch-shift responses at all durations, the greatest discrepancy between the patients and the controls occurred during the 100 millisecond trials, aimed primarily at reflexive responses. Further, several fMRI studies reveal that when a pitch-shift lasts for fewer than 300 milliseconds, participants demonstrate reflexive responses, whereas shifts exceeding 300 milliseconds elicit voluntary responses (Burnet et al., 1998; Hain et al., 2000). By exhibiting more abnormal responses during the 100 millisecond trials, patients with Parkinson's Disease demonstrated deficits in their reflexive reactions, suggesting that the general shortfall lies in automatic, knee-jerk processing rather than voluntary processing (Kiran & Larson, 2001).

Aims of Study

The present study recruited a total of 36 participants (11 NCP and 25 controls) and employed the same voice-pitch task that was able to successfully identify sensorimotor deficits

among a Parkinson's Disease population (Kiran & Larson, 2001). Because NCP is associated with psychosis risk factors, and given that individuals with psychosis also demonstrate deficits in MSI, studying sensorimotor integration among NCP is novel and promising. I hypothesized that NCP individuals would show greater difficulty integrating auditory feedback and vocal output when compared to controls. Deficits in integration would be demonstrated by abnormal performance on the voice-pitch task, similar to what was found in patients with Parkinson's Disease. Specifically, I hypothesized NCP participants to show exaggerated responses (greater magnitudes) and slower reaction times (longer latencies). Together, these data may inform the understanding of psychosis vulnerability more generally and shed light on ways in which researchers can continue to assess MSI among this group.

Method

Participants

A total of 36 participants (11 NCP and 25 controls) aged 17-25 (M = 20.50, SD = 2.04) were recruited at Northwestern University's Adolescent Development and Preventive Treatment (ADAPT) research program. Out of the 36 participants, 11 participants were recruited from the Introduction to Psychology recruitment pool and were given the opportunity to receive course credit. Additionally, an advertisement was placed online through the Northwestern Paid Registry to recruit community members. Twenty-five interested participants called the ADAPT lab and were scheduled for an in-person visit, receiving $10 per hour as compensation.

All participants initially completed the Community Assessment of Psychic Experiences (CAPE; Stefanis et al., 2002) positive symptom dimension which includes questions such as: "*Do you ever think that people can communicate telepathically"* and *"Do you ever feel as if thoughts in your head are being taken away from you?"* (See Appendix for the full

questionnaire.) For each question, participants were instructed to circle an answer on a four-item likert scale including "Never," "Sometimes," "Often," and "Nearly Always." A sum score of the positive symptom domain (20 items total) was obtained and used to determine NCP and control groups. Those scoring less than nine were named controls ($N = 25$), and those scoring greater than nine were named NCP ($N = 11$). This cut-off was determined based on the median split from the first Introduction to Psychology pool of 245 participants, who completed the positive dimension of the CAPE in 2016.

Clinical interviews. Participants then completed a clinical interview, the psychosis module of the Structured Clinical Interview for DSM-IV (SCID; First et al., 2004) in order to rule out formal psychotic disorders. This interview was given by trained assessors including staff and graduate students.

Demographic information. Participants completed the rest of the CAPE (Stefanis et al., 2002) questionnaire, which included negative (e.g., *"Do you ever feel that you are not much of a talker when you are conversing with other people?"*) and depressive (e.g., *"Do you ever feel sad?"*) symptom dimensions in addition to the positive dimension. Participants were also given the Beck Anxiety Inventory (BAI; Beck et al., 1988), a self-report questionnaire that assesses anxiety symptoms, as well as the Wide Range Achievement Test (WRAT; Wilkinson & Robinson, 2006), a tool for assessing general intelligence.

Procedure and Apparatus.

Voice-pitch task. To assess for deficits in the integration of auditory feedback and vocal output, all participants were given the voice-pitch task developed by Kiran and Larson (2001). Prior to the task, hearing problems and other difficulties that may affect prolonged vocalizations were recorded. At the start of the task, participants were instructed to sit in front of a computer

screen, wearing headphones, and to follow directions on the screen. During the ten practice trials, participants saw the word “ahhh” on the computer screen and were asked to vocalize using a natural, conversation voice until they saw the word “stop.” During their vocalizations, they heard the pitch of their voice shifted 100 cents either up or down through the headphones they were wearing. This pitch shift lasted 200 milliseconds. Participants were told to ignore what they heard in their headphones and simply to sustain vocalization until prompted otherwise, keeping a steady volume and pitch. If the participant vocalized too quietly or too loudly for the computer to pick up, the words “too quiet” or “too loud” appeared on the screen. When this happened, the task administrator clarified the instructions for the participant.

The test run included 60 trials and were divided into three blocks of 20. Within each block, ten upward shifts (hearing through the headphones an upward shift in pitch) and ten downward shifts (hearing through the headphones a downward shift in pitch) were presented. The shifts targeted participants’ involuntary (reflexive) reactions to a shift in pitch. The total task time was approximately ten minutes.

Variables of interest. The variables deduced from this task were magnitude and latency. As previously noted, magnitude refers to the extent to which participants deviated from their baseline pitch, measured in cents. Additionally, latency refers to the time it took for participants to respond to the pitch-shift, measured in milliseconds. In other words, latency is the time elapsed between a pitch-shift stimulus and the beginning of a participant’s response.

Data Analysis

SPSS Statistics 25 was used to conduct behavioral analyses. Group differences in continuous and categorical demographic variables were evaluated with independent *t*-tests and Chi-square tests. Independent *t*-tests were employed to examine group differences on voice-pitch

task performance, specifically investigating response magnitudes (pitch deviation from baseline) and response latency (time when response begins). Additionally, exploratory analyses were employed to investigate the relationships between voice-pitch task variables (magnitude and latency) and anxiety (BAI) and NCP symptoms (positive, negative, and depressive symptoms from the CAPE). These bivariate correlations were conducted within the whole group to increase sample size.

Results

Descriptive Statistics

Descriptive statistics are summarized in the Table 1. There were no significant differences between NCP and control participants in demographic characteristics, including age, $t(34) = -.09$, $p = .93$, parental education, $t(34) = -.64$, $p = .53$, anxious symptoms, $t(6.5) = -1.39$, $p = .21$, negative symptoms, $t(34) = -1.42$, $p = .17$, or depressive symptoms, $t(33) = -1.88$, $p = .07$. As expected, the NCP group reported higher scores on the positive dimension of the CAPE compared to the controls, $t(14) = -7.85$, $p \leq .001$, $d = -3.04$. Additionally, the NCP group also scored lower on the WRAT when compared to controls, $t(34) = 2.86$, $p = .007$, $d = .95$. Controlling for WRAT in the analyses did not change the direction or magnitude of the results. Therefore, analyses were conducted without controlling for WRAT.

Group Differences in Voice-Pitch Task

While none of the group differences in the variables of interest (magnitude and latency) were statistically significant, there were noteworthy descriptive differences for each. When matching (raising pitch in response to an upward stimulus), the NCP group exhibited non-significant but slightly larger magnitudes, descriptively speaking, than the control group, $t(34) = -.54$, $p = .59$, $d = .18$. This held when matching to a downward stimulus, as well, $t(34) = -1.07$, p

= .29, *d* = .32. Additionally, when compensating (i.e., lowering pitch in response to an upward stimulus), the NCP group had slightly larger magnitudes than the control group, *t*(34) = -.89, *p* = .38, *d* = .27. This also held when compensating in response to a downward stimulus, *t*(34) = .17, *p* = .40, *d* = .27. See Figure 1.

The NCP group also exhibited slightly longer latencies than the control group, both when matching and when compensating, although again, the results were not significant. Descriptively, when matching, the NCP group demonstrated longer latencies for upward stimuli, *t*(34) = -1.03, *p* = .31, *d* = .34 as well as downward stimuli, *t*(34) = 0.72, *p* = .48, *d* = .25. When compensating, the NCP group demonstrated longer latencies as well, both for upward stimuli, *t*(34) = .69, *p* = .13, *d* = .56 and downward stimuli, *t*(34) = -.40, *p* = .69, *d* = .15. See Figure 2.

Associations Between Voice-Pitch Task Variables and Clinical Symptoms

Exploratory analyses were conducted to investigate relationships between voice-pitch task variables and clinical symptoms (BAI scores, CAPE positive symptoms, CAPE negative symptoms, and CAPE depressive symptoms). No significant correlations were found. Results are summarized in Table 2.

Discussion

The results of the present study, although nonsignificant, offer preliminary descriptive findings suggesting that NCP individuals may demonstrate abnormal performance on the voice-pitch task. Because the task targets multisensory integration, specifically the integration of auditory feedback and vocal output, it is possible that NCP individuals possess a deficit in the ability to integrate the two. However, more participants are needed before definitive conclusions can be made. Descriptively, NCP participants showed greater magnitudes and longer latencies with small to medium effect sizes. The greater magnitudes suggest a pattern towards exaggerated

responses, consistent with the findings among patients with Parkinson's Disease (Kiran & Larson, 2001). Additionally, the longer latencies could be a sign that the NCP group requires more time to "detect" the mismatch, because more time elapsed between the pitch-shift stimulus and the beginning of their reflexive responses. Taken together, these results may hint towards a possible deficit in motor control among NCP groups when utilizing feedback from their own voice (Kiran & Larson, 2001). However, the noted interpretations of descriptive data are speculative, and again, more participants are needed to better understand task performance among each group. Increasing sample size would increase the likelihood of detecting small effects that may otherwise be missed without such statistical power.

Based on preliminary findings, it is possible that the descriptive differences in means in the variables of interest could be reflective of MSI deficits, specifically the sensorimotor integration of auditory feedback and vocal output. MSI deficits have been observed among patients with schizophrenia (Williams et al., 2010; Stekelenburg et al., 2013; Jacobson et al., 2010), although this specific form (auditory feedback and vocal output) has not been explored anywhere on the continuum. Given that NCP is associated with psychosis risk factors, it is possible that the known MSI deficits in schizophrenia may also underlie abnormal performance on the voice-pitch task in NCP. Identifying a possible shared vulnerability factor is an area of particular interest when considering the prevention and intervention of psychopathology. As previously noted, the goal of studying NCP is not to predict psychosis, but rather to explore variations that may exist on the continuum and relate to the onset of psychosis (Jacobson et al., 2010; Polanczyk et al., 2010). The use of a voice-pitch task in the present study highlights the feasibility of using this task among NCP to explore sensorimotor integration. This is important

for research that continues to explore possible shared MSI vulnerabilities on the psychosis continuum.

Additionally, one of the motivating factors for conducting this study was to extend the research from Kiran and Larson's (2001) study in Parkinson's Disease to the psychosis continuum. It is interesting that, although nonsignificant, the average magnitude and latency responses among the NCP group align with what was observed in patients with Parkinson's Disease. Other research has investigated abnormalities in Parkinson's disease, such as movement and gestures, and many of these abnormalities overlap with the psychosis continuum (Dean et al., 2014). Preliminary findings from the present study contribute to this work and may shed light on shared integration pathways (specifically between auditory feedback and vocal output) between Parkinson's Disease and NCP.

Further, I employed exploratory analyses to investigate relationships between task variables and anxious, positive, negative, and depressive symptoms. While I did not observe any significant relationships, future research may benefit from investigating possible links. For instance, alogia is a negative symptom that refers to the absence of speech. Additional research may explore a possible link between alogia and the integration of auditory feedback with vocal output, as both factors have implications for vocalization. Determining links with symptoms may be a useful way to tease apart questions relating to the etiology of MSI deficits.

While there are several strengths to this study, particularly its novelty, there are important limitations to consider, as well. Most notably, the current sample size is not sufficient, and more participants are needed in order to truly understand sensorimotor integration among NCP groups. Although I was able to detect small to medium effect sizes, more participants may detect smaller effects. Furthermore, while the direction of the preliminary data mimic previous findings within

the literature (Kiran & Larson, 2001), none of the findings were statistically significant, so the interpretations throughout must be read with caution (the intention of these interpretations are to offer a potential perspective of MSI and NCP for future work). The main priority is to continue recruiting additional participants until recruitment goals are met.

Additionally, future research with more participants would benefit from longer duration stimuli (to investigate voluntary responses) as well as more sophisticated types of analyses that incorporate cognitive measures and interactions among variables. Likewise, extending this task to other at-risk and psychosis populations may be useful in investigating sensorimotor integration and varying states of psychosis symptoms. Similarly, following the data of this study longitudinally may provide insights into task performance and disease progression. Together, this task may help identify a potential vulnerability marker; however, more work is needed to address some of the remaining unknown questions.

The present study is the first to investigate the integration of auditory feedback and vocal output along the psychosis continuum. Although preliminary, these data may provide information as to the underlying vulnerabilities that occur along the psychosis continuum and contribute to the understanding of the pathogenesis of psychosis. As noted, schizophrenia is not necessarily a dichotomous diagnosis, and investigating factors along the continuum may better help explain the etiology of psychosis than when restricted to the far end of the spectrum (Verdoux & Van Os, 2002). Ultimately, identifying certain characteristics along the continuum may allow for effective intervention and is crucial for understanding the prevention of psychopathology.

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Table 1
Demographic Information

	NCP	Control	Total	Statistic	*P*
Age					
Mean	20.55	20.48	20.50	$t(34) = -.09$	.93
(SD)	(1.86)	(2.14)	(2.04)		
Gender					
Male	3	7	10		
Female	8	18	26		
Total	11	25	36	$\chi 2(1) = .002$	.96
Parent Education (years)					
Mean	16.82	16.12	16.33	$t(34) = -.64$	.53
(SD)	(3.63)	(2.70)	(2.98)		
BAI Scores					
Mean	20.29	8.57	11.50	$t(6.5) = -1.39$	.21
(SD)	(21.95)	(7.70)	(13.33)		
CAPE Positive Symptoms					
Mean	13.00	3.72	6.56	$t(14) = -7.85$	<.001
(SD)	(3.58)	(2.42)	(5.15)		
CAPE Negative Symptoms					
Mean	11.91	9.44	10.19	$t(34) = -1.42$	.17
(SD)	(4.76)	(4.84)	(4.89)		
CAPE Depressive Symptoms					
Mean	7.90	5.56	6.23	$t(33) = -1.88$	.07
(SD)	(3.73)	(3.18)	(3.46)		
WRAT Scores					
Mean	102.91	119.56	114.47	$t(34) = 2.86$	.007
(SD)	(20.85)	(13.61)	(17.66)		

Table 1. Note. Parental educatio
scores are the sum of all items or
depressive) were calculated by ta

Table 2
Associations Between Voice-Pitch Task Variables and Clinical Symptoms

			BAI Sum	CAPE Pos	CAPE Neg	CAPE Dep
Up_Up Magnitude			*r* = -.252 *p* = .195 *N* = 28	*r* = -.097 *p* = .572 *N* = 36	*r* = -.164 *p* = .340 *N* = 36	*r* = -.159 *p* = .361 *N* = 35
Up_Up Latency			*r* = .235 *p* = .228 *N* = 28	*r* = .135 *p* = .431 *N* = 36	*r* = .064 *p* = .710 *N* = 36	*r* = -.125 *p* = .475 *N* = 35
Up_Down Magnitude			*r* = -.160 *p* = .416 *N* = 28	*r* = .010 *p* = .956 *N* = 36	*r* = -.001 *p* = .996 *N* = 36	*r* = -.072 *p* = .679 *N* = 35
Up_Down Latency			*r* = .309 *p* = .110 *N* = 28	*r* = .220 *p* = .197 *N* = 36	*r* = -.16 *p* = .926 *N* = 36	*r* = -.067 *p* = .704 *N* = 35
Down_Up Magnitude			*r* = -.312 *p* = .106 *N* = 28	*r* = -.031 *p* = .856 *N* = 36	*r* = -.272 *p* = .108 *N* = 36	*r* = -.153 *p* = .380 *N* = 35
Down_Up Latency			*r* = .080 *p* = .678 *N* = 28	*r* = .063 *p* = .716 *N* = 36	*r* = .174 *p* = .309 *N* = 36	*r* = .127 *p* = .466 *N* = 35
Down_Down Magnitude			*r* = -.167 *p* = .394 *N* = 28	*r* = .025 *p* = .883 *N* = 36	*r* = .042 *p* = .808 *N* = 36	*r* = -.067 *p* = .701 *N* = 35
Down_Down Latency	*r* = .334 *p* = .073 *N* = 28	*r* = .184 *p* = .281 *N* = 36	*r* = .184 *p* = .283 *N* = 36	*r* = -.061 *p* = .726 *N* = 35		

Table 2. Note. Voice-pitch task variables are represented by StimulusDirection_ResponseDirection. Up_Up and Down_Down refer to matching. Up_Down and Down_Up refer to compensating.

Figure 1

Magnitude Group Differences Between NCP and Control Participants

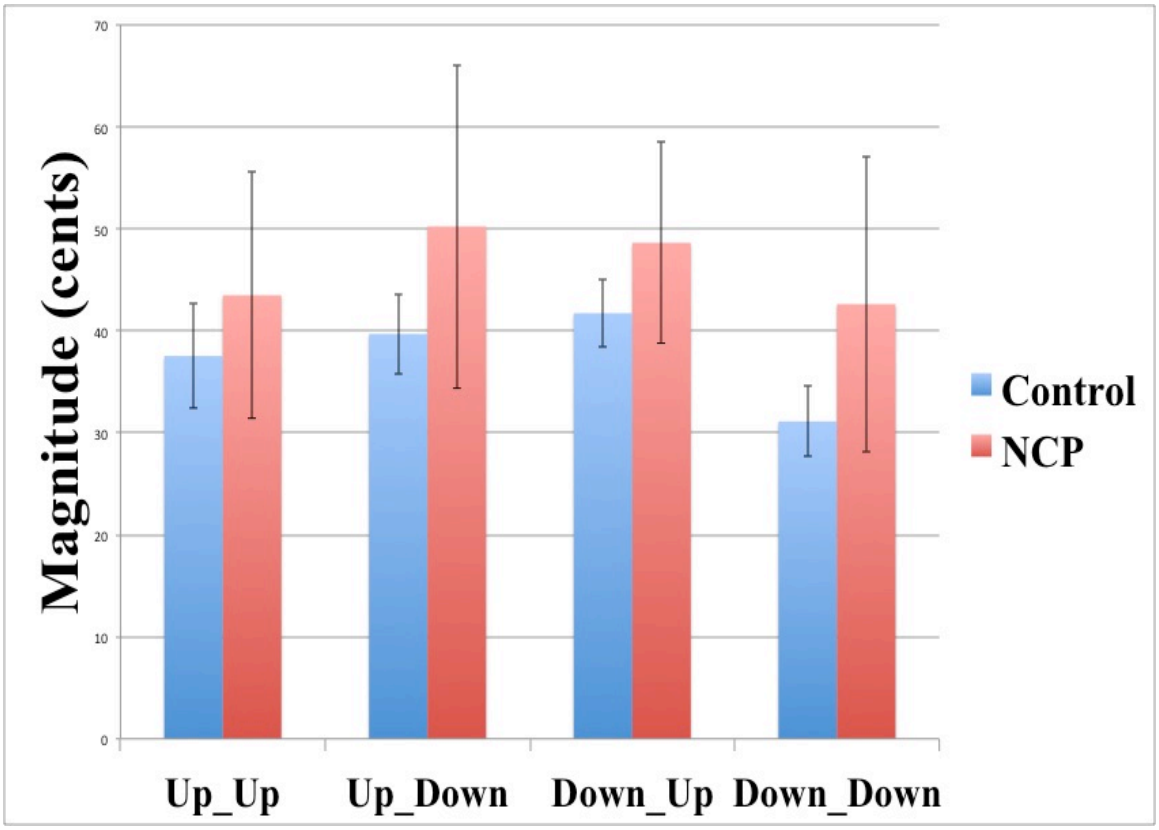

Fig. 1. Magnitude group differences between NCP and control participants on the voice-pitch task. Note. Magnitude group differences between NCP and control participants on the voice-pitch task. The four clusters represent StimulusDirection_ResponseDirection. Up_Up and Down_Down refer to matching. Up_Down and Down_Up refer to compensating. Error bars indicate mean standard error.

Figure 2

Latency Group Differences Between NCP and Control Participants

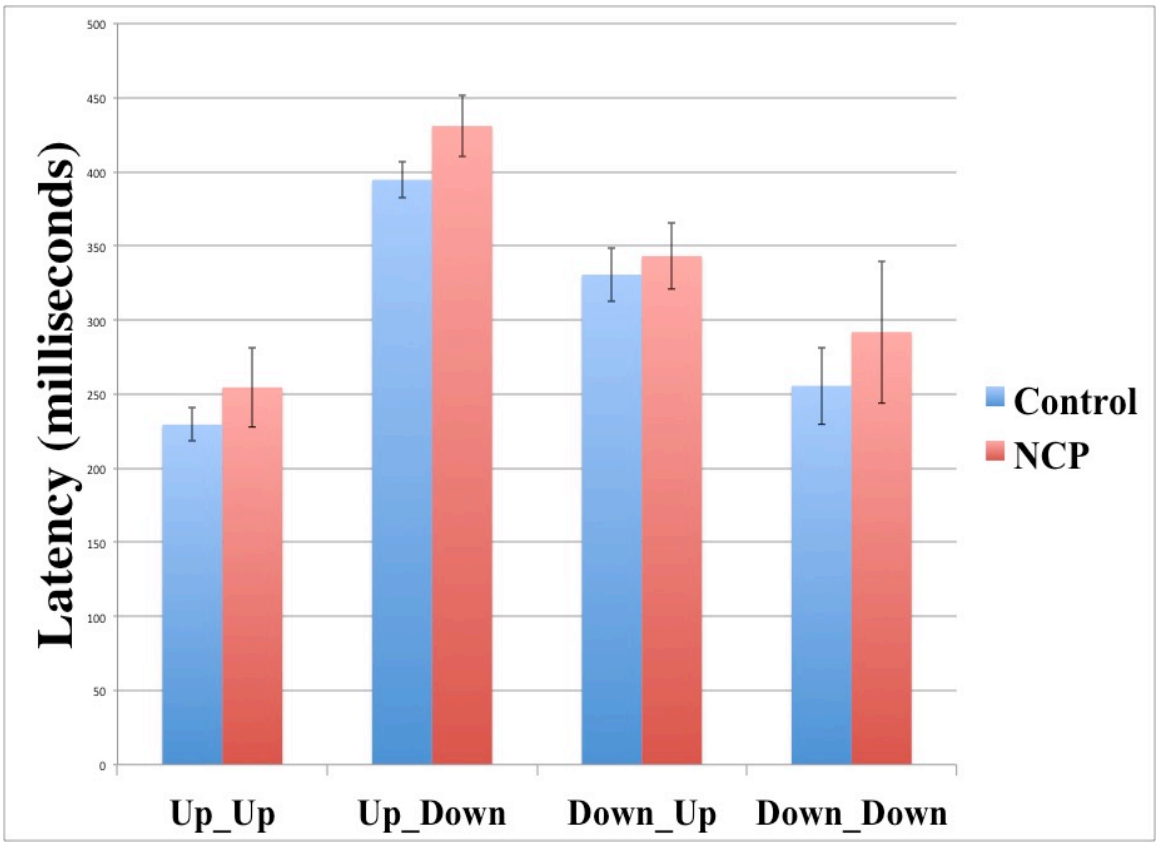

Fig. 2. Latency group differences between NCP and control participants on the voice-pitch task. Note. Latency group differences between NCP and control participants on the voice-pitch task. The four clusters represent StimulusDirection_ResponseDirection. Up_Up and Down_Down refer to matching. Up_Down and Down_Up refer to compensating. Error bars indicate mean standard error.

Appendix

Community Assessment of Psychic Experiences (CAPE; Stefanis et al., 2002)

Response scale:

1 = *never;* 2 = *sometimes;* 3 = *often;* 4 = *nearly always*

Each question includes the following follow-up question:

If you ticked "sometimes," "often," or "nearly always," please indicate how distressed you are by this experience:

1 = *not distressed;* 2 = *a bit distressed;* 3 = *quite distressed;* 4 = *very distressed*

1. Do you ever feel sad?
2. Do you ever feel as if people seem to drop hints about you or say things with a double meaning?
3. Do you ever feel that you are not a very animated person?
4. Do you ever feel that you are not much of a talker when you are conversing with other people?
5. Do you ever feel as if things in magazines or on TV were written especially for you?
6. Do you ever feel as if some people are not what they seem to be?
7. Do you ever feel as if you are being persecuted in some way?
8. Do you ever feel that you experience few or no emotions at important events?
9. Do you ever feel pessimistic about everything?
10. Do you ever feel as if there is a conspiracy against you?
11. Do you ever feel as if you are destined to be someone very important?
12. Do you ever feel as if there is no future for you?
13. Do you ever feel that you are a very special or unusual person?
14. Do you ever feel as if you do not want to live anymore?
15. Do you ever think that people can communicate telepathically?
16. Do you ever feel that you have no interest to be with other people?
17. Do you ever feel as if electrical devices such as computers can influence the way you think?
18. Do you ever feel that you are lacking in motivation to do things?
19. Do you ever cry about nothing?
20. Do you believe in the power of witchcraft, voodoo or the occult?
21. Do you ever feel that you are lacking in energy?
22. Do you ever feel that people look at you oddly because of your appearance?
23. Do you ever feel that your mind is empty?
24. Do you ever feel as if the thoughts in your head are being taken away from you?
25. Do you ever feel that you are spending all your days doing nothing?
26. Do you ever feel as if the thoughts in your head are not your own?
27. Do you ever feel that your feelings are lacking in intensity?
28. Have your thoughts ever been so vivid that you were worried other people would hear them?
29. Do you ever feel that you are lacking in spontaneity?
30. you ever hear your own thoughts being echoed back to you?

31. Do you ever feel as if you are under the control of some force or power other than yourself?
32. Do you ever feel that your emotions are blunted?
33. Do you ever hear voices when you are alone?
34. Do you ever hear voices talking to each other when you are alone?
35. Do you ever feel that you are neglecting your appearance or personal hygiene?
36. Do you ever feel that you can never get things done?
37. Do you ever feel that you have only few hobbies or interests?
38. Do you ever feel guilty?
39. Do you ever feel like a failure?
40. Do you ever feel tense?
41. Do you ever feel as if a double has taken the place of a family member, friend or acquaintance?
42. Do you ever see objects, people or animals that other people cannot see?