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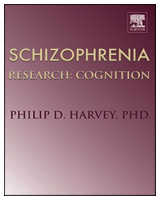

Negative auditory hallucinations are associated with increased activation of the defensive motivational system in schizophrenia

Anna R. Knippenberg, Sabrina Yavari, Gregory P. Strauss *

Department of Psychology, University of Georgia, United States of America

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ABSTRACT

Auditory hallucinations (AH) are the most common symptom of psychosis. The voices people hear make comments that are benign or even encouraging, but most often voices are threatening and derogatory. Negative AH are often highly distressing and contribute to suicide risk and violent behavior. Biological mechanisms underlying the valence of voices (i.e., positive, negative, neutral) are not well delineated. In the current study, we examined whether AH voice valence was associated with increased activation of the Defensive Motivational System, as indexed by central and autonomic system response to unpleasant stimuli. Data were evaluated from two studies that used a common symptom rating instrument, the Psychotic Symptom Rating Scale (PSY-RATS), to measure AH valence. Participants included outpatients diagnosed with SZ. Tasks included: Study 1: Trier Social Stress Task while heart rate was recorded via electrocardiography ($N = 27$); Study 2: Passive Viewing Task while participants were exposed to pleasant, unpleasant, and neutral images from the International Affective Picture System (IAPS) library while eye movements, pupil dilation, and electroencephalography were recorded ($N = 25$). Results indicated that negative voice content was significantly associated with: 1) increased heart rate during an acute social stressor, 2) increased pupil dilation to unpleasant images, 3) higher neural reactivity to unpleasant images, and 4) a greater likelihood of having bottom-up attention drawn to unpleasant stimuli. Findings suggest that negative AH are associated with greater Defensive Motivational System activation in terms of central and autonomic nervous system response.

1. Introduction

Auditory hallucinations – the most common symptom of psychosis – often involve hearing threatening voices (i.e., negative auditory verbal hallucinations) which can be distressing, increasing suicide risk, violent behavior, and disability in psychotic disorders (David, 1999; Baethge et al., 2005; Fujita et al., 2015; Larøi et al., 2019). Medications and psychosocial interventions are minimally effective at changing the valence and distress of hallucinations, and therefore reflect an important treatment target for many individuals with psychosis (Dellazizzo et al., 2022; Jenner and Van De Willige, 2001).

Limited progress in changing hallucination valence may be due to a limited mechanistic understanding. One approach for exploring mechanisms underlying AVH valence comes from the field of behavioral neuroscience. The "Defense Cascade Model" (Bradley et al., 2001) describes how stimuli of differing valence and arousal levels initiate a patterned cascade of physiological changes in the autonomic and central nervous systems to promote defensive responding. As stimulus arousal and unpleasantness increases, it signals an imminent threat, leading to physiological changes (e.g., cardiac acceleration) to prepare the organism for defense. In nonhuman animals, defensive responses include freezing to escape predator detection (Kozlowska et al., 2015). In humans, defensive responses activated in the context of perceived danger include a racing heart or sweaty palms when exposed to unpleasant stimuli (e.g., violent images). Therefore, human laboratory-based paradigms that measure the physiological responses (e.g., heart rate) to unpleasant versus neutral stimuli of differing arousal levels can be used as biomarkers of the defense cascade model. Negative stimuli of differing arousal and valence levels that result in varied physiological changes can be measured as a proxy of defensive responding.

The body's response to varying stimulus arousal levels can be measured using electrocardiology (heart rate), electrodermal activity (skin conductance), and electromyography (startle response) (Löw et al., 2015; Schupp et al., 2004; Kozlowska et al., 2015). These measures show different response patterns, such as initial deceleration and then acceleration of heart rate, decline followed by potentiation in startle

* Corresponding author at: University of Georgia, Department of Psychology, 125 Baldwin St., Athens, GA 30602, United States of America.
E-mail address: gstrauss@uga.edu (G.P. Strauss).

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response, and gradual increase in electrodermal activity (Bradley and Lang, 2000; Bradley et al., 2001; Davis, 1992). Other physiological measures, such as pupillometry, eye tracking, and electroencephalography (EEG), are also used to assess defensive responding. Pupillometry reflects emotional arousal (Carter and Luke, 2020), while eye tracking measures attention to threats (Nummenmaa et al., 2006). EEG monitors cortical pyramidal neuron activation and can indicate emotional reactivity and selective attention (Hajcak et al., 2010).

Negative emotion abnormalities in psychiatric disorders are associated with dysfunctional defense activation suggesting aberrant nervous system response to threat. For example, individuals with PTSD or borderline personality disorder (BPD) display heightened emotional reactivity to unpleasant stimuli compared to healthy controls (Selby et al., 2009; Schalinski, 2013), whereas individuals with psychopathy, a disorder marked by decreased negative emotional responses and lack of empathy, display reduced autonomic and neurophysiological response to unpleasant stimuli (Patrick, 2022). Negative emotion abnormalities are commonly observed in psychotic disorders, yet it is unclear whether abnormal defensive responding is associated with negative AVH (Horan et al., 2008).

Based on the defense cascade model (Bradley et al., 2001) and studies on other psychiatric groups (Lang et al., 1998; Selby et al., 2009), we evaluated the hypothesis that greater negative valence in AVH would be associated with increased defensive responding to emotional stimuli presented in the laboratory. We hypothesized that negative AVH would be associated with 1) increased heart rate during an acute social stressor, 2) increased pupil dilation to unpleasant images, 3) higher neural reactivity to unpleasant images, and 4) greater likelihood of having bottom-up attention drawn to unpleasant stimuli. The study evaluated outpatients with psychotic disorders as they present with a wide range of AVH severity and negative valence in their AH. Healthy controls were not included due to lack of experiencing AH, lack of negative valence in sub-threshold psychotic-like experiences, and the specificity of the question at hand regarding biological correlates of AVH valence in those with psychotic disorders.

2. Methods

2.1. Participants

Participants included 52 outpatients with schizophrenia or schizoaffective disorder (Study 1 $n = 27$; Study 2 $n = 25$; Table 1) recruited from local mental health centers. Participants were clinically stable in terms of no changes in medication type or dosage within 1 month of testing, no recent hospitalizations, and no current substance use disorders. Diagnosis was established based on medical records and the Structured Clinical Interview for DSM-IV-TR (SCID-IV; First et al., 2001). BPRS scores suggest that most patients were mild to moderately symptomatic on average. Participants provided written informed consent approved by the local Institutional Review Board.

2.2. Procedures

Data were collected across two studies examining emotional reactivity in SZ. In Study 1, participants completed a mental arithmetic task to induce psychological stress while electrocardiography (ECG) was recorded. In Study 2, participants passively viewed images from the International Affective Picture System Library (Bradley and Lang, 2007) varying in arousal and valence levels (unpleasant, pleasant, neutral) while eye tracking, pupillometry, and EEG were recorded ($N = 25$). After a clinical interview, each participant was rated on the Psychotic Symptom Rating Scale by a trained rater (PSY-RATS; Haddock et al., 1999).

Table 1
Demographic and clinical characteristics of Study 1 and Study 2.

Study 1	SZ ($N = 27$)
	M (SD)
Age	41.8 (10.5)
Personal education	13.2 (1.9)
Parental education	13.5 (2.6)
% Female	39.3 %
Race	
% White	78.6 %
% Black	3.6 %
% Hispanic	7.1 %
% Asian	0.0 %
% Other	7.1 %
BPRS positive	3.21 (1.27)
BPRS negative	2.23 (1.32)
BPRS disorganized	2.25 (0.89)
BPRS total	47.54 (11.1)

Study 2	SZ ($N = 25$)
	M (SD)
Age	43.0 (10.9)
Personal education	13.3 (1.9)
Parental education	13.5 (2.2)
% female	28.6 %
Race	
% White	72.0 %
% Black	16.0 %
% Hispanic	4.0 %
% Asian	4.0 %
% other	4.0 %
BPRS positive	3.11 (1.14)
BPRS negative	2.14 (1.32)
BPRS disorganized	2.11 (0.86)
BPRS total	46.21 (9.87)

Note. SZ = schizophrenia.

2.3. Measures

2.3.1. Study 1: Trier Social Stress Test

Upon arrival at the laboratory, participants rested for 45 min while heart rate was monitored for stress levels to return to baseline before proceeding. After resting, participants completed the mental arithmetic task of the Trier Social Stress Test (TSST) while ECG data were collected using a wireless Biopac MP150 system and standard three-electrode (lead II) setup. Data were sampled at 1000 Hz, processed/analyzed via Acqknowledge v4.2 software, and visually inspected for artifacts to manually correct/reject. Beats per minute were calculated by averaging 30-s epochs throughout the task compared to baseline, resulting in a stress reactivity BPM difference score.

2.3.2. Study 2: Passive Viewing Task

Passive viewing tasks were administered separately to collect eye tracking/pupillometry and EEG data. In both tasks, a fixation cross was presented (1000 ms) followed by an IAPS image (3000 ms; 19″ monitor, 1280 × 1024 resolution; 70 cm viewing distance; 32.6° x 21.0° visual angle). Ninety stimuli (30 pleasant, 30 unpleasant, 30 neutral) differing in valence/arousal ratings based on condition were presented in random order. By design, pleasant and unpleasant conditions did not differ in arousal. No conditions differed in lower-level visual features (e.g., complexity, luminance, red/green/blue saturation).

2.3.3. EEG recording, data reduction, and measurement

EEG and eye tracking procedures are consistent with prior studies (Foti et al., 2009; Hajcak et al., 2012; Wynn et al., 2010; Schupp et al., 2004; Sabatinelli et al., 2013; Strauss et al., 2013). EEG signal was recorded using a 64 Ag/AgCl electrode elastic cap (BrainVision actiCap

model) with an online right mastoid reference and re-referenced offline using average left and right mastoid electrodes (Hajcak et al., 2012). Eye movements measured via horizontal electrooculogram (EOG) were recorded as the voltage between electrodes placed lateral to the external canthi. Eye blinks measured via vertical EOG were recorded from electrodes above and beneath the left eye. Electrode impedance levels were maintained below 15KΩ. EEG and EOG signals were amplified by a BrainVision actiCHamp amplifier (5000 gain; 0.05 to 100 Hz band-pass filter; 60 Hz notch filter). Amplified signals were digitized at 500 Hz and averaged offline.

Matlab EEGLAB and ERPLAB toolboxes were used for signaling processing (Lopez-Calderon and Luck, 2014). A high-pass filter of 0.01 was applied offline to continuous EEG signals to prevent LPP attenuation (Hajcak et al., 2012). EEG signals containing excessive muscle activity (i.e., EMG) were manually removed, and an Independent Component Analysis (ICA) was conducted to identify/correct eye blink activity.

ICA-corrected signals were used to construct ERP variables. EEG signal was divided into epochs 200 ms pre-stimulus onset to 3000 ms post-stimulus onset. Epochs were baseline corrected using a 200 ms pre-stimulus period. LPP computed as the mean amplitude of centroparietal electrodes Cz, Pz, CP1, and CP2 from 300 to 3000 ms.

2.3.4. Eye tracking and pupillometry recording

Eye tracking was recorded from the right eye, using an SR Research Eyelink 1000 desk-mounted system (2000 Hz), and calibrated before each trial. Pupil data were collected using Eyelink 1000 set to measure pupil diameter values between approximately 1–3 mm. Participants were seated with their head in a chin-and-forehead rest to decrease motion artifacts (19" Dell monitor P190S; 70 cm viewing distance; 60 Hz refresh rate). IAPS stimuli were displayed for 3000 ms each while eye movements and pupil dilation were recorded. One-second fixation on a cross proceeded each IAPS image.

Prior to Study 2, pilot studies were conducted to identify (pilot study 1) and validate (pilot study 2) emotional areas of interest (E-AOIs) of IAPS images (see Bebko et al., 2011 or Strauss et al., 2016 for details). E-AOIs were used to compute first fixation time as the primary eye tracking dependent variable.

In pilot study 1, twelve undergraduate students with normal or corrected-to-normal vision and no history of psychiatric/neurological illness passively viewed unpleasant, pleasant, and neutral IAPS images (5 s per stimuli) while eye tracking was recorded (33.02° x 20.64° visual angle). Participants rated subjective arousal for each trial (i.e., How positive/negative/calm or excited does the picture make you feel)? Aggregate heat maps were constructed using average fixation location and times. Participants were then given unlimited time to draw shapes over the most arousing areas of each picture. *E*-AOIs were identified by combining aggregate heat maps from passive IAPS viewing and shapes from the freehand draw. Resulting heat maps represented an average fixation location and time for each image. Average placement within the shapes was turned into a two-dimensional matrix merged with eye tracking and shape placement representations, thus determining *E*-AOIs. All E-AOI were: 1) identified via eye tracking maps indicating relevant arousal of the area, and 2) rated as negative by at least 50 % of participants. E-AOIs were verified by two experimenters who reported no discrepancies.

In pilot study 2, a separate group of twelve undergraduate students were recruited to assess E-AOIs validity obtained in pilot study 1. E-AOIs most commonly identified using combined aggregates were blacked out to measure whether valence/arousal ratings differed from the original images. Valence/arousal ratings for modified images were lower, thus confirming E-OAIs validity (i.e., accurately representing arousing areas of the scenes).

First fixation time was calculated as the primary dependent variable on each trial, as a measure of motivated attention and bottom-up attention of arousing stimuli. Values reflect time (ms) at which first fixation entered an E-AOI. Faster times reflect greater bottom-up competitive advantage for emotional stimuli (Nummenmaa et al., 2006).

2.3.5. Psychotic Symptom Rating Scale

Auditory hallucinations were measured via the auditory hallucination (AH) subscale of the Psychotic Symptom Rating Scale (PSY-RATS; Haddock et al., 1999), an 11-item scale assessing varying dimensions of auditory hallucinations over the past week. A clinical rater trained to reliability standards rated each participant on the PSY-RATS. Each item is rated on a five-point Likert scale (0–4). Items 6–8 from the PSY-RATS AH subscale were included in analyses as a measure of the amount of negative content (6), degree of negative content (7), and amount of distress (8).

2.4. Data analysis

Bivariate Pearson's correlations were used to test the hypothesis that negative hallucination valence was associated with greater activation of the defensive motivational system. AVH were measured as the average rating of PSY-RATS items 6–8. Measures of autonomic and neurophysiological activation included heart rate during the TSST, 1st fixation time during eye tracking passive viewing, pupil dilation during eye tracking passive viewing, and the LPP ERP component as measured via EEG during passive viewing.

3. Results

In Study 1 and 2, participant AVH as measured via average PSY-RATS ratings (items 6–8) ranged from 0 to 4 (Study 1: M = 1.33, SD = 1.48; Study 2: M = 1.00, SD = 1.33). Correlations indicated that greater negative valence in AVH was associated with greater defensive motivational system engagement to unpleasant stimuli on measures of heart rate, eye tracking, pupil dilation, and neurophysiological response on the LPP (see Table 2).

Greater negatively valenced AVHs were also significantly associated with responses to pleasant stimuli, including: longer first fixation times and greater pupil dilation. Additionally, the associations between task variables and clinical measures of negative symptoms, disorganization, and total symptoms on the BPRS were generally nonsignificant (except for faster first fixation time to pleasant stimuli and more severe disorganization; see Supplemental Tables 1 and 2 for exploratory correlations conducted to contextualize the results).

4. Discussion

This study examined the biological correlates of negative auditory verbal hallucinations (AVH). We evaluated the hypothesis that negative AVH in individuals with SZ are associated with increased activation of the defensive motivational system (Bradley et al., 2001). Results

Table 2
Correlations between negative emotion reactivity task variables and auditory hallucination valence.

Task	DV	PSY-RATS AH negative valence
Study 1 (n = 27)		
TSST Mental Arithmetic Task	Heart rate (bpm)	0.39*
Study 2 (n = 26)		
Passive Viewing (Eye Tracking)	1st Fixation Time	−0.57*
Passive Viewing (Pupillometry)	Pupil Dilation	0.55**
Passive Viewing (ERP)	Late Positive Potential	0.45*

Note. TSST = Trier Social Stress Task; ERP = Event-related potential; PSY-RATS = Psychotic Symptom Rating Scale; AH = Auditory hallucination.

supported this hypothesis such that negative AVH were associated with greater mobilization of the defense cascade as indexed by autonomic and central system responses to unpleasant stimuli. Negative voice content was significantly associated with: 1) increased heart rate during an acute social stressor, 2) increased pupil dilation to unpleasant images, 3) higher neural reactivity to unpleasant images, and 4) greater bottom-up attention drawn to arousing aspects of unpleasant stimuli.

Several interpretations are plausible regarding these correlational results. First, AVH may lead to heightened activation via a stimulus-driven effect in which hallucinations precede defensive dysfunction. Frequent exposure to unpleasant stimuli as negative AVH may *lead to* dysfunction within the system by way of chronic ANS and CNS activation. This hypothesis is supported by evidence that negative AVH content is precipitated by acute and chronic stressors, such as trauma (Bentall et al., 2012; Read and Argyle, 1999; Read et al., 2003; Morrison and Petersen, 2003; Offen et al., 2003; Shevlin et al., 2007; Rosen et al., 2018; Corstens and Longden, 2013). Stressors may influence voice content such that voices mimic the sound of an abuser or contain themes of guilt, shame, or powerlessness (Corstens and Longden, 2013; Hardy et al., 2005; Birchwood et al., 2004). If precipitating stressors such as trauma have a causal relationship with negative AVH, it poses the question of whether such stressors lead to nervous system overactivation driving the development of negative AVH and heightened physiological responsivity to emotional stimuli. Individuals who are also genetically predisposed to nervous system dysfunction may be impacted by a so-called "2nd-hit" upon experiencing early childhood adversity similar to what has been proposed in PTSD development (i.e., Two-Hit Model of PTSD; Georgopoulos et al., 2018). Despite the relationship between negative AVH and childhood adversity, not all experiences of negative voice content are preceded by abuse or trauma, thus offering only one explanation for negative AVH phenomenology. A genetic predisposition for chronic ANS and CNS overactivation may provide another. As is observed in other disorders (e.g., PTSD), an individual may receive both hits of a genetic vulnerability and early childhood insults during development which lead to later onset of negative AVH.

Alternatively, defensive motivational system dysfunction may *lead to* negative AVH either through chronic HPA axis dysfunction associated with neurobiological features of SZ itself, a genetic predisposition to ANS and CNS dysfunction, the experience of early childhood trauma, or a combination of some or all of these pathways. Both explanations may occur within the same individual – one in which AVH *cause* defensive overactivation and another in which chronic nervous system overactivation *causes* AVH.

Research is needed to analyze person-level variability and how some mechanisms might explain negative AVH among some people but not others. Regardless of which explanation is correct, it is likely that a combination of genetic and developmental factors, as well as the stress of experiencing negative AVH, contributes to the onset and maintenance of negative content. New cluster analyses (McCarthy-Jones et al., 2014) on auditory hallucinations phenomenology support the hypothesis that different neurocognitive processes underlie hallucinations as indicated by quantifiable subtypes, suggesting the existence of multiple profiles.

Limitations should be considered when interpreting the current results. First, small sample sizes limit generalizability of the findings. Although similar results across two studies strengthen reliability, large replication studies with multiple emotional reactivity measures are needed. Second, clinically stable outpatients with SZ were evaluated resulting in milder hallucination severity than an inpatient sample. Research should include inpatient populations, specifically first episode with higher rates of negative voices as well as youth at clinical high risk for psychosis (CHR) for whom negative voices may predict psychosis conversion (Misiak et al., 2016; Marshall et al., 2019). Third, the role of medication was not clear since all patients were prescribed antipsychotics. Research should examine the influence of antipsychotic medications on autonomic responses in medicated versus unmedicated groups. Fourth, although EEG has excellent temporal resolution, poor spatial resolution limits conclusions regarding neuroanatomical substrates. While results suggest increased neurophysiological response to unpleasant stimuli is associated with negative valence AVH, brain regions or subregions involved in this relationship cannot be inferred. Finally, given that both studies were cross-sectional, longitudinal studies are needed to examine temporal associations between AVH valence and defensive responding. CHR may be a valuable group for evaluating this association, as voice valence is highly variable in this population, making it possible to observe temporal precedence among relationships as the illness progresses among those who develop a psychotic disorder. Alternatively, ecological momentary assessment (EMA) and ambulatory psychophysiology may allow observations of temporal associations on a shorter time scale as they relate to state fluctuations.

Despite limitations, findings support the association between negative auditory hallucinations and greater activation of the defensive motivational system as measured by central and autonomic nervous system response. Operating in a "fight or flight" state of preparedness often confers a competitive advantage for the state of defense necessary to respond to threats within the environment. However, in the case of psychosis, it is likely maladaptive since threatening stimuli are not actually present – rather, the threat originates from a false perception (i.e., hallucination). The defensive system is not designed to handle unpredictable false perceptions of negative stimuli; therefore, it is not surprising that reactions to negative AVH can produce extreme behaviors (e.g., withdrawal, isolation, aggression, violence). Research elucidating the temporal nature of this association is needed to identify targets for pharmacological, psychosocial, or neuro/psychophysiological feedback treatments.

Current interventions have focused on changing individuals' beliefs about the voices or regulating emotions after exposure to unpleasant stimuli (McCarthy-Jones, 2017). Still, there is a need for interventions that target negative auditory hallucinations before they occur. A limited number of interventions aim to do so either by altering the negative content of the voices themselves, decreasing the frequency of negative AVH, or increasing positive self-schema (e.g., Compassion Focused Therapy, Group Person-Based Cognitive Therapy, Competitive Memory Training, Assertiveness Training, AVATAR therapy) (Mayhew and Gilbert, 2008; Chadwick, 2006; Van Der Gaag et al., 2012; Hayward et al., 2009; Leff et al., 2014). Shifting hallucination valence to become less negative or more neutral would be a meaningful clinical outcome for many individuals and could constitute a novel treatment target, particularly in cases in which removing hallucinations altogether has not been feasible. Antecedent approaches, in conjunction with digital health tools, may be useful for delivering existing interventions at the most optimal time.

CRediT authorship contribution statement

Anna R. Knippenberg: Writing – review & editing, Writing – original draft, Visualization, Formal analysis. **Sabrina Yavari:** Writing – review & editing, Formal analysis. **Gregory P. Strauss:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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Declaration of competing interest

Authors have no conflicts of interest related to the current research.

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Appendix A. Supplementary data

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