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Schizophrenia and Corollary Discharge: A Neuroscientific Overview and Translational Implications

Rujuta Parlikar*, Anushree Bose*, Ganesan Venkatasubramanian

WISER Program, Department of Psychiatry, National Institute of Mental Health and Neurosciences, Bangalore, India

Corollary discharge mechanism refers to the suppression of sensory consequences of self-generated actions; a process that serves to distinguish between self and non-self based on discrimination of origination of action. It explains, say for example, why we cannot tickle ourselves. This review discusses how corollary discharge model is an essential neural integration mechanism central to the motor functioning of animal kingdom. In this article, research conducted in the field of corollary discharge has been reviewed to understand the neuroanatomical and neurophysiological basis of corollary discharge and gain insight into the biochemical basis of its dysfunction. This review article also explores the role of corollary discharge and its dysfunction in the presentation of symptoms of schizophrenia, discussing the findings from corollary discharge studies on schizophrenia population. Lastly, the link between schizophrenia psychopathology and corollary discharge dysfunction has been highlighted, and an attempt has been made to establish a case for correction of corollary discharge deficit in schizophrenia through neuromodulation.

KEY WORDS: Corollary discharge; Efference copy; Motor activity; Auditory hallucination; Transcranial direct current stimulation.

INTRODUCTION

When we move around in the environment, there are essentially two phenomena that occur with us. These include the sensations that we experience from our own actions, and the sensations which we perceive due to environmental forces that act on us passively. If we weren't able to differentiate these two broad groups of sensations, it would create a significant problem in our understanding of the world.[1] This ability to differentiate between self-generated activities from the ones resulting from environmental cues is extended over a wide range of sensations. Taking this for instance, when we speak and listen to ourselves, we have the ability to identify the ownership of our speech. This can easily be discriminated from speech heard from the elements of the outside world.[2]

If the organism fails to differentiate between sensory impact arising from its own activity versus changes coming from deviations in the sensory environment, this could lead to innumerable challenges in assessing the world.[1] For example, with the intentional movement of the eyes or head there would be consequential movement of the retinas. This would cause hurdling of visual scenes one after the other and certain amount of blurring of vision.[3] But in reality, the organism is required to experience a change in its visual scenario, only if there is an actual environment modification in the scene. It is certainly expected to experience visual stability when the outside spurs are not present and the organism is simply shifting its gaze voluntarily. Another very interesting phenomenon occurs when an organism is undergoing motion. Linear motion, for example specially targets the otolith organs in the inner ear, which give the organism the sense of motion. But the organism has the ability to identify whether it is moving passively (for example travelling in a train), or actively (for example running a race).[4]

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Address for correspondence: Ganesan Venkatasubramanian, MD, PhD
WISER Neuromodulation Program, Department of Psychiatry, National Institute of Mental Health and Neurosciences (NIMHANS), Hosur Road, Bengaluru 560029, Karnataka, India
Tel: +91-80-26995256, Fax: +91-80-26564830
E-mail: gvs@nimhans.ac.in
ORCID: https://orcid.org/0000-0002-0949-898X
*These authors contributed equally to this study as co-first authors.

The very fact that the brain of an organism is able to establish this differentiation is consequence of its ability to monitor its own movements. This differentiation is achieved by generation of efference copies of its motor commands by the brain and transmitting them to its sensory reception structures. When these efference copies match the incoming reception of consequences of self-generated action, there occurs a suppression of the sensation arising from perceiving one's own action, this phenomenon is known as corollary discharge.[5] Corollary discharge is the estimation made by the brain that enables it to predict the likely sensory consequences of its motor activities; this, in turn, helps the organism in establishing the difference between self and non-self.[6]

COROLLARY DISCHARGE

Definition

Corollary discharge is an integration mechanism that embodies the core principle of neural functioning in most of the species.[1] It can be defined as a neural integration mechanism with two most essential functions of the suppression of sensations resulting from the organism's own actions, and discreet learning of the ability to predict future events based on the present subset of events.[5]

It has been fervently postulated that the ability to establish the concept of self and non-self is not simply restricted to the assessing of sensory inputs in the context of motor movements, but it also incorporates complex higher mental functions like thought and consciousness that, in principle, retain the features central to motor mechanisms.[7] Studies have proven that the ownership of a thought involves a neural circuit, in which, corollary discharge plays an important part in the process of rendering the consciousness to a particular thought.[8]

Though in literature the terms 'efference copy' and 'corollary discharge' have been used sometimes interchangeably, it is pertinent to draw a distinction between the two. As elucidated previously,[9,10] any action is preceded by transmission of a blueprint of the intended action plan to sensory cortex from the frontal areas of brain responsible for planning. This blueprint carrying the details about expected sensory consequences of intended action is referred to as efference copy. Actual sensation experienced on reception of self-generated action is denoted as 'sensory reafference'. The brain, while executing the movement, effectively subtracts the 'expected' sensations from 'experienced' sensations; this manifests as a net suppression of sensory awareness when actions are self-generated. This sensory suppression is referred to as corollary discharge.[9,10]

Historical Background and Evolutionary Implication

Corollary discharge has been a topic of intrigue and research since long in the scientific community. This neural integration mechanism was extensively studied for the first time in the field of visual perception.[11] It was put forth, that there is a motor command that is sent to the sensory structures for evaluating the sensory feedback generated from the activities of a fish's eye movement. Sperry[11] named this phenomenon as "corollary discharge". Another study conducted around the same time by scientists von Holst and Mittelstaedt,[12] evaluated the concept of 'efference copy command' that is essentially similar to the corollary discharge suggested by Sperry.[11] In years that followed, several studies were conducted to understand this mechanism, directly and indirectly. For instance, one study reviewed the integration of all motor commands, its corollary discharge and the perception generated from such movements under one set up.[13] Several other studies briefly touched upon and considered the concept of corollary discharge mechanism.[14,15] The concept of this feedback circuit related to corollary discharge was studied in the context of visual perception to the most extensive degree.[16-19] Corollary discharge circuit in sensory perception was also gauged by other research groups.[20] The history of the examination of the corollary discharge phenomenon spans from the 1900s to this date; in contemporary research newer studies with substrates like eye-movement or event-related potential correlates are opening up a whole new avenue for further imploring and examination of this context.

Studies found that organisms that fail to develop this complex neural integration mechanism of corollary discharge are at an evolutionary disadvantage, since their own activities jar their perception of the outside world, lending them to extinction. The importance of the development of these complex feedback signals comprising the corollary discharge phenomenon is thus signified, extending from invertebrates to vertebrates, with an increase in degree of complexity up along the taxonomical ladder of animal kingdom.[1]

The Neurophysiology of Corollary Discharge

Neurophysiological findings indicate that animals constantly update themselves with the sensory consequences of their own motor commands so that they are able to distinguish the sensory consequences of their own activity from that of the passive sensory reception from environmental activity. This ability of the organisms on account of their corollary discharge signals is mediated by specific set of neurons.[21] For instance, among crickets, the auditory reception remains suppressed during the act of their own singing, thus preventing them from undergoing auditory damage from constant noise they create. This inhibition was found to be brought about by a neuron identified as corollary discharge interneuron (CDI).[21] These CDI mediate the presynaptic inhibition of auditory sensations and also lead to post synaptic inhibition of the auditory interneuron which leads to decreased auditory reaction to sounds produced by the organism. The process of corollary discharge prevents an organism (like cricket or bullfrog) from getting desensitized to its environment because of sensory saturation from receiving its own near constant noise. In addition to this the corollary discharge is also responsible for increasing the sensitivity of the sensory neurons to external stimuli occurring interim.[21]

Studies report that when any movement is initiated, the copy sent from the motor command is compared with the reafference or the sensory outcome of the movement. If they both are comparable with no difference, no further motor adjustment of the movement is done. But instead, if any minute change is observed, the brain tries to identify if the difference is on account of some external stimuli or is due to lack of corrective compensatory mechanism.[8] It has also been suggested that the corollary discharge mechanism has an evolutionary advantage over regular proprioceptive feedback mechanism since it also predicts future movements thus preparing the organism for a varied stimuli response pattern.[22]

Neurophysiological evidences show that when the efference copy of motor command matches the sensory feedback (reafference copy) for a particular movement, there is a resonant increase in the gamma oscillations; this increased gamma oscillations might underlie the brain's inference of this motor command as self-generated; on the contrary, if there is a mismatch between reafference and efference copies even by minute degrees, gamma oscillations decrease; this might result in the brain inferring this latter scenario as a externally generated event. Such neurophysiological changes subserve the organism's ability to differentiate between activity of self and non-self.[23] In tune with these observations, findings implicate an increase in gamma oscillations between frontal and temporal lobes during the act of talking as against the act of passive listening.[24]

Evidences indicate that corollary discharge mechanism plays a crucial role in the context of understanding of self and non-self in higher motor tasks like thought or consciousness. Corollary discharge phenomena speeds up the process whereby the attention signal generated accesses the working memory at the time when a thought is being focused there. Corollary discharge phenomena is also implicated in correction of directional attention when there is a discrepancy between the expected and the actual consequence arising from an action (for example, it plays a role in inhibiting distractors which engage an organism thereby deviating their attentional focus away from a particular object or phenomena of interest).[8]

Neuroanatomical Basis of Corollary Discharge

Several studies have probed into the understanding of the anatomical structures that could be responsible for the corollary discharge phenomenon. Most common finding is the activity in fronto-temporal circuits responsible for attenuation of auditory response to self-generated sounds.[25] Some studies have also found cerebellum to be playing an important role in the comparison between a prediction and actual activity.[25,26] It is postulated that cerebellar-parietal connection establishes the difference if any between predicted outcome of an activity and the actual outcome of the activity, so that compensatory mechanisms can be initiated. Some studies have reported evidences implicating ventrolateral and mediodorsal nuclei of the thalamus in the processing of corollary discharge phenomena in human patients with lateral thalamic lesions.[6] While the corollary discharge in the auditory pathway is mediated through the arcuate fasciculus that connects the frontal and temporal cortical areas,[27] the corollary discharge in the visual saccadic movements is brought about by superior colliculus-mediodorsal thalamus and frontal eye field pathway utilizing the process of coordinating and stabilizing vision across saccades. Findings further suggest that these circuits for corollary discharge in the visual saccadic movements extend from

extra-ocular motor neurons right up to the cerebral cortex.[28)]

Based on the putative neuroanatomical substrates, the phenomenon of corollary discharge has been classified into lower order categories (performing reflex inhibition and sensory filtration) and higher order categories (sensory analysis and sensorimotor learning/planning by experts).[1)] It was surmised that corollary discharge plays an important role in preventing an organism from undergoing a reflex activity in effect of its own actions at lower level of functioning, and this underlies its ability to acquire sensorimotor learning; for example, a bird fine tuning its tones using sensory feedback obtained from the corollary discharge, from its memory. An example of reflex inhibition and sensory filtration from corollary discharge mechanism can be seen in *Pleurobranchaea*, a gastropod, organism with simpler nervous system. *Pleurobranchaea* exhibits repulsion if the tactile mechano-receptors of its oral veil are activated on contact with an external stimulus. But activation of the same tactile mechano-receptors at the time of its feeding activity, does not lead to any repulsion. The study comments that it occurs as a result of corollary discharge interneuron activity that silences the reflex withdrawal of the gastropod during its self-generated activity, in this case being feeding.

Role corollary discharge mechanism in sensory analysis and sensorimotor learning and planning has been documented in organisms with complex nervous system as well. A study examining the neuroanatomical functioning of bats, specifically, *Rhinolophus rouxii*, which sends sonic signals for hunting and estimating obstacles in its path, indicated that corollary discharge mechanism mediated their sonic navigational system. The corollary discharge mechanism served to a) match the echo received by the receptor centre in the inferior colliculi with the efference copy generated and sent by the bat's vocalization centre, and b) examine whether the actual echo is received in the expected time window; failing which, the source of the echo is labelled as external. Such higher order analyses through corollary discharge mechanism gives this animal the ability to estimate the location, size and speed of the object (self or non-self) that is causing the echo. In higher order complex motor functions like thoughts,[7)] studies have evaluated the ballistic control model of attention, suggesting that defect in the attention is responsible for disturbance in consciousness.[8)] Areas like prefrontal cortex are responsible for goal directed thoughts while superior parietal lobule and temporal parietal junction are responsible for the generation of attention signal. These two areas are believed to be responsible for stimulating the attention signal while inhibiting the distractions. Visual cortex is responsible for focused attention on the target thought, while the corollary discharge is meant for targeting a buffer space where the attention can be temporarily focused.[8)]

Neurochemical Basis of Corollary Discharge

Analysis of population with dysfunctional corollary discharge mechanisms (patients of schizophrenia,[29)] for instance) indicates a possible role of abnormal levels of different neuro-modulators like dopamine, acetylcholine, γ-aminobutyric acid, glutamate of the many others as one of the few causative factors.[8,30)] However among this group of neurotransmitters, there is compelling evidence for acetylcholine to play a crucial role in corollary discharge feedback circuits.[31)] It has been proposed that acetylcholine aberrations might underlie the disruption of corollary discharge.[32)] Another postulates that corollary discharge, which is mediated as a feedback through the internal association fibres in the neural circuit is inhibited by cholinergic inputs.[8)] Acetylcholine is essentially balanced in the brain, but if increased, may lead to complete shutting of the corollary discharge feed forward feedback, leading to dispersion of attention to varied stimuli and loss of the ability of identification of an activity as an action of self.[33)]

COROLLARY DISCHARGE IN THE CLINICAL DISORDERS

As a neural integration mechanism, the study of corollary discharge mechanism has not just been restricted to physiology; its contribution to clinical pathology has also been examined. Several studies have found this feed forward phenomenon to be integral to appropriate perception and prediction, and its malfunction manifests itself in the form of several diseases.

Among movement disorders, bradykinesia has been studied. Bradykinesia is a slowing of the limb movements as seen in parkinsonism, a neurological disorder which is categorized by the disintegration of dopaminergic neurons of the basal ganglia.[34)] One study found that the

movement disorder occurs on account of misjudgment during the limb movement. In these patients, there is inappropriate coordination between the proprioceptive sensory feedback and the corollary discharge mechanism operating at the higher centres. As the patient is unable to predict the position of the limb, it results in slowing of his limb movement leading to bradykinesia.[35]

Cerebellar disorders have also been studied to evaluate if corollary discharge plays any role in the typical presentation like usage of the index finger or performing actions of opposition.[36] Actions like apposition of thumb require well-coordinated, sequential functioning of muscle movements. It has been suggested that disruption to pyramidal tracts to the cerebellum responsible for corollary discharge dysfunction, can account for lack of practiced, harmonized progressive movement tasks.

Certain ownership disorders like anarchic hand syndrome in which there is perceived loss of control over a limb.[37] Utilization syndrome in which the patient is completely dependent on the external stimuli for perception of the world,[38] and alien hand syndrome in which the patient completely denies the ownership of his/her limb have been studied.[39] One of the studies probed into the possible reasoning behind the specific presentation of these disorders and postulated that it could be on account of lack of coordination between the sensory feedback mechanism and the corollary discharge feed-forward circuit.[40]

In one neurophysiological study conducted on the phrenic nerve activity of a cat, observations indicated that corollary discharge mechanism plays an important role in moderating the activity of the phrenic nerve outflow rhythms.[41] The study found that in spite of splitting the brain stem of the cat, rhythms of the two phrenic nerves could be harmonized. This is believed to be due to the circuit that integrates with sensory feedback from the contralateral phrenic nerve activity.

A common neurodegenerative condition, Alzheimer's disease,[42] has been found to have notable memory deficits in its primary phase.[43] Few studies have suggested that one of the features of memory impairment in Alzheimer's disease is that inherent (implicit) memory is used to recall explicit (freshly sort out information) memory.[44,45] It has now been put forth, that sensorimotor integration errors in the form of prediction errors in task outcomes are common presentation of these patients during the act of recall of memories.[46] This is believed to be on account of feed-forward or corollary discharge mechanism dysfunction.

In psychiatric disorders, potential corollary discharge dysfunction in autism spectrum disorder has been studied. Autism is a neuro-developmental disorder which presents with uncharacteristic development in socialization, interaction and mannerisms and conduct.[47] A study conducted in understanding its symptomatology has found that the one of the possible pathogenetic basis for autism is that the neural integration of sensations from multiple inputs is impaired in these individuals.[46] This defect is responsible for failure in comprehending and predicting patterns of movements at the higher centres. Perhaps, secondary to these corollary discharge deficits, patients find it difficult to anticipate the consecutive actions of their initiated movement. The study emphasizes on the possible role of dysfunction of the feed forward circuit.

Undoubtedly, all the above-mentioned disorders and physiological dysfunctions occurring on account of impaired corollary discharge mechanism are noteworthy. But it requires special mention that lack sense of self, inability to identify activities self-generated by the body, and the sense of attention for tasks at hand are perhaps most profoundly affected in the psychiatric disorder schizophrenia.[8] Many empirical studies have evaluated the impact of corollary discharge impairment on schizophrenia patients (Table 1).

COROLLARY DISCHARGE IN SCHIZOPHRENIA

Brief Overview

Schizophrenia is a complex psychiatric disorder that is known to affect approximately 0.3% to 0.7% of the population worldwide.[48] Inability to distinguish between thoughts and actions generated from self and others, which is a core component of schizophrenia[8] has been postulated to be secondary to the failure of corollary discharge mechanism.[15,49] Corollary discharge dysfunction in schizophrenia has been explored through numerous methods in the last few decades. Methods primarily employed for tracking this corollary discharge circuit include functional magnetic resonance imaging[50] and event related potentials.[51-53] Few studies have used the smooth muscle pursuit task for studying the prediction factor of

Table 1. Summary of studies on corollary discharge deficit in schizophrenia (SCZ) patients with auditory hallucination in comparison to healthy controls

Study	Factor	Healthy control	SCZ	Implication
Ford *et al.*, 2002; ERP study	Gamma coherence between frontal & temporal lobes	Greater frontal-temporal coherence during talking than listening	Not seen	Frontal-temporal disconnection in SCZ
Ford *et al.*, 2001; ERP study	N100 amplitude to self-talk	Attenuated	Inadequate attenuation	Lack of suppression of PAC during self-speech in SCZ
Heinks-Maldonado *et al.*, 2007; ERP & DTI study	Feed forward model of self-initiated activity	Graded suppression of N100: N100 Sup. to unaltered voice >altered voice	Absence of Graded suppression of N100	Deficient forward model in SCZ; AVH result from "misperception"
	Structural deficits in arcuate fasciculus	Insignificant	Pronounced	Damage to brain's architecture adds to CD dysfunction in SCZ
Ford *et al.*, 2008; EEG study	Pre-movement / action synchrony in response to button press	Maximal gamma-beta synchrony over contralateral sensori-motor cortex	Reduced gamma-beta synchrony over sensori-motor cortex	Efference copy/CD deficit across modalities in SCZ with AVH
Whitford *et al.*, 2011; Neuro-physiological and diffusion tensor imaging study	Delayed feedback (graded) to self-initiated act	CD disrupted in response to delayed feedback	CD corrected upon delayed feedback (at 50 ms)	Time lag in arrival of efference copy to sensory cortex in SCZ; result-CD deficit
	Structural deficits in arcuate fasciculus for cortical sup. across delayed feedback conditions	Linear relationship between FA in the arcuate and pattern of N1 suppression across delay conditions	Inadequate FA in the arcuate fasciculus	Evidence supports fronto-temporal disconnection hypothesis in SCZ
Spering *et al.*, 2013; Eye movement study	Effect of abnormal pursuit on Prediction performance	Better trajectory prediction with pursuit; performance enhanced with increase in duration of stimulus presentation; correlation between pursuit gain & pursuit judgment	Poor trajectory prediction; No pursuit advantage with increased presentation; no correlation	SCZ patients have intact early sensory (visual processing), but fail to use efference copy signal. Sensory systems have impaired efference signal
Shergill *et al.*, 2014; fMRI study	Attenuation of self-processing	Attenuated activation in secondary somatosensory cortex; sensation and action were synchronous	Absence of or inadequate attenuation of activity in secondary somato-sensory cortex	SCZ patients are unable to predict the sensory consequence of their own actions; deficient CD

ERP, event related potential; PAC, primary auditory cortex; DTI, diffusion tensor imaging; AVH, auditory verbal hallucinations; CD, corollary discharge; EEG, electroencephalography; FA, factional anisotropy; fMRI, functional magnetic resonance imaging.

corollary discharge.[54] Though most neurophysiological studies have focused on role of corollary discharge dysfunction in the pathophysiology of auditory hallucinations in schizophrenia (summarized in Supplementary Table 1; available online only), these studies on corollary discharge in schizophrenia are diverse in the questions they posed to answer.

One of the studies that was conducted to understand the self-disturbances in schizophrenia enlisted two primary elements of distortions, namely i) hyper flexibility which leads to excessive attention towards self-features as though they were external, and ii) reduction is self-affection, which is diminished understanding of one's presence.[55] From the perspectives of operationalized clinical manifestations, these self-disturbances are described as positive symptoms such as delusions and hallucinations and negative symptoms like affective flattening, avolition, alogia, anergia, and anhedonia constitute the major diagnostic criteria. However, apart from these pivotal presenting symptoms, schizophrenia patients are also known to suffer from cognitive deficits like disturbances in attention, working memory, and certain executive functions. This cluster of mental and physical manifestations leaves a patient in a state of personal and social jeopardy which is associated with a life long suffering.[56]

Contextually, it is interesting to note that studies have found that disturbances in stream of consciousness or derealization with loss of contact from self are also on account of disturbed corollary discharge activity. Corollary discharge buffer activity is required to focus all attention on an object of interest giving the individual a conscious focused experience. It has been put forth that reduction in

the activity of this discharge buffer is responsible for causing distortion and bringing to attention varied stimuli that the person finds difficult to assemble into one level of consciousness (Fig. 1).[8]

Studies investigating possible causes for the disruption of corollary discharge in disorders like schizophrenia, have postulated that the possible role of decreased myelination of tracts in the brain could be the cause of delayed corollary discharge mechanism in neural circuitry.[30] Several studies have probed into understanding the link between auditory hallucinations and deficient corollary discharge. It has been demonstrated that in healthy people, corollary discharge released from the motor speech areas of frontal lobe, act on the auditory cortex preparing it for the sensations of its self-generated activity that gives an individual the ability to establish its self from non self.[27] Recent findings suggest that in schizophrenia patients this process is disrupted as reflected in specific differences observed in structural architectural and neurophysiological activity of the schizophrenia patients in comparison to healthy controls (Table 1). Based on these observations, it has been surmised that such disruption of this corollary discharge is responsible for the auditory hallucinations in schizophrenia.

Delusions are false beliefs that suggest of an abnormality in the affected individual's thought process and judgement, which cannot be accounted for through logical reasoning. The most important part of it is the degree to which the individual holds the belief as true, in spite of presenting contrary evidence.[57] Studies have now found that these occur on account of disruption of the corollary discharge,[58] which, under normal circumstances is responsible for higher motor planning, prediction and learning.[1] As there is loss of ability to use learned memory to infer understanding of current situation, patients engage in convenient explanations[59-61] that bring them relief, hence they hold on to them.[62] These beliefs are then used to deduce future experiences of the patient that constitute a part of the psychopathology itself.[63]

Patients suffering from schizophrenia may present with language disturbances like incoherence and pressure of speech, tangentiality or derailment. Studies examining these features report that the disruption of corollary discharge, which is responsible for the inhibition of distractions during attention focusing, leads to disintegrated emphasis on a singular biased object. The patient finds oneself subjected to varied stimuli that bring about the classic presentation of formal thought disorder in these patients.[8] Symptoms like thought insertion present in schizophrenia are thus postulated to be a result of defective corollary discharge mechanism.[64] A study claimed that the lack of ownership of a thought occurs on account of lack of efference copy to the attention bias. Since patients experience a thought or a sensation from an activity that is self-generated, but there is no efference copy, they misconstrue their thought or activity as a result of outside forces or as non-self.[64]

Another intriguing symptom of schizophrenia is that of somatic passivity or passivity phenomena. Essentially, in a healthy individual, an act of holding a pen and writing on

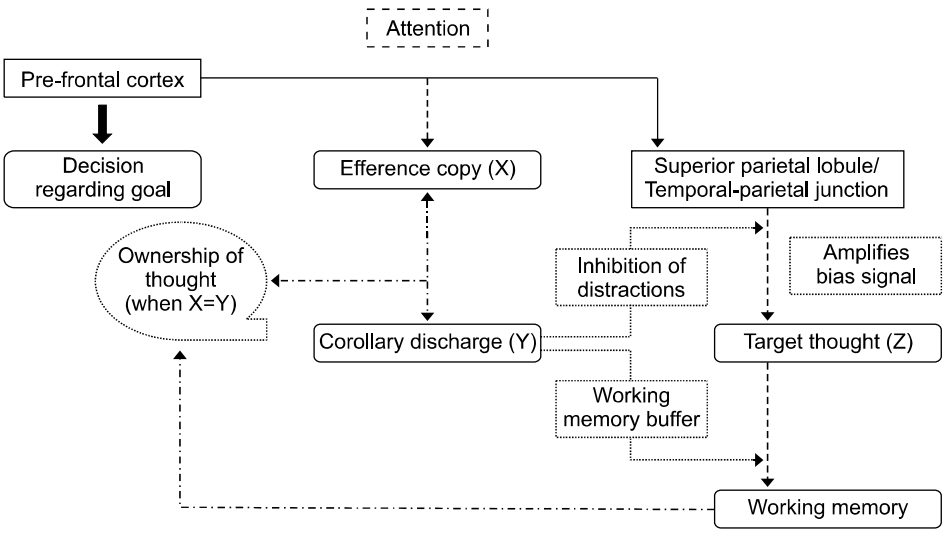

Fig. 1. Corollary discharge feedback mechanism. If X equals Y, efference copy (estimation) and afference feedback complement; so, no neural activity is changed and the activity is identified as 'Self'. If X does not equal Y, assessment is done to evaluate if there is (i) inappropriate compensatory mechanism, or (ii) external influence on activity.[8]

a piece of paper has two components. One that, he has the desire to conduct such an activity, and when it is being accomplished, he has the acknowledgement of the activity being self-generated. In the context of passivity experience, schizophrenia patients will attribute the agency of their own volitional act to an external force; thus, patients with passivity symptoms will report that a non-self agent control their actions, thoughts and sensations. For instance, they would use the pen to write something, but wouldn't be able to acknowledge that it was their volitional act, but rather an activity of an external agency. The study postulated that, on account of lack of an efference copy to the sensory system, the patient is in an unfortunate position to unable to predict the positional errors of its limb and correct them before the peripheral feedback arrives. Hence, though the patient is aware of the executed action, he is unaware of being the initiator of the same.[65,66] To understand the neural correlates of passivity symptoms, proprioceptive function has been examined in schizophrenia patients suffering from passivity phenomena. It has been demonstrated that proprioception (as examined by self-induced attempt at tickle) too is affected in schizophrenia patients in comparison to healthy controls who were able to tickle themselves unlike the healthy individuals.[26]

Studies have delved into examining the causative factors believed to be responsible for corollary discharge involving prediction and its anatomical and physiological basis. Patients demonstrating corollary discharge dysfunction have disturbances in neural myelination. Decreased myelination in white matter leads to delay in the conduction of impulse to sensory areas thus contributing to lack of sensory feedback eventually affecting person's concept of "self".[30] Alongside decreased myelination, aberrations in acetylcholine level have been postulated to explain both symptoms of schizophrenia and corollary discharge dysfunction.[31] Abnormally increased levels of acetylcholine might underlie symptom presentation in schizophrenia; a related conceptualization of the neural model in schizophrenia posits an interaction between cholinergic aberrations and thalamus.[8] Thalamus relays both attentive stimuli and distractions. Acetylcholine acting on the nicotinic receptors of the thalamus regulates the neural activity of information processing by engaging attention. Acetylcholine acting on the muscarinic receptors of the association fibres, suppresses their activity, hence preventing the inhibition of distractor stimuli. Corollary discharge is inhibited by such excess of acetylcholine that leads to gradual 'loss of self' experienced by schizophrenia patients with the genesis of Schneiderian first rank symptoms (this includes thought alienation, somatic passivity and related similar experiences).[67] The increased acetylcholine contributes to attention deficit by inhibiting monitoring activity of prefrontal cortex; this in turn sets up the platform for the onset of negative symptoms and cognitive decline on account of prefrontal inhibition. With on-going increase in the acetylcholine neuromodulator, complete disintegration of the "concept of self" occurs as the disorganization syndrome presents itself.[8]

Corollary Discharge, Dreams and Hallucinations

Similarities between sleep-related experiences and hallucinations have been a matter of legitimate scientific curiosity that has led to examination of two processes in relation to each other.[68] Dreams and other sleep related experiences like hypnagogia share some overlapping features with hallucinations—1) subjective descriptions like complexity, emotional charge and acceptance of bizarre or implausible scenarios, etc.; and 2) underlying neural mechanism.[69] However, there exist crucial and clear differences between the two on phenomenological features and neural mechanisms. While dreams and hypnagogia are multisensory experiences with visual modality dominating the presentation, in schizophrenia, hallucinations are mostly auditory. In dreams, the experience happens in external world and the person attributes the process of dreaming to self. In schizophrenia, hallucination is cloaked with veridical perception; they are concurrent with the ongoing mental processing. Dreams are often not recalled upon wakefulness and they rarely affect a person's experience of himself; hallucinations occur in wakefulness, and they are not only remembered well but they often interfere with a person's understanding of himself and world he lives in (for details see review[68]). Interestingly, corollary discharge, being integral to the sensory-motor systems of consciousness, is common to both of these experiences—dreams and hallucinations. It has been reported that corollary discharge or feed forward circuits are temporarily suppressed during the act of sleeping in normal healthy brains.[69] It has been further posited that the level of cortical suppression (that occurs

through execution of corollary discharge) accounts for the differing experience of the dreamer; the greater the suppression of corollary discharge or feed forward circuits during sleep, more vivid and immersive is the dreaming experience.[69] In both dreaming (along with associated sleep related experiences) and hallucinations, thalamus activation regulated by acetylcholine activity plays a crucial role. Acetylcholine directs the focus of mind inwards, creating an attentional bias towards internal experiences that is required to perpetuate experiences like dreaming and hallucinations.[70] Similar limbic activity has been shown to underlie affective components of hallucinations and emotional component of dreaming.[70] These evidences can guide fine tuning of clinical definition of hallucinations as "overlaid on veridical perceptions" (because dreams are non-veridical perceptions and unbidden experience too),[68] and provide leads for novel clinical treatments[69] that target neural mechanisms responsible for such unbidden, vivid and affective perceptual experiences.

Neuromodulation of Corollary Discharge in Schizophrenia

Transcranial direct stimulation (tDCS) operates on the concept of utilizing weak intensity direct current to modulate cortical excitability. Several studies have examined and reported effectiveness of tDCS neuromodulation treatment for refractory symptoms of schizophrenia (see review[71] for details), particularly refractory auditory hallucination.[72,73] Two studies among these have explored effect of neuromodulation with tDCS on corollary discharge mechanism among schizophrenia patients with persistent auditory hallucination. In schizophrenia patients with persistent auditory verbal hallucinations, add-on tDCS (in open-label design) resulted in significant reduction of auditory hallucination severity with concurrent amelioration of corollary discharge deficit.[74] This observation was replicated recently in randomized, double-blind, sham-controlled design; importantly, the magnitude of cortical suppression during corollary discharge had a significant positive correlation with reduction in auditory hallucination scores.[75] These observations are in conformation with previous report of neuroplastic effect of tDCS enhancing auditory processing efficiency.[76] It is possible that fronto-temporo-parietal tDCS reduces auditory hallucination severity by addressing aberration in corollary discharge mechanism possibly through adaptive modulation of neuroplasticity; potentially, both anodal stimulation to left dorsolateral prefrontal cortex and cathodal stimulation to temporo-parietal junction facilitates these changes.[75]

FUTURE RECOMMENDATIONS

Corollary discharge is a crucial neural integration that gives an organism the ability to identify active undertakings from passive activities. It is released from the motor areas of the brain, carrying efference copies of its expected sensory outcomes. Despite the extensive body of research available on corollary discharge mechanism, certain areas require further understanding.[1] The corollary discharge activities in olfactory and gustatory components of sensory apparatus haven't been explored adequately. As far as vestibular feedback is concerned, corollary discharge is known to provide stability to an organism and predict changes on external instigation, but how brain analyses external cues during active motion has not yet been studied in detail.[4] Also, studies have found superior-colliculus-mediodorsal thalamus frontal eye-field pathway as an important corollary discharge circuitry. However, further research is required to assess the contribution of this pathway to corollary discharge dysfunction especially with regards to pathogenesis of symptoms and disorder.[28]

Though the dysfunction of corollary discharge has been studied in several disorders, the exact circuits are yet to be elucidated definitively. Further research at the cellular and molecular level of corollary discharge phenomena and yield to better understanding of its mechanistic basis of action. Research on the developmental aspects of corollary discharge feedback circuits is warranted to facilitate insight into the genetic component of feed forward dysfunction model of psychopathology. Studies are required to delineate if there are different types of corollary discharge circuits employed for different modalities, or one particular corollary discharge circuit is meant for one typical function.

Since the exact neural circuitry basis of corollary discharge induced cortical suppression is yet to be delineated, it is difficult to rule out confounding concurrent processes. For example, let's consider cortical suppression during talking out loud.[50] Active talk involves a) con-

traction of the temporalis muscle that spans over the frontal-temporal region and b) demands more attention than passive. The contribution of these factors to cortical suppression needs to be examined so that contribution of corollary discharge to cortical suppression could be established.[77] Advanced brain imaging techniques can answer such questions by allowing examination of neural circuits specific to corollary discharge by studying the interplay between different brain structures.[78]

CONCLUSION

The purpose of this review was to elucidate neural basis of corollary discharge phenomena, summarize existing research on it, and build arguments that support its candidature for explaining schizophrenia symptoms with focus on auditory hallucinations. To this extent, neurophysiological, neuroanatomical and neurochemical explanations of mechanistic basis of corollary discharge phenomena were reviewed and examined.[8,28]

Corollary discharge is integral to the ability of an organism to recognize itself and its own actions like proprioception[1] and underlies highest forms of neural assessments like thought and ipseity.[7] Contribution of corollary discharge in attenuation of sensory feedback arising from the self-generated activity of the organism is imperative and unambiguous.[50] Extensively studied in audition[51] proprioception,[1] and higher motor complex process like thought,[8] corollary discharge of thought in healthy brain involves attenuation of auditory cortex, the somatosensory cortex and thalamus, as a result of match between the efference copy and sensory re-afference. This essentially gives the organism a feeling of 'self'. Another important utility of corollary discharge is its role in predictive functions; like visual stability continues to be because of corollary discharge phenomena. To enable this, the corollary discharge signals anticipate extra retinal pre saccades before they occur, and corollary discharge signal thusly keep an organism prepared for the change in field of vision.[79] Corollary discharge is implicated in learning and memory processes as well and it helps an organism in self-monitoring in skill acquisition phase using the copies of corollary discharge stored in it memory to make better approximations of intended activity.[1] A deficiency in corollary discharge thus manifests as altered in perceptual experiences (dreams and hallucinations), disrupted sense of self (schizophrenia), difficulty in learning and memory (source monitoring) and other cognitive problems. Extensively studied in schizophrenia in the context of auditory hallucinations, neurophysiological and neuroimaging findings on corollary discharge establish that its dysfunction plays a crucial role in hallucination pathophysiology.[9] However, to ascertain contribution of corollary discharge to other symptoms of schizophrenia like delusion and disorganization[8] robust empirical evidences are required. With reports of correction of corollary discharge deficiency with tDCS neuromodulation, a new avenue for examination of its neural circuitry, role in psychiatric symptoms and contribution dream/sleep-related experiences opens up. As schizophrenia manifests as disruption of the self, understanding of schizophrenia aetiology can benefit from deeper exploration of corollary discharge phenomena that is integral to the experience of the self.

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Supplementary Table. Neurophysiological Studies of Corollary Discharge dysfunction in schizophrenia

STUDY	BACKGROUND	METHODOLOGY	KEY FINDINGS WITH IMPLICATIONS
(Zheng et al., 2015)	**Premise** ⌖ Understanding neural functioning under Octave illusion could further understanding of AVH in SCZ ⌖ Because of CD dysfunction in SCZ, under octave illusion, activation patterns from frontal to temporal areas differ between HC and SCZ **Aim** ⌖ To examine activation and synchronization within frontal and temporal brain regions in HC and paranoid SCZ patients with AVH ⌖ To explore the association between clinical symptoms and brain activation and synchronization patterns during octave illusion in SCZ group	**Methods** ⌖ **Location**: Hangzhou, China ⌖ **Sample**: SCZ (first episode with AVH)=23, HC=23 ⌖ **Instructions**: The participants were asked to focus on a white cross mark displayed on a computer screen of black background at 1 m distance from them. The paradigm was administered through earphones. Three minutes of silence preceded the administration of two sequences of octave illusion, separated by a gap of 10 sec from each other. ⌖ **Deutsch Octave Illusion ERP Paradigm Details** – Two tone of 400 Hz and 800 Hz presented to either ear at a time together in a manner when the right ear received the high tone, the left ear received the low one, and vice versa. After first presentation, the auricular inputs were reversed for the second presentation such that each ear alternately received each of the 400 Hz and 800 Hz sequence. The tones were of 70 dB sound pressure level	**Results** ⌖ Task-related theta power change values of frontal and temporal areas were significantly lower for both HC and SCZ groups with no between group difference ⌖ Values of the task-related theta coherence difference between the ipsilateral frontal-temporal areas were increased in SCZ group ⌖ Negative correlation between task related power changes in between the hemispheres ⌖ Task related coherence differences in the right hemisphere positively correlated with hallucination scores of SCZ participants **Implications** ⌖ Increased ipsilateral synchronization in SCZ during octave illusion is similar to activation patterns during AVH. This stronger mobilization or disinhibition implies a monitoring deficit. ⌖ In SCZ the frontal area monitoring of the temporal part was not powerful enough, even if the synchronized pathway was ‘over-mobilized', accounting for corollary discharge dysfunction hypothesis ⌖ the dysfunctional right hemisphere linked to the corollary discharge might result in a relatively active left hemisphere, which is often the case

		and 250 ms sine waves at equal amplitude and followed each other without pause.	for an ongoing hallucination in schizophrenia
(Ford et al., 2014)	**Premise** ⌖ Motor acts are preceded by efference copy of plan of execution which facilitates CD of the expected sensation in sensory cortex even when the causal chain between the act and its sensory consequence is indirect and dependent on external devices ⌖ Established dysfunction of efference copy and CD in SCZ **Aim** ⌖ To examine pre-movement activity of button press to hear tone (a paradigm translatable to less vocal animals) for similar patterns of CD dysfunction in SCZ as seen in 'talking' paradigm	**Methods** ⌖ **Location**: California, USA ⌖ **Sample**: SCZ=26, HC=22 ⌖ **Instructions**: During button tone condition, the participants were asked to deliver tones to themselves in the self-paced manner they were trained at. In the Pay tone condition they listened to the tone play back. ⌖ **ERP Paradigm**: Participants pressed a button, every 1–2 seconds, to deliver 1000 Hz, 80 dB sound pressure level, tones with zero delay between press and tone onset (Button Tone). After 100 tones had been delivered, the task was stopped. The temporal sequence of tones was preserved for play back (Play Tone). In addition, subjects pressed a button at approximately the same pace, and no sound occurred (Button Alone)	**Result** ⌖ HC had larger Lateralized readiness potential (LRP) – a measure of motor plan – preceding button press for tone delivery and N1 suppression than SCZ ⌖ Positive correlation between LRP amplitude and N1 suppression across both the groups **Implication** ⌖ Even when efference copy is indirectly related to the sound associated with it and the sound does not strictly come from "self" SCZ exhibit efference copy and CD dysfunction ⌖ Pre-movement activity reflects the efference copy ⌖ Self-delivery of tones also produces robust N1 suppression and involves auditory cortex ⌖ Using this paradigm CD mechanism can be studies in lab animals. Neurophysiological recordings used with lab animals and humans are comparable, making both the 'talk' and 'button press' paradigms and the physiological assay translatable across species
(Nawani et al., 2014)	**Premise** ⌖ CD dysfunction in SCZ evidenced as inadequate N100 suppression ⌖ tDCS causes adaptive	**Methods** ⌖ **Location**: Karnataka, India ⌖ **Sample**: SCZ=05 ⌖ **ERP Paradigm**: The Talk-Listen Paradigm as described by Ford et al.,	**Result** ⌖ Before tDCS, N1 amplitude of Talk condition didn't significantly differ from N1 amplitude of Listen condition; post tDCS this difference was significant.

	neuromodulation of N100 **Aim** ⌖ Examining effect of tDCS in corollary discharge dysfunction in SCZ with AVH	2010. Talk condition: Participants made short crisp vocalizations of the phoneme "ah" repeatedly in a self-paced manner, about every 1–2 s, for 100 s. This speech was recorded using a microphone connected to the stimulus presentation computer and transmitted back to subjects through headphones in real time (zero delay). Listen condition: The recording from the Talk condition was played back, participants had simply listen to it. ⌖ **Intervention**: Add-on tDCS therapy	**Implication** ⌖ Add-on tDCS therapy was observed to modify and correct corollary discharge dysfunction in SCZ participants with AVH ⌖ Neuroplasticity modulating effects of tDCS address auditory processing deficits in SCZ
(Ford et al., 2012)	**Premise** ⌖ CD dysfunction in SCZ is manifested as inability to distinguish self from externally generated experience **Aim** ⌖ To examine if CD dysfunction is specific to SCZ by exploring it in other psychotic illness and first degree relatives of psychotic patients ⌖ To examine if reduced N1 suppression during talking is an endophenotype of	**Methods** ⌖ **Location**: Chicago, USA ⌖ **Sample**: SCZ =30, SA=19, BPP=39, HC=43, SCZ Relatives=30, SA Relatives=23, BPP Relatives=50 ⌖ **ERP Paradigm**: The Talk-Listen Paradigm. Talk condition: Participants made short (<300ms), sharp vocalizations of the phoneme "ah" repeatedly in a self-paced manner, about every 1–2 s, for 180 s. This speech was recorded using a microphone connected to the stimulus presentation computer and transmitted back to subjects through headphones in real time (zero delay). Listen condition: The recording from	**Result** ⌖ Inadequate N1 suppression in SCZ, SA and BPP group of psychotic patients ⌖ N1 suppression failure among the patient groups didn't correlate with current positive symptoms; though the patients group differed in degree of psychotic symptoms they didn't in N1 suppression ⌖ N1 suppression among first degree relative didn't differ significantly from HC or probands **Implication** ⌖ Efference copy / CD dysfunction as reflected in failure of N1 suppression can be observed in SCZ and other psychotic disorders; it could be a trait marker for psychosis ⌖ N1 suppression is not a robust endophenotype for psychosis risk nor is it a heritable trait in the family

	psychosis	the Talk condition was played back, participants had simply listen to it.	of psychotic proband
(Perez et al., 2012)	**Premise** ⌖ CD dysfunction in SCZ is manifested as inability to distinguish self from externally generated experience **Aim** ⌖ To examine if putative CD modulation of auditory cortex responsiveness during speech is compromised early in the course of SCZ and CHR	**Methods** ⌖ **Location**: Chicago, USA ⌖ **Sample**: CHR =40, ESCZ =41, HC=86 ⌖ **ERP Paradigm**: The Talk-Listen Paradigm. Talk condition: Participants made short (<300ms), sharp vocalizations of the phoneme "ah" repeatedly in a self-paced manner, about every 1–2 s, for 180 s. This speech was recorded using a microphone connected to the stimulus presentation computer and transmitted back to subjects through headphones in real time (zero delay). Listen condition: The recording from the Talk condition was played back, participants had simply listen to it.	**Result** ⌖ Inadequate N1 suppression in SCZ and ESCZ ⌖ N1 suppression abnormalities are not affected by illness duration ⌖ No significant age effects were observed over N1 suppression from adolescence into adulthood ⌖ CHR group could not be statistically distinguished from either the HC or ESCZ groups ⌖ Attenuated speech-related N1 suppression was not related to positive or negative symptoms in early illness or CHR patients **Implication** ⌖ Inadequate N1 suppression in SCZ is present since early state (2.5 years of illness) and cannot be attributed to chronicity of the illness ⌖ Inadequate N1 suppression is not a progressive feature of SCZ; it exists before many of the sequelae of chronic illness like - chronic disability, medication exposure, long-standing social and occupational dysfunction ⌖ The putative CD mechanism is present since adolescence and shows no further age-related changes into midlife
(Whitford et al.,	**Premise** ⌖ CD dysfunction in SCZ	**Methods** ⌖ **Location**: California, USA	**Result** ⌖ In SCZ, CD deficit was not observed at 50 ms delay

2011)	underlies their inability to distinguish between internally and externally generated experience **Aim** ⌖ To examine Investigating if corollary discharges among SCZ subjects are delayed at in arriving at sensory cortex	⌖ **Sample**: SCZ=21, HC=25 ⌖ **ERP Paradigm**: Participants trained to vocalize "Ah". A sample "Ah" selected from their recording presented to them at button press at a lag of 0 ms, 50 ms and 100 ms. ⌖ Diffusion Tensor Imaging: 15 SCZ and 17 HC were scanned, fractional anisotropy of their arcuate fasciculus was examined for level of cortical suppression across all three conditions of the CD paradigm	condition only, out of all the three conditions. At 0 ms, N1 suppression was subnormal, at 50 ms it was supranormal and at 100 ms comparably lower than that. ⌖ In HC unlike in SCZ, delayed stimulus delivery led to inadequate N1 suppression ⌖ Inadequate FA of arcuate fasciculus was observed in SCZ group. Further, a linear relationship was between observed between participants' FA in the arcuate and their pattern of N1 suppression across delay conditions ⌖ Evidence supports disconnection hypothesis **Implication** ⌖ In SCZ there is a time lag in arrival of efference copy to sensory cortex causing CD. As with externally imposed delay in self-initiated stimulus delivery inadequate N1 suppression deficit looks corrected. ⌖ Structural damage of white matter tracts in SCZ perhaps explain the conduction delays captured in ERP paradigm assessing CD. ⌖ Aberrant physiological interactions that underlie faulty discrimination between internally and externally generated actions are perhaps mediated by WM abnormalities in fibers that connect action initiation and action reception regions of brain
(Ford et al., 2008)	**Premise** ⌖ Phase synchronization precedes the transmission	**Methods** ⌖ **Location**: Chicago, USA ⌖ **Sample**: Psychotic patients=23	**Result** ⌖ In HC, prepress gamma band neural synchrony was maximal over the contralateral sensory-motor

	of a copy of motor commands to sensory cortex, where the expected sensation (CD) is generated ⌖ If prepress PLF (phase locking factor) reflects the activity in motor cortex, it should be contralateralized. If it reflects the action of the efference copy, it should be related to subsequent sensory suppression **Aim** ⌖ To quantify the synchrony of neural events associated with the hypothesized efference copy during initiating a button press ⌖ To relate possible measures of the efference copy to subsequent suppression of sensory re-afference	(SCZ=20, SA=3); HC=25 ⌖ **ERP Paradigm**: Subjects were asked to press a button with the index finger of their right hand, at will, every 1 to 2 seconds. They were stopped after about 2 minutes of pressing or after about 50 to 60 presses. This was a control task for the talk-listen paradigm. In Talk condition, the participants made short (<300ms), sharp vocalizations of the phoneme "ah" repeatedly in a self-paced manner, about every 1–2 s, for 180 s. This speech was recorded using a microphone connected to the stimulus presentation computer and transmitted back to subjects through headphones in real time (zero delay). In listen condition, the recording from the Talk condition was played back, participants had simply listen to it.	cortex in healthy subjects, and correlated with the ipsilateralized somatosensory ERP amplitude evoked by the press ⌖ Prepress gamma and beta band (especially those with avolition/apathy) neural synchrony reduced in patients. **Implication** ⌖ Premovement asynchrony may reflect an elemental deficit in SCZ, as observed in both prespeech and prebutton-press data. This reflects efference copy/corollary discharge mechanism dysfunction across modalities.
(Ford et al., 2007)	**Premise** ⌖ CD prepares the auditory cortex for incoming sound by minimizing its responsiveness and allowing the incoming sound to be tagged as self-generated	**Methods** ⌖ **Location**: California, USA ⌖ **Sample**: Psychotic patients=27 (SCZ=23, SA=4); HC=26 ⌖ **ERP Paradigm**: It had four conditions – **Talk condition:** Participants made short (<300ms), sharp vocalizations of	**Result** ⌖ N1 suppression during talking is more compared to expectancy effects based on visual warning, or simple agency effects based on self-delivery, of speech sounds ⌖ In HC group only agency and expectancy had a modest suppression effect on N1 suppression

	Aim ⌖ To explore if predictive information from expectancy (i.e., warning) and agency (i.e., self-stimulation) modulates auditory cortex in the same way and to the same degree as talking ⌖ To explore of these modulatory effects of expectancy and agency are deficient in SCZ in comparison to HC	the phoneme "ah" repeatedly in a self-paced manner, about every 1–2 s, for 180 s. This speech was recorded using a microphone connected to the stimulus presentation computer and transmitted back to subjects through headphones in real time (zero delay). **Agency Condition:** Speech sounds were delivered by a self-paced button press (zero delay) and heard instantaneously. A sample of speech recorded at the beginning of the session was used as the auditory stimulus. **Expectancy Condition:** During warned condition, a visual warning prepared subjects for the impending speech sound. A sample of speech recorded at the beginning of the session was used as the auditory stimulus, same as agency condition. **Listen condition**: The recording from the Talk condition was played back, participants had simply listen to it.	⌖ Deficits in either expectancy- or agency-related N1 suppression effects in the patients did not contribute to their diminished suppression of N1 during talking **Implication** ⌖ Agency and expectancy conditions do not amount to robust cortical suppression as does the talk condition. This could be because in the talk condition, "ah" sounds produced were produced in the moment and resultantly advance knowledge regarding its characteristics was close to perfect. This implies that degree of perfection regarding the knowledge of content affects N1 suppression. ⌖ Though expectancy and agency effects may contribute to the N1 suppression seen during talking, they do not completely explain CD phenomena ⌖ Auditory cortical suppression effects during talking may be primarily a reflection of the specific ways motor systems are wired to send a corollary discharge of the planned actions to the specific sensory cortical areas that receive the sensory consequences of these actions
(Judith M. Ford et al., 2007)	**Premise** ⌖ Neural synchronization preceding self-generated actions underlie the feed forward models of actions ⌖ Pre-action synchrony should	**Methods** ⌖ **Location**: California, USA ⌖ **Sample**: SCZ=24, HC=25 ⌖ **ERP Paradigm**: Cued talk-listen paradigm - The subjects uttered "ah" while a cue (yellow X) was on the	**Result** ⌖ Increase in phase synchrony of neural oscillations preceding speech onset in both HC and SCZ ⌖ Smaller prespeech neural synchrony in SCZ ⌖ No association between prespeech signal and N1 suppression (sign of cortical responsiveness to

	be related to subsequent sensory suppression; this feature is deficient in SCZ **Aim** ⌖ To quantify the neural correlates of the efference copy associated with speaking, assess group differences in this neural signal and relate it to AVH	screen (1.66 seconds). This was repeated five times in each of the six talk blocks. During the talk task, utterances were recorded for playback during the listen task. During the listen task, the visual display was similar to the talk task; cues were seen as before, but the instruction "TALK" was replaced with "LISTEN."	speech sounds) in SCZ unlike HC ⌖ Prespeech intertrial coherence was larger during talking than listening ⌖ Degree of prespeech synchrony in SCZ inversely correlated with auditory hallucination severity **Implication** ⌖ The observed premovement burst of synchronous neural activity reflects forward model in action, preparing the CNS for the sensory consequences of self-initiated actions ⌖ Apart from dampening irrelevant sensations resulting from our own actions, the forward model aids in differentiating between internally and externally generated percepts ⌖ "If an efference copy of a thought or inner experience does not produce a corollary discharge of its expected sensory consequences, internal experiences may be experienced as external".
(Heinks-Maldonado et al., 2007)	**Premise** ⌖ Feed forward model posits that impressions of self-generated actions prepare sensory cortex for incoming sensation from those actions ⌖ CD dysfunction in SCZ may contribute to the misperception of inner experiences and thoughts as AVH	**Methods** ⌖ **Location**: California, USA ⌖ **Sample**: SCZ=23, HC=23 ⌖ **ERP Paradigm**: Modified talk-listen paradigm was used. **Talk condition:** Participants made short (<300ms), sharp vocalizations of the phoneme "ah" repeatedly in a self-paced manner, about every 1–2 s, for 180 s. This speech was recorded using a microphone connected to the	**Result** ⌖ In HC, N100 suppression during talk to unaltered voice feedback > N100 suppression during talk to altered voice feedback, especially over left hemisphere ⌖ N1 suppression produced in SCZ did not show a graded pattern to altered feedback unlike HC group ⌖ The pattern of auditory cortical responses in non-hallucinators was heterogeneous ⌖ The strength of the N100 amplitude suppression

	Aim ⌖ To assess the precision of the forward model in SCZ using the N1 component of the auditory event-related potential to spoken speech in real time. ⌖ To explore the relationship between AVH and CD	stimulus presentation computer and transmitted back to subjects through headphones in real time (zero delay). **Listen condition**: The feedback-voice heard via headphones was varied randomly between their own unaltered voice (self, unaltered), their own voice pitch-shifted downward by 2 semitones (self, pitch-shifted), the alien unaltered voice (alien, unaltered), and the alien voice pitch-shifted downward by 2 semitones (alien, pitch-shifted). **Behavioral Response**: After each trial, subjects were prompted to indicate via button press whether the feedback heard was their own voice or the alien voice or whether they were unsure. Response window was ser within 1.5 seconds after the prompt.	effect correlated with AVH severity ⌖ Hallucinators had the smallest N1 suppression effect (to unaltered voice feedback); non-hallucinators were comparable to controls **Implication** ⌖ Better N1 suppression to unaltered voice feedback reflects a precise forward model mechanism that enables the auditory system to distinguish between internal and external sources of auditory information ⌖ CD in SCZ is not very precise and, instead, is more generally dysfunctional ⌖ heterogeneous pattern of cortical suppression in non-hallucinators may reflect differences in hallucination history, and raises the question about whether the pattern of N100 suppression reflects hallucination state or trait ⌖ The smaller the N100 suppression effect, the more errors and the more severe the hallucinations. ⌖ "Misattribution" is not part of the hallucinatory experience, but part of the delusional system used to explain the aberrant experience while "misperception" more accurately describes auditory hallucinations
(Ford et al., 2002)	**Premise** ⌖ Auditory cortical responsiveness is reduced during talking due to CD from frontal speech	**Methods** ⌖ **Location**: California, USA ⌖ **Sample**: SCZ=07, HC=07 **ERP Paradigm:** Three equiprobable probes, each 250 m in duration, were	**Result** ⌖ An interdependence between frontal and temporal areas was observed during talking in HC, this feature was somewhat disrupted in SCZ, especially those prone to auditory hallucinations.

	producing areas to the speech reception areas in the temporal lobe ⌖ Coherence is a frequency-dependent measure of the degree of relatedness between EEG recorded over two different brain areas **Aim** ⌖ To assess the role of frontal speech area involvement in CD mechanism, by calculating the degree of inter-relatedness between frontal and temporal lobes during talking compared to listening, using EEG coherence algorithms	presented at random ISIs (0.8, 1.0 or 1.2 s): speech syllable [ba], broadband noise, and square checkerboard. The probe sequence was presented to subjects continuously during both Listening and Talking conditions. The first prerecorded hallucinatory statement was repeated back to the subject for 30 sec (about seven repetitions). Next, the subject repeated the same statement aloud for another 30 sec. They did so silently as well in silent condition. This Listen/Talk sequence was repeated for each of the seven hallucinatory statements, for a total of 7 min. **Analysis of Coherence**: Data was translated data from time to frequency domain via Fast Fourier Transform (FFT) at all points. Coherence was calculated for each frequency band of interest (delta: 1–3 Hz; theta: 4–7 Hz; alpha: 8–12 Hz; beta: 13–20 Hz; gamma: 30–50 Hz). Coherence is the spectral cross-correlation between two electrodes normalized by their power spectra	**Implication** ⌖ The greater frontal-temporal coherence during talking than listening in HC may reflect the action of a corollary discharge from frontal brain structures preparing auditory cortex for speech ⌖ In HC, high levels of activation in the frontal speech areas could be related to high levels of excitation or inhibition in the temporal lobes ⌖ Increased coherence during talking implies a continuous dialogue between neural systems responsible for producing speech and those involved in perceiving its effects ⌖ N1 suppression that reflects suppression of the auditory cortex responsiveness is perhaps due to a connection between the frontal and temporal lobes that is broken or disrupted in SCZ ⌖ Normal effects of talking and inner speech are disrupted in SCZ
(Ford et al., 2001a)	**Premise** ⌖ CD dysfunction of auditory system in SCZ underlies	**Methods** ⌖ **Location**: California, USA ⌖ **Sample**: SCZ=07, HC=07	**Result** ⌖ HC group produced smaller N1s to uttered than played back vowels

	certain positive symptoms like AVH **Aim** ⊕ To examine CD in SCZ by ruling out the possibility of differential attention effects on N1 on speech by using 'talk' as the probe	⊕ **ERP Paradigm:** Talk-listen paradigm. Subjects uttered syllables [*a*] and [ei] for about 3 min. Each subject's self-generated vowel sequence was recorded and played back to them through head phones, after first adjusting the gain to equalize loudnesses during playback and talking	⊕ SCZ group did not show this reduction in N1 to their own utterances **Implication** ⊕ In normal individuals a speech-related CD suppressing responsiveness of auditory cortex to self-generated speech sounds occurs ⊕ CD mechanism of auditory cortex suppression is dysfunctional in schizophrenia
(Ford et al., 2001b)	**Premise** ⊕ CD dysfunction of auditory system in SCZ mediated as failure to recognize self-generated action underlies certain positive symptoms of SCZ like AVH **Aim** ⊕ To examine auditory cortical responsiveness to probes presented to participants while they sat silently (Baseline), spoke out loud (Talking), and while they heard their speech played back to them (Listening)	**Methods** ⊕ **Location**: California, USA ⊕ **Sample**: SCZ=10, HC=12 ⊕ **ERP Paradigm:** Three equiprobable probes, each 250 m in duration, were presented at random ISIs (0.8, 1.0 or 1.2 s): speech syllable [ba], broadband noise, and square checkerboard. **Baseline Condition**: Subjects sat upright heard the sounds, and faced a video monitor to see the checkerboard with eyes focused on a fixation point on the screen throughout the experiment. The presentation of stimuli lasted 2 min and 42 sec. **Talk and listen condition**: The same sequence of probe stimuli was presented while subjects alternated between listening to their own prerecorded voice repeating a	**Result** ⊕ Talking affected N1 to acoustic but not to visual probes ⊕ In HC, N1 to acoustic probes was reduced during Talking compared to Baseline. In SCZ, N1 to acoustic probes was not smaller during Talking compared to Baseline. ⊕ Baseline N1 amplitude was smaller in SCZ than in HC. ⊕ Inner speech reduced responsiveness of auditory cortex in HC group; compared to baseline, N1 to acoustic probes was reduced when control subjects were repeating sentences silently to themselves ⊕ Inner speech didn't reduce auditory cortical responsiveness in SCZ **Implication** ⊕ N1 suppression effects are modality specific ⊕ Corollary discharge happens automatically, without motivation or effort

	hallucinatory statement (e.g., "Get off your duff and do something") for 30 s followed by repeating that same statement for another 30 s. This alternation was repeated for seven different hallucinatory statements. This Listen/Talk sequence lasted a total of seven min. **Inner speech and listen Condition:** Following the presentation of probes during the silent baseline condition, self-recorded hallucinatory statements, repeated for the 30 s recording were alternated with the subjects repeating that same statement silently to themselves for 30 s. This listen/inner speech sequence was repeated seven times, once for each of seven different statements and lasted seven minutes.	⌖ As a measure of cortical responsiveness, N1 is minimally affected by attention, and these attention effects on N1 could possibly obscure our ability to detect the action of the corollary discharge ⌖ Corollary discharge may signal speech reception areas that speech-related activations are self-generated, avoiding misperceptions that these thoughts have an external source.

SCZ – Schizophrenia / schizophrenia patients, HC – Healthy Controls, CD – Corollary discharge, ERP – Event related potential, AVH – Auditory verbal hallucination, SA – Schizo-affective patients, BPP – Bipolar disorder patients, CHR – Clinically high risk for psychosis (Individuals with attenuated Positive Symptoms, brief Intermittent Psychotic States, and/or Genetic Risk with Deterioration in social/occupational functioning); ESCZ – Early illness SCZ (individuals who fall within 2.5 years of initial hospitalization for psychosis or initiation of antipsychotic medication), FA – Fractional Anisotropy

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