Published in final edited form as:

Cell. 2016 March 10; 164(6): 1269–1276. doi:10.1016/j.cell.2016.02.039.

Insights into the Neural and Genetic Basis of Vocal Communication

Genevieve Konopka[*] and **Todd F. Roberts**[*]

Department of Neuroscience, UT Southwestern Medical Center, Dallas, TX 75390-9111

Abstract

The use of vocalizations to communicate information and elaborate social bonds is an adaptation seen in many vertebrate species. Human speech is an extreme version of this pervasive form of communication. Unlike the vocalizations exhibited by the majority of land vertebrates, speech is a learned behavior requiring early sensory exposure and auditory feedback for its development and maintenance. Studies in humans and a small number of other species have provided insights into the neural and genetic basis for learned vocal communication and are helping to delineate the roles of brain circuits across the cortex, basal ganglia and cerebellum in generating vocal behaviors. This Review provides an outline of the current knowledge about these circuits, the genes implicated in vocal communication, and a perspective on future research directions in this field.

Keywords

speech; language; sensorimotor circuits; human brain; songbird; FOXP2

INTRODUCTION

The insights discussed in this Review have been largely attained through the study of developmental disorders affecting speech and analysis of neuronal circuits in songbirds and mice. Genetic screens of individuals with inherited forms of speech disorders, like verbal dyspraxia, stuttering and some types of autism, have allowed for the identification of a number of genes (*FOXP2*, *CNTNAP2*, *FOXP1*, *GNPTAB*, *GNPTG*, *NAGPA*) involved in speech and/or social-cognitive development that can now be studied using animal models (Konopka and Roberts, 2016; Lepp et al., 2013). Of these, the transcription factor *FOXP2* has been the most intensively studied. Mutations of *FOXP2* in humans are associated with an inherited verbal dyspraxia, a speech disorder that results from difficulties in controlling orofacial muscles. The study of FOXP2 is now providing significant insights into the underpinnings of vocal motor learning and the development of neuronal circuits.

[*]Correspondence: genevieve.konopka@utsouthwestern.edu (G.K.), todd.roberts@utsouthwestern.edu (T.F.R.).

Songbirds have long been the predominant model for studying the neural circuit mechanisms for vocal learning (Doupe and Kuhl, 1999; Mooney et al., 2008). Like human speech, birdsong is learned during a developmental sensitive period and requires early sensory exposure to a vocal model (song tutor) and auditory feedback for its normal development and maintenance. Studies in songbirds have revealed a well delineated neural circuit spanning from the cortex to the brainstem that is necessary for song learning and song production. The organization of this song circuit is similar to the core cortical and basal ganglia circuits involved in speech (Doupe and Kuhl, 1999; Jarvis, 2004). In addition, knockdown of the transcription factor FoxP2 in songbirds disrupts song development in a manner similar to disruptions seen in human speech development, indicating analogous circuit and gene regulatory mechanisms for song and speech (Fisher and Scharff, 2009; Haesler et al., 2007; Haesler et al., 2004; Lai et al., 2001; Murugan et al., 2013). Despite these important behavioral and neurobiological parallels between birdsong and speech, studies in songbirds have been limited by the lack of methods for efficiently and precisely editing the avian genome; however, the recent development of transgenic songbirds (Abe et al., 2015; Agate et al., 2009; Liu et al., 2015; Scott et al., 2010), advances in viral vector methods and gene editing tools (Betley and Sternson, 2011; Heidenreich and Zhang, 2016; Roberts et al., 2012; Roberts et al., 2010), and the sequencing of the avian genome (Warren et al., 2010; Zhang et al., 2014) all promise to enrich the continued use of songbirds in the study of speech disorders.

The genetic accessibility of mice and the wide range of molecular and genetic tools available for studying the mouse brain provides a powerful platform for examining how genetic disorders affect the central nervous system, and how genes implicated in speech and social/cognitive disorders impact neuronal circuit development and synaptic function. Mice exhibit both neonatal calls as well as adult vocalizations pertinent to social interactions (Scattoni et al., 2009). However, it should be appreciated that unlike speech and birdsong, vocal behaviors in mice are not learned from social models using auditory feedback. For instance, deaf mice can develop normal vocalizations (Portfors and Perkel, 2014). This lack of vocal learning limits the use of mice for modeling speech development. However, their vocalizations still allow studying motor and auditory brain circuits involved in vocal communication (Holy and Guo, 2005).

Overall, it is important to note that comparing vocalizations among humans, songbirds and mice will always be challenging. While there is significant conservation of brain structures and genes among these divergent species, human language, characterized by speech and sign-based forms of communications in deaf communities, has a level of complexity and abstraction that may well be unique and thus difficult to model. Furthermore, vocal behaviors in mice and some species of songbirds are sexually dimorphic and sensitive to sex-steroids, further underscoring the different evolutionary trajectories associated with vocal communication. However, by focusing on brain structures associated with speech: the cortex, basal ganglia, and cerebellum, we here provide touchstones for comparing and integrating genetic and neural circuit data from songbirds and mice with data from humans.

Cortex

The observation that brain lesions of the inferior frontal cortex lead to a disruption in speech production (expressive aphasia) in the late 1800s heralded the study of brain functions out of the dark ages of phrenology and provided one of the first insights into the brain mechanisms for vocal communication (Dronkers et al., 2007). This work by Paul Broca was soon followed by that of Karl Wernicke who found that lesions of the superior temporal gyrus (STG) led to a deficit in speech perception (receptive aphasia) (Mathews et al., 1994). These early descriptions, along with later accounts provided by the pioneering work of the neurosurgeon Wilder Penfield who carried out stimulation and recording of specific neocortical areas in awake patients (known as electrocorticography, ECoG, or intracranial electroencephalography, iEEG) (Penfield and Rasmussen, 1949), laid the basis for attributing neural mechanisms to speech and language. Modern approaches have additionally used non-invasive techniques such as magnetoencephalography (MEG), transcranical magnetic stimulation (TMS), and functional MRI (fMRI) to study speech in both patient and neuro-typical populations (for an in-depth discussion and primary references please see (Cattaneo, 2013; Chang et al., 2015; Devlin and Watkins, 2007; Poeppel, 2012; Price, 2010)).

The use of these techniques has revealed that the early divisions of speech production and perception into independent cortical regions were overly simplistic (see references in (Hickok et al., 2011)). For example, premotor cortex may modulate speech perception, and auditory areas (e.g. STG) are thought to influence speech production. The integration of these feedback loops among speech-related cortical areas permits ongoing learning, maintenance, and refinement of speech. Interestingly, a bilateral ECoG study directly demonstrated the existence of sensory-motor integration during speech and also provided evidence for bilateral neural activity in contrast to much of the work focusing on left hemisphere lateralization of language (Cogan et al., 2014).

Researchers have recently begun to parse the neuronal substrates for perceiving and producing the basic elements of speech. Application of ECoG allowed the determination of the neural responses to specific phonemes, or units of sound, during speech perception, showing that there are discrete and localized invariant responses to specific phonemes in the STG (Mesgarani et al., 2014). In addition, MEG of the cortex was recently used to identify the timescales of linguistic structure in a study of speech perception (Ding et al., 2015). Multi-electrode recordings have also recently helped map the spatial representation of phonetic features for speech production in the ventral sensorimotor cortex (adjacent to the so-called “Broca’s area” in the inferior frontal cortex) (Bouchard et al., 2013). Building upon over a century of work, these and other studies are redefining areas of the cortex important for speech.

These insights into speech production and comprehension are pertinent to the understanding of genetic and neuropsychiatric disorders that affect speech and language. Structural imaging of individuals with *FOXP2* mutations have identified both increases and decreases in gray matter in several cortical regions associated with speech such as the STG and the inferior frontal gyrus (Belton et al., 2003; Watkins et al., 2002). fMRI studies of some of

these individuals have also found decreases and/or alterations in cortical brain activity during word and non-word repetition paradigms (Liegeois et al., 2003; Liegeois et al., 2011), suggesting that deficits in cortical function may be associated with language difficulties imposed by this mutation possibly as a consequence of altered cortico-cerebellar or cortico-striatal circuitry. Disruption of corollary discharge pathways linking motor and auditory cortical circuits are speculated to contribute to auditory hallucinations and "imaginary inner speech" in schizophrenia ((Heinks-Maldonado et al., 2007; Horga et al., 2014) and references in (Hugdahl, 2015)). Deficits in vocal communication are also associated with autism spectrum disorders (ASD). Patients with ASD related syndromes or the more severe diagnosis of intellectual disability often have speech delay or can even be completely non-verbal. At the functional level, a reduction in left hemispheric lateralization of language has been observed in ASD patients as well as changes in prosody, verbal fluency and activation of non-typical language areas ((Kleinhans et al., 2008) and references in (Dichter, 2012)). Recent fMRI work has demonstrated hypoactivation of the STG in patients with ASD who exhibit language problems, suggesting that this fMRI signature could be used as a biomarker for ASD patients who will progress to poor outcomes and presenting an opportunity for therapeutic intervention (Lombardo et al., 2015).

Studies in songbirds have provided important insights into the architecture and function of cortical circuits for vocal communication. First, cortical song circuits involved in production of learned song are separable from those involved in vocal plasticity (Aronov et al., 2008; Brainard and Doupe, 2000; Charlesworth et al., 2012; Olveczky et al., 2005). Song-related cortical pathways associated with cortico-basal-ganglia-cortical loops are essential for feedback dependent changes in vocal behavior, like tutor song imitation in juvenile birds and the deterioration of song structure following the loss of hearing. Yet, lesions in cortical portions of this circuit do not disrupt the birds' ability to produce previously learned vocal behaviors (Bottjer et al., 1984; Brainard and Doupe, 2000; Olveczky et al., 2005; Scharff et al., 2000). Second, researchers have identified the song premotor region HVC as a critical structure for encoding learned song and considerable research effort has begun to elucidate how stereotyped vocal sequences are organized and represented in HVC (Amador et al., 2013; Hahnloser et al., 2002; Kosche et al., 2015; Long and Fee, 2008; Long et al., 2010; Markowitz et al., 2015; Okubo et al., 2015; Peh et al., 2015; Wang et al., 2008). Third, recent studies have revealed an essential role of song motor circuits, including HVC, in learning from sensory experience of a vocal model (Roberts et al., 2012) and indicate that sensory experience of the tutor and learning of vocal motor sequences both have a profound influence on shaping the functional organization of song motor programs during development (Adret et al., 2012; Bolhuis and Moorman, 2015; Mooney, 2014; Okubo et al., 2015; Prather et al., 2010; Roberts et al., 2012; Roberts et al., 2010; Shank and Margoliash, 2009; Vallentin et al., 2016). Songbirds have been intensively studied and we point the reader to a number of excellent reviews of songbird neurobiology (Bloomfield et al., 2011; Brainard and Doupe, 2002, 2013; Brawn and Margoliash, 2015; Doupe and Kuhl, 1999; Kuebrich and Sober, 2015; Mooney, 2014; Roberts and Mooney, 2013; Schneider and Mooney, 2015; Tschida and Mooney, 2012)

The role of the cortex in mouse vocalizations is less clear and mice genetically altered to lack large parts of the cortex still produce normal adult vocalizations (Hammerschmidt et al.,

2015). However, it has recently been shown that alteration of the gene *SRPX2* specifically in the cortex, results in changes to neonatal mouse pup vocalizations (Sia et al., 2013). *SRPX2* is of particular interest to the study of speech as it is associated with speech dyspraxia caused by rolandic seizures (Roll et al., 2006) and it is a transcriptional target of FOXP2 (Roll et al., 2010). Therefore, further studies are required to determine whether other brain regions and circuits developmentally compensate for the function of cortex in mouse vocalizations and how specific genes are involved in cortical function and circuits that affect vocalizations.

Basal ganglia

The basal ganglia are a set of interconnected forebrain nuclei that are critically involved in the control of motor behaviors and learning. These brain regions include the striatum (caudate and putamen in primates), globus pallidus or pallidum, the subthalamic nucleus and the substantia nigra. The basal ganglia receive input from the cortex and provide feedback to the cortex via a series of pathways that loop through the thalamus. A number of excellent reviews of the general role of basal ganglia function in motor control, learning and disease states have been published (Enard, 2011; Graybiel, 2008; Gunaydin and Kreitzer, 2015; Nelson and Kreitzer, 2014; Redgrave et al., 2010; Shepherd, 2013; Tritsch and Sabatini, 2012); here we briefly review the role of the basal ganglia in vocal control.

Basal ganglia circuits play a critical role in motor control and the learning of sequential motor behaviors, including speech production in humans (Watkins, 2011). Ischemic strokes affecting the caudate nucleus can result in language impairment and damage to thalamic nuclei linking the basal ganglia with the cortex are consistently linked with disruptions of speech (Barbas et al., 2013; Gronholm et al., 2015). Interestingly, stuttering is associated with functional abnormalities in the basal ganglia circuits, and other forms of inherited speech disorders have been linked to disruptions of the dorsal striatum (Alm, 2004; Belton et al., 2003; Craig-McQuaide et al., 2014; Watkins, 2011; Watkins et al., 2002). Further evidence for a role of the basal ganglia in speech comes from insights into diseases that disrupt the functioning of basal ganglia circuits, such as Huntington's disease, which also affects the motor control of speech. Huntington's disease results in cell death in the striatum and is characterized by progressive development of involuntary movements and can lead to problems with sequencing of motor movements for speech and problems swallowing. Although the vocal disruptions associated with Huntington's disease have not yet been modeled in mice (Pouladi et al., 2013), transgenic songbirds expressing the human mutant Huntington gene have recently been shown to also develop disruptions in the vocal control of their learned song, including progressive disruptions in song sequencing, and stuttering (Liu et al., 2015).

Perhaps the greatest insights into the role of the basal ganglia for vocal control in mammals have emerged from the study of FOXP2. As previously mentioned, FoxP2 mutations results in deficits in coordinated movements required for speech. We point the reader to several reviews on FOXP2 (Enard, 2011; Fisher and Scharff, 2009; French and Fisher, 2014; Newbury and Monaco, 2010; Scharff and Petri, 2011) and we here highlight what has been gleaned about basal ganglia circuits from its study.

FOXP2 is highly expressed in the human dorsal striatum during development, as well as the dorsal striatum of other mammals, songbirds and reptiles (Haesler et al., 2004; Teramitsu et al., 2004). Imaging studies have shown that the dorsal striatum is severely impacted in individuals with mutations of *FOXP2*, implicating both the dorsal striatum and FOXP2 in speech learning and production. Within the dorsal striatum, FoxP2 is highly expressed in medium spiny neurons (MSNs) (Schulz et al., 2010; Takahashi et al., 2003). Striatal MSNs integrate glutamatergic inputs from the cortex and dopaminergic inputs from the midbrain, and function in the control of motor behaviors, action selection and learning of motor sequences. Heterozygous *Foxp2* mice exhibit decreased synaptic plasticity at corticostriatal synapses and increased levels of extracellular dopamine in the striatum. In contrast, expression of human FOXP2 in the mouse is associated with increased synaptic plasticity at corticostriatal synapses, decreased levels of extracellular dopamine, and enhanced transitions from declarative to procedural learning (Enard et al., 2009; Groszer et al., 2008; Schreiweis et al., 2014). These data are in line with the transcriptional program of human FOXP2 regulating genes involved in brain and craniofacial development (Konopka et al., 2009).

In songbirds a specific region of the dorsal striatum, termed Area X, plays a dedicated and exclusive role in song learning. Lesions to Area X during the sensitive period for vocal learning prevent accurate vocal imitation of a tutor's song and the normal development of stereotyped vocalizations by adulthood (Scharff and Nottebohm, 1991). FoxP2 is strongly expressed in Area X, and knockdown (KD) of FoxP2 in Area X of young zebra finches, using hairpins against FoxP2 mRNA, causes an increase in vocal variability and prevents birds from accurately copying the song of their tutor (Haesler et al., 2007). The disruption in vocal learning and vocal-motor variability in songbirds following FoxP2-KD is reminiscent of the orofacial motor disruptions seen in humans carrying a nonfunctional *FOXP2* allele (Haesler et al., 2007; Murugan et al., 2013). Moreover, KD of FoxP2 in Area X of zebra finches renders MSNs insensitive to dopamine receptor (DR1) agonists or antagonists (Murugan et al., 2013), and leads to decreased spine density on MSNs (Schulz et al., 2010). Male zebra finches produce a song with less trial-by-trial variability when singing directly to a female bird than when practicing their song in isolation or singing in the presence of male birds. This context-dependent ability of male birds to sing more stereotyped song in the presence of a female is abolished following infusion of a D1R antagonist into Area X or following FoxP2-KD in Area X (Haesler et al., 2007; Leblois and Perkel, 2012; Leblois et al., 2010; Murugan et al., 2013). This effect on song production is thought to result from a disruption in the timing of information flow through the basal ganglia and D1R antagonists and FoxP2-KD both result in shortening of synaptic delays through the basal ganglia circuits (Murugan et al., 2013). These data indicate that FoxP2 impacts vocal behavior by regulating postsynaptic dopaminergic signaling, synaptic plasticity, and the flow of signals through the striatum.

The highly homologous gene *FOXP1* is among the group of 71 significant recurrent *de novo* mutations associated with ASD (Sanders et al., 2015). In the mouse, *Foxp1* is one of the 100 most abundant striatal-enriched genes, and therefore, its dysfunction is likely to have a significant impact on that structure (Heiman et al., 2008). Brain-wide deletion of *Foxp1* in

mouse results in autism-relevant behaviors (Bacon et al., 2014), and patient-relevant haploinsuffienct *Foxp1* mice exhibit altered vocal communication, dysregulation of known ASD and Foxp2 target genes in the striatum, and changes in medium spiny neuron excitability (Araujo et al., 2015). Future studies that dissect out the distinct and overlapping contributions of FoxP1 and FoxP2 to basal ganglia function should be informative for understanding vocal communication.

Cerebellum

The cerebellum functions at multiple levels of sensorimotor integration including sensory acquisition, processing timing information and prediction of motor output (Manto et al., 2012). The role of the cerebellum in sensorimotor integration has been known for decades based on lesions and degenerative disorders in patients (see references in (Murdoch, 2010)). However, there has been relatively less research into sensorimotor integration in the cerebellum as it pertains to speech and language (compared to the cortex or basal ganglia), and this is surprising given a centuries old literature of patients with cerebellar lesions having speech alterations (Holmes, 1917; Murdoch, 2010). In patients with *FOXP2* mutations, one of the areas of the brain with significantly altered amounts of grey matter is indeed the cerebellum (Watkins et al., 2002).

We refer the reader to a recent consensus paper on the role of the cerebellum in language (Marien et al., 2014), but outline some key points from that consensus here. Similar to the cortex, the cerebellum is involved in speech perception and participates in parsing phonetic information. In particular, the evaluation of timing information from incoming signals appears to require cerebellar function. The cerebellum is also important for speech production and is involved in the rate of production, particularly at the phonological level. The coordination of the vocal tract in speech is also at least partly dependent on cerebellar input. Another critical facet of the cerebellum is the differential functional localization of speech attributes at a regional level. For example, specific lobules and specific medial-lateral portions of lobules can be dissected into those contributing to sensorimotor control versus those involved in other cerebellar functions.

Beyond sensorimotor control, the cerebellum is also important for the higher order components of speech and language, as there is growing evidence for cerebellar involvement in cognition and affect (Stoodley and Schmahmann, 2010) with functional connectivity studies supporting connections between the cerebellum and cortical areas (Habas et al., 2009; Krienen and Buckner, 2009). Such evidence fits with disruption in cerebellar function in numerous neuropsychiatric diseases. For example, the cerebellum has been increasingly recognized as a key brain region in ASD pathophysiology (see references in (Becker and Stoodley, 2013; Hampson and Blatt, 2015; Mosconi et al., 2015)). In particular, Purkinje neurons, which are the sole motor output of the cerebellum, appear to be particularly vulnerable in ASD and relevant to vocal production. Of note, FOXP2 expression in the cerebellum is limited to the Purkinje neurons and mouse models of Foxp2 exhibit striking cerebellar defects as well as altered vocal behavior (see references in (Usui et al., 2014)). The ASD-related gene *Tsc1* has also been specifically deleted in Purkinje neurons in mice leading to ASD-relevant behaviors including changes in vocalizations (Tsai et al., 2012). It

remains to be seen if the other cell types in cerebellum, like granule cells for example, are vulnerable in disorders impacting speech or if Purkinje neurons play a particularly privileged role. Together, these data support an important role for the cerebellum in speech across numerous levels from genes to neural activity to circuits. Interestingly, the role of the cerebellum in birdsong learning and song motor control is underexplored and a research area requiring more attention given the behavioral findings in humans and mice.

Future Directions

With only a handful of genes studied to date and a limited understanding of the brain circuits contributing to specific aspects of speech, there have nonetheless been significant inroads made toward understanding the genetic and neural basis of speech. However, there is clearly a need for the identification of additional genes and further use of model systems to better understand the neural circuits underlying vocal communication.

The use of human patients with damage in speech-related brain areas has served as both a historical and modern basis for understanding brain regions, circuits and neural functions underlying speech. More recently, studies of patients undergoing surgery for epilepsy have allowed a more detailed understanding of the neural substrates of speech. However, such invasive procedures will always be limited to patient populations, and there is a need to increase the resolution of non-invasive approaches such as MEG and fMRI to study speech in neuro-typical populations. For example, recent fMRI work has been able to extricate overlapping sets of neural activity in the auditory cortex to identify distinct responses to either speech or music (Norman-Haignere et al., 2015). Furthermore, correlations between resting-state fMRI and gene expression have also recently been uncovered (Hawrylycz et al., 2015; Richiardi et al., 2015; Wang et al., 2015), but future studies that combine such approaches with speech-related task-based imaging, including investigations beyond traditional areas of speech and language (Blank et al., 2015) should provide insights into genomic correlates of speech and a deeper understanding of speech-related brain networks.

There are ongoing efforts to identify additional genes important for speech through genome wide association studies of both endophenotypes in patient populations and neuro-typical populations. While the majority of these studies have been underpowered in terms of numbers of samples, a few hits have been observed that might make sense in terms of brain expression or function such as *SCN11A* or *ROBO2* (see references in (Graham and Fisher, 2015)). As has been seen in other fields, additional large-scale studies will need to be carried out to definitively identify other relevant genes. Once the gene list is in hand, the field will be able to dissect out the requirements for these genes at the neuronal and circuit level. There is also still much to be learned about *FOXP2*, 15 years after its initial association with speech. The use of animal models will be critical for the continuation of these studies and will likely require introduction of advanced genetic methods in songbirds and other species that exhibit complex vocal behaviors. For example, the study of vocal circuits in non-human primates, including the particularly social marmoset monkeys, is a burgeoning field. Indeed, recent studies in marmosets have shown that normal vocal development can be influenced by parental feedback (Takahashi et al., 2015). With the help of a ten year Brain/MINDS (Brain Mapping by Integrated Neurotechnologies for Disease Studies) project in Japan

focused on marmoset research (Okano et al., 2015), these small monkeys might play an important role in the future of neuroscience research aimed at better understanding neural circuits for vocal development.

Acknowledgments

We apologize for being unable to cover and cite all of the relevant literature due to space constraints. We thank Dr. Brad Lega for comments on the manuscript. Work from the authors' laboratories is supported by the UT Southwestern Jon Heighten Scholar in Autism Research (G.K.) and Thomas O. Hicks Scholar in Medical Research (T.F.R.), grants from the National Institutes of Health (R01DC014702, R01MH102603, R01MH103517, R21MH107672 to G.K. and R01DC014364 to T.F.R), National Science Foundation (IOS-1451034 to G.K. and T.F.R. and IOS-1457206 to T.F.R.), UT BRAIN Initiative Seed Grants (366582 to G.K. and 362808 and 365231 to T.F.R.), Friends of the Alzheimer's Disease Center at UT Southwestern (G.K.), and Klingenstein-Simons Award in Neuroscience (T.F.R.). T.F.R is an Essel Investigator supported by a NARSAD Young Investigator Grant from the Brain and Behavior Research Foundation.

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