Chapter 17

Aging auditory cortex: the impact of reduced inhibition on function

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List of abbreviations

EEG Electroencephalography
fMRI Functional magnetic resonance imaging
HG Heschl's gyrus
MEG Magnetoencephalography
PV Parvalbumin-expressing interneuron
SST Somatostatin-expressing interneuron
STG Superior temporal gyrus

Mini-dictionary of terms

Auditory cortex: Region of the cerebrum that is sensitive to features of sound.
Electro-/magnetoencephalography: Noninvasive measurement of neural activity with high temporal resolution, recorded at or close to the scalp.
Frequency tuning: Sensitivity of a neuron to certain sound frequencies.
Functional magnetic resonance imaging: Noninvasive measurement of brain activity with high spatial resolution based on blood oxygen levels.
Hyperexcitability: Increase in spontaneous or stimulus-evoked neuronal activity.
Neural adaptation: Reduction in neural response due to sustained stimulation.
Neural inhibition: Suppression of one neuron's activity by another neuron.
Neural plasticity: Experience-related development and/or reorganization of connections between cells in the brain.
Periodicity processing: Sensitivity of neurons to regular or quasi-regular fluctuations in amplitude and frequency of sounds.
Sensorineural hearing loss: Impaired hearing abilities due to cochlea damage.

Introduction

About 40% of people aged 50 years or older live with some form of hearing loss (Cruickshanks et al., 1998). This number is likely an underestimation, because hearing difficulties are often experienced decades before a diagnosis is made (Pichora-Fuller & Levitt, 2012), and some changes, such as a loss of synapses connecting hair cells in the cochlea to auditory nerves, may not be detected with current assessment tools (Liberman & Kujawa, 2017). The most common type of hearing loss (sensorineural) involves damage to the cochlea, where sound waves are converted to electrical signals. However, it is becoming increasingly clear that age-related hearing loss is a dysfunction of the entire auditory system. Damage to the auditory periphery induces neuroplastic changes throughout the auditory pathway, including auditory cortex (Salvi et al., 2017). Manifestations of this maladaptive plasticity may include phantom sound percepts (tinnitus), perceiving sounds as being too loud (hyperacusis), increased distractibility by sound, impaired sound localization, and difficulties with understanding speech in noisy situations such as a crowed restaurant.

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Here we review age and hearing loss–related changes in auditory cortex, with a specific focus on how reduced neural inhibition that occurs subsequent to hearing loss affects auditory functions. Since most older individuals have some form of hearing loss, we include literature related to age-related hearing loss and aging more generally. Moreover, we include research undertaken in animal models, where neurophysiological changes have been described in detail, in order to present plausible mechanisms by which behavioral deficits observed in humans may arise. We begin by describing auditory cortex anatomy and function in young, normal-hearing individuals and then reflect on changes associated with aging and hearing loss.

Anatomy and function of auditory cortex

Anatomical organization

Auditory cortex is located on the supratemporal plane of the superior temporal gyrus (STG) and is macroscopically divided into Heschl's gyrus (HG), planum polare, and planum temporale (Moerel, De Martino, & Formisano, 2014). HG is a diagonal structure of one or more convolutions extending laterally into the STG and medially to insular cortex (Clarke & Morosan, 2012). HG is bordered anteriorly by planum polare and posteriorly by planum temporale (Moerel et al., 2014, Fig. 17.1A). Human auditory cortex has been further subdivided into primary and nonprimary fields by mapping variations in cell type and density, chemical patterns, transmitter receptors, and myelin (Brewer & Barton, 2016; Clarke & Morosan, 2012, Fig. 17.1B). Primary auditory cortex receives the bulk of its sensory inputs from the thalamus and projects further to nonprimary regions. These areas, in turn, project to prefrontal cortex via anteroventral and posterodorsal pathways (Rauschecker & Scott, 2009). Descending connections project back from auditory cortex to subcortical and peripheral auditory structures (Lesicko & Llano, 2017).

Auditory cortex is comprised of 80% excitatory neurons and 20% inhibitory interneurons (Ouellet & de Villers-Sidani, 2014), and maintaining this balance is crucial for normal cortical network dynamics. As we describe further, a loss of inhibition likely underlies many age-related changes in auditory function. The most prominent inhibitory interneurons are those expressing either parvalbumin (PV) or somatostatin (SST). Parvalbumin interneurons tend to form synapses at the cell body of excitatory neurons and inhibit their output. Somatostatin interneurons synapse preferentially on dendrites of excitatory neurons, thereby suppressing neuronal inputs (Fig. 17.2A; Hattori, Kuchibhotla, Froemke, & Komiyama, 2017; Ouda, Profant, & Syka, 2015).

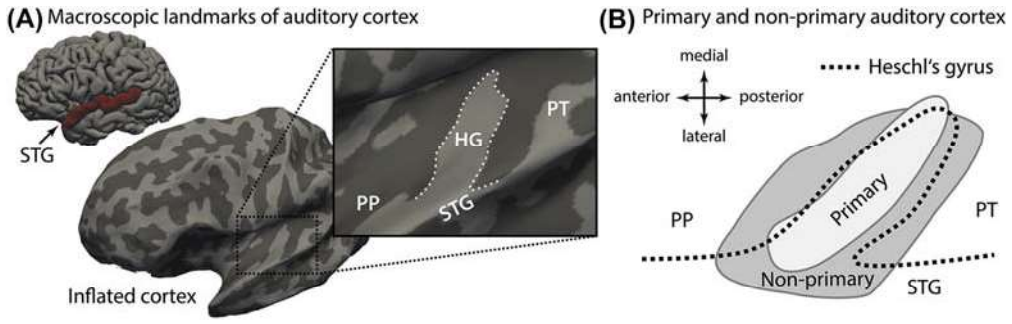

FIGURE 17.1 **Anatomy of human auditory cortex.** (A) Macroscopic landmarks. (B) Parcellation of auditory cortex (Brewer & Barton, 2016; Clarke & Morosan, 2012). *HG*, Heschl's gyrus; *PP*, planum polare; *PT*, planum temporale, *STG*, superior temporal gyrus.

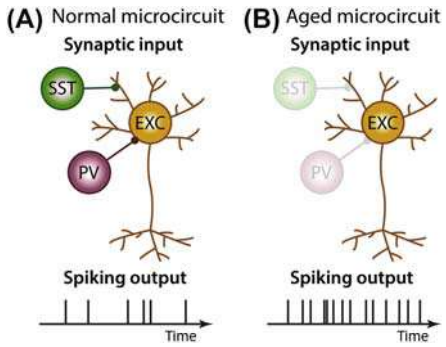

FIGURE 17.2 **Excitatory–inhibitory microcircuit.** (A) Inhibitory interneurons (PV, SST) regulate excitatory (EXC) neurons. (B) Loss of inhibitory neurons in aging.

Functional organization

Neurons in primary auditory cortex are most sensitive to pure tones (sounds comprised of a single frequency), whereas spectrally rich sounds (e.g., speech) most strongly activate nonprimary auditory cortex (Wessinger et al., 2001). Moreover, auditory cortex is organized tonotopically, such that neurons sensitive to similar sound frequencies tend to cluster together spatially (Moerel et al., 2014). Multiple tonotopic gradients with high-to-low-to-high-frequency organization have been identified in human auditory cortex, but the number of gradients and their spatial extent and orientation are still debated (Brewer & Barton, 2016; Moerel et al., 2014; Saenz & Langers, 2014). In addition to frequency, neurons in auditory cortex are also sensitive to sound periodicity (Brewer & Barton, 2016), the patterns of amplitude and frequency fluctuations in sounds to which neurons may synchronize their output (Goossens, Vercammen, Wouters, & van Wieringen, 2016). Neurons in human primary auditory cortex can encode periodicities up to about 150 Hz, whereas neurons in nonprimary regions have an upper cutoff of approximately 50 Hz (Billig et al., 2019).

As noted earlier, nonprimary auditory cortex sends ascending projections along both posterodorsal and anteroventral pathways: the former are thought to preferentially process a sound's spatial information, whereas the latter are thought to preferentially process nonspatial information (Rauschecker & Scott, 2009; Woods & Alain, 2009). Critically, each of these feature representations is crucial for auditory behaviors such as speech comprehension and is orchestrated by a balance between neural excitation and inhibition (Wehr & Zador, 2003).

Anatomy in the aged auditory cortex

Cortical morphometry

Aging is associated with a reduction in cortical surface area, cortical thickness, and gray matter volume throughout the human brain. Auditory cortex is no exception, but the decline with age seems to be less prominent compared to frontal and parietal cortex (Salat et al., 2006; Sowell et al., 2003). In addition, gray matter density also decreases with age; while the overall numbers of neurons and glial cells remain relatively stable, there are noticeable changes in their morphology over the life span (Conde & Streit, 2006; Ouda et al., 2015; Palmer & Ousman, 2018). How these cellular changes relate to macroscopic changes in cortical morphometry is still debated.

Peripheral hearing loss is associated with a decrease in gray matter volume in aged human auditory cortex beyond that which could be attributed to aging alone (Eckert, Cute, Vaden Jr., Kuchinsky, & Dubno, 2012), although this effect is not always reported (Ouda et al., 2015). In contrast, consistent changes in the myelinated fibers (white matter) that connect other brain regions to human auditory cortex have not been observed subsequent to age-related hearing loss (Ouda et al., 2015).

Loss of inhibition

A loss of inhibition in auditory cortex may be the most prominent change associated with aging and hearing loss (Caspary, Ling, Turner, & Hughes, 2008; Hattori et al., 2017; Salvi et al., 2017). Neurophysiological work in rats suggests that the number of PV and SST interneurons in primary auditory cortex decreases with age (Ouellet & de Villers-Sidani, 2014; other types of inhibitory neurons remain constant in number). Aging and hearing loss may thus lead to a disruption in the balance of excitation and inhibition in auditory cortex, reflecting reduced inhibition of inputs to and outputs from excitatory neurons due to a loss of SST and PV interneurons, respectively (Fig. 17.2B).

Magnetic resonance (MR) spectroscopy in older adults with and without hearing loss further suggests that a loss of inhibition in auditory cortex is associated hearing impairment (Gao et al., 2015). Estimations of the loss of specific types of inhibitory neurons in humans are rare, but one study examining PV interneurons found no age-related change (Bu, Sathyendra, Nagykery, & Geula, 2003). However, in this study the "young" brains examined comprised a sample of five adults with a median age of 50, such that the magnitude of age-related changes may have been underestimated. As we discuss in the next section, age and hearing loss related changes in human auditory cortex function closely resemble those observed in animal models demonstrating a loss of inhibition. We thus argue that decreased inhibition may underlie many of the behavioral changes in auditory function that have been observed in studies of aging and hearing loss.

Function in the aged auditory cortex

Changes in anatomy are expected to affect function. In this section, we focus on functional manifestations that may result from a loss of inhibition in auditory cortex following aging and hearing loss.

Hyperexcitability

Hearing phantom sounds (tinnitus) and perceiving sounds as being inordinately loud (hyperacusis) are common experiences reported by older people. A likely mechanism underlying these experiences is enhanced neuronal excitability—or hyperexcitability—that occurs subsequent to a reduction in inhibition (Eggermont, 2015; Knipper, Van Dijk, Nunes, Rüttiger, & Zimmermann, 2013).

Spontaneous activity (i.e., spiking of auditory cortical neurons in the absence of experimental sounds) increases in animals following acute traumatic (>100 dB SPL; Eggermont, 2015) and weeks-long nontraumatic noise exposure (~70 dB SPL; Munguia, Pienkowski, & Eggermont, 2013). Critically, humans experience this nontraumatic noise level commonly in their everyday lives (e.g., restaurants, streets). Aged, nonexposed animals show similar increases in spontaneous activity in the primary auditory cortex (Hughes, Turner, Parrish, & Caspary, 2010; Overton & Recanzone, 2016).

Neurons in the primary auditory cortex of aged and noise-exposed rodents (compared to young animals without hearing loss) also respond more strongly to sound (Knipper et al., 2013; Salvi et al., 2017). These observations have been corroborated for human auditory cortex using electro-/magnetoencephalography (EEG/MEG), showing sound-evoked response enhancements in older compared to younger adults and in older adults with hearing loss compared to older adults without (Herrmann, Henry, Scharinger, & Obleser, 2013; Tremblay, Piskosz, & Souza, 2003, Fig. 17.3).

Increased spontaneous activity in aging and hearing loss may be related to a loss of PV interneurons that regulate the output of excitatory neurons and, to a lesser degree, SST interneurons that regulate synaptic inputs (Natan, Rao, & Geffen, 2017). Moreover, we speculate that this same reduction of inhibition may contribute to sound-evoked hyperexcitability. Increased spontaneous activity may contribute to tinnitus, whereas sound-evoked hyperexcitability may manifest as hyperacusis (Eggermont, 2015; Knipper et al., 2013).

Reduced neural adaptation

Closely related to sound-evoked hyperexcitability is the observation that populations of neurons in auditory cortex recover faster from adaptation following sound stimulation in older compared to younger human adults. That is, the aged brain requires shorter refractory periods between successive stimulus-evoked responses (Herrmann, Henry, Johnsrude, & Obleser, 2016). Electrophysiological research in rodents demonstrates similar age-related changes at the single neuron level (Cisneros-Franco, Ouellet, Kamal, & de Villers-Sidani, 2018; de Villers-Sidani et al., 2010). Neural adaptation is a mechanism by which previous sensory information is "remembered" within neural circuits. Faster recovery from adaptation implies that previous sound information is less well represented in the response to a current sound. This reduced "memory" may impair the detection of change (de Villers-Sidani et al., 2010) and the dynamic fine-tuning of the auditory system based on statistical information in the environment (Herrmann, Maess, & Johnsrude, 2018).

Pharmacologically restoring inhibition in auditory cortex has been shown to restore normal adaptation in aged rodents (Cisneros-Franco et al., 2018). Moreover, optogenetic work in mice suggests that SST interneurons are crucial for regulating stimulus-specific neural adaptation, whereas PV interneurons regulate neural responses unspecific to previous

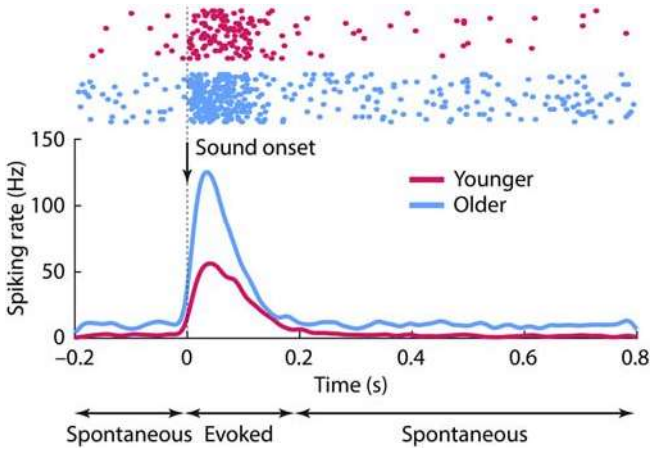

FIGURE 17.3 Age-related increase in spontaneous and sound-evoked activity. Simulated spiking activity for individual trials (top; *each dot* represents one spike) and the resulting time courses (bottom).

sound information (Natan et al., 2015, 2017). Hence, age-related loss of SST interneurons is a likely mechanism underlying changes in neural adaptation that impact the system's ability to capitalize on the environmental information to optimize sound processing.

Periodicity processing

Synchronization of neural activity with amplitude or frequency modulations that occur naturally in sounds is an important mechanism by which periodicity is represented in the brain. In speech processing, for example, the degree to which neural activity is synchronized to the amplitude envelope at the syllable/word rate (<10 Hz) is related to how well the speech signal is represented neurally.

EEG/MEG studies in humans demonstrate that neural synchronization with slow amplitude modulations (~4 Hz) in nonspeech sounds (Goossens et al., 2016) and with the amplitude envelope of speech (Presacco, Simon, & Anderson, 2016) is enhanced in auditory cortex of older compared to younger adults (Fig. 17.4). This enhanced synchronization may lead to an overrepresentation of unattended sounds in auditory cortex and has been hypothesized to underlie the common experience by older people that speech comprehension in the presence of other sounds is difficult (Millman, Mattys, Gouws, & Prendergast, 2017). Corroborating psychophysical work indicates that individuals with unilateral hearing impairment perceive slow amplitude modulations in sounds to fluctuate stronger in their hearing-impaired compared to normally functioning ear (Moore, Wojtczak, & Vickers, 1996).

Synchronization with faster modulation rates (>70 Hz) is decreased in aged nonhuman primates (Overton & Recanzone, 2016). Similar results have been observed in humans (Goossens et al., 2016; Purcell, John, Schneider, & Picton, 2004), although the EEG/MEG measurements utilized may pick up subcortical responses to these fast modulation rates in addition to those arising from auditory cortex. Critically, the reduction in neural synchronization with fast periodicities may indicate challenges in temporal fine-structure processing, a feature of sound that is important for pitch perception, sound localization, and identifying speech among fluctuating background noise (Hopkins, Moore, & Stone, 2008).

Inhibition is thought to be crucial for regulating sensitivity to sound periodicity (Caspary, Palombi, & Hughes, 2002), and changes observed in aged neural circuits may thus originate from a loss of inhibition (Rabang et al., 2012). This may reflect direct effects of changes to the balance of excitation and inhibition that regulates neurons encoding periodicity. Alternatively, it may represent a knock-on effect of inhibition-dependent changes in the properties of neural adaptation, such as recovery times (Augustin, Ladenbauer, & Obermayer, 2013). The role of specific subtypes of inhibitory neurons (e.g., PV, SST) in periodicity processing is unknown, and an investigation of the link between neural synchronization and inhibition in human auditory cortex—for example, assessed via MR spectroscopy—has not yet been undertaken.

Spectral processing

As described earlier, neurons in auditory cortex are frequency-tuned, responding preferentially to certain sound frequencies and less so to others. Narrow frequency tuning (in which neurons show an extreme preference for their characteristic frequency) may support speech processing in noisy situations by enabling the segregation of multiple concurrent auditory streams with different spectral profiles (e.g., male and female voices). Aging and traumatic sound exposure in animals are associated with broadened or unspecific frequency tuning and with tonotopic map reorganization, where neurons that previously responded to high frequencies shift their sensitivity to lower frequencies (de Villers-Sidani et al., 2010; Norena, Tomita, & Eggermont, 2003; Turner, Hughes, & Caspary, 2005). Even exposure to sounds at nontraumatic levels,

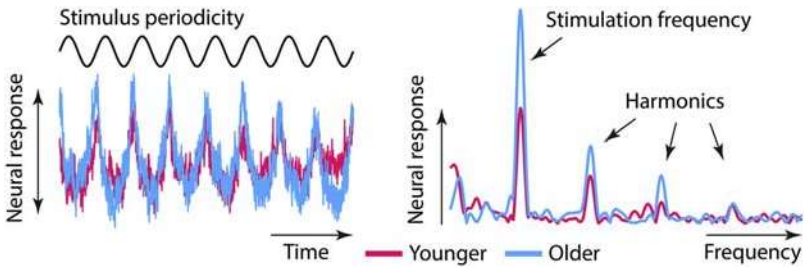

FIGURE 17.4 Age-related increase in sensitivity to low-frequency periodicity. Simulated neural responses in the time (left) and frequency domain (right; Fourier spectrum).

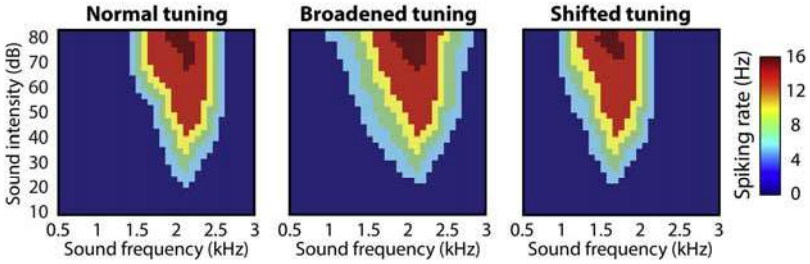

FIGURE 17.5 **Frequency tuning.** Schematic of normal, broadened, and shifted tuning.

including those humans encounter regularly, broadens the frequency tuning of auditory cortex neurons in animals (Thomas, Friedman, Cisneros-Franco, Ouellet, & de Villers-Sidani, 2019).

Neural inhibition shapes frequency tuning in auditory cortex via a process called "sideband inhibition" in which responses to frequencies just above and below the stimulus frequency are inhibited (Isaacson & Scanziani, 2011). Thus, reducing inhibition broadens tuning (Wang, McFadden, Caspary, & Salvi, 2002), whereas increasing inhibition narrows tuning (Aizenberg, Mwilambwe-Tshilobo, Briguglio, Natan, & Geffen, 2015). Optogenetic manipulations of auditory cortex neurons further suggest that narrow frequency tuning is mediated by PV and SST interneurons (Aizenberg et al., 2015; Kato, Asinof, & Isaacson, 2017) and that increased PV inhibition improves behavioral frequency discrimination (Aizenberg et al., 2015; SST interneurons were not tested). The loss of PV and SST interneurons with age (Ouellet & de Villers-Sidani, 2014) may thus broaden frequency tuning and may underlie challenges older people experience segregating multiple concurrent streams in noisy environments (Fig. 17.5).

FMRI has revealed that human auditory cortex is organized tonotopically, although some details are still debated (Moerel et al., 2014). The effects of age and hearing loss on tonotopic organization and frequency tuning in humans are largely unexplored using fMRI. One study observed a shift of neural sensitivity from high- to low-frequency sounds and an absence of clear tonotopic gradients in auditory cortex of people with severe hearing loss compared to people with normal hearing (Wolak et al., 2017). Nevertheless, previous reports also highlight the inconsistency of described age and hearing loss–related changes in tonotopic map organization and frequency tuning across human fMRI studies (Ouda et al., 2015; Saenz & Langers, 2014). EEG/MEG studies have reported tonotopic map reorganization (Mühlnickel, Elbert, Taub, & Flor, 1998) and broadened frequency tuning (Sekiya, Takahashi, Murakami, Kakigi, & Okamoto, 2017) in individuals with tinnitus, but the results are not always consistent across participants. Interestingly, although older participants show sound-evoked hyperexcitability in auditory cortex as measured by EEG (indicating reduced inhibition), they do not show evidence of changes in frequency-specific adaptation—a form of frequency tuning (Herrmann et al., 2013). Thus, while animal work clearly demonstrates age and hearing loss–related changes in spectral processing in auditory cortex, the available human data are insufficient to infer systematic changes in tonotopic organization or frequency tuning.

Spatial processing

The ability to spatially localize the origin of a sound is crucial for sound segregation in everyday life such as identifying the direction from which a car is approaching on a noisy street. However, spatial hearing abilities degrade as individuals age and acquire hearing loss (Koehnke & Besing, 2001).

Spatial information is processed preferentially in posterior-dorsal parts of auditory cortex (Rauschecker & Scott, 2009; Woods & Alain, 2009). Data from aged monkeys demonstrate that hyperexcitability (i.e., enhanced spontaneous and sound-evoked activity) is not limited to primary auditory cortex, but extends to spatially tuned posterior auditory fields (Juarez-Salinas, Engle, Navarro, & Recanzone, 2010). These data provide indirect evidence that neural inhibition decreases in spatially sensitive fields in posterior auditory cortex as individuals age (Juarez-Salinas et al., 2010).

Narrow tuning of neurons in posterior auditory cortex to specific sound locations is crucial for accurate sound localization. Yet, posterior fields in the aged auditory cortex exhibit broadened spatial tuning in addition to hyperexcitability (Juarez-Salinas et al., 2010; Fig. 17.6). This suggests that a loss of inhibition may drive the posterior field to respond similarly to many spatial locations instead of responding preferentially to one specific location, in turn, impairing sound localization. How different interneuron subtypes affect spatial tuning is unknown, but given the role of SST interneurons in spectral tuning, we expect them to be important for spatial tuning as well.

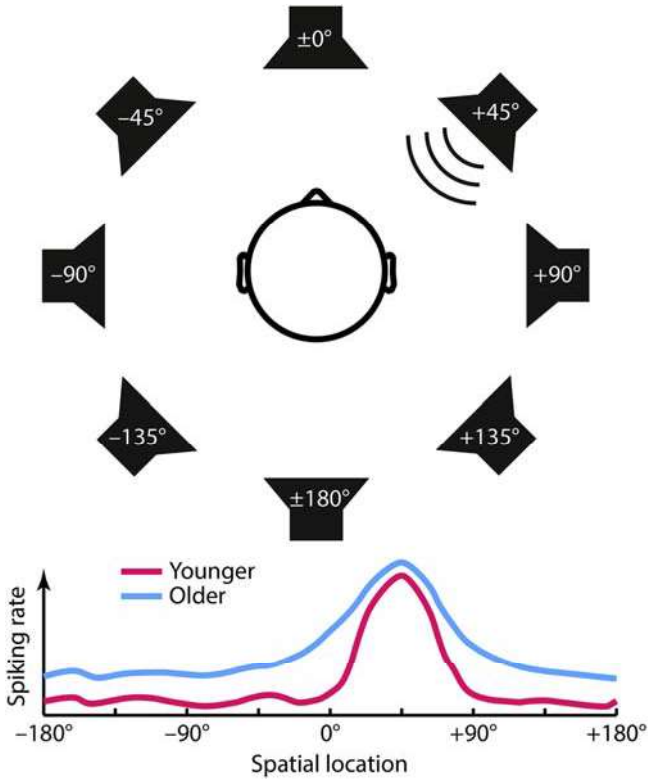

FIGURE 17.6 **Spatial tuning.** Schematic of age-related changes in neural sensitivity to spatial location.

Loss of inhibition facilitates neural plasticity

A loss of inhibition and subsequent hyperexcitability associated with aging and hearing loss are commonly thought to restore homeostasis and to compensate for peripheral decline of auditory sensitivity, such that reduced peripheral inputs are augmented subcortically and cortically (Caspary et al., 2008). However, recent work suggests that a loss of inhibition instead returns brain regions within the auditory pathway to a state of increased neural plasticity that resembles the period early in development during which inhibitory circuits have not yet matured (Cisneros-Franco et al., 2018). Since auditory cortex projects to nonauditory regions such as prefrontal cortex, this may have broad-reaching consequences unspecific to audition. We thus propose that in addition to the neural and perceptual dysfunctions described earlier, reduced inhibition in auditory cortex associated with aging and hearing loss may induce neural plasticity and potentially impact neural circuits beyond auditory cortex.

Discrepancies between work in animals and humans

Research in animals has revealed several age and hearing loss–related changes in functions of auditory cortex. These results are not always corroborated by noninvasive techniques in humans. The reasons for these discrepancies are likely diverse. Research in humans may lack sufficient power as it often relies on small participant samples. Moreover, large-scale population recordings (EEG/MEG, fMRI) may be insensitive to subtle and slowly evolving changes associated with aging and hearing loss. Critically, exposure to the types of sounds humans commonly encounter in everyday life may be less traumatic than the unstructured noises often used to induce hearing loss in animals (Thomas et al., 2019). Moreover, the rich sound environments in which humans live may reduce the effects of traumatic noise events compared to the quiet environments in which laboratory animals are housed (Norena & Eggermont, 2005). Differences in the natural auditory habitats of humans and laboratory animals may thus contribute to the discrepancies between observations.

Conclusion and future directions

In this chapter, we have reviewed anatomical and functional changes that occur within auditory cortex following aging and hearing loss, with a focus on the role of reduced inhibition in age and hearing loss–related dysfunction. We suggest that

reduced inhibition manifests functionally in complex ways, not all of which are response enhancements, and that a loss of inhibition may underlie many hearing difficulties experienced by older people. The role of specific subtypes of inhibitory neurons is unclear for many auditory functions (e.g., periodicity processing). It is further unknown how a loss of inhibition affects descending auditory pathways that are crucial for experience- and attention-related plasticity. Most critically, the translation of the detailed emerging picture of excitatory–inhibitory function in animal microcircuits to human function is a major challenge. Fruitful research avenues will (1) focus on experiments in humans that specifically test hypotheses derived from the detailed evidence of functional changes observed in animals; and (2) relate activity in neuronal microcircuits to global signals such as scalp recordings in rodents and monkeys to ultimately enable the translation to human electrophysiology and the study of perception. Finally, large cohort studies and longitudinal approaches in humans may be required to elucidate systematic, but likely subtle, changes in human auditory cortex associated with aging and hearing loss.

Applications to other areas of aging

A loss of neural inhibition associated with aging is not limited to auditory cortex but has been observed throughout the brain. The extent to which the loss of inhibition in nonsensory brain regions is linked to age-related degradations of sensory functions (exacerbating loss of inhibition) is currently unknown. Critically, reduced cortical inhibition is associated with a wide range of age-related pathologies including cognitive decline, with some evidence suggesting that hearing impairment may accelerate cognitive decline in older people (Livingston et al., 2017). Accordingly, sound-evoked hyperexcitability—an index of reduced inhibition in auditory cortex—has been observed in older people with mild cognitive impairment compared to older people with normal cognitive function (Bidelman, Lowther, Tak, & Alain, 2017).

Understanding the age-related reduction of inhibition in the auditory system may help elucidate the consequences of a loss of inhibition in the brain more broadly. Noninvasive electrophysiological recording techniques are highly sensitive to sound-evoked neural activity in humans and, when paired with relevant psychophysical measures, may provide a model system in which to study the relationships between aging, neuronal responsivity, and behavior. Moreover, well-designed studies relating changes in these measures to shifts in the balance of excitation/inhibition (analogous to animal studies reviewed here) may provide insights into the mechanisms underlying diverse deficits observed in aging. That sound-evoked hyperexcitability predicts cognitive decline (Bidelman et al., 2017) suggests that approaches capitalizing on auditory processing deficits to understand cognitive decline in aging provide a unique avenue for studying neural function across systems and morbidities.

Key facts of auditory aging

- Most older adults live with some form of hearing impairment.
- Hearing problems include being overly sensitive to sounds, having problems with localizing sounds, and experiencing difficulty understanding speech in noisy situations.
- First signs of hearing impairment often occur when people are in their 30–50s.
- The causes of the experienced hearing difficulties likely reflect dysfunction of the whole auditory system from the periphery to cortex.
- Treatments of hearing impairment via prostheses target dysfunction within the auditory periphery, while more central deficits may limit their effectiveness.

Summary points

- This chapter focuses on the anatomy and function of auditory cortex.
- Aging and hearing loss are associated with reduced neural inhibition in auditory cortex.
- Reduced inhibition leads to hyperexcitability, altered periodicity processing, and changes in spectral and spatial tuning.
- The hearing difficulties experienced by older people may reflect functional manifestations of reduced inhibition.
- Reduced inhibition in auditory cortex may facilitate neural plasticity in the brain.

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