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Sensation and Psychiatry: Linking Age-Related Hearing Loss to Late-Life Depression and Cognitive Decline

Bret R. Rutherford, M.D., **Katharine Brewster, M.D.**, **Justin S. Golub, M.D.**, **Ana H. Kim, M.D.**, and **Steven P. Roose, M.D.**

New York State Psychiatric Institute, New York; and the Department of Psychiatry and the Department of Otolaryngology—Head & Neck Surgery, Columbia University College of Physicians and Surgeons, Columbia University, New York

Abstract

Recent research has linked age-related hearing loss to impaired performance across cognitive domains and increased risk for dementia diagnosis. The data linking hearing impairment to incident late-life depression are more mixed but suggest that diminished hearing does increase risk for depression. Behavioral mechanisms may explain these associations, such as the withdrawal of older adults from situations in which they may have difficulty hearing and communicating, which may contribute to the development of social isolation, loneliness, and consequent cognitive decline and depression. At a neural level, chronic hearing loss leads to reduced activation in central auditory pathways, resulting in compensatory increased activation in the cognitive control network, dysfunctional auditory–limbic connectivity, and deafferentation-induced atrophy in frontal brain regions. These pathologic changes decrease cognitive performance and increase depression risk by reducing cognitive reserve, increasing executive dysfunction, and disrupting normative emotion reactivity and regulation. Based on the available data and informed by this model, evidence-based suggestions are proposed for clinicians treating older adults, and a research agenda is advanced to facilitate the development of rationally designed and age-appropriate psychiatric treatments for older adults with age-related hearing loss. First and foremost, treating hearing loss should be investigated as a means of improving cognitive and depressive outcomes in well-designed studies incorporating comprehensive psychiatric assessments, randomization, objective documentation of compliance, and analyses of treatment mediators that will facilitate further therapeutic development. Multimodal neuroimaging studies integrating audiometric, neuropsychological, and clinical assessments also are needed to further evaluate the model proposed.

Age-related hearing loss is the third most common health condition affecting older adults after heart disease and arthritis (1). The prevalence of significant hearing impairment rises steeply with age, from 3% among adults 20–29 to 49% of adults ages 60–69 and over 80% in individuals 85 years of age and older (2, 3). Twenty-eight million Americans are hearing impaired among an estimated 500 million hearing-impaired individuals globally, with the

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Address correspondence to Dr. Rutherford (brr8@cumc.columbia.edu).

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45- to 64-year-old demographic containing the greatest absolute number of hearing-impaired individuals (4). While historically considered a benign effect of aging or exclusively a quality of life issue, age-related hearing loss is in fact associated with significant psychological and medical morbidity, including social isolation, frailty, and falls (5). Hearing loss is now the fifth leading cause of years lived with disability worldwide and will climb higher as aging of the population causes its associated functional limitations to affect more people (6).

The negative health outcomes associated with age-related hearing loss include the development and/or exacerbation of neuropsychiatric conditions such as dementia and late-life depression (3,7). In the following, we critically review available research on the relationships of age-related hearing loss to these predominant neuropsychiatric disorders of later life, present a model linking progressive hearing loss to cognitive decline and depression, and offer evidence-based suggestions to practicing clinicians regarding the optimal evaluation and treatment of hearing impairment in their patients. This approach follows successful previous efforts in geriatric psychiatry to identify and treat comorbid medical disorders (e.g. obstructive sleep apnea, erectile dysfunction, respiratory disorders) in order to optimize the treatment responsivity of psychiatric conditions. It also reflects contemporary efforts to develop personalized treatments for late-life psychiatric conditions, many of which are etiologically complex and poorly responsive to empirically applied, as opposed to rationally designed, treatments.

PATHOPHYSIOLOGY, CLINICAL PRESENTATION, AND EVALUATION OF AGE-RELATED HEARING LOSS

Age-related hearing loss is characterized by an elevated hearing threshold (i.e., the volume at which a sound can be detected) and reduced speech perception (especially in noisy or complex listening environments) (8). The hearing loss is likely due to an aggregation of risk factors that influence its time of onset, severity, and course rather than having a single etiology (e.g., excessive noise exposure, ototoxic medications, prior ear infections, and genetic predisposition). Age-related hearing loss initially affects high-frequency sounds (>2 kHz) where the consonants in spoken English that are important for speech clarity are clustered. It may progress to encompass mid frequencies (500 Hz to 2 kHz) characteristic of human speech in quiet environments and low frequencies (<500 Hz) important for music appreciation (9). Although hearing loss can start at any age, it typically comes to medical attention in the sixth decade of life and progresses slowly and symmetrically over decades. Individuals most often report difficulty understanding speech in noisy environments and constant tinnitus, and sometimes experience a paradoxical hypersensitivity to loud sounds.

The pathophysiology of hearing loss encompasses both degenerative changes to inner ear structures (e.g., loss of inner and outer hair cells, deterioration of spiral ganglion cells, stria vascularis atrophy) and altered neural processing of auditory input. However, the causes of these developments and the precise ratio of peripheral versus central contributions remain unclear (10). The severity and extent of hearing loss can be measured using pure tone audiometry, which tests the overall integrity of the hearing pathway from 125 Hz to 8 KHz.

Central auditory dysfunction results in reduced speech perception disproportionate to the degree of hearing loss as well as loss of temporal coding fidelity (11). More complex behavioral and/or electrophysiologic assessments are required to measure central auditory processing deficits, such as tests requiring the listener to process different information simultaneously presented to each ear (12).

AGE-RELATED HEARING LOSS, COGNITIVE DECLINE, AND DEMENTIA

Several recent reviews (13), meta-analyses (14), and a National Institute on Aging workshop on the topic (15) have linked peripheral age-related hearing impairment to cognitive decline in older adults, though discrepant results do exist. The strongest studies couple standard objective audiometric assessments (where <25 dB indicates normal hearing, 25–40 dB indicates mild loss, 41–70 dB indicates moderate loss, and >70 dB indicates severe loss) with reliable measures of global cognition (e.g., Mini-Mental State Examination). While the large samples analyzed in many studies permit statistical significance to be achieved despite generally small effect sizes, it is notable that more severe hearing impairment has been associated with greater subsequent decline across cognitive domains and increased risk for dementia diagnosis, even after adjusting for multiple covariates as well as baseline cognitive function (16).

To cite the strongest of these studies linking hearing loss with cognitive decline, Lin et al. analyzed data from 639 participants in the Baltimore Longitudinal Study of Aging who had evaluable audiometric testing data and were dementia free at baseline (7). Followed over 17 years, participants who later developed dementia experienced significantly greater hearing loss per year compared with those who did not. The hazard ratio for incident dementia increased with more severe hearing loss, rising to 4.9 in individuals with severe hearing loss compared with those with normal hearing. Lin and colleagues also connected worsening hearing loss with decreasing processing speed and executive functioning in data from the National Health and Nutritional Examination Survey (17). In the Health Aging and Body Composition Study (N=1,984 subjects with available audiometric and cognitive data), participants having baseline hearing impairment (pure tone average >25dB) demonstrated significantly greater rates of cognitive decline on the Modified Mini-Mental State Examination and Digit Symbol Substitution Test over 6 years' follow-up (3). Other studies investigating performance within specific cognitive domains (e.g., processing speed, executive functioning, episodic memory) as opposed to global measures of cognition have reported modest associations with hearing loss across all domains (18).

The few available studies focusing on central auditory dysfunction analyze data from longitudinal population-based studies, such as the Framingham Heart Study (19) and Adult Changes in Thought study (20). These reports indicate that central auditory processing dysfunction as measured by dichotic listening tests is strongly associated with subsequent dementia diagnosis. However, unraveling the specific contribution of central (as opposed to peripheral) hearing loss to cognitive decline is complicated, since coexisting peripheral deficits may confound measurements of downstream central auditory processing dysfunction. In addition, overlap between constituent elements of central auditory processing and executive functioning, particularly response inhibition, obscures the direction

of causality within central auditory processing–dementia associations, since executive dysfunction also is observed in patients with mild cognitive impairment and dementia. Central auditory dysfunction may be most appropriately conceptualized as a marker for incipient dementia rather than a cause of cognitive decline.

These data linking age-related hearing loss to cognitive decline must be interpreted in light of significant limitations, which most notably include their inability to characterize the precise nature of the association between hearing and cognition. While the temporal precedence of hearing loss and the risk it poses for subsequent dementia in cognitively normal subjects argues for hearing loss causing cognitive decline, it may be the case that degraded hearing influences certain neuropsychological assessments rather than cognition per se or that hearing loss brings older adults to medical attention more frequently, resulting in over-diagnosis of cognitive impairment (21). Upstream common causes such as inflammation, vascular pathology, and other systemic neuro-degenerative processes may lead to hearing loss, depression, and cognitive impairment via central nervous system–wide functional decline, making it the case that none of these conditions is causally related to the others (11). In that case, greater sensitivity of tests in one domain (hearing or cognition) could identify deficits in that domain prior to the other, leading to the appearance of an illusory causal relationship. More broadly, hearing impairment may introduce a systematic bias into neuropsychological assessments, many of which were designed and validated using verbal explanations of instructions and/or presentation of stimuli. Finally, the global cognitive screening tools most often used in population-based longitudinal studies may capture only limited variability in a normally aging population, with ceiling effects that could potentially lead to an underestimation of the true relationship between age-related hearing loss and cognitive decline.

AGE-RELATED HEARING LOSS AND LATE-LIFE DEPRESSION

Initial surveys suggested that older individuals reporting difficulty hearing also had higher scores on depressive symptom scales (22), a finding that also was borne out in a meta-analysis aggregating data across seven studies surveying N=17,767 subjects in the United States and internationally (23). Despite this initial support and the intuitive appeal of hypothesized links between depression and hearing impairment, higher quality studies obtaining objective information on peripheral hearing capacity through audiometric testing are substantially fewer and more mixed in their results than in the case of hearing and cognition. Moreover, there are no available studies of the risk central auditory dysfunction in older adults poses for the development of late-life depression.

Gopinath et al. found that depressive symptoms were associated with mild but not moderate or severe hearing loss (24), while Lee and colleagues found that hearing thresholds measured with pure tone audiometry (but not self-reported hearing impairment) were associated with depressive symptomatology in a community-dwelling older Chinese population (N=914) (25). In another analysis of National Health and Nutritional Examination Survey data, hearing impairment was not significantly associated with major depressive disorder defined by a minimum score on the Patient Health Questionnaire, but self-reported hearing aid use (at least 5 hours per week) was associated with significantly

lower odds of depression (26). Finally, Contrera et al. related audiometric data obtained at year 5 of the Health Aging and Body Composition Study to a construct of “emotional vitality,” which they defined as greater self-reported happiness at year 1 and higher personal sense of mastery, happiness, and low depressive and anxious symptomatology as measured at year 6 (27). These investigators found that compared with individuals with no hearing impairment, participants with at least moderate (i.e., >40 dB) impairment had 23% lower odds of emotional vitality. Methodological issues such as the different evaluation time points used as well as the unclear relation of emotional vitality to clinical entities such as major depressive disorder limit generalizability of these particular data.

Several additional limitations pertain to the above-reviewed studies of age-related hearing impairment and depression, possibly explaining the lack of consistent findings. First, reliance on self-report data for hearing capacities and depressive symptoms may introduce significant bias. Stigma around hearing loss may cause individuals not to self-identify as experiencing hearing impairment, and late-life depression often manifests more in somatic symptoms and lethargy (so-called “depression without sadness”) that may not be ascertained by general screening tools. Second, by virtue of their cross-sectional, “moment in time,” design, these studies cannot determine causality and may not evaluate an individual at the precise time he or she is symptomatic. Age-related hearing loss develops insidiously over a period of years, and there may be particular vulnerable periods or important thresholds for the development of depression within this course that escape sampling at one particular time point. Additionally, given its episodic nature, it is possible that hearing impairment could cause a depressive episode that then resolves or else is treated prior to evaluation in a study.

CLINICAL AND BEHAVIORAL MEDIATORS LINKING HEARING IMPAIRMENT TO COGNITIVE DECLINE AND DEPRESSION

Many hearing-impaired older adults avoid or withdraw from social contexts in which background noise will make it difficult to communicate (28), resulting in social isolation and reduced communication with family and friends (29, 30). Observational studies of older adults worldwide have reported associations between age-related hearing loss and impaired social functioning, reduced social support, and increased role limitation due to emotional problems (31, 32). The presence of hearing problems has been shown to reduce the amount of time older adults spend outside their homes and increase the odds of withdrawing from leisure activities (33). This finding obtains in both community settings as well as in nursing homes, where residents with severe hearing loss have been shown to have 1.4 times greater odds of demonstrating low social engagement and 1.3 times greater odds of spending little time participating in facility activities (34).

As has been noted in the National Academy of Medicine’s recent report on age-related hearing loss, the impact of hearing impairment on social functioning is likely to depend greatly on the demands of an individual’s environment (35). The same magnitude of hearing loss may pose much more difficulty for a person working in a noisy office and socializing in busy restaurants than for a retired person who meets friends in a quiet home. In like fashion, it has been hypothesized that hearing loss may affect women’s social functioning more

severely because in many cultures women rely more heavily on verbal communication to give and receive emotional support. A population-based survey of community-dwelling Canadians aged 45 and older found that the odds of reporting social isolation increased with the severity of the hearing impairment among women (but not among men), even when sociodemographic factors, medical comorbidities, and functional limitations were taken into account (36). Cross-sectional analysis of an independent data set showed that greater hearing loss was associated with increased odds of social isolation in older women, while hearing loss was not significantly associated with social isolation in other age groups or in men (29).

Social isolation, typically indexed by objective measures such as having a small social network, being unmarried, and participating in few activities, has been linked to reduced quality of life as well as numerous adverse physical and mental health outcomes in older adults (see Figure 1) (37). Reduced social interaction increases the risk for incident dementia, even after controlling for confounding variables (38, 39), and has been linked to development of depressive symptoms (40). Self-reported loneliness, which is often closely related to social isolation, has been shown to more than double the risk of Alzheimer's dementia diagnosis in longitudinal studies (41). In addition, several studies have reported that older adults with significant hearing loss report lower physical activity levels, perform worse on the Short Physical Performance Battery, and have slowed gait speeds (42). Frailty characteristics such as slowed gait speed exhibit significant reciprocal relationships with both cognition (43) and mood (44) and thus may represent an intermediate step in the progression of some older adults from hearing loss to cognitive decline and late-life depression.

Beyond contributing to social isolation, hearing loss also may unmask or hasten cognitive decline by depleting cognitive reserve, which represents the brain's ability to mitigate the consequences of pathologic insults by using pre-existing cognitive processes or by enlisting compensatory processes (45). Decreased cognitive reserve, as indexed by educational level, occupational attainment, or participation in leisure activities, is associated with significantly increased risk of dementia (46). Studies suggest more cognitive processing resources must be allocated to process degraded auditory inputs caused by age-related hearing loss, leaving fewer resources available for nonperceptual cognitive processes (47). In fact, even after controlling for age and subjects' abilities to receive verbal instructions, hearing loss is associated with poorer performance on working memory tasks, particularly on more demanding tests that overwhelm available processing capacity (48).

Finally, age-related hearing loss co-occurs with tinnitus (most commonly perceived as a steady ringing, rushing, or static sound) in roughly 40% of cases and with vestibular/balance difficulties 20% of the time (49). As these conditions are associated with increased risk for cognitive decline (50) and mood/anxiety disorders (51), they likely play a role in transducing the risk for neuropsychiatric dysfunction observed with age-related hearing loss. Current neurobiological models of tinnitus suggest that, similar to depression, it is associated with serotonin depletion and/or limbic dysfunction (52). Chronic tinnitus may also cause stress and life disruption that increase risk for development of depression. Similarly, mobility problems and balance disorders are strongly associated with frailty in older adults (53) and constitute major risk factors for falls (54), which are a leading cause of

morbidity and mortality in older adults. Fear of falling is strongly associated with diminished physical activity, reduced social activity, loss of functional independence, and depression (55).

NEURAL MEDIATORS LINKING HEARING IMPAIRMENT TO COGNITIVE DECLINE AND DEPRESSION

Neuropathologic studies have reported the presence of pathophysiologic features of Alzheimer's disease (i.e., plaques and tangles) in multiple central auditory regions, such as the cochlear nuclei, inferior colliculi, thalamus, and primary auditory cortex, while sparing peripheral structures (56). During auditory processing, information ascends from the cochlear nerve to the ventral and dorsal cochlear nuclei before crossing via the trapezoid body to synapses in the superior olivary complex or inferior colliculus. Projections from the central nuclei of the inferior colliculi then are relayed via the lateral lemniscus through the medial geniculate nucleus of the thalamus before continuing to the superior temporal gyrus (primary auditory cortex) and surrounding associated secondary regions for further processing. Thus, it is conceivable that amyloid plaques, neurofibrillary tangles, and other Alzheimer's-type pathology may interrupt processing of auditory information in parallel to more widespread cognitive decline, but it is unlikely that such lesioning of the auditory system explains the full extent of the hearing–cognition association.

Rather, recent neuroimaging studies of age-related hearing impairment have begun to elucidate compensatory and neuroplastic changes associated with degraded auditory input that provide plausible pathways by which chronic hearing loss may cause cognitive dysfunction and affective dysregulation. As depicted in Figure 2, deterioration of the peripheral hearing apparatus over time decreases input to primary auditory cortex, secondary association cortices, and the auditory thalamus, which is visualized in functional neuroimaging tasks as decreased neural activations to auditory stimuli (57). In the short term, blunted neural responses to sounds alters resting functional connectivity in the default mode network (58) and leads to compensatory increased activations in the cognitive control network to support effortful listening (Figure 2, pathway B1) (59). This compensation may tax the network's capacity and cause manifest executive dysfunction (60), which is common in older depressed patients (61) and portends poor response to antidepressant medication as well as more chronic, recurrent course of illness (62).

Chronic deafferentation of auditory and cognitive control networks is associated with atrophy of their constituent nodes, including primary auditory cortex, prefrontal cortex, and anterior cingulate cortex (Figure 2, pathway B3) (57, 63). Lin et al. analyzed the change in brain volume measurements over time in 126 normal and hearing-impaired older adults participating in the Baltimore Longitudinal Study of Aging (63). Subjects with at least mild hearing impairment had accelerated volume declines in whole brain and specifically the right temporal lobe after adjustment for multiple confounders. Such deafferentation-induced atrophy may serve to magnify the deleterious effects of acute compensatory shifts, creating a vicious cycle of declining hearing capacity, worsening executive function, and increasing risk for depression and dementia.

In addition, reduced peripheral input is associated with deactivations in key limbic structures that exhibit strong reciprocal connections with the auditory network (Figure 2, pathwayB2) (64). The medial geniculate nucleus of the thalamus and the auditory cortex are reciprocally connected to the basolateral amygdala (65), allowing acoustic features of stimuli to be encoded in forward connections from auditory regions to the amygdala and the emotional valence of auditory stimuli to be carried in backward projections from the amygdala to the auditory cortex (66). In the only available neuroimaging study of hearing impairment and emotion processing, Husain et al. found that hearing-impaired subjects exhibited decreased amygdala and parahippocampal responses to emotionally valenced sounds from the International Affective Digital Sounds database, as well as prolonged reaction times to pleasant and unpleasant (but not neutral) auditory stimuli (64).

These findings suggest that dysfunctional auditory–limbic connectivity and diminished functionality of key nodes such as the anterior cingulate cortex may impair both emotion reactivity (default neural and behavioral responses to emotional stimuli) and emotion regulation (the reshaping of emotion based on directed, goal-compatible thoughts), since these functions are supported by automatic responses in subcortical regions such as the amygdala modulated by top-down feedback from medial (ventromedial prefrontal cortex and anterior cingulate cortex) and lateral (dorsolateral and ventrolateral prefrontal cortex) regions (67). However, since the currently available study has only examined neural responses to emotional information presented aurally, it is unclear whether the neuropathology associated with age-related hearing loss also impairs processing of emotional information presented to other sensory modalities. One might expect that emotional responses to visual, taste, smell, and touch sensations would be likewise affected given the consumption of cognitive resources by effortful listening as well as the atrophy occurring in the setting of chronic unremediated hearing loss, but more studies are needed to examine emotion reactivity and regulation in older adults with hearing loss across sensory modalities.

TREATING HEARING IMPAIRMENT TO PREVENT OR REVERSE ADVERSE NEUROPSYCHIATRIC CHANGES

The obvious therapeutic implication of the above-reviewed evidence for the association between age-related hearing loss, cognitive decline, and the development of late-life depression is to treat the hearing loss to avoid these adverse outcomes. An appropriately fitted hearing aid amplifies speech for improved communication, resulting in improved psychosocial functioning and increased quality of life (68, 69). A trial of hearing amplification is indicated when high-frequency thresholds are greater than 40 dB on a patient’s audiogram. When hearing loss is severe to profound, hearing aids may not adequately provide clarity of speech, especially in noisy environments, resulting in amplification of noise only (70). For such individuals, cochlear implants that convert acoustic sound to electrical signals that stimulate the cochlear nerve directly have become the gold standard for treatment. The great majority of patients undergoing cochlear implantation for age-related hearing loss achieve significant functional improvement, similar to that seen in younger patients (71, 72).

Emerging evidence suggests that the positive effects of restoring auditory input using hearing aids or cochlear implants extends beyond improved hearing to better cognitive functioning and reduced depressive symptoms. Naturalistic assessments of neuropsychiatric status before and after hearing treatment have shown improvement on short-and long-term global cognition, memory tasks, depressive symptom scores, and social functioning (73–75). An open series of 40 older adults assessed before and after hearing aid fitting showed a significant decline in reported loneliness after 4–6 weeks of hearing aid use; patients with more severe hearing loss reported the greatest improvements (76). Benefits of cochlear implantation on neuropsychiatric outcomes may be even greater: a recent prospective observational study found that cochlear implantation but not hearing aid treatment was associated with significant decreases in self-reported loneliness, particularly in those with high levels of baseline loneliness (77). Cochlear implantation also has been reported to improve cognitive performance, personal well-being, and life satisfaction (78, 79).

Although these preliminary studies suggest hearing treatment may show promise for preventing incident cognitive decline and late-life depression as well as improving already present neuropsychiatric dysfunction, there are important limitations to such findings. Most of the available studies enrolled small sample sizes and did not select participants based on the presence of mild cognitive impairment, major depressive disorder, or even threshold symptom scores. Only one study has attempted to compare hearing treatment to a control group. Mulrow and Aguilar randomized 194 subjects to receive hearing aids versus a wait list control and reported significantly increased self-reported quality of life and cognitive function as well as decreased depressive symptoms at 6 weeks and 4 months post–hearing aid prescription (68). Wait list groups are in general weak controls that may result in an overestimation of treatment effects, particularly in medical device studies where placebo effects are likely to be large. Finally, analyses of hearing aid use and depressive symptoms are plagued by inaccurate reporting, as studies only rarely use objective measures of when hearing aids are in place, turned on, and actually being used. In general, hearing aid use by older adults is surprisingly low (80), and few data are available on the association between actual hearing aid use and self-reported use.

In combination with restoration of hearing, it additionally may be therapeutically valuable to target the neural mediators and behavioral outputs proposed in Figure 3 to maximize functionality. For example, computerized cognitive training might be used to target executive dysfunction in older adults with chronic hearing loss (81). Relying on adaptive neuroplastic changes in the cognitive control network induced by repetitive cognitive exercises, computerized training has been associated with improved memory, processing speed, task shifting, response inhibition, and dual-task processing (82). Treatment with antidepressant medication has been shown to normalize pathological decreases in prefrontal cortex and striatal function, increases in limbic activity, and disordered connectivity between these regions (83). Evidence-based psychotherapies such as cognitive-behavioral therapy may improve emotion regulation by augmenting lacking top-down modulation of emotions (84). To date, none of these treatment strategies has been studied systematically in patients with comorbid hearing loss and cognitive decline or late-life depression.

CLINICAL IMPLICATIONS FOR PRACTICING PSYCHIATRISTS

Given the links between age-related hearing loss and later-life neuropsychiatric disorders, the evaluation of a patient presenting with subjective memory problems or depressive symptoms may be an important opportunity to diagnose and treat hearing impairment. Simple screening tests may be conducted for peripheral auditory deficits, such as determining if an individual can detect whispered speech, fingers being rubbed together, or a watch ticking next to each ear. Individuals may be asked, “Do you have difficulty with your hearing?” or formal screening questionnaires may be administered, such as the Hearing Handicap Inventory for the Elderly—Screening Version (85). Individuals screening positive may then be referred for audiometric testing with pure tone hearing thresholds in each ear, word recognition, and/or more complex behavioral or electrophysiologic assessments to measure central auditory processing deficits (e.g., tasks presenting speech items to both ears either simultaneously or in an overlapping manner) (12).

Detection of a significant hearing impairment provides psychiatrists important prognostic information regarding cognitive functioning and risk for depression that can be conveyed to patients. While controlled studies examining the effectiveness of hearing aids and cochlear implantation for cognitive and affective outcomes have not yet been performed, treatment of age-related hearing loss is indicated, given its deleterious effects on social functioning, behavioral activation, and of course, hearing itself. Psychiatrists may be in a particularly useful position with respect to helping patients accept their hearing loss condition, understand that hearing loss is treatable and not an inevitable part of aging, and elicit information regarding the obstacles to obtaining treatment for hearing loss, including fear of stigmatization or discomfort due to wearing hearing aids.

DIRECTIONS FOR FUTURE RESEARCH

Given that the current treatments for both late-life depression and cognitive decline/dementia are significantly limited in efficacy, research has focused on the causative aging-related processes involved in an effort to disentangle the disorders’ etiological complexity. Identifying risk factors or pathophysiologic subtypes based on neurobiological and or behavioral data may facilitate the development of novel treatment approaches tailored to the underlying pathophysiology. For these reasons in combination with the data reviewed above, age-related hearing loss merits further empirical attention as a causal and/or precipitating factor for the development of dementia and depression. To begin, observational studies incorporating comprehensive neuropsychiatric evaluations are needed to quantify the prevalence and incidence of late-life depression and cognitive impairment in older adults with hearing impairment as well as, conversely, the frequency of hearing impairment among patients presenting with depression or memory problems. Next, it would be instructive to know whether standard treatments for depression (e.g., antidepressant medication) as well as treatments designed to delay progression to dementia are less effective in the setting of age-related hearing loss.

However, the most urgent research need is for rigorously designed research to determine whether hearing treatment is effective for the prevention of incident cognitive/affective

problems and for the treatment of current cognitive impairment and depression. Acute, prospective, randomized, controlled studies of hearing remediation for older adults with comorbid hearing loss and cognitive decline and/or syndromal late-life depression may be most feasible to begin. Studies could be conducted of whether hearing treatment is as effective as monotherapy for cognitive impairment and depression or as augmentation of standard of care treatments (e.g., memory enhancers or antidepressants). Treatment studies must incorporate comprehensive psychiatric assessments for patient ascertainment, rigorous methodology including randomization and objective documentation of compliance, and analyses of treatment mediators that will facilitate further therapeutic development and translation to clinical practice and population health. While modern hearing aids can objectively track usage with built-in data logging features, this has rarely been used for research purposes (86).

A key consideration will be the composition of the control groups for such studies, as positive expectancies instilled by discernible changes in hearing are likely to lead to substantial placebo effects. While sham-controlled studies of hearing aids have been performed (87), it is difficult to maintain blinding when participants are aware of whether or not they are hearing better. In the case of hearing aids, one option may be to compare full-amplification versus low-amplification hearing aid treatment, with the latter providing noticeably increased auditory volume without being sufficient for adequate speech discrimination. Incorporating neuroimaging assessments into such clinical trials will be important, as increasing auditory input via hearing assistive devices (such as hearing aids) or cochlear implantation (for severe hearing loss) theoretically should restore more normative activation patterns and increase availability of the cognitive control network for higher-order processing.

Finally, multimodal neuroimaging studies, likely incorporating audiometric, neuropsychological, and clinical data, are needed to further evaluate the model proposed in Figure 2. Is reduced neural reactivity to auditory stimuli associated with dysfunctional activation patterns and volumetric differences in the cognitive control network as well as measurable cognitive control deficits on behavioral tests? If so, what is the direction of causation in the auditory–cognitive control network relation-ship, and how do other important factors such as vascular lesions fit in? Similarly, do older adults with hearing loss exhibit reduced top-down modulatory capacities in the processing of affective information? Such a finding would be interesting, given that on average compared with younger adults, older individuals have fewer negative emotional experiences, are less likely to report unhappiness, and feel a greater sense of well-being (86). Hearing loss may be a sentinel developmental event that pushes some individuals off of a normal, healthy aging trajectory toward increased risk for depressive disorders and cognitive decline.

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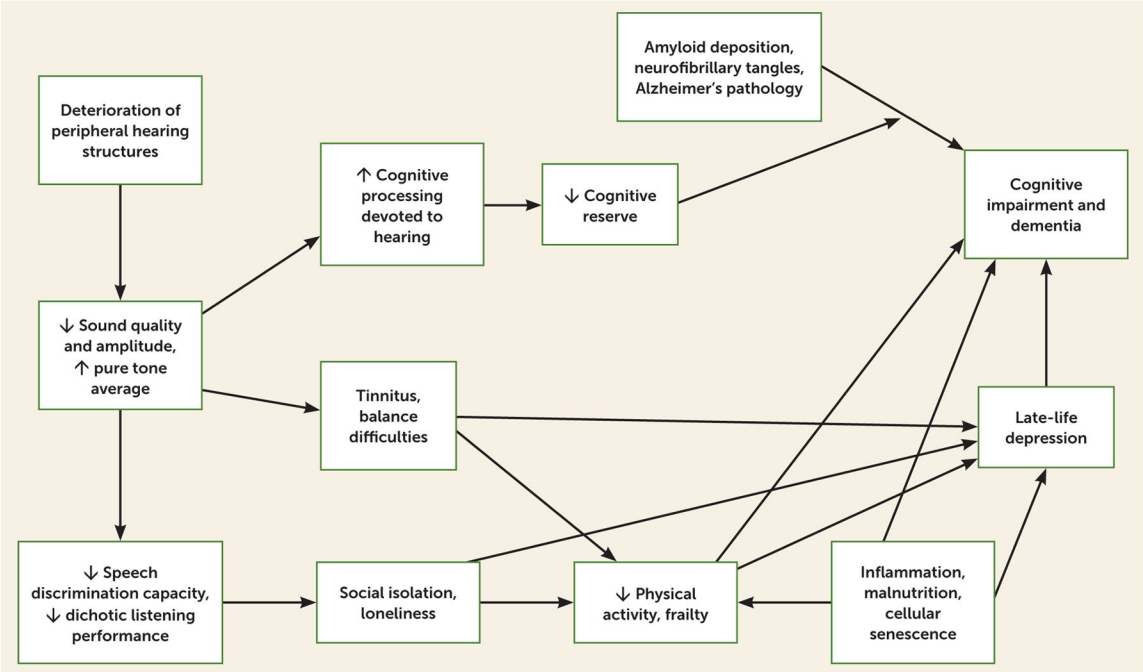

FIGURE 1.

Clinical and Behavioral Mediators Linking Age-Related Hearing Loss to Neuropsychiatric Dysfunction

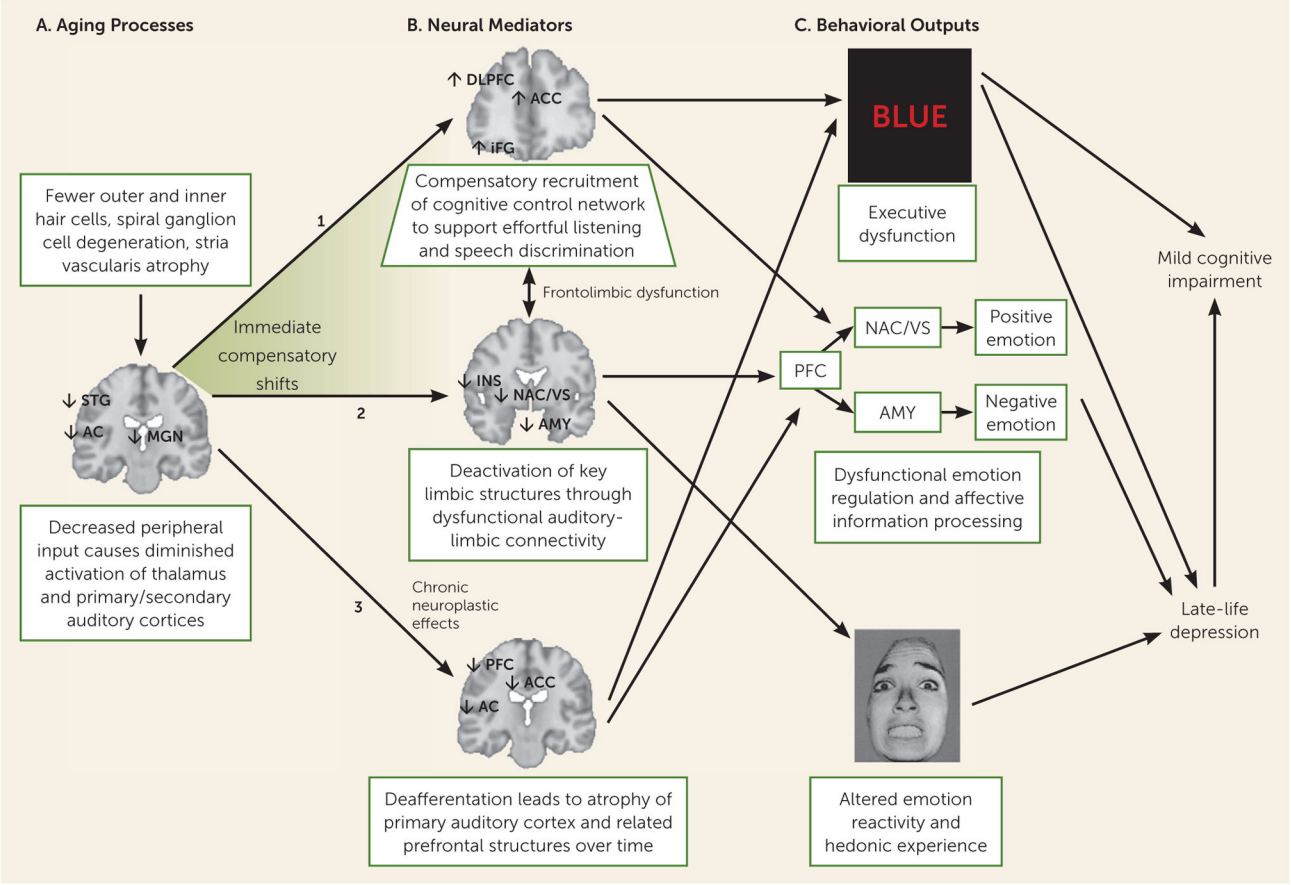

FIGURE 2. Putative Causal Mechanisms Linking Age-Related Hearing Loss to Cognitive Decline and Late-Life Depression[a]

[a]A. Degenerative changes associated with age lead to peripheral sensorineural hearing loss. B. Neural mediators include compensatory recruitment of the cognitive control network to support effortful listening (B1), activation changes in limbic regions sharing connectivity with the auditory network (B2), and deafferentation-induced atrophy (B3). C. These acute activation changes and longer-term adverse neoplastic sequelae of hearing loss may lead to executive dysfunction and impaired affective information processing, leading to increased risk of late-life depression and mild cognitive impairment., AC=primary auditory cortex, ACC=anterior cingulate cortex, AMY=amygdala, DLPFC=dorsolateral prefrontal cortex, iFG=inferior frontal gyrus, INS=insula, MGN=medial geniculate nucleus of the thalamus, NAC=nucleus accumbens, PFC=prefrontal cortex, STG=superior temporal gyrus, VS=ventral striatum.

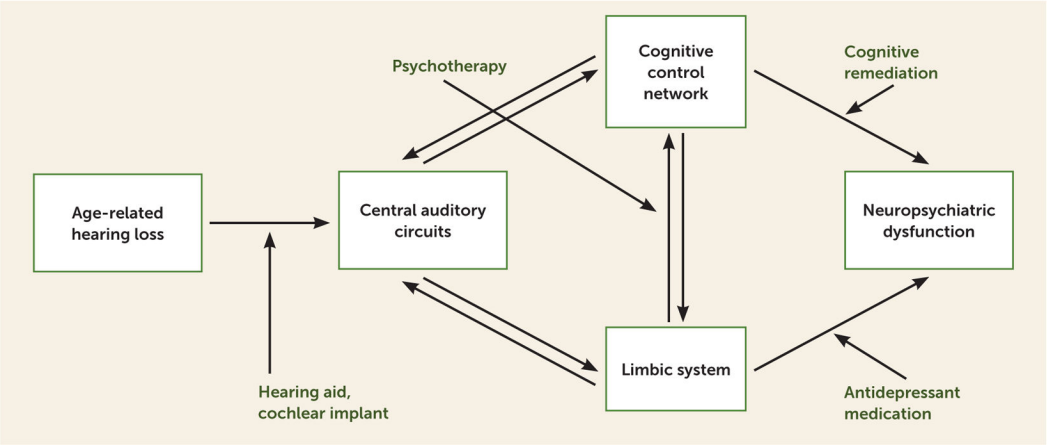

FIGURE 3.

Possible Targets of Therapeutic Intervention for Patients With Hearing Loss Who Are at Risk of Neuropsychiatric Dysfunction