The association between hearing impairment and dementia has emerged as a major public health challenge, with significant opportunities for earlier diagnosis, treatment and prevention. However, the nature of this association has not been defined. We hear with our brains, particularly within the complex soundscapes of everyday life: neurodegenerative pathologies target the auditory brain, and are therefore predicted to damage hearing function early and profoundly. Here we present evidence for this proposition, based on structural and functional features of auditory brain organization that confer vulnerability to neurodegeneration, the extensive, reciprocal interplay between 'peripheral' and 'central' hearing dysfunction, and recently characterized auditory signatures of canonical neurodegenerative dementias (Alzheimer’s disease, Lewy body disease and frontotemporal dementia). Moving beyond any simple dichotomy of ear and brain, we argue for a reappraisal of the role of auditory cognitive dysfunction and the critical coupling of brain to peripheral organs of hearing in the dementias. We call for a clinical assessment of real-world hearing in these diseases that moves beyond pure tone perception to the development of novel auditory ‘cognitive stress tests’ and proximity markers for the early diagnosis of dementia and management strategies that harness retained auditory plasticity.

Introduction: scope and nature of the problem

Hearing impairment in later life is a major clinical issue and a leading association of cognitive decline (Gates and Mills, 2005; Lin et al., 2011; Loughrey et al., 2018), presenting significant potential opportunities for dementia diagnosis, treatment and prevention (Dawes et al., 2015; Taljaard et al., 2016; Livingston et al., 2017). But how are hearing impairment and dementia related? Hearing loss of any...
cause tends to limit social engagement and quality of life (Graydon et al., 2019), amplifies the effects of cognitive impairment and may confound or delay diagnosis of dementia (Panza et al., 2015; Wayne and Johnsrude, 2015). Conversely, diagnosis of hearing loss and compliance with hearing aids are hindered by cognitive impairment (Dawes et al., 2015). There may, however, be a more fundamental pathophysiological basis for the association: hearing is a complex cognitive function that, alongside other cognitive functions, is directly vulnerable to the pathophysiological processes that cause dementia (Wayne and Johnsrude, 2015; Hardy et al., 2016).

Recent studies addressing the link between hearing impairment and dementia have focused predominantly on audiometric pure tone detection, the ability to detect quiet sounds (Lin et al., 2011; Loughrey et al., 2018). However, most natural auditory environments or ‘scenes’ comprise mixtures of sounds that change over time, and listening—perception and understanding of sounds—is a highly active cognitive process (Bendixen, 2014) (Fig. 1). Consider, for example, the everyday scenario of following a conversation in a crowded room. After substantial ‘pre-cognitive’ processing in the auditory brainstem (Cope et al., 2015), the incoming auditory signal must be deconstructed (by ‘auditory scene analysis’) (Goll et al., 2012a; Golden et al., 2015c; Hardy et al., 2016) into discrete and stable percepts or ‘auditory objects’ corresponding to voices and speech features, separate from background noise (Griffiths and Warren, 2004; Goll et al., 2010b). Such auditory objects must be matched to stored representations and expectations to achieve recognition and ultimately, an appropriate behavioural response. These processes collectively constitute ‘auditory cognition’ (Fig. 1) and depend critically on neural computations in auditory cortical and linked processing networks: the auditory brain (Fig. 2).

Evidence that neurodegenerative pathologies target the auditory brain and produce ‘central’ hearing deficits disproportionate to any peripheral hearing loss was first produced some time ago (Kurylo et al., 1993; Strouse et al., 1995). More recently, a diverse array of ‘central’ auditory deficits has been described in these diseases (Mahoney et al., 2011; Rohrer et al., 2012; Fletcher et al., 2015, Golden et al., 2015c; Grube et al., 2016; Hardy et al., 2016; Eversfield and Orton, 2019; Jafari et al., 2020), ranging widely beyond ‘deafness’ (impaired sound detection) to encompass altered auditory perception, understanding and behavioural responses, with far-reaching consequences for hearing function in daily life. To date, however, the role of the auditory brain in linking hearing impairment to cognitive decline has been largely overlooked.

Here we argue that the auditory brain is integral to the development and expression of hearing impairment in dementia. Our case rests on three interwoven lines of evidence: the structural and functional characteristics of auditory brain organization targeted by neurodegenerative diseases; the known extensive interplay between so-called ‘peripheral’ and ‘central’ hearing mechanisms; and mounting data on auditory cognitive dysfunction as a prominent, early and specific manifestation of canonical dementia syndromes. We propose a roadmap for future work directed toward developing novel auditory cognitive tests, biomarkers and therapies.

The auditory brain: structural and functional substrates for neurodegeneration

The auditory system has evolved to allow adaptive behavioural responses to complex, dynamic acoustic environments (Griffiths et al., 2001; Pickles, 2015). However, its structural and functional characteristics confer specific vulnerabilities to neurodegenerative pathologies.

Anatomically, the hierarchy of auditory processing relays and in particular the large-scale cerebral networks that process sound information (Fig. 2) are highly distributed. The spread of pathogenetic proteins in neurodegenerative dementias (Fig. 2) targets these networks rather than the peripheral organs of hearing. Though histopathological data remain limited, neurodegenerative pathologies may preferentially involve auditory association cortex and cortico-cortical projections rather than primary sensory cortex (Esiri et al., 1986; Lewis et al., 1987), thereby striking the integrative mechanisms that are most critical for auditory object analysis.

Accurate auditory signal transduction (for example, during spatial hearing or speech perception) depends on precise integration of frequency-based (spectral) and time-based (temporal) information (Griffiths et al., 2001; Bizley et al., 2009): any pathology that damages relevant neural circuits is likely to disrupt such processing early in its course. As the auditory signal passes up the processing hierarchy, it is transformed non-linearly such that it is no longer a direct replica of the incoming signal encoded at the periphery (Wang, 2007; Gaucher et al., 2013); due to the intrinsically temporal nature of sound, this transformation of auditory information is particularly evident in the time domain and supports the extraction of invariant auditory object features and cross-modal integration. The resulting percept is normally robust to noisy variations in the sensory signal; however, its non-linear nature means that even small perturbations of neural circuit function due to neurodegenerative disease may have disproportionately large perceptual and behavioural consequences.

Two additional, related guiding principles of auditory system operation that are critical for adaptive functioning in complex, dynamic auditory environments are functional plasticity and reciprocity. Reciprocity is mediated by recursive, afferent-efferent feedback that supports auditory change detection and top-down tracking of behaviourally relevant sound sources (Shamma and Micheyl, 2010; Zion Golumbic et al., 2013), as well as predictive decoding and ‘filling-in’ of ambiguous and varying auditory inputs, such as degraded speech (Malmierca, 2014; Simon, 2015; Donhauser and Baillot, 2020) (Fig. 1).
Plasticity (for example, perceptual learning of degraded speech) (Hardy et al., 2018) enables dynamic neural adaptation to auditory experience. These functional principles are evident throughout the auditory system (Russo et al., 2005; Barascud et al., 2016; Guinan, 2018) and are highly sensitive to synaptic neurochemical (particularly cholinergic) modulation, especially under challenging listening conditions (Dhanjal et al., 2013; Kuchibhotla et al., 2017; Minces et al., 2017). They are therefore potentially highly susceptible to neurodegenerative pathologies that disrupt synaptic and neurotransmitter pathway integrity. Moreover, the characteristics of non-linear stimulus coding, extensive efferent regulation of afferent pathways and pervasive plasticity (though not specific to audition) are much more marked in the auditory system than in other sensory systems, notably vision (King and Nelken, 2009). Impaired functional adaptation of auditory brainstem pathways has perceptual consequences in patients with mild cognitive impairment (Bidelman et al., 2017), suggesting that indices of auditory plasticity may be sensitive and dynamic markers of neurodegenerative pathologies.

‘Peripheral’ and ‘central’ hearing: a false dichotomy and a double hit

The anatomical and functional interactions of auditory processing stages (Figs 1 and 2) suggest that any sharp distinction between ‘peripheral’ and ‘central’ hearing is likely to be a false dichotomy. Pure tone audiometry (PTA), the
mainstay of standard clinical audiological assessment, is generally interpreted as an index of ‘peripheral’ (cochlea and auditory nerve) hearing. However, PTA thresholds are affected by attention (Musiek et al., 2017), executive function (Gates et al., 2010) and brainstem pathologies that do not directly involve the cochlea (Cope et al., 2015), reflecting the known role of top-down influences on cochlear sensitivity (Terreros and Delano, 2015). Furthermore, PTA does not fully predict ability to hear speech in noise (the principal hearing complaint of older listeners) (Anderson et al., 2011; Guest et al., 2018; Holmes and Griffiths, 2019). Conversely, ‘central’ hearing functions that rely on high-fidelity signal coding at brainstem level (such as speech intelligibility) are tuned by efferent synaptic functional adaptation at the cochlea (Pressnitzer et al., 2008) and auditory agnosia is modulated by peripheral hearing loss (Coebergh et al., 2020).

Neurodegenerative diseases that principally involve cortical and subcortical pathways may therefore significantly impact hearing functions canonically attributed to the peripheral sense organs; indeed, elevated PTA thresholds have recently been documented in the non-fluent agrammatic variant of primary progressive aphasia (nfvPPA), a primary cortical degeneration (Hardy et al., 2019). On the other hand, anatomical involvement of subcortical auditory relays by neurodegenerative pathology does not necessarily lead to a perceptual deficit (Hughes et al., 2014).

Moreover, neurodegenerative diseases typically target the ageing brain, and healthy ageing itself affects multiple stages...
Table 1 Auditory phenotypes of some major dementia syndromes

| Syndrome       | Core clinical features                                      | Key auditory symptoms                                      | Auditory deficits                       | Proposed auditory diagnostic test   | Pathological neuroanatomy         |
|----------------|-------------------------------------------------------------|------------------------------------------------------------|-----------------------------------------|-------------------------------------|----------------------------------|
| Alzheimer’s disease | Typical: Episodic/topographical memory loss, parietal deficits | Difficulty tracking sound sources/information in busy acoustic environments, auditory disorientation, difficulty understanding less familiar accents, auditory agnosia, increased sound sensitivity | Scene analysis, localization, attention, melody contour, accents, environmental sound recognition, working memory | Auditory stream separation, sound localization/motion detection, DLT | Posterior cingulate, precuneus, lateral tempo-parietal cortex |
|                 | PCA: Visuo-perceptual/visuospatial, other parietal deficits | Similar or more severe than typical AD                     | More severe involvement of auditory scene/spatial processing | Auditory stream separation, sound localization/motion detection | Phono discrimination |
|                 | LPA: Anomia, phonological and verbal working memory deficits | Auditory hallucinations                                   | Pure tone detection, complex tone perception, auditory scene analysis, rhythm perception, speech loudness perception | Sinewave speech comprehension | Cortico-subcortical circuits |
|                 | LBD: Fluctuating alertness/attention/executive deficits, visuo-perceptual, visual hallucinations, REM sleep behaviour disorder, parkinsonism | | | | |
| FTD            |               |                |                | Temporal pattern discrimination | Peri-Sylvian networks, prefrontal cortex |
| nfvPPA         | Speech production deficits, agrammatism | Agnosia for environmental sounds/accents, word deafness | Pure tone detection, perception of pitch interval/timbre/rhythm/prosody, accent comprehension | | |
| svPPA          | Anomia and vocabulary loss, visual agnosias, behavioural changes similar to bvFTD | Musicophilia/sound aversion, tinnitus, phonagnosia/nonverbal sound agnosia | Environmental sound/voice recognition, emotional recognition/reactivity, hedonic valuation, integration of semantic/affective information | Environmental sound recognition | Auditory/multimodal association cortex in anterior temporal lobe, orbitofrontal cortex, insula |
| bvFTD          | Socio-emotional, executive dysfunction with disinhibition, apathy, loss of empathy, obsessions and rituals, dietary and other behavioural abnormalities | Sound aversion/musicophilia, phonagnosia | Emotional recognition/reactivity, hedonic valuation, voice recognition, integration of semantic/affective information | Vocal emotion recognition | Auditory/multimodal association cortex in anterior temporal lobe, orbitofrontal cortex, insula, anterior cingulate, striatal circuits |

The table summarizes major clinical features, and auditory cognitive deficits, candidate auditory cognitive tests for early diagnosis and neuroanatomical associations in canonical dementia syndromes for which adequate data are available (see also Fig. 3).

1Auditory domains affected based on behavioural test performance. 2Based currently on experimental studies (examples referenced below) with a view (particularly for Alzheimer’s disease) to potential scalability, e.g. online administration, but provisional and require further clinical validation. 3Major distribution of pathological changes in brain networks relevant to auditory deficits, as assessed using voxel-based morphometry, functional neuroimaging (chiefly functional MRI) and/or post-mortem material. 4Underpinned by Alzheimer pathology in majority of cases; Includes dementia with Lewy bodies and Parkinson’s disease dementia; 5Not usually severe; 6Particularly associated with right temporal lobe atrophy; 7Can be delivered via headphones using virtual space stimuli; 8Other auditory abnormalities analogous to typical Alzheimer’s disease; 9Processing of degraded (e.g. sinewave-transformed) speech that is subject to perceptual learning and modulated by neurotransmitter function, by analogy with tests on degraded visual stimuli that show promise for diagnosis of LBD.

AD = Alzheimer’s disease; bvFTD = behavioural variant frontotemporal dementia; DLT = dichotic listening test; FTD = frontotemporal dementia; LBD = Lewy body disease; LPA = logopenic aphasia; nfvPPA = non-fluent agrammatic variant of progressive non-fluent aphasia; PCA = posterior cortical atrophy; svPPA = semantic variant of primary progressive aphasia.

Examples of experimental studies using proposed tests: 7Goll et al., 2012a; 8Golden et al., 2015c; 9Tuwaig et al., 2017; 10Gates et al., 2011; 11Hardy et al., 2020; 12Johnson et al., 2020; 13Weil et al., 2017; 14Hardy et al., 2017c; 15Grube et al., 2016; 16Golden et al., 2015b; 17Omar et al., 2011.
of auditory processing, ranging from cochlea to cortex (Bendixen, 2014; Bidelman et al., 2014; Roth, 2015; Henry et al., 2017; Zhao et al., 2019). Some of these effects (in particular, degeneration of synapses between inner hair cells and auditory nerve fibres) are undetectable or ‘hidden’ on standard PTA and may therefore be underestimated (Wu et al., 2019); other effects (such as attentional suppression of irrelevant sensory information) may only emerge under challenging listening conditions or for particular tasks, such as tracking fine-grained temporal information in speech (Henry et al., 2017). Increased cognitive effort and engagement of task-relevant capacities (in auditory cortex or executive control systems) may compensate to a degree for the widespread effects of ageing on auditory signal processing (Profant et al., 2015; Meister et al., 2016; Glick and Sharma, 2017; Bidelman et al., 2019); however, if compensatory mechanisms are compromised by neurodegenerative pathology, this ‘double hit’ may cause hearing loss to become functionally significant. Such decompensation would be relatively more likely under adverse listening conditions.

**Figure 3** A pathophysiological synthesis of hearing impairment and dementia. This figure schematizes proposed relations between development of peripheral hearing loss (blue), changes in auditory cognition (gold) and general cognitive function (red) and underlying neurodegeneration (black), based on emerging epidemiological and pathophysiological evidence. Hearing loss can be considered a potential causal risk factor for cognitive decline (Risk), a proximity marker for incipient dementia (Proximity) or a feature of the established dementia syndrome (Phenotype), according to the time window in which it occurs; the mechanisms of these effects are distinct but likely to be interdependent. Alzheimer’s disease has been the major focus of epidemiological studies assessing the risk of developing dementia in association with hearing loss (Lin et al., 2011; Taljaard et al., 2016; Livingston et al., 2017; Loughrey et al., 2018), though the distinction from cerebrovascular and other pathologies is problematic; midlife hearing loss may account for ~10% of all cases of dementia, and has been proposed to have a direct potentiating effect (arrow) on the evolution of neurodegeneration. Though the mechanism of this linkage is unclear, animal models suggest it could occur via cellular effects such as oxidative stress or altered gene expression (Frenzilli et al., 2017; Park et al., 2018), changes in neural circuit function (Oxtoby et al., 2017; Bidelman et al., 2019) or a complex interaction between aberrant circuit activity and protein spread (Griffiths et al., 2020). However, a direct causal effect has not been established: for example, peripheral hearing function was not associated with brain amyloid deposition (a relatively specific preclinical marker of Alzheimer’s disease) in a large cohort of cognitively healthy older people (Parker et al., 2020) and such an effect would still not account for the majority of cases of dementia with hearing alterations. Here we suggest that alterations in ‘central’ hearing or auditory cognition may constitute an early warning signal of incipient dementia, due to the computational demands imposed by listening in challenging everyday acoustic environments. In support of this idea, predominantly central auditory deficits (involving, for example, dichotic listening) have been shown to predict CSF tau levels and regional atrophy profiles consistent with Alzheimer’s disease pathology in cross-sectional studies (Tuwaig et al., 2017) and longitudinal development of a clinical syndrome compatible with Alzheimer’s disease (Gates et al., 2011), while large genetic and neuropathological surveys have suggested changes in hearing (in particular, speech-in-noise perception) may be a preclinical marker of neurodegeneration (Brenowitz et al., 2020a, b). We emphasize that deficits of peripheral and central hearing and more general cognitive functions are likely to interact strongly, with ‘vicious cycling’.
In this context, neurodegenerative effects on auditory brain function might act as ‘proximity makers’ for incipient, more generalized cognitive decline.

**Major dementias have diverse auditory phenotypes**

The neurodegenerative diseases that cause canonical dementia syndromes have specific profiles of large-scale, cortico-subcortical network involvement, determined by the patterns of spread of pathogenic proteins (Seeley et al., 2009; Warren et al., 2013) (examples in Fig. 2). These pathologies have correspondingly diverse clinical phenotypes including prominent auditory cognitive deficits (Table 1).

**Alzheimer’s disease**

Alzheimer’s disease produces a core impairment of auditory scene analysis, not attributable to more elementary deficits of sound perception or generic cognitive capacities (Idrizbegovic et al., 2011). Auditory scene processing deficits may predate onset of more generalized cognitive decline in people at risk of developing Alzheimer’s disease (Golob et al., 2009; Gates et al., 2011) and in both the typical amnestic and posterior cortical (visuospatial) syndromic presentations of Alzheimer’s disease (Goll et al., 2012a; Golden et al., 2015c; Hardy et al., 2020), suggesting that such deficits are a functional marker of Alzheimer’s disease pathology. This interpretation would corroborate neuroanatomical findings linking impaired auditory scene analysis to dysfunction and atrophy of the temporo-parietal ‘default mode’ network that is essential to Alzheimer’s disease pathogenesis (Warren et al., 2012; Goll et al., 2012a; Golden et al., 2015a, c) (Fig. 2).

More generally, auditory phenotypic features of Alzheimer’s disease may signify a unifying deficit in encoding sound sources and patterns as distinct auditory objects (Griffiths and Warren, 2004; Goll et al., 2010b, 2011; Hailstone et al., 2012; Hardy et al., 2017b). Such a deficit might ultimately underpin environmental sound agnosia in Alzheimer’s disease (Cobergh et al., 2020) and impaired phonological processing (most saliently in the logopenic variant) (Johnson et al., 2020), amplified by abnormalities of auditory working memory (Dhanjal et al., 2013).

**Lewy body disease**

Auditory dysfunction is prevalent in the Lewy body disease (LBD) spectrum (Parkinson’s disease and dementia with Lewy bodies) and may be a marker of disease onset, evolution and severity (Seifan et al., 2019; Jafari et al., 2020). Diverse auditory phenomena have been reported, ranging from auditory hallucinations to impairments of auditory scene analysis, tone and rhythm processing (Mollaei et al., 2019; Cochen De Cock et al., 2020; Jafari et al., 2020). Electrophysiologically, there is evidence of impaired auditory startle, deviance detection, habituation and sensory filtering (Perriol, 2005; Jafari et al., 2020) as well as olivocochlear efferent pathway dysfunction (De Keyser et al., 2019). The unifying deficit may be dynamic disruption of synaptic transmission at multiple levels of the auditory hierarchy (Jafari et al., 2020), due to abnormal top-down, neuromodulatory (principally dopaminergic) regulation.

**Frontotemporal dementias**

Auditory perceptual dysfunction is emerging as a core feature of nfvPPA (Goll et al., 2010a, 2011; Golden et al., 2016; Grube et al., 2016; Hardy et al., 2019), including deficits of rhythm, pitch and timbre perception (Goll et al., 2010a, 2011; Grube et al., 2016) and sound detection (Hardy et al., 2019). The key mechanism is likely to be impaired auditory pattern analysis in peri-Sylvian and connected prefrontal regions that govern expectations about incoming sensory traffic (Cope et al., 2017; Hardy et al., 2017a, b) (Fig. 2).

In contrast, semantic variant PPA typically spares elementary auditory pattern perception, leading instead to degraded semantic analysis of environmental sounds, voices and affective auditory signals (Bozeat et al., 2000; Goll et al., 2010a, b, 2012b; Hailstone et al., 2011; Fletcher et al., 2015; Golden et al., 2015b; Muhammed et al., 2018). This profile reflects selective degeneration and functional reorganization of antero-medial temporal lobe (Fig. 2) and its connections, including orbitofrontal cortices and auditory thalamus.

In the behavioural variant of frontotemporal dementia, inappropriate emotional reactions to voices, environmental sounds and music are often prominent (Omar et al., 2011; Fletcher et al., 2015): these are likely to be driven by impaired valuation and regularity decoding in complex auditory environments, linked to dysfunction of neural circuits mediating reward and rule processing (Clark et al., 2017, 2018).

**Hearing impairment: cause, canary or corollary of dementia?**

The complex pathophysiological relations between hearing impairment and dementia (schematized in Fig. 3) remain to be fully defined. Impoverished sensory fidelity due to peripheral hearing loss or disturbed subcortical auditory trafficking will potentially have effects both on auditory cognition and more general cognitive functions such as attention, executive processing and perceptual learning (Loughrey et al., 2018; Fig. 1), leading to ‘vicious cycling’. Hearing loss might therefore produce both syndromic and generic cognitive signatures. The balance of these is likely to depend on stimulus and task demands as well as the particular neurodegenerative process. Emerging epidemiological evidence suggests
that hearing impairment may potentiate neurodegeneration, perhaps via an interaction of aberrant auditory activity with culprit proteinopathies in vulnerable neural circuits (Griffiths et al., 2020) (Fig. 3). Indeed, hearing impairment might constitute a facilitating cause of neurodegenerative disease evolution, an early warning ‘canary’ for impending cognitive disaster or an accompaniment of established dementia: these non-exclusive mechanisms would have mutually reinforcing consequences for auditory brain function.

**Conclusions: a synthesis and future view**

The balance of neuroanatomical, physiological and clinical evidence suggests that the auditory brain plays a key role in the increasingly well documented association between dementia and hearing impairment. Degeneration of central auditory processing mechanisms (in particular, auditory cognitive dysfunction) will tend to amplify any degree of peripheral deafness and reduce compensatory capacity under natural (noisy) listening conditions. This reflects the extensive reciprocal interplay between afferent and efferent auditory processing pathways, exquisitely vulnerable to neurodegenerative proteinopathies. Moreover, neurodegenerative pathologies have distinct and relatively specific auditory cognitive phenotypes as well as generic effects on cognitive functions relevant to hearing, in line with the large-scale neural network signatures of these diseases. The synthesis we propose has neurobiological, diagnostic and management implications that should be tackled in future work.

Neurobiologically, central auditory dysfunction is likely to be a fundamental, early consequence of neurodegenerative dementias, due both to direct involvement of susceptible auditory processing networks by pathogenic protein spread and remote effects on highly interconnected structures. This requires substantiation using physiologically grounded neuroimaging techniques such as functional MRI and magnetoencephalography that may also help clarify the neural mechanisms of compensatory and therapeutic effects. Detailed, longitudinal disease phenotyping with biomarker and ultimately histopathological support (accounting for healthy auditory ageing and comorbid disease) will be required to elucidate the auditory pathophysiological signatures of particular proteinopathies, to assess the relative importance of hearing impairment in different diseases and to clarify the role of peripheral hearing deficits in potentiating the neurodegenerative process (Griffiths et al., 2020).

Diagnostically, hearing impairment might plausibly constitute a proximity marker for incipient cognitive decline and dementia, reflecting the heavy computational demands that auditory signal processing imposes on failing neural circuits. If substantiated in longitudinal studies of at-risk populations, this would raise the exciting prospect of novel auditory ‘cognitive stress tests’ for detecting the early stages of neurodegeneration and identifying dynamic, physiological biomarkers of disease evolution, residual plasticity and therapeutic response (Hardy et al., 2018). Such markers could represent red flags for targeting population-based screening and recruitment into dementia prevention trials from primary care settings and could be developed into ‘digital biomarkers’ that are highly scalable. For example, headphone-based tests of spatial hearing, degraded speech perception and dichotic listening could be performed online (Gates et al., 2011; Golden et al., 2015c). In addition, developing a toolkit of novel tests to quantify the relative contributions of peripheral and central auditory deficits would allow accurate characterization of auditory phenotypes in individual patients and could facilitate diagnosis of particular neurodegenerative pathologies (Table 1). It will be crucial to capture the real-world impact of central hearing impairment, which is likely to be more profound than would be predicted by the degree of any peripheral hearing loss.

Management approaches that focus solely on peripheral sound amplification are likely to be of limited efficacy for improving hearing function in dementia. There is a clear practical and pathophysiological motivation to address any potentially reversible component of peripheral hearing loss and ensuring compliance with hearing aids (Proctor et al., 2020). Ultimately, however, the goal of management should be to minimize hearing-related disability in the complex listening environments of daily life—to treat the patient, not the audiogram or the neuropsychological test score. Personalized interventions directed to central auditory mechanisms such as ‘smart’ hearing aids (Koohi et al., 2017), hearing-based behavioural therapies and auditory cognitive rehabilitation (Russo et al., 2005) should be combined with education and environmental modification supported by a detailed assessment of functional disability. Pharmacological modulation of cholinergic and dopaminergic function to harness auditory plasticity has shown early promise in Alzheimer’s disease and LBD (Dhanjal et al., 2013; Hardy et al., 2017c; Jafari et al., 2020): such approaches could herald a new era of physiologically informed, integrated management focussing on retained capacity rather than deficits and embracing both central and peripheral auditory impairment in dementia.

**Funding**

The Dementia Research Centre is supported by Alzheimer’s Research UK, Brain Research Trust, and The Wolfson Foundation. This work was supported by the Alzheimer’s Society, Alzheimer’s Research UK and the National Institute for Health Research University College London Hospitals Biomedical Research Centre. J.C.S.J. is supported by an Association of British Neurologists Clinical Research Training Fellowship, funded by Guarantors of Brain. C.R.M. is supported by a grant from Bart’s Charity. R.S.W. is supported by a Wellcome Clinical Research Career Development Fellowship (201567/Z/16/Z). D.E.B. is supported by a Wellcome Clinical Research Career Development Fellowship (201567/Z/16/Z). D.E.B. is
supported by a BRC Hearing and Deafness grant. C, J.D.H. is supported by an Action on Hearing Loss-Dunhill Medical Trust Pauline Ashley Fellowship. J.D.W. receives grant support from Action on Hearing Loss, Alzheimer’s Research UK, Alzheimer’s Society, Guarantors of Brain, Brain Research UK, MRC, Wellcome Trust, and the Wolfson Foundation.

Competing interests
The authors report no competing interests.

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