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Social factors may mediate the relationship between subjective age-related hearing loss and episodic memory

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ABSTRACT

Objectives: To investigate whether the relationship between subjective age-related hearing loss (SARHL) and episodic memory functioning is mediated by measures of social functioning. **Methods:** Using data from 8,163 adults over 50 that participated in the Irish Longitudinal Study of Ageing (three waves, each two years apart), we used a multiple mediation model within a Structural Equation Modelling framework to explore potential social mediators of the relationship between

SARHL and episodic memory functioning, controlling for demographic and health covariates. **Results:** Neither the direct effect of self-reported hearing difficulties on memory functioning $(\beta = -.03)$, nor the total effect $(\beta = .01)$, were significant. A small inconsistent indirect effect

of self-reported hearing difficulties on episodic memory via weekly social activity engagement ($\beta = -.002$) was found. **Conclusions:** Self-reported hearing difficulties may exert an indirect effect on episodic memory via

weekly social activity engagement. The findings may have implications for identification of individuals at risk of memory decline in later life. ARTICLE HISTORY Received 22 August 2019 Accepted 5 February 2020

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KEYWORDS

Age-related hearing loss; cognitive decline; cognitive impairment; dementia; episodic memory; causal mechanism

Introduction

Increasing attention is being paid to age-related hearing loss (ARHL) as an important modifiable risk factor for dementia in the older population (Albers et al., 2015; Livingston et al., 2017; Loughrey, Kelly, Kelley, Brennan, & Lawlor, 2018). ARHL is a widely prevalent condition, with one-third of older adults afflicted with a disabling loss (World Health Organisation, 2018). It is estimated that effective management of ARHL could reduce the global burden of dementia by 9%, more than any other modifiable risk factor (Livingston et al., 2017). Additionally, ARHL has been associated with a wide range of psychiatric disorders (Blazer & Tucci, 2018).

Understanding how ARHL may affect cognition has important implications for its effective treatment and management. However, the causal basis for the association between ARHL and cognitive decline is not clear (Wayne & Johnsrude, 2015). Several pathways have been posited, including a common causal antecedent such as vascular risk factors (Panza, Solfrizzi, & Logroscino, 2015) or a mechanistic pathway such as sensory deprivation (Lin et al., 2014).

One mechanism through which ARHL may impact cognitive functioning is through its potential effect on social functioning and loneliness. Severity of hearing loss is linearly associated with an increased risk of restriction in social engagement (Mick, Kawachi, & Lin, 2014). Additionally, observational studies show an association between ARHL and social isolation (Mick et al., 2014) and loneliness (Sung, Li, Blake, Betz, & Lin, 2016). Social factors such as loneliness, social isolation, and social support are, in turn, considered to be risk factors for cognitive decline in later life (Boss, Kang,

& Branson, 2015; Livingston et al., 2017; McHugh Power, Tang, Lawlor, Kenny, & Kee, 2016; Shankar, Hamer, McMunn, & Steptoe, 2013) through plausible biological mechanisms such as HPA axis dysfunction, inflammation, blood pressure, physical activity, and gene expression (Cacioppo & Hawkley, 2009). There may be a cascade effect whereby ARHL leads to difficulties in speech and social engagement with consequences for cognitive function (Fulton, Lister, Bush, Edwards, & Andel, 2015). Research has demonstrated that the additional cognitive effort to understand speech with a hearing loss may disrupt encoding processes (McCoy et al., 2005; Tun, McCoy, & Wingfield, 2009). This is supported by neuro-imaging research which suggest a shift in cortical activation to frontal brain regions from the temporal lobes in response to auditory stimuli (Campbell & Sharma, 2013; Peelle, Johnsrude, & Davis, 2010; Peelle, Troiani, Grossman, & Wingfield, 2011; Wingfield & Grossman, 2006). Some research from epidemiologic data has suggested that this may have longer-term consequences for episodic memory (Rönnberg, Hygge, Keidser, & Rudner, 2014; Rönnberg et al., 2011; 2013). This cognitive domain is of primary importance in diagnosing the presence and type of dementia and in predicting its onset, particularly Alzheimer's disease (Salmon, 2012).

Previously, Amieva and colleagues showed that a longitudinal association between hearing loss and cognitive decline disappeared once social isolation and depression were controlled for as covariates (Amieva et al., 2015). A crosssectional study using data from the English Longitudinal Study of Ageing reported that social isolation mediated the association between hearing loss and cognitive functioning (Ray, Popli, & Fell, 2018). Ray and colleagues explored the impact of objectively measured age-related hearing loss on cognitive functioning with social isolation as a potential mediator (with items on marital status, contact with family and friends, and engagement in social organisations) in adults aged over 50. Since this study was cross-sectional in nature, using a Sobel style mediation test (in which the putative mediator is excluded and then included in the regression of the dependent variable upon the independent variable, and changes in the model observed), bias is highly likely in its results (Maxwell, Cole, & Mitchell, 2011), since temporal order of variables is a critical component of mediation analysis. As such, these analyses did not examine mediation in a strict sense, which limits inferences regarding the causal relationship between these factors, but the findings are consistent with such an effect (Imai, Keele, Tingley, & Yamamoto, 2011). While Ray and colleagues measured hearing loss objectively, it is also of interest to explore the impact of subjective age-related hearing loss on clinical outcomes such as cognitive functioning. Though pure-tone audiometry is the gold standard for assessment of peripheral hearing, there is a role for self-reported difficulty, since this likely also encompasses factors such as difficulties in sound segregation, in using indexical cues such as differences in voices of others, and the ability to spatially locate sounds in space - all of which may affect the compensatory cognitive resources required to successfully hear and understand, particularly speech (Gates & Mills, 2005; Helfer, Merchant, & Wasiuk, 2017; Hornsby & Kipp, 2016; Humes, 2013; Kim et al., 2017; Moore et al., 2014; Wayne & Johnsrude, 2015). Thus subjective or self-reported agerelated hearing loss may be an important functional measure for older adults.

In this study we examined whether social factors, namely loneliness, weekly social activity engagement, and a count of friends and relatives that individuals felt close to, mediated the relationship between subjective age-related hearing loss (SARHL) and episodic memory function. We used three waves of data to allow for temporal assumptions necessary to conduct a mediation analysis (MacKinnon, Fairchild, & Fritz, 2007). If it were the case that SARHL impacts memory because of disuse, then social disengagement, which is likely to occur with SARHL for the reasons specified above, should partly mediate the relation-ship between SARHL and memory functioning.

Methods

Participants

Data from the first three waves of the Irish Longitudinal Study on Ageing (TILDA) were utilised for the current analysis. Wave one of data collection was conducted between 2009 and 2011, with follow-up waves conducted every two years thereafter. The study is a representative cohort study of ageing adults in Ireland, and is described in terms of its design in detail elsewhere (Kenny et al., 2010). Participants were recruited using a stratified random sampling approach based on all geographical units of Ireland, and for the purposes of the current study, only data collected from participants aged over 50 (n = 8,163) were considered (the remainder were spouses or partners of each participant who were also invited to participate in the study despite being under the age of 50).

Participants had a mean age of 63.83 (age range 50-105, standard deviation = 9.78) and 45.8% were male. Sample size was calculated a priori to be 8,000 in order to be nationally representative (Kenny et al., 2010). Participants completed a home-based interview with a TILDA assessor, as well as completing a questionnaire. Depending on their ability, participants were also invited to visit the TILDA health centre at waves 1 and 3 to take part in a more comprehensive health assessment. Individuals who wore hearing aids and who reported a diagnosis of dementia, Alzheimer's disease, or serious memory impairment at baseline were removed from analyses, yielding a final sample size for the current analysis of 8040.

Between waves 1 and 2, 1180 participants were lost to follow-up (of these, 205 were deceased, 166 were not traceable, and 809 refused). Between waves 2 and 3, a further 304 had deceased, 89 were not traceable, and 56 were lost to follow-up for other reasons.

Measures

The outcome of interest was episodic memory functioning, which was specified as a latent variable with two indicators of episodic memory (immediate and delayed word recall). This latent variable of episodic memory was measured both at wave 2 and wave 3. Immediate and delayed word recall were evaluated in a manner replicated from the Health and Retirement Study (HRS) whereby ten nouns (e.g.child, king, market) were read aloud to the participant, whose task was to repeat as many as possible immediately following the initial reading (to give a score of immediate word recall) and again following a distractor task (to give a score of delayed word recall).

The main predictor of interest in the current study was subjective age-related hearing loss (SARHL), specified as a latent factor with two components. A third indicator, whereby individuals reported whether or not they had difficulty following a conversation, was removed due to poor factor loading (following guidelines provided by Little, Lindenberger, and Nesselroade (1999)). The first indicator was self-rated deafness. Initially, participants were asked to rate their own hearing with the question "Is your hearing (with or without a hearing aid) a) excellent, b) very good, c) good, d) fair, or e) poor?" Scores were reversed to yield a measure of self-rated deafness, in order to make the values of this variable consistent in direction with the other indicator of hearing quality. The second indicator was a question about whether the participant experienced hearing problems (participants responded yes or no). All variables were coded numerically to allow for the measurement model to calculate factor loadings. Of these measures, the first indicator has previously been shown to be the best self-report measure of hearing decline (as validated against the Whispered Voice test (Gibson, Cronin, Kenny, & Setti, 2014); and in the same study, the second indicator also demonstrated acceptable performance in terms of diagnostic value for hearing deficits. While pure-tone audiometry remains the clinical gold standard for evaluating hearing loss objectively among older adults, it is difficult to integrate within large-scale epidemiological studies, and thus subjective reports of hearing loss have been validated for use in many such studies (Ferrite, Santana, & Marshall,

2011; Gibson et al., 2014) due to their high degree of correlation with such objective measures. Measures of hearing difficulty were available only in wave 1.

Mediators of interest for the current study were social in nature, although we acknowledge that there may be other mediators of the relationship between self-reported hearing difficulties and memory functioning. Loneliness at waves 1 and 2 was specified as two separate latent variables, one indicating loneliness at each wave. For both variables, there were four indicators: items from the 5-item modified version of the UCLA loneliness scale (Russell, 1996), with one item removed because of poor factor loading (Item 4: "Are you in tune with others around you?"). A second mediator was social activity engagement, which was measured here as a yes or no response to the question "Do you engage in social activity at least once a week?". This was measured at waves 1 and 2. A third mediator, also measured at waves 1 and 2, was a count of the number of friends and relatives the participant reported feeling close to.

Covariates were also included in the model. These were age, sex, education (categorised as having received no education, having received a second level qualification, or having received at least a third level qualification), physical health status (a count of chronic conditions reported by the participant), functional status (activities of daily living measure), and depressive symptomatology (measured as a continuous score of the Centre for Epidemiological Studies 20-item depression scale (Radloff, 1977) with the item asking about loneliness removed, in order to avoid multicollinearity).

Data analysis

A Structural Equation Modelling (SEM) framework was used for all analyses. This framework is flexible in terms of missingness and can model multiple pathways at once, making it particularly suitable for exploration of multiple mediation pathways. The model is illustrated in Figure 1 (below), with results included. Loneliness, weekly social activity engagement, and number of friends and relatives that the participant felt close to were the mediators of interest. The mediation model in SEM was created using the lavaan package in R software (Rosseel, 2012), with a maximum likelihood estimator, and full information maximum likelihood approach to missingness. Covariates (measured at wave 1) were specified to predict the latent memory function factor (measured at wave 3). Baseline levels of the mediators and outcomes (baseline being wave 1 for the mediators, and wave 2 for the outcomes) were controlled for in the analyses. Covariates at wave 1 also were included as predictors of each mediator at wave 2. Same-wave residuals at baseline were constrained to correlate. All three cognitive indicators were tests based on the same list of ten words and as a result residuals would be expected to correlate.

Results

Descriptive statistics

The sample is characterised below, in Table 1. Mean levels of loneliness were low. Additionally, SARHL seemed to affect a minority of the population. Bivariate correlations between variables of interest were mostly small but statistically significant (see Table 2).

Structural equation model

The model converged normally after 124 iterations and fit was acceptable $[\chi^2_{236} = 3817, p < .001; CFI = 0.92, TLI = 0.90, RMSEA = 0.057 (Cl_{90} = 0.056; 0.059), SRMR = 0.086].$ Items loaded relatively well onto the latent factors (see Table 3 where values ranged from 0.79 to 0.96) and reliability for each latent variable was high (omegas are reported in Table 3 whereby a value of > 0.70 indicates high reliability). Factor loadings for memory at waves 2 and 3 were high, ranging from 0.76 to 0.87. A similar pattern of high factor loadings was observed for the latent variable of loneliness at waves 1 and 2 (ranging from 0.73 to 0.81), and for hearing at wave 1 (0.69, 0.95).

The structural component of the model showed that the association between SARHL at wave 1 and memory functioning at wave 3 was small ($\beta = .012$) and did not reach statistical significance (see Table 4), controlling for memory



Figure 1. Graphical representation of the model described in Table 4, above. Mediators are Loneliness, Weekly Social Activity Engagement, and Social Connectedness; Independent Variable is SAHL, and Criterion Variable is Memory Functioning. Figures depicted are betas (standardised coefficients) whereby * = p < 0.05; ** = p < 0.01; *** = p < 0.001; n.s. = not significant at the p < 0.05 level.

Table 1. Sample characteristics at wave 1, n = 8,163.

Variable Name	Standard Deviation	
Age	63.83 (range 50-105)	9.78
Sex	45.8% male (n = 3738) 54.2% female (n = 4425	
Education Level	30.7% no qualifications 39.9% second level qualification 29.4% third level qualification	
Immediate Recall (Trial 1)	5.69 (range = $0-10$)	1.74
Immediate Recall (Trial 2)	7.44 (range = $0-10$)	1.89
Delayed Recall	5.87 (range = $0-10$)	2.35
Self-rated Hearing	27.35% (n = 2233) – Excellent 30.45% (n = 2486) – Very good 27.7% (n = 2289) – Good 11.77% (n = 961) – Fair 2.37% (n = 194) - Poor	
Problems with Hearing	85.86% (n = 7019) - No problems 14.14% (n = 1156) - Problems	
Difficulty Following Conversations	93.44% (n = 7639) – No difficulty 6.56% (n = 116) – Difficulty	
Loneliness (scores calculated using 5 items)	1.24 (range $= 0$	
Social Activity Engagement	29.3% (n = 2028) no weekly activity engagement 70.67% (n = 4887) weekly activity engagement	
Depressive Symptomatology	4.79 (range 0-60)	6.22
Number of Chronic Illness Conditions Reported	Median = 1 (mean = 1.71); range 0-0	1.45
Activities of Daily Living Limitations	$ \begin{array}{l} \mbox{Median} = 0 \ 0 \ \mbox{limitations: } 91.45\% \ (n = 7476) \ 1 \ \mbox{limitations: } 5.53\% \ (n = 452) \ 2 \ \mbox{limitations: } 1.47\% \ (n = 120) \ 3 \ \mbox{limitations: } 0.76\% \ (n = 62) \ 4 \ \mbox{limitations: } 0.42\% \ (n = 34) \ 5 \ \mbox{limitations: } 0.21\% \ (n = 17) \ 6 \ \mbox{limitations: } 0.17\% \ (n = 14) \end{array} $	ins:

Table 2. Correlation Matrix containing all relevant variables to above analyses (T1 = Time 1; T2 = Time 2; T3 = Time 3; SE = Social Engagement). All correlations listed are Spearman correlations. (* p < 0.05; ** p < 0.01; *** p < 0.001).

	-				•							
		1	2	3	4	5	6	7	8	9	10	11
1	Self-rated Deafness T1	1										
2	Problems Hearing T1	0.70***	1									
3	Problems with Conversation T1	0.28***	0.29***	1								
4	Loneliness T1	0.12***	0.07***	0.07***	1							
5	Loneliness T2	0.10***	0.07***	0.05***	0.65***	1						
6	Social Engagement T1	-0.07***	-0.04***	-0.03*	-0.09***	-0.09***	1					
7	Social Engagement T2	-0.10***	-0.07***	-0.05***	-0.11***	-0.10***	0.37***	1				
8	Immediate Recall T1	-0.20***	-0.15***	-0.14***	-0.11***	-0.09***	0.09***	0.09***	1		1	
9	Immediate Recall T2	-0.19***	-0.12***	-0.15***	-0.12***	-0.11***	0.09***	0.08***	0.51***	1		1
10	Delayed Recall T1	-0.19***	-0.14***	-0.12***	-0.11***	-0.09***	0.09***	0.07***	0.69***	0.55***	1	
11	Delayed Recall T2	-0.17***	-0.17***	-0.13***	-0.11***	-0.09***	0.10***	0.08***	0.51***	0.73***	0.58***	1

 Table 3. Measurement component of Structural Equation Model, describing results for three latent factors: Hearing Problems, Loneliness, and Memory Functioning.

	Factor Loading	SE	Z	р	Omega
Memory wave 2 \sim					0.85
Immediate Recall 1	0.77	0.02	58.38	<.001	
Immediate Recall 2	0.87	0.02	68.25	<.001	
Delayed Recall	0.80	0.03	62.89	<.001	
Memory wave 3 \sim				<.001	0.87
Immediate Recall 1	0.76	0.02	51.41	<.001	
Immediate Recall 2	0.87	0.02	57.11	<.001	
Delayed Recall	0.79	0.02	56.12	<.001	
Loneliness wave 1 \sim					0.79
1	0.76	0.01	56.83	<.001	
2	0.74	0.01	54.98	<.001	
3	0.77	0.01	57.25	<.001	
5	0.73	0.01	53.85	<.001	
Loneliness wave 2 \sim					0.81
1	0.75	0.01	51.13	<.001	
2	0.77	0.01	51.49	<.001	
3	0.81	0.01	53.65	<.001	
5	0.74	0.01	50.81	<.001	
Hearing Problems \sim					0.96
Self Rated Deafness	0.95	0.04	23.85	<.001	
Problems Hearing	0.69	0.01	22.55	<.001	

functioning at wave 2. Loneliness ($\beta = -.03$), weekly social activity engagement ($\beta = .04$) but not number of close friends and relatives ($\beta = -.00$), all at wave 2 had small but significant associations with memory functioning at wave 3, such that better memory was related to lower levels of loneliness, and evidence of weekly social activity engagement. Increasing age, female sex, lower levels of education (but not depression, chronic disease count, or ADL impairments) were also associated with poor memory functioning (see Table 4).

SARHL at wave 1 was also associated with weekly social activity engagement ($\beta = -.05$) at wave 2, but not with loneliness ($\beta = .01$) or number of close friends and relatives ($\beta = 0.01$; see Table 4), controlling for wave 1 levels of each outcome. Weekly social activity engagement ($\beta = -.002$), but not loneliness ($\beta = -.00$) or number of close friends or relatives ($\beta = -0.00$), were found to be significant and inconsistent partial mediators¹ of the relationship between SARHL and memory functioning. Neither the direct effect of SARHL on memory functioning ($\beta = -.03$), nor the total effect ($\beta = .01$), were significant. Because of this, the examined "mediation" effect should be better described as an indirect effect (Holmbeck, 1997). As such, it is possible to state that SARHL exerted an indirect effect on memory functioning via weekly social activity engagement.

Discussion

It was hypothesised that loneliness, weekly social activity engagement and number of close friends and relatives would mediate the association between self-reported hearing difficulties and episodic memory functioning. No total effect of hearing difficulties on episodic memory was found, nor was there a direct effect. An indirect effect of SARHL on episodic memory functioning via weekly social activity engagement was found, but no evidence was found that it or the other social factors (loneliness, number of close friends and relatives) were mediators of this relationship. While social factors have been hypothesised to mediate the association between SARHL and cognitive decline (Pichora-Fuller, Mick, & Reed, 2015), to the best

Table 4. Structural component of SEM.

· · · ·	Beta	SE	Z	р
Memory wave 3 as an outcome				
Memory wave 2	0.642	0.026	34.45	<.001
Loneliness wave 2	-0.032	0.013	-2.379	0.017
Social Activity Weekly Engagement wave 2	0.036	0.040	2.923	0.003
Number of close friends relatives wave 2	-0.000	0.003	-0.011	0.991
Hearing	0.012	0.019	0.878	0.380
Age	-0.213	0.002	-16.059	<.001
Sex	-0.094	0.036	-7.514	<.001
Education	0.118	0.024	9.175	<.001
Depressive Symptomatology	-0.016	0.003	-1.228	0.220
CVD conditions	-0.010	0.039	-1.084	0.278
ADL impairments	-0.009	0.042	-0.734	0.463
Loneliness wave 2 as an outcome				
Loneliness wave 1	0.700	0.028	35.604	<.001
Hearing wave 1	0.008	0.019	0.629	0.529
Age	0.000	0.002	0.001	0.999
Sex	-0.020	0.036	-1.584	0.113
Education	-0.012	0.024	-0.930	0.352
Depressive Symptomatology	0.139	0.003	10.753	<.001
CVD conditions	0.010	0.040	0.812	0.417
ADL impairments	-0.006	0.042	-0.437	0.662
Social Activity Weekly Engagement wave 2 as an outcome				
Social Activity Weekly Engagement wave 1	0.363	0.014	26.823	<.001
Hearing wave 1	-0.047	0.006	-3.302	.001
Age	-0.016	0.001	-1.167	0.243
Sex	-0.068	0.012	-5.030	<.001
Education	0.106	0.008	7.666	<.001
Depressive Symptomatology	-0.061	0.001	-4.385	<.001
CVD conditions	-0.008	0.013	-0.585	0.559
ADL impairments	-0.042	0.014	-3.004	0.003
Number of close friends and relatives wave 2 as an outcom	e			
Number close friends relatives wave 1	0.424	0.013	31.49	<.001
Hearing wave 1	0.007	0.085	0.509	0.611
Age	0.019	0.009	1.328	0.184
Sex	-0.031	0.166	-2.313	0.021
Education	-0.003	0.112	-0.231	0.817
Depressive Symptomatology	-0.041	0.013	-2.926	0.003
CVD conditions	-0.001	0.184	-0.047	0.962
ADL impairments	0.000	0.194	0.004	0.997
Mediators				
Loneliness	-0.000	0.001	-0.608	0.543
Social Activity Weekly Engagement0.991	-0.002	0.001	-2.189	0.029
Number of close friends and relatives wave 2	-0.000	0.000	-0.011	0.028
Sum of Mediators	-0.002	0.001	-2.197	0.466
Total effect	0.010	0.019	0.729	

of our knowledge this is the first study to directly assess this hypothesis using a longitudinal mediation approach with multiple waves, considered a pre-requisite to properly evaluate mediation pathways (MacKinnon et al., 2007). Additionally, a nationally representative population-based design was used.

SARHL does not, according to our findings, have an effect on episodic memory functioning in older adults in a direct fashion. This finding differs from the study by Amieva et al., (2015), although they explored global cognitive functioning as an outcome, and the timeframe was much longer than in the current analysis (25 years). Agerelated cognitive decline is a slow process and it is possible that the timeframe in the current analysis was not sufficiently long to observe direct associations between hearing difficulties and memory. In our analysis, the beta coefficient obtained for the association between selfreported hearing difficulties and episodic memory was small and non-significant. However, previous epidemiological studies researching this relationship have reported modest beta values (Deal et al., 2015, 2016; Harrison Bush, Lister, Lin, Betz, & Edwards, 2015). This may be a reflection of hearing loss having a cumulative effect on cognition over time with detrimental effects only observable in the later stages of this process or with increasing severity of hearing impairment (Lin et al., 2013; Wayne & Johnsrude, 2015). It is also possible that the current analytic strategy had sufficient statistical power to demonstrate a mediation effect (via social activity engagement) but not a direct effect between SARHL and episodic memory functioning (MacKinnon, Krull, & Lockwood, 2000), which can manifest in the pattern of results shown.

The current findings, which show how SARHL may exert a small indirect effect on episodic memory functioning via social factors warrants further research. The pattern of results is consistent with the notion of a small negative indirect effect (albeit not statistically significant) between SARHL and memory functioning, and that engagement in weekly social activity offsets this by a small amount. Thus, the relationship between hearing loss and cognitive decline in older adults may be better understood by examining change in social functioning with hearing loss over different temporal intervals.

Furthermore, our results indicate that the impact of SARHL on social factors and subsequently cognitive functioning may be complex and vary with each social factor. Prior research has suggested that different social factors themselves may make unique contributions to cognitive function and dementia outcomes in the general ageing population (Kelly et al., 2017; Kuiper et al., 2015). Hearing loss in turn may have differential effects on these social factors. For example, hearing loss (assessed by audiometry) was associated with greater loneliness or perceived social isolation independently of objective social participation (Weinstein & Ventry, 1982). Thus hearing loss may directly impair the quality of socialisation, with later consequences for participation. Hearing loss may also have differing effects on social functioning in different demographic groups. In their analysis, which used a similar measure of social connectedness, Mick et al. (2014) reported a significantly increased risk of social isolation with hearing loss for women aged 60-69 but not for other included demographic groups. Further research is required to untangle this relationship and assess how social factors may alter over time following onset of hearing loss with possible consequent changes in cognitive functioning.

Our study has some limitations which future observational and clinical studies may consider. Future studies need to assess these mechanisms in a population of older adults with more prevalent hearing loss, since our sample showed generally good hearing according to self-reports. However, we frame our results as a potential explanatory model rather than a predictive model. Future studies should also examine social factors as mediators for the relationship between hearing loss and dementia and with subtypes such as Alzheimer's disease or vascular dementia. Social factors affecting conditions such as depression and cognitive activity have been reported to be associated with age-related decline independent of neuropathological burden (Wilson et al., 2013; 2014). The age range of the participants involved in TILDA was very broad (55 years). Hearing loss in adults of this age range is a potentially critical issue, with researchers recommending that opportunistic screening for objective hearing loss is undertaken among those over the age of 50 (Ray et al., 2018). Thus, conducting research into the antecedents of SARHL among populations of this age is warranted.

An additional limitation is that as we used a self-report measure we could not assess the impact of the degree of hearing loss, and this may have contributed to the nonsignificant direct effect. In previous studies, a moderate to severe hearing loss was associated with a significant decline in memory whereas a mild hearing loss was not (Deal et al., 2015, 2016). It would be of interest to explore mediation effects in a study which had obtained objective measures of hearing loss, to see if the effect via social functioning persists.

Another limitation is that while we examined the mediating influence of social factors we cannot infer from our findings what the aetiological mechanisms underpinning this mediation may be (VanderWeele, 2009; Westhorp, 2018). We have highlighted above several potential biological mechanisms (Cacioppo & Hawkley, 2009) which may be examined in future experimental studies. Tying specific social changes following hearing loss to underlying biological changes would give further support to their hypothetical link to neurocognitive changes. Additionally, the methods used in our study to measure social factors were quantitative and may not have fully measured these factors. An experimental approach or an approach using mixed methods may better capture these variables and more appropriately establish a social mechanism between hearing loss and cognitive decline (Spencer, Zanna, & Fong, 2005). The relationship between hearing loss and cognitive decline most likely involves multiple aetiological mechanisms (Panza et al., 2015; Uchida et al., 2018) which make differential contributions to cognitive outcomes. Further research is required to examine if the social factors examined in this study offer potential pathways for developing therapies which aim to prevent cognitive decline among those with a hearing loss (Kelly & Russo, 2018).

Another consideration is the impact of hearing loss on speech understanding. Decline in speech perception and subsequent impairment of social function and concomitant loneliness may represent a pathway through which hearing loss progressively disrupts cognitive function. Several neuro-imaging studies have reported decreased activation and atrophy in neural regions associated with speech perception (Campbell & Sharma, 2013; Lin et al., 2014; Peelle et al., 2011). Additionally, such studies have also reported increased activation of the other regions including the frontal lobes in response to auditory stimuli, possibly reflecting recruitment of higher cognitive functions to promote perception of speech (Campbell & Sharma, 2013; Husain et al., 2011; Peelle et al., 2010, 2011; Wingfield & Grossman, 2006). There also appears to be cortical reorganisation of the auditory cortex in response to visual stimuli in the early stages of hearing loss possibly as a compensatory mechanism (Campbell & Sharma, 2014). Future studies could assess this as this dataset had no measures of speech understanding. This is of clinical importance as improving speech perception and modifying social factors may potentially supplement audiological treatment for hearing loss to reduce risk for cognitive decline and dementia.

Our study indicates that further research into modifiable social mechanisms mediating the link between hearing loss and cognitive functioning is warranted. As social functioning may be optimised through various therapeutic strategies, intervention trials to assess this in the hearing loss population are required.

Note

 Mediation refers to the putative mechanism through which one variable can impact another, via a third variable. A partial mediation occurs if the third ("mediator"/M) variable accounts for only a proportion of the total effect of the independent variable ("X") on the dependent variable ("Y"). Partial mediation can be contrasted with complete mediation, which occurs when M accounts for the full total effect of X on Y. Mediation effects, whether complete or partial, can be consistent or inconsistent. An inconsistent mediation effect occurs when the mediated effect has a different sign to the overall effect (MacKinnon, Krull, & Lockwood, 2000).

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Ethical standards

The Irish Longitudinal Study of Ageing was approved by the Faculty of Health Sciences Research Ethics Committee in Trinity College Dublin prior to starting. The study was conducted in accordance with the 1964 Helsinki declaration, and its later amendments. Informed consent was obtained from all individual participants included in the study.

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