# Hearing loss mediates executive function impairment in sleep disordered breathing

Catherine M Hill BM MSc1&2, Romola S Bucks PhD3, Colin R Kennedy MD1, Dawn Harrison BSc4, Annette Carroll BSc5, Nicolas Upton1 Alexandra M Hogan PhD MBBS6&7

**Affiliations**

Division of Clinical Experimental Sciences, Faculty of Medicine, University of Southampton, UK

 Southampton Children’s Hospital, Southampton, UK.

School of Psychological Science, University of Western Australia, Perth, Australia

Research Governance, Tayside Medical Science Centre, Ninewells Hospital, Dundee, UK

Sleep Disorders Unit, Canberra Hospital, Australia

Cognitive Neuroscience & Psychiatry, University College London, Great Ormond St Institute of Child Health; UK

1. Department of Anaesthetics, Addenbrookes Hospital, Cambridge University Hospitals Trust, Cambridge

**Address correspondence to**

Dr Catherine Hill, Division of Clinical Experimental Sciences, Mail point 803CB, G-Level, University Hospital Southampton, Tremona Road, Southampton, SO16 6YD, United Kingdom. Tel +4423 8120 6091, e mail cmh2@soton.ac.uk

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**Abbreviations:**

CMEDHQ: childhood middle ear disease and hearing questionnaire

dB: decibels

ECG: electrocardiogram

EEG: electroencephalogram

EF: executive function

EMG: electromyogram

GEC: global executive composite

OAHI: obstructive apnea/hypopnea index

OSA: obstructive sleep apnea

RMSEA: Root Mean Square Error of Approximation

SDB: sleep disordered breathing

SDQ: strengths and difficulties questionnaire

**Contributors' Statement:**

Dr Hill conceptualized and designed the study, contributed to data collection and analysis, drafted the initial manuscript, and approved the final manuscript as submitted.

Dr Bucks carried out the data analysis, reviewed and revised the manuscript,

Professor Kennedy assisted with the design of the study and approved the final manuscript as submitted

Ms. Harrison undertook data collection and data entry and critically reviewed and approved the final manuscript as submitted

Mrs Carroll undertook polysomnography data collection and scoring, assisted with data entry and critically reviewed and approved the final manuscript as submitted.

Mr. Upton assisted with data entry, drafting of the manuscript and approved the final manuscript as submitted.

Dr Hogan conceptualized and designed the study, contributed to data collection and analysis, contributed to and approved the final manuscript as submitted.

**Abstract**

**Background:** Sleep disordered breathing (SDB) is often co-morbid with conductive hearing loss in early childhood, due to a shared aetiology of adenotonsillar hypertrophy. Hearing loss is independently associated with impairment of executive function and behavioural difficulties. We hypothesised that these impairments in children with SDB may be mediated via hearing loss.

**Methods:** Fifty-eight children including 37snorers awaiting adenotonsillectomy and 21 healthy non-snoring controls, aged 3-5 years, were assessed with pure tone audiometry, Strengths and Difficulties (SDQ), Behavior Rating of Executive Function (BRIEF-P) and Childhood Middle Ear Disease and Hearing questionnaires. Polysomnography in snoring children generated an obstructive apnea/hypopnea index (OAHI). Two regression models examined the effect of SDB and the mediating impact of hearing loss on BRIEF and SDQ.

**Results:** Snoring children had significantly poorer hearing, greater past exposure to hearing loss and higher total SDQ and BRIEF-P scores than non-snoring controls. The first regression model, including all children, demonstrated that the impact of snoring on BRIEF\_P, but not SDQ, was entirely mediated by history of hearing loss exposure, but not same day audiometry. The second model examined snoring children only, categorising the group into 12 with obstructive sleep apnea (OSA) (OAHI > 5) and 25 without. OSA had a direct effect on SDQ scores but this was not mediated by history of hearing loss.

**Conclusion:** In early childhood, conductive hearing loss mediates the relationship between SDB, irrespective of severity, and parent report of executive function but not behaviour. Treatment of hearing loss in pre-school SDB might improve executive function.

**Introduction**

Obstructive sleep disordered breathing (SDB), from simple primary snoring to sleep apnea (OSA), affects up to 10% and 5.7% of children respectively[[1]](#endnote-1),[[2]](#endnote-2). Childhood SDB is associated with impaired cognitive function, general intelligence, learning, school performance, language skills and behaviour[[3]](#endnote-3),[[4]](#endnote-4),[[5]](#endnote-5). Furthermore, recent data from a cross-sectional study of over 1000 children have suggested a ‘dose-response’ relationship between SDB and cognitive and behavioural function5. The pre-frontal cortex sub-serving executive function (EF), appears to be particularly vulnerable to the sleep fragmentation and intermittent hypoxia that characterize OSA[[6]](#endnote-6),[[7]](#endnote-7).

Upper airway lymphoid hypertrophy is a key cause of SDB in otherwise healthy, non-obese, pre-school children. Hence, first line treatment is adenotonsillectomy1. However, adenotonsillar hypertrophy also causes Eustachian tube dysfunction and otitis media with effusion[[8]](#endnote-8),[[9]](#endnote-9), a leading cause of conductive hearing loss, affecting up to 20% of 2 year olds and 8% of 8 year olds[[10]](#endnote-10). Not surprisingly, given this shared aetiology, hearing loss is a recognised co-morbidity in SDB[[11]](#endnote-11). Children aged 2 to 12 years with OSA have a 5-fold risk of otitis media with effusion compared to non-snoring controls[[12]](#endnote-12) while habitual snorers have a 1.3-fold risk[[13]](#endnote-13).

Importantly, hearing loss in childhood is independently associated with behavioural and EF impairment. Parent reported EF was significantly worse than normative data in 214 children (5-18 years) with hearing impairment[[14]](#endnote-14). Similarly, meta-analysis confirms greater behavioural difficulties in children with hearing loss, including those with sensorineural hearing loss unrelated to Eustachian tube dysfunction.[[15]](#endnote-15) With respect to conductive hearing loss, 4-5 year olds with recurrent otitis media are more than twice as likely as healthy peers to have abnormal/borderline Strengths and Difficulties questionnaire (SDQ) scores [[16]](#endnote-16) and Total Difficulties scores are around 25% higher[[17]](#endnote-17). Furthermore, behavioural difficulties in children with hearing loss are greatest in those with greater language impairment[[18]](#endnote-18).

We aimed to explore the extent to which hearing loss contributes to parent report of EF and behavioural problems in typically developing, otherwise healthy young children with SDB. We hypothesised that the impact of SDB on EF and behaviour would be wholly or partially mediated by hearing loss.

**Patients and methods**

Setting: Children aged 3-5 years inclusive, with a history of snoring were recruited from Southampton Children’s Hospital and Queen Alexandra Hospital, Portsmouth, UK. Children were listed for adenoidectomy and/or tonsillectomy. Non-snoring, similar aged, control children with no otolaryngology history were recruited from pre-school nurseries. Children were assessed at the NIHR Wellcome Trust Clinical Research Facility, Southampton.

Children with cranio-facial abnormalities, moderate or severe learning disabilities, attention deficit hyperactivity disorder, chronic respiratory/cardiac conditions, significant allergy, or where parents were not conversant in English were excluded.

The study was approved by the UK National Research Ethics committee (06/Q1702/12) and parents signed a consent form on behalf of their child.

*Measures*

Socio-economic status: Mothers reported age of completion of education in 4 categories: completed secondary education to age 16 years; ‘A’ levels (to 18 years) or a college diploma; undergraduate degree, or post-graduate degree.

The Behavior Rating of Executive Function-Preschool questionnaire (BRIEF-P): this comprises 86 parent-rated items based on a 3-point scale (never; sometimes; often). Sub-scales reflect core domains of EF(Inhibition, Working Memory, Emotional Control, Shift and Plan/Organise) and generate a Global Executive Composite (GEC), where clinically significant T scores are > 65[[19]](#endnote-19).

The Strengths and Difficulties questionnaire: this 25 item behavioural screener, with established reliability and validity[[20]](#endnote-20), comprises 5 subscales (emotional symptoms, conduct problems, hyperactivity/inattention, peer relationship problems, and pro-social behaviour), each with 5 questions answered with a 3-point rating scale (0, not true; 1, somewhat true; 2, certainly true). A total difficulties score is generated by summing all subscales, except prosocial behaviour. Total scores of 0-13 are considered normal; 14-16 borderline and 17-40 abnormal.

The Childhood Middle Ear Disease and Hearing Questionnaire (CMEDHQ)[[21]](#endnote-21) comprises 19 questions about life-time hearing problems and treatment. Eleven questions generate a total score (maximum 79). The questionnaire was developed by the UK Medical Research Council Hearing Institute as a screening tool. Compared to standard, pure-tone audiometry it has sensitivity of 77.3%, versus 89.7%, and specificity of 70.9%, versus 71.5%, to predict hearing loss requiring interventionwith a threshold score of >18 indicative of a past history of significant hearing loss21.

Assessment of snoring*:* Snorers were actively recruited from surgical waiting lists based on a positive snoring history. Snoring status was further confirmed using a question in the CMEDHQ ‘Has your child snored?’ with potential responses ‘Only with colds, never, rarely, often or always’. Children were considered snorers if they snored ‘often or always’ and non-snorers if they snored rarely or never or only with colds.

Pure tone audiometry*:* Children able to cooperate were tested using an abbreviated SWEEP test (Grayson-Stadler, MN) according to British Society of Audiology guidelines. Pure tones were presented unilaterally via headphones in sequence: 1000Hz, 2000 Hz, 4000Hz, 500 Hz and 250Hz, initially at 40dB. If a positive response was achieved, the child’s hearing threshold was determined by sequentially reducing the tones by 10dB. Conversely, if no response was achieved, sound levels were increased to a maximum of 80dB. The best sound level detected with confidence in the speech range was recorded in each ear. Hearing impairment is defined by the [World Health Organisation](https://en.wikipedia.org/wiki/World_Health_Organisation) as a hearing loss with thresholds higher than 25dB in one or both ears.

Polysomnography**:** All snorers had a single night of polysomnographic evaluation using computerised systems (Alice 5 –Respironics, Chichester UK) according to recognised standards[[22]](#endnote-22). Montage included EEG (C3/A2, O1/A2, C4/A1, O2/A1), right and left EOG, bipolar submental EMG; thoracic and abdominal excursions; nasal airflow (Protech, Mukilteo, WA), finger pulse oximetry (Masimo Inc, Irvine, CA), ECG and synchronous video-recording. Sleep staging[[23]](#endnote-23) and respiratory events[[24]](#endnote-24) were scored according to standard criteria using Alice 5 Sleepware. Specifically, obstructive apnoea was defined by >90% drop in airflow for 2 breaths or more and hypopnoea as > 50% drop in airflow for 2 breaths of more accompanied by either ≥ 4% oxygen desaturation or an EEG arousal, in both cases accompanied by continued respiratory effort. The obstructive apnea/hypopnea index (OAHI) was defined as the number of obstructive apnoeas, hypopnoeas and mixed apnoeas per hour of total sleep time.

Anthropometric assessment: Height and weight were measured and body mass index computed.

*Statistical methods*

Continuous variables were inspected for normality. Group differences were explored using *t*-tests or Mann Whitney U tests, according to data distribution. Two mediation models examined the effect of hearing on the relationship between SDB and EF and behavioural measures, using Mplus v7.4 (Muthén & Muthén, Los Angeles, CA) indirect regression method[[25]](#endnote-25). The maximum-likelihood estimator was used and bootstrapping analysis was used to calculate 90% bias corrected confidence intervals (CI) using 10,000 bootstrap samples. Model fit was evaluated with chi square, the Root Mean Square Error of Approximation (RMSEA) with 90% CIs, and the Comparative Fit Index (CFI). A RMSEA value of <.05 indicates good model fit, whereas RMSEA >.05 but with a CI that includes .05 indicates adequate fit. A CFI >.90 indicates adequate and >.95 good model fit[[26]](#endnote-26). Given the potential impact of socio-economic status on snoring, hearing and the outcomes, mother’s education was entered as a predictor into each model. Statistical significance was set at 0.05. Effect sizes were expressed as Cohen’s *d* and standardised beta coefficients*.*

**Results**

Thirty-seven snorers (24 boys) and 21 non-snoring controls (12 boys) were recruited. Demographic variables are reported in Table 1. There were no differences between groups in age, gender distribution or body mass index and no child was obese. There was a significant difference in maternal educational level, [Χ2 (3) = 31.76, *p*< .001], with control mothers having higher educational attainment.

As would be predicted, snoring children had significantly greater hearing loss exposure (CMEDHQ scores), higher sound level thresholds on audiometry, higher total SDQ scores and BRIEF-P GEC scores than non-snoring control children (Table 1). Of the non-snoring control children, just 1/17 (6%) had a positive history of hearing loss, compared to 9/27 (33%) in the snoring group. Based on available pure tone audiometry data, 8/32 (25%) of snoring and none of the non-snoring control children had hearing loss as defined by a threshold of > 25dB in either ear. Only 1/21 (5%) of control children had an abnormal total SDQ score compared to snoring children of whom 5/37 (14% were classified as borderline and a further 12 (32%) as abnormal; similarly, while all non-snoring control children had normal BRIEF-P GEC scores, 7/37 (19%) snoring children had abnormal scores.

*Snoring versus non-snoring as a predictor of BRIEF-P and SDQ scores*

The first model, examined the presence of snoring as a predictor of BRIEF-P GEC score and SDQ total score and produced good fit, Χ2 (3) = 5.50, *p* = .139, RMSEA = 0.12, 90% CI 0-0.28, CFI = .97. Overall, being in the snoring group was significantly associated with greater EFproblems measured on the BRIEF-P (total effect size 0.42, 90%CI 0.25-0.60). However, 0.31/0.42 (73.8%) of this relationship was through mediating variables (standardised indirect effect estimate 0.31, 90% CI 0.12-0.51, p .008). Specifically, the impact of snoring on BRIEF-P scores was mediated by CMEDHQ scores (specific, standardised indirect effect estimate 0.23, 90%CI 0.6-0.41, p=.029), representing 0.23/0.31 (74.2%) of the total indirect effect, but was not mediated by best-ear pure tone audiometry (specific, standardised indirect effect estimate 0.08, 90%CI -0.4-0.19. p=.258). That is, snoring was associated with a history of poorer hearing, which predicted greater EF problems. Since the direct effect of snoring group on BRIEF-P scores was no longer significant, once the indirect effects had been considered (direct effect estimate 0.11, 90%CI -0.14-0.36, p=.454), the impact of snoring group on BRIEF-P performance was wholly mediated by a history of poorer hearing (CMEDHQ scores).

The total effect of snoring group on SDQ scores was also significant (total standardised effect 0.53, 90%CI 0.37-0.69, p<.001), but the indirect effect was not (total standardised indirect effect 0.18, 90%CI 0.03-0.354, p=.093). That is, being a snorer was associated with more problem behaviours on the SDQ, but this effect was independent of hearing. Table 2 reports the unstandardized estimates and confidence internals for this model, whilst, for ease of interpretation, figure 1 graphically presents the standardised paths.

(Figure 1 here)

*OSA as a predictor of BRIEF-P and SDQ scores within the snoring sample.*

To explore the impact of OSA on EF and behavioural problems, habitual snorers were divided into OSA or non- OSA groups based on 2 thresholds: OAHI of >2/hour and then > 5/hour. There were no significant differences in CMEDHQ scores, BRIEF-P scores, or best-ear pure tone audiometry (Table 1) between OSA and non-OSA children using a diagnostic threshold of OAHI > 2/hour and > 5/hour. That is snorers, irrespective of OAHI severity, did not differ with respect to measures of exposure to hearing loss or of executive function. However, when OSA was defined by OAHI of >5 /hour, individuals with OSA had higher SDQ behavioural difficulties scores (Table 1).

**Discussion**

We report, for the first time, that EF in children with sleep disordered breathing may be mediated by their lifetime exposure to hearing loss, as assessed by parental report but not by same day pure tone audiometry assessment. This suggests a specific role for hearing loss exposure to the development of EF difficulties in these children. The developmental time course of EF is protracted[[27]](#endnote-27) and life-time exposure to hearing impairment is therefore likely to be more relevant than a single assessment. Indeed, guidelines for treating otitis media with effusion emphasize the importance of serial assessment in children, because of the fluctuation of hearing loss in this condition[[28]](#endnote-28).

Behavioural difficulties, by contrast, are associated with obstructive sleep apnea severity and not mediated by children’s exposure to hearing loss.

Animal models of OSA, exposed to intermittent hypoxia, demonstrate localisation of neural dysfunction to the pre-frontal cortex and hippocampus6. Magnetic resonance spectroscopy data in children with OSA[[29]](#endnote-29) support similar localisation of neuronal injury, providing a potential neuropathophysiological pathway to executive dysfunction in SDB. Auditory pathways underpinning auditory perception myelinate during infancy[[30]](#endnote-30) , before OSA is generally a concern. However, the auditory cortex, which modulates the categorisation of auditory stimuli according to experience, namely ‘listening and learning’[[31]](#endnote-31), matures gradually during the pre-school years[[32]](#endnote-32), [[33]](#endnote-33), that is, the same time period in which adenotonsillar growth peaks. Importantly, this maturation involves improved connectivity between the auditory cortex and the frontal lobes. Language acquisition, specifically the development of an ‘internal language’, has been hypothesised to underpin the development of EFin early childhood[[34]](#endnote-34). Thus SDB and co-morbid hearing impairment at critical periods of brain maturation could synergistically constrain EF development through both alteration of pre-frontal cortex neuronal integrity, impaired hippocampal-dependant memory consolidation and impaired language development. Despite an extensive literature highlighting deficits in EF in children with SDB and documenting the fact that surgical correction of OSA is not followed by resolution of executive dysfunction[[35]](#endnote-35), measures of hearing loss have been overlooked. This is surprising, given their shared aetiology, and may have overlooked a potentially treatable cause.

*Limitations*

The small sample size and cross-sectional nature of this study mean that these results should be treated with caution and examined in future studies.

Although we did not confirm the absence of SDB in the non-snoring control children with polysomnography, the significant difference in pure tone audiometry measures between the groups lends some objective support to group assignment. Furthermore, in a large population based study of young children5, all 90 non-snoring children were confirmed to have an OAHI < 1/h of total sleep time on polysomnography, lending support to the specificity of a negative clinical history.

The BRIEF-P and SDQ questionnaires were all parental report measures and may have been subject to reporter bias. Furthermore, the BRIEF-P has been shown in previous studies to over-estimate working memory deficit compared to objective measures particularly in children with mild sleep disordered breathing[[36]](#endnote-36). However, bias for parents to score snoring children more highly on the BRIEF-P scale would predict a direct relationship between snoring and BRIEF-P, not the indirect relationship we have demonstrated via hearing loss exposure. It is likely that parental report captures important, ecologically valid, behaviours that represent complimentary dimensions of EF not measured in direct assessments[[37]](#endnote-37).

 Future studies should include objective measures of executive function, alongside parental report, to capture multiple dimensions of function.

### Summary

These observations suggest that hearing loss may mediate the relationship between SDB and EF in early childhood. Our interpretation is that hearing loss is likely to exert its effects on EF via language development. Further research could usefully test this hypothesis in a larger sample and assess whether targeted screening and treatment of hearing loss in SDB provides therapeutic benefit.

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