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Risk Factors For Hearing Decline From Childhood To Early Adolescence

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Objective: To identify risk factors of hearing decline between 9 and 13 years of age. The risk factors examined included sociodemographic, health, and lifestyle-related factors.

Methods: This study was embedded within a population-based prospective cohort study from fetal life onwards in the Netherlands. Pure-tone audiometry and tympanometry were performed at the age of 9 and 13 years. The hearing decline was defined as an increase in low-frequency or high-frequency pure-tone average of at least 5 dB in one of both ears. Multivariable logistic regression was performed to examine the association of possible risk factors with hearing decline. The study was conducted from April 2012 to October 2015, and from April 2016 to September 2019.

Results: Of the 3,508 participants included, 7.8% demonstrated a hearing decline in the low frequencies, and 11.3% in the high frequencies. Participants who reported alcohol consumption were more likely to have a hearing decline in the low frequencies (OR 1.5, 95% CI 1.1; 2.0). Moreover, a lower educational level was associated with an increased odds of having a hearing decline in the high frequencies (OR 1.4, 95% CI 1.0; 1.8). Age, sex, household income, personal music player use, and body mass index were not associated with hearing decline.

Conclusion: Educational level and risky behavior were significantly associated with hearing decline from childhood to early adolescence. The findings of the present study can help in the design of public health interventions to prevent hearing loss at a young age.

Key Words: adolescent, audiometry, child, hearing loss, risk-taking.

Level of Evidence: 2 (prospective cohort study)

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INTRODUCTION

As the third leading cause of years lived with disability globally, hearing loss (HL) can have a major impact on daily life.[1,2] Although the prevalence of HL increases with age, several health behaviors that contribute to HL present early in life.[3,4] For example, many young people voluntarily expose themselves to loud music in their leisure- time activities.[5] Cumulative exposure to excessive noise is known to damage the cochlea, which eventually results in irreversible HL.[6] As hearing damage at a young age can lead to a considerable burden in later life, prevention is of great importance.[4]

In the design of interventions aimed at the prevention of HL, it is essential to identify factors associated with acquired HL as early in life as possible. The previous cross-sectional studies have identified multiple risk factors for HL in childhood, including demographic factors like male sex and low socio-economic status (SES), as well as health and lifestyle-related factors such as exposure to excessive noise and obesity.[7–12] To date, no longitudinal investigations have been published as there are limited cohort studies with multiple assessments of audiometry from childhood to early adolescence. To elucidate the risk factors and ultimately possible causal pathways of HL in childhood, a large population-based longitudinal study design would be of added value.

Adolescence is a period of transition between childhood and adulthood. Compared to other life periods it is characterized by many physical and psychological changes,

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amongst others an increase in risky behavior.[13] About half of the teenagers engage in one or more risky behaviors, such as smoking, alcohol, and drug use, which are important determinants of disease.[14–16] While it is known that such risky behavior can jeopardize health, no study has examined the associations of risky behavior with changes in hearing in childhood to early adolescence.

Here we present the findings from a longitudinal population-based study on hearing from childhood to early adolescence. The aim of the current study was to investigate which potential risk factors are associated with a decline in hearing between 9 and 13 years of age. Besides the previously identified sociodemographic, health, and lifestyle-related factors, we examined the association between risky behaviors and hearing decline.

MATERIALS AND METHODS

Study Design and Population

This study was embedded in the Generation R study, a population-based prospective birth cohort study from fetal life onwards in Rotterdam, the Netherlands. The design and population have been described previously.[17] Participants and their parents were invited to the research center in the Erasmus Medical Center-Sophia Children's Hospital to undergo various measurements and received extensive questionnaires. A hearing assessment was performed for the first time around the age of 9 years (baseline), and for the second time around the age of 13 years (follow-up). A total of 5355 participants completed pure-tone audiometry at baseline, and 4774 during follow-up. Participants were eligible for inclusion if they completed pure-tone audiometry both at baseline and during follow-up ($n = 4139$). Due to time constraints, masking was not applied, and bone conduction thresholds were not measured. Tympanometry served as a measure of middle ear function, and was used to differentiate between conductive HL (CHL) and sensorineural HL (SNHL). Any HL, defined as an increase in low-frequency or high-frequency pure-tone average > 15 decibels hearing level (dB HL), in combination with a normal tympanogram was considered SNHL. Participants with HL in combination with abnormal or missing tympanometry were excluded ($n = 631$). The final sample included 3508 participants (Fig. 1). Data at baseline were collected from April 2012 to October 2015, and at follow-up from April 2016 to September 2019. The study protocol was approved by the Medical Ethical Committee of Erasmus MC University Medical Center in Rotterdam, the Netherlands.

Hearing Assessment

Audiometric testing was conducted in a sound-treated booth meeting the maximum permissible ambient sound pressure levels of ISO standard 8253-1. Pure-tone audiometry was performed using a clinical audiometer (Decos audiology workstation; version 210.2.6 with AudioNigma interface) and TDH-39P headphones with MX-41/AR ear cushions, calibrated every 12 months. Air conduction thresholds were obtained at frequencies 0.5, 1, 2, 3, 4, 6, and 8 kHz. Testing began at 20 decibel dB HL. The level was reduced by 10 dB HL or increased by 5 dB HL according to the presence or absence of a response. The actual threshold was set after 2 out of 3 responses were consistent, according to the shortened ascending method based on ISO standard 8253-1.[18] The right and left ears were alternately tested first. Tympanometry was performed with an Interacoustics AT235h tympanometer with a 226-Hz probe tone to assess middle ear function. Tympanograms were categorized as described by Jerger (1970), assessing a value of at least 0.25 ml as normal compliance and a value between −100 and 100 decapascals (daPa) as normal middle ear pressure.[19] Abnormal tympanometry could present external or middle ear pathology.

Definitions of Hearing Decline

The low-frequency pure-tone average (LPTA) was calculated by averaging thresholds at 0.5, 1, and 2 kHz, and the high-frequency pure-tone average (HPTA) by averaging thresholds at 3, 4, 6, and 8 kHz. The hearing decline was defined as an increase in LPTA or HTA of at least 5 dB in one of both ears between baseline and follow-up. In an additional analysis, the hearing decline was defined as an increase in LPTA or HPTA of at least 10 dB in one of both ears.

Covariates

Covariates were selected based on previous literature and data available from the Generation R study. Information on participants' age, sex, educational level, personal music player (PMP) use, and monthly household income were obtained via parental questionnaires. All covariates were assessed at baseline, except for educational level which was only available during follow-up. In the Netherlands, secondary education is offered at three different

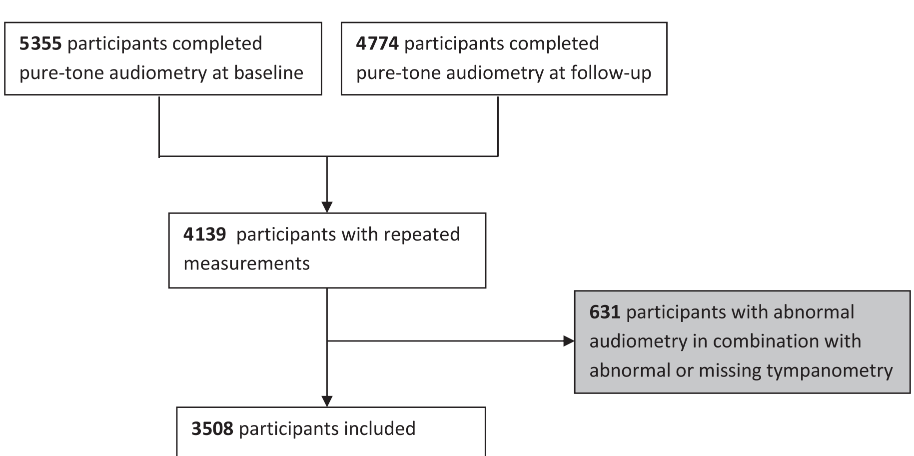

Fig. 1. Flow chart of the study population.

levels. The choice of the educational path is made at the end of primary school, and it is based on the student's academic level and interests. The educational level of the participant was classified as low (primary education only or preparatory secondary vocational education), intermediate (senior general secondary education), or high (university preparatory education). The questionnaire on PMP use was completed by the parent at baseline and included categorical questions on average listening time a day and volume level. In analyses, listening time a day was categorized into <0.5 h a day, 0.5–1 h a day, and more than 1 h a day, and volume level into low, moderate, and high based on total study population tertiles. Household income, which was used as a marker of SES, was classified as below the national average (<€2800), or equal to or above the national average (≥€2800). At baseline, we measured participants' weight and height to calculate the body mass index (BMI) (weight (kg)/height $(m)^2$).

Participants were asked to complete a computerized interview on risky behavior in private at the research center during follow-up. The research assistant made clear that all data would be anonymized to reduce the risk of social desirability bias. The questionnaire included questions on tobacco use, alcohol consumption, and cannabis use. Smoking on average more than 1 cigarette a month, ever having multiple glasses of alcohol on one occasion, and/or smoking cannabis were considered risky behaviors.

Statistical Analyses

Descriptive statistics were used to evaluate the characteristics of the study population. Between-group differences were analyzed using independent samples *t*-tests and Pearson's Chi-square tests. In a non-response analysis, the baseline characteristics of the included participants were compared with those excluded. Multivariable logistic regression analysis was performed to examine whether the covariates age, sex, household income, educational level, listening time and volume level of PMP use, BMI, tobacco use, alcohol consumption, and cannabis use were associated with a hearing decline in the low and/or high frequencies. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. Interactions between the covariates up to the third-order were examined and included in the multiple logistic regression model when significant. The correlation between the covariates was assessed using Pearson's and Point-Biserial correlation, to ensure multicollinearity was not present. As demonstrated in Table S1, there were only weak correlations (all correlation coefficients ≤0.4). Missing data on covariates were handled using multiple imputations by chained equations. Ten imputed datasets were generated. As no major differences in the magnitude or direction of the effect estimates were observed between analyses with imputed missing data and complete cases only, we only present the results of the model based on imputed datasets. Two-tailed tests were used for all analyses with statistical significance accepted at $p < 0.05$. Statistical analyses were performed using SPSS version 24.0 for Windows software (SPSS Inc).

TABLE I.
Characteristics of the Included Study Population ($n = 3508$).

Baseline characteristics	
Age at baseline, mean (±SD)	9 yrs. and 9 mo (4 mo.)
Age at follow-up, mean (±SD)	13 yrs. and 7 mo. (4 mo.)
Sex, *n* (%)	
Male	1720 (49.0)
Female	1788 (51.0)
Educational level participant, *n* (%)	
Lower	704 (20.1)
Intermediate	671 (19.1)
Higher	1610 (45.9)
Missing	523 (14.9)
Household income, *n* (%)	
<€2800	944 (26.9)
≥€2800	1981 (56.5)
Missing	583 (16.6)
Body mass index (kg/m^2), mean (±SD)	17.5 (2.6)
Listening time personal music player use	
<0.5 h a day	723 (20.6)
0.5–1 h a day	326 (9.3)
>1 h a day	97 (2.8)
Unknown	2362 (67.3)
Volume level personal music player use, *n* (%)	
Low	317 (9.0)
Moderate	633 (18.0)
High	47 (1.3)
Missing	2511 (71.6)
Cigarette smoking, *n* (%)	
No	2765 (78.8)
Yes	219 (6.2)
Missing	524 (14.9)
Alcohol consumption, *n* (%)	
No	2338 (66.6)
Yes	641 (18.3)
Missing	529 (15.1)
Drugs, *n* (%)	
No	2932 (83.6)
Yes	47 (1.3)
Missing	529 (15.1)

Note: Missing data on the covariates were imputed using multiple imputations by chained equations.
Abbreviations: mo., month; ***n***, number; yrs., year.

Non-Response Analyses

The non- response analysis showed that participants excluded ($n = 631$) more often attended a lower educational level (25.0% vs. 20.1%, $X^2(2) = 8.0$, $p = 0.019$) as compared to participants who were included ($n = 3508$) (Table S2).

RESULTS

Study Population

The sociodemographics of the study population ($n = 3508$) are presented in Table I. The majority of participants attended a higher educational level (45.9%) and had a higher net monthly household income (56.5%). The mean length of follow-up was 3 years and 10 months (with an SD of 5 months).

Audiological Results

The cohort's mean hearing thresholds of right and left ears at baseline and follow-up are shown in Figure 2. In Table II, the distribution of the degree HL at baseline

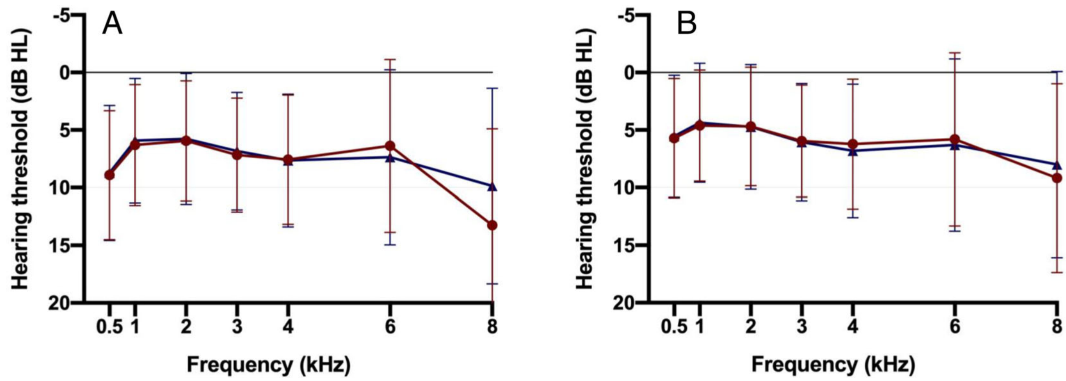

Fig. 2. Mean hearing thresholds of the cohort at baseline (A) and follow-up (B). The error bars represent ±1 standard deviation. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

TABLE II.
Distribution of Degree Low-Frequency and High-Frequency Pure-Tone Averages At Baseline and Follow-Up.

	Low-frequency pure-tone average (dB HL)		High- frequency pure-tone average (dB HL)	
	Baseline	Follow-up	Baseline	Follow-up
Normal hearing (≤15 dB HL), *n* (%)	3389 (96.6)	3453 (98.4)	3227 (92.0)	3332 (95.0)
Slight (16–25 dB HL), *n* (%)	111 (3.2)	45 (1.3)	260 (7.4)	158 (4.5)
Mild (26–40 dB HL), *n* (%)	6 (0.2)	8 (0.2)	17 (0.5)	12 (0.3)
Moderate (41–55 dB HL), *n* (%)	0	0	3 (0.1)	3 (0.1)
Moderately severe (56–70 dB HL), *n* (%)	2 (0.1)	2 (0.1)	1 (0.0)	2 (0.1)
Severe (71–90 dB HL), *n* (%)	0	0	0	1 (0.0)

Note: The ear with the poorest pure-tone average is presented.

TABLE III.
Low-Frequency and High-Frequency Pure-Tone Average At Baseline of Participants With and Without Hearing Decline.

	No hearing decline in the low frequencies *n* = 3235	Hearing decline in the low frequencies *n* = 273
Low- frequency pure-tone average at baseline	6.2 (3.9) dB HL	2.1 (5.3) dB HL
High- frequency pure-tone average at baseline	7.2 (4.5) dB HL	5.6 (4.4) dB HL
	No hearing decline in the high frequencies *n* = 3112	Hearing decline in the high frequencies *n* = 396
Low- frequency pure-tone average at baseline	6.1 (4.0) dB HL	4.3 (5.2) dB HL
High- frequency pure-tone average at baseline	7.6 (4.2) dB HL	4.1 (5.3) dB HL

Note: The ear with the greatest decline is presented.
Abbreviation: dB HL, decibels hearing level.

and follow-up are presented. The ear with the poorest PTA is presented.

A total of 273 (7.8%) participants demonstrated a hearing decline in the low frequencies, with a mean (±SD) decline of 8.4 (2.8) dB. The hearing decline in the low frequencies was unilateral in 87.9% (37.0% right and 50.9% left) and bilateral in 12.1%. In total, 396 (11.3%) participants demonstrated a hearing decline in the high frequencies, with a mean (±SD) decline of 8.4 (3.2) dB. The hearing decline in the high frequencies was unilateral in 85.1% (37.6% right and 47.5% left) and bilateral in 14.9% of the participants. Participants with hearing decline, demonstrated better (lower) hearing thresholds at baseline, as shown in Table III.

Univariate analyses of the baseline data showed that participants with hearing decline more often attended a lower educational level compared to participants whose hearing did not change (23.4% vs. 19.8% X^2 (2)=6.2, $p = 0.045$ for hearing decline in the low frequencies, and 29.0% vs. 26.6% $X^2(2) = 6.9$, $p = 0.037$ for the high frequencies). No differences in age, sex, and household income were observed (Table S3 and S4).

TABLE IV. Multivariable Logistic Regression.			
		Hearing decline in the low frequencies	Hearing decline in the high frequencies
Variable	Categories	OR (95% CI)	OR (95% CI)
Age at baseline (years)		1.0 (0.7; 1.6)	1.2 (0.8; 1.6)
Age during follow-up (years)		1.0 (0.7; 1.4)	1.1 (0.8; 1.5)
Sex	Female	*Reference*	*Reference*
	Male	0.9 (0.7; 1.1)	1.1 (0.9; 1.4)
Household income	<€2800	1.0 (0.7; 1.4)	1.0 (0.8; 1.3)
	≥€2800	*Reference*	*Reference*
Educational level	Low	1.3 (1.0;1.9)	1.4 (1.0;1.8)*
	Middle	1.3 (0.9; 1.9)	1.1 (0.8; 1.4)
	High	*Reference*	*Reference*
Body mass index (kg/m^2)		1.0 (1.0;1.1)	1.0 (0.9; 1.0)
Listening time personal music player use	<0.5 hr a day	*Reference*	*Reference*
	0.5–1 hr a day	1.0 (0.6;1.4)	0.8 (0.6; 1.1)
	>1 hr a day	0.8 (0.5; 1.5)	0.9 (0.6; 1.4)
Volume level personal music player use	Low	*Reference*	*Reference*
	Moderate	1.0 (0.7; 1.2)	1.0 (0.7; 1.5)
	High	1.0 (0.8; 2.3)	1.2 (0.6; 2.2)
Cigarette smoking	No	*Reference*	*Reference*
	Yes	0.9 (0.5; 1.5)	1.2 (0.8; 1.9)
Alcohol consumption	No	*Reference*	*Reference*
	Yes	1.5 (1.1; 2.0)*	0.9 (0.7; 1.3)
Drugs use	No	*Reference*	*Reference*
	Yes	1.2 (0.5; 3.2)	1.5 (0.7; 3.3)

Note: Models were constructed using multivariable logistic regression. Values are odds ratios (OR) and 95% confidence intervals (95%) relative to the adolescents without hearing decline. There were no missing data on these variables as they were imputed using multiple imputation methods. *$p < 0.05$. **$p < 0.01$.

Risk Factors of Hearing Decline

Table IV presents the results of the multivariable logistic regression analysis. Participants who reported alcohol consumption had an increased odds of having hearing decline in the low frequencies as compared to participants who do not report alcohol consumption (OR 1.5, 95% CI 1.1; 2.0). Age, sex, household income, educational level, BMI, PMP use, cigarette smoking, and drug use were not associated with hearing decline in the low frequencies. Participants with a lower educational level, as compared to participants with a higher educational level, were more likely to have a hearing decline in the high frequencies (OR 1.4, 95% CI 1.0; 1.8). Age, sex, household income, BMI, PMP use, and risky behaviors were not associated with a hearing decline in the high frequencies. None of the interaction terms examined were significant. Between baseline and follow-up, an increase in PMP use was observed. However, neither listening time nor volume level at follow-up were significantly associated with a hearing decline in the low or high frequencies.

In additional analyses, we examined potential risk factors of hearing decline of more than 10 dB. In total, 35 participants (1.0%) had a decline in hearing of more than 10 dB in the low frequencies, and 56 (1.6%) in the high frequencies. As shown in Table S5, a lower educational level (OR 3.9, 95% CI 1.5; 9.9) and cigarette smoking (OR 4.3, 95% CI 1.4; 12.8) were associated with a higher odds of having hearing decline of more than 10 dB in the low frequencies. No risk factors for the hearing decline of more than 10 dB in the high frequencies were identified. None of the interaction terms tested were significant.

DISCUSSION

To our knowledge, this is the first population-based prospective cohort study to examine risk factors of hearing decline from childhood to early adolescence. Between ages 9 and 13 years, 7.8% of the participants had a hearing decline in the low frequencies and 11.2% in the high frequencies. Among the participants with hearing decline, the majority still had hearing thresholds within normal limits. We found that hearing decline was affected by participants' educational level and risky behaviors.

Prevalence estimates of childhood HL vary widely in literature, which to some extent may be due to methodological differences between studies. The prevalence of SNHL in the current study is lower than that reported for US children aged 12–19 years.[20] Although the same definition of SNHL was used in the study of Lalwani et al., participants were older, which may explain the higher prevalence rate found. As mentioned previously, the hearing decline in the present study was more often observed in the high frequencies and was mostly unilateral. Interestingly, participants with hearing decline demonstrated better (lower) hearing thresholds at baseline as compared to participants without hearing decline.

Adolescents with a lower educational level were more likely to have a hearing decline in the high frequencies as compared with their peers with a higher educational level. An inverse association between educational level and hearing acuity has previously been found in adults.[21,22] It could be related to the general SES, which is partly determined by the educational level. Research has shown that individuals with a lower SES are more likely to engage in unhealthy behaviors such as smoking, which in turn may contribute to the risk of HL.[23–25] In addition, Widen and Erlandsson found that adolescents with a higher SES are more concerned about the risk of HL due to excessive noise exposure and use ear protection to a greater extent than those with a lower SES.[26]

Many risky behaviors associated with adverse health outcomes have their origin in adolescence.[27] In the present study, adolescents who reported alcohol consumption were found to be more likely to have a hearing decline in the low frequencies. Previous cross-sectional studies among adults have reported an inverse association between moderate alcohol consumption and the prevalence of HL.[28–30] It has been suggested that alcohol may decrease the prevalence of HL through its effect on the vasculature supply of the cochlea.[31] However, these changes can only be expected after long-term moderate alcohol consumption, and not after incidental alcohol consumption as in the present study. There is increasing evidence that risky behaviors are interrelated[32,33]; When adolescents take one risk, they tend to take multiple. For example, adolescents who engage in unsafe music listening habits, report more substance use.[34,35] Thereby, there are multiple pathways through which risky behavior can affect hearing acuity. More research is needed to further substantiate our knowledge of the relationship between risky behavior and HL.

There was no evidence of a relationship between the listening time and volume level of PMP use and hearing decline in the current study. The effect of excessive PMP use on hearing was not examined due to the low number of participants listening for an extended time and at high volume levels. Over the past years, many studies have investigated the risk of hearing damage related to PMP use. Whereas some studies found that hearing thresholds are significantly elevated in young people using PMPs,[36–41] others did not observe an association between PMP use and hearing acuity.[42–45] It is known that noise-induced HL develops slowly over the years of exposure. Thereby, it could be that the effect of PMP use is not evident yet in this young population with a relatively short time of PMP use. The analyses in the present study were limited by a large amount of missing data on PMP use. Future research is needed to determine the long-term impact of PMP use and other sources of recreational noise exposure on young people's hearing.

Emerging evidence suggests that obesity increases the risk of HL in adolescence.[11,12,46] Mechanisms of inflammation, oxidative stress, and reduced vascular supply to the cochlea have been proposed.[12,46,47] In the present study, body mass index was not associated with hearing decline, which could be explained by our relatively healthy and young population.

In this study, the hearing decline was defined as an increase in PTA of more than 5 dB between baseline and follow-up. In a meta-analysis, Mahomed et al. evaluated the test-retest reliability of manual audiometry, reporting differences ranging from −0.4 to 2.3 dB HL for individual frequencies.[48] When hearing decline was defined as an increase in PTA of more than 10 dB, lower educational level and cigarette smoking were associated with a higher odds of having a hearing decline in the low frequencies. However, as the number of participants with hearing decline of more than 10 dB was relatively low, these results should be interpreted with caution.

Strengths and Limitations

The strengths of this study are the large population-based sample with detailed hearing assessments over time using a prospective study design. Testing was performed by dedicated research assistants with a small variance. A limitation is that otoscopic examination and bone-conduction audiometry were not performed due to time constraints. Since our main interest was SNHL, we decided to exclude participants with abnormal tympanometry in case of elevated thresholds. Second, information on sociodemographic covariates and PMP use was obtained via parental questionnaires. Recall bias cannot be excluded and the parent proxy to measure PMP use might be unreliable. Third, whereas all covariates were assessed at baseline, data on the educational level and risky behaviors were only available during follow-up. A lower educational level and highly risky behavior were assumed to be precursors to hearing decline. However, as several studies reported an association of HL with behavioral problems and academic underachievement, reverse causality cannot be ruled out.[49,50] Fourth, there is potential for residual confounding. There are many more determinants of HL that were not examined in this study as the data were not available such as genetic susceptibility, other sources of recreational and environmental noise exposure, the use of hearing protection, otologic diseases, and the use of ototoxic drugs. Last, the cohort selection towards a relatively Dutch and highly educated population over time may affect the generalizability of our findings.[17]

CONCLUSIONS

In this 4- year follow-up study, 7.8% of the children were estimated to have a hearing decline of more than 5 dB in the low frequencies, and 11.2% in the high frequencies. Interventions to prevent HL should be offered in particular to children with a lower educational level and higher risky behavior as these children have an increased odds of having a hearing decline. The hearing decline was not affected by age, sex, household income, PMP use, and BMI in early adolescence. Future research is required to examine the long-term impact of these factors on hearing acuity.

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