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Modifiable determinants of hearing impairment in adults

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ABSTRACT

Objective. To identify factors contributing to the declining prevalence of hearing impairment in more recent generations.

Methods. We used data on hearing thresholds and potential risk factors of hearing impairment collected from studies in Beaver Dam, Wisconsin, the Epidemiology of Hearing Loss Study (1993–1995, n = 3753; 1998–2000, n = 2800 and 2003–2005, n = 2395), the concurrent Beaver Dam Eye Study on the same cohort, and a subgroup (n = 2173) of the Beaver Dam Offspring Study (2005–2008).

Results. Educational attainment significantly reduced the odds ratio (OR) of the birth cohort effect on hearing impairment from 0.90 to 0.93, while a history of ear infection had a reverse effect on the decreasing trend (significantly changing the OR from 0.93 to 0.94). Occupational noise exposure, smoking, and a history of cardiovascular disease, while associated with hearing impairment, did not attenuate the cohort effect. The cohort effect remained significant after known risk factors were adjusted (OR = 0.93; 95% confidence interval, 0.90, 0.97)

Conclusion. These data provide strong evidence that environmental, lifestyle, or other modifiable factors contribute to the etiology of hearing impairment and add support to the idea that hearing impairment in adults may be prevented or delayed.

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Introduction

Rapid change in disease prevalence over time is a vital indicator that a disorder has environmental or other modifiable risk factors. Understanding these changes may provide important insights into ways to improve population health. Previously, Zhan et al. analyzed the trend of hearing impairment in older adults and found that people born in more recent years were less likely to have hearing impairment at a given age than those born in earlier years (Zhan et al., 2010). Over a typical generational span of 20 years, the prevalence of hearing impairment declined by 42% and 23% for men and women, respectively. This birth cohort effect suggested that environmental and modifiable factors may be associated with the development of hearing impairment because human genetic changes are extremely slow and unlikely to happen in less than 20 years.

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During the 20th century there were many positive changes in the environment (e.g., cleaner air and water, institution of noise-reduction efforts in the work place) and behavioral factors (e.g., higher education, recent decreased rates of smoking), improvements in health care (e.g., immunizations and antibiotics), and declines in other disorders (e.g., cardiovascular disease (CVD), hypertension, and hypercholesterolemia), that may have beneficial effects on overall health and reduced morbidity, but obesity and sedentary lifestyles have become more common (Flegal et al., 2002; Garte, 2007; Hill and Needham, 2006; Middendorf, 2004).

Noise exposure, smoking, drinking, diabetes, CVD and its risk factors and socioeconomic factors (Agrawal et al., 2008; Cruickshanks et al., 2010; Gates et al., 2000; Helzner et al., 2005) have been associated with hearing impairment. However, which of these factors may be related to improvements in hearing health is unknown. Therefore, the purpose of this paper was to evaluate the associations of modifiable factors reported to be associated with hearing impairment to determine if these factors contributed to the observed decreasing temporal trend in a population-based cohort and a subgroup of their adult offspring.

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Methods

Subjects

Methods used in the Epidemiology of Hearing Loss Study (EHLS) and the Beaver Dam Offspring Study (BOSS) have been reported in detail elsewhere (Cruickshanks et al., 1998; Zhan et al., 2010). The EHLS cohort consisted of adults who participated in the population-based Beaver Dam Eye Study (BDES) and were alive as of March 1, 1993. This cohort was examined in 1993-1995 (n = 3753), 1998-2000 (n = 2800) and 2003-2005 (n = 2395). Adult offspring of participants in the EHLS participated in the BOSS (n=3285). This analysis excluded 1112 BOSS participants younger than 45 years because all EHLS participants were older than 45 years. A signed informed consent was obtained from all study participants. Characteristics of participants and nonparticipants have been described in the previous reports (Nondahl et al., 2006; Zhan et al., 2010). In general, eligible people who did not participate in the EHLS were older, more likely to be male, and had a lower socioeconomic status as indicated by education level than participants; eligible people who did not participate in the BOSS were slightly younger and more likely to be male than participants.

Data collection

Data were collected through the use of examinations, questionnaire interviews and laboratory tests for blood samples. The same standardized hearing examination procedures (including an otoscopic evaluation, screening tympanogram, and pure-tone air- and bone-conduction audiometry) except minor modifications were applied to the three EHLS cycles and the BOSS. Pure-tone air-conduction thresholds were measured for each ear at 0.5, 1, 2, 3, 4, 6 and 8 kHz (American Speech-Language-Hearing Association (AHSA), 1987) in a sound-treated booth using clinical audiometers. Bone-conduction thresholds were measured at 0.5 and 4 kHz at the baseline EHLS, while at each follow-up and BOSS examination, bone-conduction thresholds were measured at 0.5, 2 and 4 kHz. Masking procedures were used when necessary. At each examination, hearing impairment was defined as a pure-tone average of air-conduction thresholds at 0.5, 1, 2 and 4 kHz greater than 25 dB HL (hearing level) in either ear.

The questionnaire was administrated as an interview, focusing on ear and hearing-related medical history, noise exposure (occupational noise exposure, military service and leisure-time noise exposure), hearing perception, socioeconomic status including educational attainment, lifestyle factors, general medical history and medication use. Some variables for the EHLS participants were provided by the concurrent BDES on the same cohort (Klein et al., 2006). Birth cohort was defined as the year of birth. Educational attainment was divided into four categories: less than high school, high school, some college, college graduate and above. History of ear infection was based on the question, "Has a doctor ever told you (or your parents) that you had an ear infection?" Smoking status was classified as non-smoker, past smoker and current smoker, and history of CVD was defined as reporting a history of angina, heart attack, or stroke.

Analysis techniques

All statistical analyses were conducted by using SAS software, version 9 (SAS Institute Inc., Cary, North Carolina). The alternating logistic regression (ALR) proposed by Carey et al. (1993) was first applied to determine factors associated with hearing impairment, accounting for correlations from the repeated measurements and familial aggregations. ALR was then used to examine how each significant modifiable factor changed the birth cohort effect on hearing impairment by comparing the odds ratios (ORs) and regression coefficients for the birth cohort effect in the fully-adjusted model to those in the model with one factor removed.

Statistical significance (p<0.05) of the change of the birth cohort effect was tested using the bootstrap approach (Miller, 2004). To account for the correlation within families and within subjects, we resampled families as a whole. A total of 1000 bootstrap data sets were generated and 95% confidence interval (CI) for the change of the regression coefficient was calculated. A 95% CI excluding zero indicated a statistically significant change of the birth cohort effect.

As a sensitivity analysis to reduce heterogeneity in hearing impairment, 751 hearing impairment cases were excluded if they had self-reported onset of hearing impairment at <30 years old, a history of ear surgery, a conductive

hearing impairment without any evidence of decreased hearing sensitivity if the conductive hearing impairment was resolved, or the difference in the PTA between two ears was >20 dB.

Results

Participants included in these analyses were ages 45–100 years, with birth years between 1902–1946 (EHLS) and 1922–1962 (BOSS). Shown in Table 1 are the characteristics of baseline EHLS participants and a subgroup of the BOSS participants. The overall prevalence of hearing impairment at each baseline was 45.9% (EHLS) and 18.9% (BOSS), respectively. Only 30.2% EHLS participants had received education higher than high school, while 63.6% of the BOSS subjects attended college. Occupational noise exposure was common (51% and 43.5% of EHLS and BOSS participants, respectively).

Table 2 shows age-adjusted associations between potential risk factors and hearing impairment. Results from sex-specific (stratifying on sex) models were similar to those from sex-adjusted models for most factors. Higher education was significantly associated with lower odds of hearing impairment. History of heavy drinking was significantly associated with higher odds of hearing impairment. Occupational noise exposure was significant only in men while ear infection and CVD were significant only in women. Leisure time noise exposure, serum HDL cholesterol, serum total cholesterol, diabetes, hypertension and body mass index were not associated with hearing impairment.

In a stepwise analysis (model 1, Table 3), age, sex, occupational noise exposure (men only), smoking, CVD (women only) and a history of ear infection (women only) remained significantly associated with the higher prevalence of hearing impairment, and higher education was associated with lower odds of hearing impairment.

The previous study (Zhan et al., 2010) conducted in the same population observed a significant birth cohort effect (adjusting for age

Table 1Distributions of factors and hearing impairment in the Epidemiology of Hearing Loss Study (EHLS, 1993–1995) and a subgroup of the Beaver Dam Offspring Study (BOSS, 2005–2008), Beaver Dam, Wisconsin.

| | EHLS (1993-1995) | | BOSS (2005-2008) | |
|--|------------------|-------------------------|------------------|-------------------------|
| | n | % | n | % |
| Hearing impairment | 1631 | 45.9 | 332 | 18.9 |
| Education | | | | |
| Less than high school | 847 | 23.8 | 54 | 3.1 |
| High school | 1632 | 45.9 | 583 | 33.4 |
| Some college | 549 | 15.4 | 584 | 33.4 |
| College or greater | 527 | 14.8 | 527 | 30.2 |
| Smoking | | | | |
| Never | 1600 | 45.9 | 902 | 51.3 |
| Past | 1374 | 39.4 | 572 | 32.6 |
| Current | 513 | 14.7 | 283 | 16.1 |
| History of heavy drinking | 584 | 16.8 | 334 | 19.0 |
| Diabetes | 343 | 10.0 | 162 | 9.2 |
| Cardiovascular disease | 504 | 14.5 | 74 | 4.2 |
| Hypertension | 1760 | 50.8 | 804 | 45.8 |
| History of ear infection | 1104 | 32.1 | 923 | 54.2 |
| Occupational noise exposure | 1769 | 50.8 | 764 | 43.5 |
| Leisure time noise exposure | 2356 | 66.3 | 1419 | 80.8 |
| | n | Mean (SD ^a) | n | Mean (SD ^a) |
| Age (years) | 3556 | 65.1 (10.5) | 1758 | 54.4 (7.2) |
| Birth year ^b | 3556 | 1928 (10) | 1758 | 1951 (7) |
| Serum total cholesterol (mg/dl) | 3430 | 238.7 (45.3) | 1734 | 206.7 (39.5 |
| Serum high density lipoprotein cholesterol (mg/dl) | 3423 | 52.4 (16.7) | 1733 | 50.2 (15.2) |
| Body mass index (kg/m ²) | 3396 | 29.6 (5.5) | 1744 | 30.9 (6.5) |

^a Standard deviation.

^b The birth year for EHLS and the subgroup of BOSS participants ranged from 1902–1946 and 1922–1962, respectively.

Table 2Odds ratio (95% confidence interval) between potential modifiable risk factors and hearing impairment controlling for sex in the pooled sample (n = 10,159 observations) from the EHLS (1993–1995, 1998–2000, 2003–2005) and BOSS (2005–2008), Beaver Dam, Wisconsin.

| Risk factor | Age-adjusted, sex-specific association | | Age- and sex-adjusted association ^a | |
|--|--|------------------|--|--|
| | Men | Women | | |
| Education (per level) | 0.67 (0.62-0.74) | 0.82 (0.75-0.90) | Men: 0.68 (0.62–0.74); Women: 0.82 (0.75–0.90) | |
| Occupational noise exposure | 1.68 (1.37-2.05) | 1.12 (0.95-1.33) | Men: 1.67 (1.38-2.04); Women: 1.12 (0.94-1.32) | |
| Leisure time noise exposure | 1.21 (0.98-1.49) | 0.96 (0.76-1.22) | 1.08 (0.93-1.26) | |
| History of ear infection | 0.95 (0.79-1.13) | 1.38 (1.16-1.64) | Men: 0.95 (0.79-1.13); Women: 1.39 (1.17-1.65) | |
| Smoking status | | | | |
| Non-smoker | 1.00 | 1.00 | 1.00 | |
| Past smoker | 1.31 (1.09-1.58) | 1.11 (0.93-1.32) | 1.19 (1.05-1.36) | |
| Current smoker | 1.70 (1.34-2.17) | 1.24 (0.96-1.58) | 1.44 (1.21–1.71) | |
| History of heavy drinking | 1.18 (1.00-1.39) | 1.31 (1.00-1.70) | 1.21 (1.05–1.39) | |
| Diabetes | 1.03 (0.84-1.26) | 1.24 (1.00-1.54) | 1.11 (0.96–1.29) | |
| History of cardiovascular disease | 1.14 (0.94-1.38) | 1.60 (1.28-2.00) | Men: 1.16 (0.96-1.39); Women: 1.57 (1.25-1.96) | |
| Hypertension | 0.95 (0.84-1.08) | 0.98 (0.86-1.12) | 0.97 (0.88-1.06) | |
| Serum total cholesterol (per 10 mg/dl increase) | 1.02 (1.00-1.03) | 1.00 (0.99-1.02) | 1.01 (1.00-1.02) | |
| Serum high density lipoprotein cholesterol (per 10 mg/dl increase) | 1.00 (0.94-1.06) | 0.97 (0.93-1.02) | 0.99 (0.95-1.02) | |
| Body mass index (per 1 kg/m ²) | 1.01 (1.00-1.03) | 1.00 (0.99-1.02) | 1.01 (1.00–1.02) | |

^a The interactions between sex and education, sex and occupational noise exposure, sex and history of ear infection, and sex and history of cardiovascular disease were statistically significant.

and sex) on hearing impairment. Model 2 in Table 3 demonstrated that this birth cohort effect (in the unit of 5-year increase) remained (OR: 0.93, 95% CI: 0.89, 0.97) after adjusting for significant risk factors.

The effect of a single risk factor on the birth cohort effect was then examined by comparing regression coefficients and odds ratios for the birth cohort effect in the fully-adjusted model to those in the model with one factor removed (Table 4). Educational attainment significantly attenuated the odds ratio of the birth cohort effect from 0.90 to 0.93. On the other hand, a history of ear infection significantly strengthened the odds ratio from 0.94 to 0.93. Inclusion of other modifiable risk factors in the model did not significantly change the birth cohort effect on hearing impairment.

Table 3 Associations between significant modifiable risk factors and hearing impairment in the multivariate model with/without birth cohort included in the pooled sample (n = 9566 observations) from the EHLS (1993–1995, 1998–2000, 2003–2005) and BOSS (2005–2008), Beaver Dam, Wisconsin.

| OR (95% CI) in model 1 ^a | OR (95% CI) in model 2 ^b |
|-------------------------------------|---|
| Not included | 0.93 (0.89-0.97) |
| | |
| 0.70 (0.64-0.76) | 0.71 (0.65-0.78) |
| 0.83 (0.75-0.92) | 0.85 (0.77-0.95) |
| | |
| 1.43 (1.16-1.77) | 1.42 (1.15-1.77) |
| 1.03 (0.86-1.23) | 1.05 (0.87-1.25) |
| | |
| 1.04 (0.87-1.24) | 1.07 (0.89-1.29) |
| 1.43 (1.20-1.71) | 1.49 (1.24-1.79) |
| | |
| 1.00 | 1.00 |
| 1.15 (1.01-1.32) | 1.16 (1.01-1.33) |
| 1.34 (1.13-1.60) | 1.34 (1.12-1.60) |
| | |
| 1.06 (0.87-1.28) | 1.05 (0.86-1.28) |
| 1.52 (1.21-1.91) | 1.51 (1.20-1.90) |
| | Not included 0.70 (0.64–0.76) 0.83 (0.75–0.92) 1.43 (1.16–1.77) 1.03 (0.86–1.23) 1.04 (0.87–1.24) 1.43 (1.20–1.71) 1.00 1.15 (1.01–1.32) 1.34 (1.13–1.60) 1.06 (0.87–1.28) |

Abbreviation: OR, odds ratio; CI, confidence interval.

The sensitivity analysis conducted in a subset of participants with a reduced heterogeneity in hearing impairment showed that each risk factor, except history of ear infection, had a similar effect on the birth cohort effect as that in Table 4 In this subset, the birth cohort effect remained significant (p<0.0001) after controlling for covariates, and a history of ear infection did not significantly change the birth cohort effect.

Discussion

In this study, educational attainment attenuated the temporal trend of declining prevalence of hearing impairment, suggesting that increasing educational attainment over time contributed to this declining trend in more recent generations. The link between educational attainment or other indicators of socioeconomic status and hearing impairment has been observed in other populations (Davis, 1989; Helvik et al., 2009) and the influence of education on hearing impairment is likely to be complex and multi-factorial. Higher socioeconomic status has also been associated with lower risk of atherosclerosis and CVD, asthma and other chronic conditions, yet the

Table 4Comparisons of odds ratios and regression coefficients for birth cohort (5-year interval) in the model with and without a significant risk factor in the pooled sample (n = 9566 observations) from the EHLS (1993–1995, 1998–2000, 2003–2005) and BOSS (2005–2008). Beaver Dam. Wisconsin.

| | Odds ratio | Regression coefficient (p-value) | Percent change of regression coefficient (95% confidence interval of the change ^a) |
|---|---------------|--|--|
| Full model ^b Impact of removing one factor from the full model | 0.93 | -0.075 (0.001) | |
| Education (per level) | 0.90 | -0.109 (<0.0001) | 45.1% (24.3% to 105.3%) |
| Occupational noise exposure | 0.93 | -0.076 (0.001) | 0.9% (-4.1% to 6.6%) |
| Smoking | 0.93 | -0.075(0.001) | 0.7% (-5.2% to 5.8%) |
| History of ear infection | 0.94 | -0.063 (0.004) | -16.4% (-36.2% to -3.8%) |
| Cardiovascular disease | 0.93 | -0.075 (0.001) | 0.5% (– 7.7% to 3.5%) |

^a 95% confidence interval was obtained according to the bootstrap approach.

^a Model 1 included age, age², age³, sex, education, smoking, occupational noise exposure, history of ear infection, cardiovascular disease (CVD), interaction between sex and education, interaction between sex and occupational noise exposure, interaction between sex and history of ear infection, and interaction between sex and CVD. Regression coefficients (p values) for within subcluster (Alpha1) and between-subcluster (Alpha2) association were 4.77 (p<0.0001) and 0.14 (p=0.18), respectively.

^b Model 2 included all terms in model 1, plus birth cohort, which was centered at year 1937 in the unit of 5-year increase. Regression coefficients (p values) for within subcluster (Alpha1) and between-subcluster (Alpha2) association were 4.77 (p<0.0001) and 0.15 (p=0.18), respectively.

^b The full model include cohort, age, age², age³, sex, education, smoking, occupational noise exposure, history of ear infection, cardiovascular disease (CVD), interaction between sex and education, interaction between sex and occupational noise exposure, interaction between sex and history of ear infection, and interaction between sex and CVD. Birth cohort was centered at year 1937 in the unit of 5-year increase.

exact mechanisms for these protective effects remain uncertain (Albert et al., 2006; Shankardass et al., 2007). Explanations for the underlying mechanisms commonly emphasize lifestyle, behavioral and psychological factors and material conditions (Madsen et al., 2010). Individuals with higher education are more likely to have a healthy lifestyle (Hill and Needham, 2006), such as less smoking and noise exposure, which may lead to a lower risk of hearing impairment. However, in the present study, these two factors were controlled for, suggesting that other mechanisms may be involved. People with more education may have better nutrition, access to health care and less workplace stress or may be more resilient to the biological effects of stress and infection (Albert et al., 2006; Cruickshanks et al., 2010). While educational attainment in older adults is difficult to modify, factors related to educational attainment may be modifiable. Thus more research is needed to identify the factors associated with education which may contribute to protective health effects.

Although occupational noise exposure, smoking and CVD were significantly, independently associated with hearing impairment, they did not change the birth cohort effect. There are several possible explanations. First, the prevalence of these factors did not change across the birth cohort, or the change may show a symmetrical curved pattern. For example, some cohorts may have an increasing prevalence of risk factors, while others may have a decreasing prevalence, with the overall impact being no change to the birth cohort effect on hearing impairment. The curve relationship between birth cohort and smoking has been observed in a study conducted among 125,707 American radiologic technologists (Freedman et al., 2002). Second, an imprecise measurement of the factors may underestimate the association between the factor and hearing impairment. For example, the use of hearing protection devices was not taken into account when defining the noise exposure. However, the use of hearing protection devices in the studied population was low (Nondahl et al., 2006) and thus is unlikely to have impacted these results. Third, since smoking and CVD are related to the mortality (Critchley and Capewell, 2003), participants may be less likely to smoke or have CVD than nonparticipants. Consequently, the association between smoking/CVD and hearing impairment may be diluted.

A history of ear infection reduced the magnitude of the decreasing trend of hearing impairment, suggesting that the prevalence of ear infection was increasing. The introduction of antibiotics may have changed the types of ear disorders, with an increase in the prevalence of serous otitis media (Ruben, 2009). It has been reported that ear infections might affect hearing more before the introduction of antibiotics than during the following years (Tambs et al., 2004). An increase in less harmful infections coupled with a decrease in harmful infections may contribute to the finding. A history of ear infection did not change the birth cohort effect when excluding those observations in which hearing impairment was not consistent with presbycusis, suggesting that the effect of ear infections on the observed birth cohort effect was more likely to be due to conductive problems. It should be noted that the increasing trend of ear infections may be artificial. For example, because of the availability of effective treatment, people whose ear infections occurred after the introduction of antibiotics might be more likely to visit a doctor and recall a history of infection, particularly in case of a mild or moderate infection, than those with ear infections before the introduction of antibiotics.

Strengths and limitations

This study has numerous strengths, including but not limited to, the large population-based study design representing multiple generations of people aged 45 years and older, use of the standardized protocol for audiometric testing and covariate collection across the EHLS and the BOSS, and use of the ALR to adjust for correlations

among repeated measurements within individuals that may be further nested within families.

Nevertheless, there are some limitations to this study. First, most of the potential modifiable risk factors were self-reported which may be subject to measurement errors. Second, risk factors may have differentially affected the relationship between participation and prevalence of hearing impairment in different birth cohorts. The EHLS participants were younger and healthier than non-participants, while the BOSS participants were slightly older than non-participants. We do not know whether the BOSS participants were less healthy than non-participants. Third, the definition of hearing impairment, while often used in clinical and epidemiological studies, does not represent hearing impairment from a single etiological pathway. We chose to replicate our findings in a subset analysis excluding those with early onset, a history of ear surgery, a conductive hearing impairment without any evidence of decreased hearing sensitivity if the conductive hearing impairment was resolved, and asymmetric hearing impairments as an attempt to address the problem of heterogeneity. The results were consistent with the overall model suggesting that these findings reflect pathways important in sensorineural hearing impairment.

Conclusion

Educational attainment attenuated the observed birth cohort effect, while a history of ear infection partly countered the decreasing birth cohort effect on hearing impairment. Occupational noise exposure, smoking and a history of CVD were significantly associated with hearing impairment, but these factors did not change the birth cohort effect. The birth cohort effect remained significant after adjusting for known risk factors associated with hearing impairment, suggesting that other unknown factors may contribute to the decreasing trend. Our data provide strong evidence that environmental, lifestyle, or other modifiable factors contribute to the etiology of hearing impairment and add support to the idea that hearing impairment in adults may be prevented or delayed.

Conflict of interest statement

The authors declare that there are no conflicts of interest.

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