

## The Association between Impaired Fasting Glucose and Noise-induced Hearing Loss

Tae-Won JANG<sup>1</sup>, Beom-Gyu KIM<sup>2</sup>, Young-Jun KWON<sup>1</sup> and Hyoung-June IM<sup>3</sup>

<sup>1</sup>Department of Occupational and Environmental Medicine, <sup>2</sup>Department of Otolaryngology-Head and Neck Surgery, Hangeang Sacred Heart Hospital, Hallym University College of Medicine and <sup>3</sup>Department of Occupational and Environmental Medicine, Hallym University Sacred Heart Hospital, Hallym University College of Medicine, Korea

**Abstract: The Association between Impaired Fasting Glucose and Noise-induced Hearing Loss: Tae-Won JANG, et al. Department of Occupational and Environmental Medicine, Hangeang Sacred Heart Hospital, Hallym University College of Medicine, Korea—Objectives:** This study was performed to determine whether there is an association between impaired fasting glucose and noise-induced hearing loss. **Methods:** The study subjects were workers in one automobile manufacturing company. The data were obtained from results of health examinations during 2005 and 2009. The factors analyzed were age, smoking and alcohol history, work duration, environmental noise level, hearing thresholds, blood pressure, serum creatinine, initial hearing threshold and fasting glucose. **Results:** The hearing thresholds at 4,000 Hz frequencies for both ears were significantly higher in 2009 than those in 2005. The changes in the hearing thresholds of the subjects with an impaired fasting glucose (100–125 mg/dl) and diabetes ( $\geq 126$  mg/dl) were greater than those of the normal ( $< 100$  mg/dl) group. After adjusting for variables such as age, smoking and alcohol history, environmental noise, hypertension and serum creatinine, fasting glucose was found to be a significant variable. Impaired fasting glucose (100–125 mg/dl) was significant ( $\beta=1.339$ ,  $p=0.002$ ) for the right ear, whereas it was not significant ( $\beta=0.639$ ,  $p=0.121$ ) for the left ear. **Conclusions:** Impaired fasting glucose, as well as diabetes, might be risk factors for hearing loss in individuals with exposure to certain noise levels. The results of this study suggest that impaired fasting glucose should be considered a risk factor for hearing loss.

(J Occup Health 2011; 53: 274–279)

**Key words:** Diabetes, Hearing loss, Impaired fasting glucose, Noise

Noise-induced hearing loss is defined as sensorineural hearing loss due to environmental noise exposure. It is usually bilateral and develops slowly over a period of several years<sup>1</sup>. Early noise-induced hearing loss can be detected by pure tone audiometry. Typically, the first sign of hearing loss due to noise exposure is a dip or notch in the audiogram at 4,000 Hz<sup>2</sup>. The notch broadens with increasing noise exposure and may be difficult to distinguish from hearing loss due to aging (presbycusis), which shows a gradual deterioration at high frequencies.

Several factors are related to noise-induced hearing loss. Nonmodifiable risk factors include age, genetics, gender and race. Of these factors, age is the most significant. Modifiable risk factors include the use of hearing protection, cigarette smoking, lack of exercise, low dietary intake of foods containing vitamins and minerals, the presence of diabetes or heart disease and poor oral health<sup>3</sup>.

Many studies have reported an association between diabetes and hearing loss; however, most prior studies have some limitations such as data from animal studies, small numbers of subjects or a cross-sectional study design. To date, there is no evidence associating impaired fasting glucose and hearing loss. Therefore, the goal of this retrospective study was to determine whether there is an association between impaired fasting glucose and hearing loss in a large group of subjects.

### Subjects

The study subjects were male workers in one automobile manufacturing company. The number of workers that had health examinations including screening tests for noise-induced hearing loss was 2,612 in 2005; these workers were considered a historical cohort. The workers underwent health examinations including follow-up

Received Dec 27, 2010; Accepted May 13, 2011

Published online in J-STAGE Jun 13, 2011

Correspondence to: Y.-J. Kwon, Hangeang Sacred Heart Hospital, 94-200, Yeongdeungpo-Dong, Yeongdeungpo-Gu, Seoul 150-719, Korea (e-mail: kwon5966@hallym.or.kr)

screening for noise-induced hearing loss in 2009; the number of workers was 2,208. Health examinations were performed from 8:00 am to 12:30 pm; the workers were instructed to fast prior to the evaluation. If the worker did not fast and had a high glucose level, his glucose level was measured again, while fasting. Subjects lost to follow-up and workers with a past medical history of ear diseases such as otitis media were excluded from the analysis. There were 1,706 subjects included in the final study.

## Methods

### Data collection

The data from the health examinations performed in 2005 and 2009 was collected. Age, smoking and alcohol drinking history, systolic and diastolic blood pressure, serum creatinine and fasting glucose were obtained from the data collected in 2009. The hearing threshold at the frequency of 4,000 Hz was obtained from the screening tests performed in 2005 and 2009. A GSI 61 Audiometer and GSI TymStar (Grason-Stadler) were used to measure the subjects' hearing thresholds and identify middle ear diseases. Background noise level was maintained to below 20 dB, and hearing tests in both years were done by the same tester.

The subjects were classified into three groups according to their fasting glucose using the criteria of the American Diabetes Association<sup>4)</sup>. Subjects with a fasting glucose below 100 mg/dl were defined as the normal group; subjects with a fasting glucose between 100 and 125 mg/dl were defined as the impaired fasting glucose group. The subjects with a fasting glucose over 125 mg/dl and those that were taking medication for diabetes were defined as the diabetes group. Blood pressure was divided into two categories, normal and hypertensive (systolic blood pressure  $\geq 140$  mmHg or diastolic blood pressure  $\geq 90$  mmHg or medications for hypertension). The serum creatinine level was divided into two categories using a cutoff of 1.2 mg/dl. Age was divided into three categories, <40, 40–49 and  $\geq 50$ . Smoking history was divided into three categories: never a smoker, ex-smoker and current smoker. Alcohol history was divided into three categories: social (drinking alcohol less than once a week), moderate (drinking alcohol one or two times a week and more than one bottle of Soju, which is the name of a Korean alcohol, in a week) and heavy (drinking alcohol more than two times a week and two bottles of Soju). The hearing threshold was treated as a continuous variable, and age was treated as a continuous variable in the analysis of covariance.

### Evaluation of noise levels

The environmental noise levels were evaluated with a noise dosimeter. Two to 10 subjects were selected at each work station, and the noise dosimeters were attached to the ear for six hours. The sound equivalent levels were

measured using noise dosimeters with frequency weighting A, a 90-dB criterion, a 80-dB threshold and 5-dB exchange rate. The highest sound level among the measured sound levels for each station was selected and defined as the environmental noise level. The number of selected subjects varied according to the scale of each station and workplace. Environmental noise was divided into three categories: <80 dB, 80–84.9 dB and  $\geq 85$  dB. In the analysis of covariance, environmental noise was treated as a continuous variable.

### Statistical analysis

Chi-square analysis was performed to compare the variables among the normal, impaired fasting glucose and diabetes groups. Kruskal-Wallis tests were performed to compare hearing thresholds among the normal, impaired fasting glucose and diabetes groups. The Wilcoxon signed-rank test was used to determine whether the hearing thresholds had significantly changed. Analysis of covariance (ANCOVA) was performed to identify the factors related to the hearing threshold changes and adjust the factors other than the fasting glucose. The dependent variable was the hearing threshold change, and the independent variables were age, smoking and alcohol history, environmental noise, hypertension, serum creatinine, initial hearing threshold and fasting glucose (model 1). Hearing threshold changes, age and environmental noise were continuous variables, whereas smoking and alcohol history, hypertension, serum creatinine and fasting glucose were categorical variables. In order to evaluate whether or not the interaction between fasting glucose and environmental noise was associated with hearing loss, the interaction term (fasting glucose  $\times$  environmental noise) was entered in ANCOVA model 2. SAS for Windows version 9.10 was used for the analysis. *p* values lower than 0.05 were considered to indicate a statistical significance.

## Results

### General and work-related characteristics of subjects

Table 1 shows the general and work-related characteristics of the subjects. The number of subjects in the: normal, impaired fasting glucose and diabetes group were 1,106 (64.8%), 501 (29.4%) and 99 (5.8%), respectively. Age, smoking and alcohol history, environmental noise and serum creatinine did not differ among the three groups. Hypertension was significantly different among the three groups; the numbers of hypertensive subjects in the normal, impaired fasting and diabetes groups were 179 (16.2%), 132 (26.3%) and 42 (42.4), respectively ( $p < 0.05$ ).

### Hearing thresholds among normal, impaired fasting glucose and diabetes subjects

Kruskal-Wallis tests and Wilcoxon signed rank tests

**Table 1.** General and work-related characteristics of the subjects in 2009 (N=1,706)

Characteristics		n (%)		
		Normal <sup>a</sup> (n=1,106)	Impaired <sup>b</sup> (n=501)	Diabetes <sup>c</sup> (n=99)
Age (yr) <sup>d*</sup>		42.1 ± 6.3 (27–59)	42.6 ± 6.5 (29–59)	45.4 ± 6.5 (29–59)
Smoking	Never smoker	268 (24.2)	105 (21.0)	24 (24.2)
	Ex-smoker	373 (33.7)	178 (35.5)	30 (30.3)
	Current smoker	465 (42.1)	218 (43.5)	45 (45.5)
Alcohol drinking	Social drinker	499 (45.1)	191 (38.1)	43 (43.4)
	Moderate drinker	266 (24.1)	131 (26.2)	24 (24.2)
	Heavy drinker	341 (30.8)	179 (35.7)	32 (32.3)
Environmental noise (dB)	<80	137 (12.4)	50 (10.0)	15 (15.2)
	80–84.9	589 (53.2)	287 (57.3)	54 (54.5)
	≥85	380 (34.4)	164 (32.7)	30 (30.3)
Hypertension <sup>e*</sup>	No	927 (83.8)	369 (73.7)	57 (57.6)
	Yes	179 (16.2)	132 (26.3)	42 (42.4)
Serum creatinine (mg/dl)	<1.2	1,094 (98.9)	490 (97.8)	96 (97.0)
	≥1.2	12 (1.1)	11 (2.2)	3 (3.0)

<sup>a</sup> Fasting glucose <100 mg/dl. <sup>b</sup> Fasting glucose 100–125 mg/dl. <sup>c</sup> Fasting glucose ≥126 mg/dl or medication for diabetes.

<sup>d</sup> The values are expressed as mean ± SD (range). <sup>e</sup> Systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or medication for hypertension. \*  $p < 0.05$ .

**Table 2.** Hearing thresholds (4,000 Hz) among the normal, impaired fasting glucose and diabetes groups (dB, Mean ± SD)

		All subjects (n=1,706)	Normal <sup>a</sup> (n=1,106)	Impaired <sup>b</sup> (n=501)	Diabetes <sup>c</sup> (n=99)
Right ear	2005*	24.7 ± 19.5	23.7 ± 18.9	26.0 ± 20.1	28.8 ± 21.9
	2009*	26.9 ± 20.9	25.4 ± 20.1	29.1 ± 21.9	32.4 ± 23.2
	Changes <sup>d*</sup>	2.2 ± 8.1**	1.7 ± 7.8**	3.1 ± 8.7**	3.6 ± 8.5**
Left ear	2005*	25.7 ± 19.4	24.8 ± 19.1	26.9 ± 19.9	28.9 ± 20.2
	2009*	27.8 ± 21.0	26.6 ± 20.5	29.4 ± 21.5	32.9 ± 21.9
	Changes <sup>d*</sup>	2.1 ± 7.6**	1.7 ± 7.6**	2.4 ± 7.7**	4.0 ± 7.0**

<sup>a</sup> Fasting glucose <100 mg/dl. <sup>b</sup> Fasting glucose 100–125 mg/dl. <sup>c</sup> Fasting glucose ≥126 mg/dl or medication for diabetes.

<sup>d</sup> Hearing threshold changes during 4 yr (2005–2009). \*  $p < 0.05$  by Kruskal-Wallis test. \*\*  $p < 0.05$  by Wilcoxon signed-rank test.

were performed; Table 2 shows the results. The hearing thresholds among the subjects for both ears in 2005 and 2009 were significantly different among the normal, impaired fasting glucose and diabetes groups by Kruskal-Wallis tests ( $p < 0.05$ ). In addition, hearing threshold changes among subjects for both ears over the four years were also significantly different among the three groups ( $p < 0.05$ ). The hearing thresholds among the subjects for both ears changed significantly from 2005 to 2009 ( $p < 0.05$  by Wilcoxon signed rank tests). These results show that the hearing thresholds of the subjects in 2009 were significantly changed compared with 2005, and the changes were significantly different among the three groups.

#### Factors affecting the change of hearing thresholds

Analyses of covariance were performed to adjust for the factors affecting the change in the hearing thresholds. Table 3 shows the results of the analysis of variance models 1 and 2. In ANCOVA model 1, impaired fasting glucose (100–125 mg/dl) was significant ( $\beta = 0.434$ ,  $p = 0.002$ ) for the right ear, whereas it was not significant ( $\beta = 0.412$ ,  $p = 0.121$ ) for the left ear. In ANCOVA model 2, the interaction term (fasting glucose × environmental noise) was not a significant variable for either ear.

#### Discussion

Many studies have reported that diabetes is associated with hearing loss. Kakarlapudi *et al.* reported that

**Table 3.** Factors associated with hearing loss: results of the analysis of covariance with hearing threshold changes as the dependent variable

Variables <sup>a</sup>	Right ear			Left ear		
	$\beta$	SE <sup>b</sup>	<i>p</i> -value	$\beta$	SE <sup>b</sup>	<i>p</i> -value
Model 1: ANCOVA test for fasting glucose						
Age	0.219	0.034	<0.001	0.151	0.033	<0.001
Smoking	0.425	0.257	0.099	0.12	0.244	0.624
Alcohol drinking	-0.242	0.234	0.302	0.215	0.222	0.332
Environmental noise	0.141	0.051	0.006	0.124	0.049	0.011
Hypertension	0.6	0.489	0.22	0.127	0.464	0.784
Serum creatinine	1.927	1.589	0.225	1.293	1.507	0.391
Initial hearing threshold <sup>c</sup>	-0.043	0.011	<0.001	-0.019	0.01	0.067
Impaired fasting glucose	1.339	0.434	0.002	0.639	0.412	0.121
Diabetes	1.25	0.853	0.143	1.854	0.809	0.022
Model 2: ANCOVA test for fasting glucose and interaction between fasting glucose and environmental noise						
Age	0.216	0.034	<0.001	0.076	0.032	0.017
Smoking	0.423	0.258	0.101	0.089	0.243	0.714
Alcohol drinking	-0.229	0.234	0.327	0.208	0.221	0.346
Environmental noise	0.116	0.142	0.413	0.101	0.134	0.451
Hypertension	0.584	0.489	0.233	0.224	0.462	0.627
Serum creatinine	1.959	1.591	0.219	1.23	1.502	0.413
Initial hearing threshold <sup>c</sup>	-0.042	0.011	<0.001	0.042	0.01	<0.001
Fasting glucose	-2.368	6.789	0.727	-1.43	6.411	0.824
Fasting glucose $\times$ environmental noise	0.017	0.082	0.837	0.009	0.077	0.912

In the statistical analyses, the significance level for the *p*-value was 0.05. <sup>a</sup> Continuous variables included age, environmental noise and initial hearing threshold; categorical variables included smoking (never, ex- and current smoker), alcohol drinking (social, moderate and heavy drinking), hypertension (no hypertension and existing hypertension), serum creatinine (<1.2 and  $\geq$ 1.2 mg/dl) and fasting glucose (<100, 100–125 and  $\geq$ 126 mg/dl or medication for diabetes). <sup>b</sup> SE means standard error. <sup>c</sup> Hearing threshold of the subjects measured in 2005.

sensorineural hearing loss was more common in diabetic patients and that the severity of the hearing loss appeared to correlate with the progression of disease<sup>5</sup>). Wu *et al.* reported on an animal study in which diabetic rats had impaired recovery from noise-induced temporary hearing loss<sup>6</sup>). McQueen *et al.* found that type 2 diabetes mellitus alone did not cause significant capillary changes in the cochlea of genetically induced diabetic rats; the changes were observed only in combination with noise exposure and/or obesity<sup>7</sup>). Several studies have reported on the possible pathogenesis of diabetes-associated hearing loss. The suggested pathogenesis includes cochlear microangiopathy, hyperglycemia of the cerebrospinal fluid or perilymph, auditory neuropathy and diabetic encephalopathy<sup>8</sup>). Fukushima *et al.* described a diabetes-associated pathology in the cochlea that includes thickened vessels of the stria vascularis, atrophy of the stria vascularis and loss of outer hair cells<sup>9</sup>). Many other studies have reported that diabetes is a risk factor for hearing loss<sup>10–13</sup>).

Austin *et al.* reported diabetes was associated with an increased risk of hearing loss; this difference was observed particularly in adults below 50 yr of age<sup>8</sup>). In a 5-year

prospective study reported by Vaughan *et al.*, diabetic patients 60 yr of age or younger had early high-frequency hearing loss similar to early presbycusis; in addition, the difference in hearing loss between diabetic and nondiabetic patients was reduced after the age of 60<sup>14</sup>). According to two studies using National Health and Nutrition Examination Surveys in the United States, persons with diabetes had a higher prevalence of hearing impairment<sup>15, 16</sup>). Elamin *et al.* reported that hearing loss occurred early in diabetic children and was related to the duration of the disease and metabolic control<sup>17</sup>).

Although prior studies have shown an association between diabetes and hearing loss, an association between impaired fasting glucose and hearing loss has not yet been identified. Therefore, impaired fasting glucose and noise-induced hearing loss were compared in this study. The audiometric notch at 4,000 Hz is a well-established clinical sign of noise-induced hearing loss and may be valuable in confirming the diagnosis; pure tone thresholds at 4,000 Hz were used in this study as a screening test for noise-induced hearing loss<sup>2</sup>). The environmental noise levels in the workplace were measured to control for the

confounding effects of noise exposure. Other risk factors including age, smoking and alcohol history, hypertension and serum creatinine were also investigated. All subjects were male and Korean; the risk factors of gender and race were not considered. In addition, all subjects used hearing protection during work according to the rules of their company; use of hearing protectors was not considered.

In this study, the hearing thresholds were elevated, and the changes were significantly different among the groups of subjects with normal and impaired fasting glucose and diabetes. The changes in hearing thresholds were greater in the impaired fasting glucose and diabetes groups than in the normal fasting glucose group. The analysis of covariance showed that the fasting glucose was a significant variable after adjusting for the other variables. Bainbridge *et al.* reported diabetes-related hearing changes had been found at low or mid-frequencies and high frequencies<sup>8)</sup>, but another study reported the changes were different between type 1 and type 2 diabetes<sup>15)</sup>. We did not measure the subjects' hearing thresholds at low or medium frequencies and did not identify whether the subjects' diabetes were type 1 or type 2 diabetes, so we could not identify the hearing threshold changes at low or medium frequencies or the differences between type 1 and type 2 diabetes.

In this study, impaired fasting glucose was significant in the analysis of variance for the right ear; however, it was not significant for the left ear. Several factors might be considered to account for this disparity in the results between the right and left ears. First, the follow-up period (4 yr) was relatively short to identify the association between fasting glucose and the hearing threshold changes. Second, many subjects who had undergone health examinations in 2005 were excluded due to loss of follow-up. Third, confounding factors such as genetics and use of organic solvents were not considered in this study, although these factors were reported to be associated with hearing loss<sup>18)</sup>. Fourth, it had been reported that type 1 and type 2 diabetes have different effects on hearing loss<sup>8)</sup>, but we could not identify whether the subjects had type 1 or type 2 diabetes. Fifth, unlike hemoglobin A1c or fructosamine, the fasting glucose of the subjects did not account for their long-term status. Further studies considering the above factors are needed to confirm the association between fasting glucose and noise-induced hearing loss.

To evaluate the effect of occupational noise exposure on hearing thresholds, adjustment for risk factors that influence hearing function is important<sup>19)</sup>. Risk factors other than fasting glucose that affect noise-induced hearing loss include age, genetics, gender, race, hearing protection, cigarette smoking, low dietary intake of foods rich in antioxidants, vitamins and minerals, and heart disease<sup>3)</sup>. An analysis of variance was performed in order to adjust for other risk factors such as age, smoking, environmental

noise, hypertension and serum creatinine. Among these factors, age and environmental noise were found to be significant variables. The variable of interaction term (fasting glucose  $\times$  environmental noise) was entered in the analysis of covariance to evaluate the synergistic effect between fasting glucose and environmental noise. The results for this did not show any significant variable of interaction term. Therefore, there was no synergistic effect between fasting glucose and environmental noise in this study.

Aging and noise exposure are common causes of sensorineural hearing loss in adults<sup>20)</sup>. According to the results of recent studies, the interaction of age and noise exposure is not simply additive<sup>21, 22)</sup>. In individuals exposed to noise, age-related hearing loss is not as much of a risk factor as those not exposed to noise<sup>22)</sup>. In this study, age and noise exposure were identified as risk factors for noise-induced hearing loss.

Cardiovascular disease can increase the risk of hearing loss; there was a significant association reported between hypertension and hearing loss<sup>23-25)</sup>. The severity of hearing loss in patients with diabetes might correlate with the progression of disease as reflected by serum creatinine<sup>5)</sup>. However, in this study, hypertension and the serum creatinine were not significant variables.

This study had several limitations. First, a selection bias could not be controlled for with regard to loss to follow-up for the screening tests for noise-induced hearing loss. Second, only the 4,000-Hz threshold was measured; the association between fasting glucose and low or medium pure tone frequency thresholds was not studied. Third, risk factors for noise-induced hearing loss such as genetics, leisure time noise exposure and use of organic solvents were not considered. Fourth, although all workers were instructed to use hearing protectors, it was not clear whether they really used the hearing protectors. Further study is needed to confirm an association between fasting glucose and hearing loss.

In conclusion, a retrospective study was performed to determine whether there is an association between impaired fasting glucose and hearing loss. According to the results of this study, impaired fasting glucose might be a risk factor for hearing loss in individuals exposed to noise in the workplace. Therefore, impaired fasting glucose should be considered a risk factor that can be associated with noise-induced hearing loss.

## References

- 1) Krishnamurti S. Sensorineural hearing loss associated with occupational noise exposure: effects of age-corrections. *Int J Environ Res Public Health* 2009; 6: 889-99.
- 2) McBride DI, Williams S. Audiometric notch as a sign of noise induced hearing loss. *Occup Environ Med* 2001; 58: 46-51.

- 3) Daniel E. Noise and hearing loss: a review. *J Sch Health* 2007; 77: 225–31.
- 4) Genuth S, Alberti KG, Bennett P, et al. Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care* 2003; 26: 3160–7.
- 5) Kakarlapudi V, Sawyer R, Staecker H. The effect of diabetes on sensorineural hearing loss. *Otol Neurotol* 2003; 24: 382–6.
- 6) Wu HP, Cheng TJ, Tan CT, Guo YL, Hsu CJ. Diabetes impairs recovery from noise-induced temporary hearing loss. *Laryngoscope* 2009; 119: 1190–4.
- 7) McQueen CT, Baxter A, Smith TL, et al. Non-insulin-dependent diabetic microangiopathy in the inner ear. *J Laryngol Otol* 1999; 113: 13–8.
- 8) Austin DF, Konrad-Martin D, Griest S, McMillan GP, McDermott D, Fausti S. Diabetes-related changes in hearing. *Laryngoscope* 2009; 119: 1788–96.
- 9) Fukushima H, Cureoglu S, Schachern PA, Paparella MM, Harada T, Oktay MF. Effects of type 2 diabetes mellitus on cochlear structure in humans. *Arch Otolaryngol Head Neck Surg* 2006; 132: 934–8.
- 10) Diniz TH, Guida HL. Hearing loss in patients with diabetes mellitus. *Braz J Otorhinolaryngol* 2009; 75: 573–8.
- 11) Mitchell P, Gopinath B, McMahon CM, et al. Relationship of Type 2 diabetes to the prevalence, incidence and progression of age-related hearing loss. *Diabet Med* 2009; 26: 483–8.
- 12) Rozanska-Kudelska M, Chodynicky S, Kinalska I, Kowalska I. Hearing loss in patients with diabetes mellitus type II. *Otolaryngol Pol* 2002; 56: 607–10.
- 13) Celik O, Yalcin S, Celebi H, Ozturk A. Hearing loss in insulin-dependent diabetes mellitus. *Auris Nasus Larynx* 1996; 23: 127–32.
- 14) Vaughan N, James K, McDermott D, Griest S, Fausti S. A 5-year prospective study of diabetes and hearing loss in a veteran population. *Otol Neurotol* 2006; 27: 37–43.
- 15) Bainbridge KE, Hoffman HJ, Cowie CC. Diabetes and hearing impairment in the United States: audiometric evidence from the National Health and Nutrition Examination Survey, 1999 to 2004. *Ann Intern Med* 2008; 149: 1–10.
- 16) Cheng YJ, Gregg EW, Saaddine JB, Imperatore G, Zhang X, Albright AL. Three decade change in the prevalence of hearing impairment and its association with diabetes in the United States. *Prev Med* 2009; 49: 360–4.
- 17) Elamin A, Fadlallah M, Tuevmo T. Hearing loss in children with type 1 diabetes. *Indian Pediatr* 2005; 42: 15–21.
- 18) Rabinowitz PM, Galusha D, Slade MD, et al. Organic solvent exposure and hearing loss in a cohort of aluminium workers. *Occup Environ Med* 2008; 65: 230–5.
- 19) Agrawal Y, Niparko JK, Dobie RA. Estimating the effect of occupational noise exposure on hearing thresholds: the importance of adjusting for confounding variables. *Ear Hear* 2010; 31: 234–7.
- 20) Mills JH, Dubno JR, Boettcher FA. Interaction of noise-induced hearing loss and presbycusis. *Scand Audiol Suppl* 1998; 48: 117–22.
- 21) Rosenhall U. The influence of ageing on noise-induced hearing loss. *Noise Health* 2003; 5: 47–53.
- 22) Albera R, Lacilla M, Piumetto E, Canale A. Noise-induced hearing loss evolution: influence of age and exposure to noise. *Eur Arch Otorhinolaryngol* 2010; 267: 665–71.
- 23) Zhang J, Zhou H, Zhang G, Xu Y. A study on early hearing impairment with essential hypertension. *Lin Chung Er Bi Yan Hou Tou Jing Wai Ke Za Zhi* 2009; 23: 731–3.
- 24) de Moraes Marchiori LL, de Almeida Rego Filho E, Matsuo T. Hypertension as a factor associated with hearing loss. *Braz J Otorhinolaryngol* 2006; 72: 533–40.
- 25) Rosenhall U, Sundh V. Age-related hearing loss and blood pressure. *Noise Health* 2006; 8: 88–94.