Traffic noise and other determinants of blood pressure in adolescence

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ABSTRACT

Background: Exposure to traffic noise has been associated with hypertension in adults but the evidence in adolescents is limited. We investigated long-term road traffic noise exposure, maternal occupational noise during pregnancy and other factors in relation to blood pressure and prehypertension at 16 years of age.
Methods: Systolic and diastolic blood pressure were measured in 2597 adolescents from the Swedish BAMSE birth cohort. Levels of road traffic noise were estimated at home addresses during lifetime and for the mother during pregnancy as well as maternal occupational noise exposure during pregnancy. Exposure to NO_x from local sources was also assessed. Associations between noise or NO_x exposure and blood pressure or prehypertension were analysed using linear and logistic regression.
Results: The prevalence of prehypertension was higher among males and in those with overweight, low physical activity or overweight mothers. No strong or consistent associations were observed between pre- or postnatal exposure to road traffic noise and blood pressure at 16 years of age. However, inverse associations were suggested for systolic or diastolic blood pressure and prehypertension, which reached statistical significance among males (OR 0.80 per 10 dB L_{den}, 95% CI 0.65–0.99) and those with maternal occupational noise exposure ≥ 70 dB L_{Aeq8h} (OR 0.60, 95% CI 0.41–0.87). On the other hand, occupational noise exposure during pregnancy tended to increase systolic blood pressure and prehypertension risk in adolescence. No associations were seen for NO_x exposure.
Conclusion: No conclusive associations were observed between pre- or postnatal noise exposure and blood pressure or prehypertension in adolescents.

1. Introduction

Environmental noise exposure from sources such as road traffic has emerged as an important health hazard, estimated to cause at least 1 million healthy life-years lost each year in Western Europe, attributed primarily to sleep disturbance, annoyance, and cardiovascular diseases (WHO, 2011). A recent systematic review found road traffic noise exposure to be associated with hypertension in adults based on a meta-analysis of 26 cross-sectional studies, however, the quality of the evidence was regarded as low (Kempen et al., 2018). There were only a few studies focusing on traffic related noise exposure and blood pressure in children, overall showing non-significant associations. Furthermore, there is evidence on air pollution exposure and blood

pressure in children. A recent review identified 14 such studies, mostly of cross-sectional design (Sanders et al., 2018). Significant associations between pollutants such as $PM_{2.5}$ and blood pressure were observed in three cohort studies and most of the cross-sectional studies reported significant associations. However, the results varied with regard to associations with systolic and/or diastolic blood pressure as well as type of air pollutants. Overall, no clear conclusions on causality can be drawn concerning exposure to traffic related noise or air pollution and blood pressure in childhood or adolescence. Besides environmental determinants, factors like age, sex, height, obesity and physical activity have been associated with hypertension in childhood and adolescence (Durrani and Fatima, 2012). It is not known to what extent environmental, genetic and lifestyle factors interact in relation to blood

pressure in adolescence.

Maternal noise exposure during pregnancy may cause adverse health effects in the offspring. For example, occupational noise exposure during pregnancy has been related to hearing dysfunction in children (Selander et al., 2016), whereas residential road traffic noise exposure during pregnancy was associated with a higher risk for childhood overweight (Christensen et al., 2016). Traffic related noise exposure during pregnancy was found to have an inverse relation to birth weight (Gehring et al., 2014). Both birth weight and gestational age are inversely associated with blood pressure later in life (Jarvelin et al., 2004; Steinthorsdottir et al., 2013). Elevated blood pressure during childhood can persist into adulthood and thus constitute a risk factor for various cardiovascular diseases (Bilenko et al., 2015; Falkner et al., 2008). However, it is not known if prenatal exposure to noise affects blood pressure in adolescence.

The aim of the present study was to investigate associations between road traffic noise exposure during pregnancy or childhood and blood pressure among 16-year-olds of a Swedish birth cohort. Furthermore, we examined the association with maternal occupational noise exposure during pregnancy. Analyses were also performed to assess possible interactions, particularly with exposure to ambient air pollution.

2. Methods

2.1. Study population

This study was conducted among individuals from the birth cohort BAMSE (Swedish acronym for children, allergy, milieu, Stockholm, epidemiology), which is described in detail elsewhere (Wickman et al., 2002). In brief, the cohort consisted of 4089 children born between 1994 and 1996 in predefined areas of Stockholm County. Shortly after birth and at the ages 1, 2, 4, 8, 12 and 16 years of age questionnaires were sent to the parents to obtain information on environmental exposures, lifestyle, and health of the children. At the 12 and 16- year follow-up the study subjects additionally filled in a questionnaire themselves. At the ages 4, 8 and 16 years the participants were invited to medical examinations. At the 16- year follow-up blood pressure measurements were made in a total of 2597 adolescents (63.5% of those originally recruited).

2.2. Noise exposure

Residential histories of the study subjects from birth to adolescence, as well as of their mothers during pregnancy, were obtained from the Swedish Tax Agency. Linkage with a national property register provided geographical coordinates for the home addresses. Road traffic noise exposure assessment was performed by estimating noise levels at the study subject's residential address based on a modification of the Nordic prediction method for road traffic noise (Jonasson and Nielsen, 1996). The method takes into account three dimensional terrain characteristics, ground surface and buildings as well as traffic flow, speed and proportion of heavy vehicles, and calculates the equivalent noise level ($L_{Aeq,24h}$) at the most exposed façade of the dwelling. Noise data were available every fifth year from 1990 to 2010 and linear interpolation was used to estimate noise levels between these years. Time-weighted average noise exposure was estimated based on the residential history of each study subject during follow-up. Noise levels were recalculated to the European noise indicator L_{den} to facilitate international comparisons (Jonasson and Gustafson, 2010). Our noise model output has been compared with the Nordic method and preliminary results indicate that 95% of the predictions are within ± 5 dB, with an average difference of less than 0.5 dB. A more detailed description of the methodology for estimating noise levels from road traffic is provided by Ogren and Barregard (2016).

To assess maternal occupational noise exposure during pregnancy information on the mother's occupation from the baseline questionnaire

was combined with a job-exposure-matrix. The job-exposure-matrix listed more than 300 occupations and groups of occupations. Occupations were classified according to the Nordic occupation code and each occupation and/or occupation group was assigned a noise level based on multiple measurements over time. Noise was assessed in five-year intervals from 1970 to 2014 and categorised in five levels. A detailed description of the original job-exposure-matrix for noise can be found elsewhere (Sjostrom et al., 2013). For the present study this matrix was further developed, primarily by expanding the number of noise exposure categories.

2.3. Outcome definition

At the 16-year follow-up data on height and weight were collected during a medical examination. Resting systolic and diastolic blood pressure was measured by trained nurses using an automatic blood pressure meter (Omron M6, AJ Medical, Lidingö, Sweden). This device has been validated for use in children and adolescents (Stergiou et al., 2006). Blood pressure was assessed three times for each individual with a 1 min pause between the measurements, following a standard protocol. The cuff (sizes S/L) was placed on the upper part of the right arm. The mean blood pressure of all three measurements of each study subject was used in the analysis. A systolic or diastolic blood pressure $\geq 90^{th}$ percentile for age, sex and height and/or $\geq 120/80$ mmHg was considered as prehypertension (NIH, 2004; Rosner et al., 2013).

2.4. Covariates

The covariates assessed included sex (dichotomous), age at blood pressure measurement ($\leq 16.5/ > 16.5$ years), height ($\leq 1.70/ > 1.70$ m), gestational age ($< 37/\geq 37$ weeks), birth weight ($\leq 2500/ > 2500$ g), pubertal stage (late/post pubertal) (Petersen et al., 1988), Body-Mass-Index (BMI) at blood pressure measurement ($< 25/ \geq 25$ kg/m^2), exclusive breastfeeding ($< 4/\geq 4$ months), maternal BMI during pregnancy assessed at first visit at the antenatal clinic ($< 25/ \geq 25$ kg/m^2), municipality at birth (Stockholm, Järfälla, Solna, Sundbyberg), parental occupation (blue collar, low/medium white collar, high white collar worker), maternal education (university/ > 2 years high school/middle school or less), physical activity ($< 5/\geq 5$ h/week), sleep duration ($< 8/\geq 8$ h/night), maternal smoking during pregnancy (yes/no), parental smoking during infancy (yes/no), nicotine use at 16 years (yes/no), and maternal occupational noise exposure during pregnancy ($< 70/70–79/\geq 80$ dB$_{LAeq,8h}$). Data on birth weight, gestational age, exclusive breastfeeding, maternal BMI, maternal education, parental occupation, parental smoking and municipality at birth were obtained from the questionnaire shortly after birth and the remaining information was extracted from the questionnaire answered by the study subjects at 16 years of age. Furthermore, information on height and weight was taken from the medical examination at the 16-year follow-up and used to calculate the BMI.

Exposure to locally emitted air pollutants, using nitrogen oxides (NO$_x$) as indicator, was estimated through dispersion models and time-weighted average outdoor levels during lifetime were calculated at residential addresses. Briefly, dispersion models were combined with emission data bases to calculate average annual levels of NO$_x$ for the years 1990, 1995, 2000, 2002, 2003, 2004, 2006, 2010 and 2015. Linear interpolation was used to obtain NO$_x$ concentrations between the years with emission data bases. Time-weighted average exposure to NO$_x$ was estimated for each individual depending on the duration of time spent at all addresses from birth to the 16-year follow-up. Air pollution concentrations were estimated for geocoded residential addresses, as well as day care and school addresses. A detailed description of the assessment is provided by Schultz et al. (2016). The estimated NOx concentrations generated by the dispersion models were regularly validated against measurements at air quality monitoring stations. During the period 1998–2005 the R^2-values for the comparison of time-

series calculations with annual measurements of mean NO_x concentrations at the urban background station in Stockholm ranged from 0.74 to 0.80 for the different years (Johansson et al., 2008).

2.5. Statistical methods

Proportions of prehypertensive individuals among the study participants were calculated in relation to selected background characteristics and one-way ANOVA tests were used to assess differences between categories. Associations between time-weighted average road traffic noise exposure of the study subjects from birth and onwards, as well as maternal occupational or residential noise exposure during pregnancy, and systolic or diastolic blood pressure at 16 years of age were explored with linear regression models, estimating beta coefficients and 95% confidence intervals. Logistic regression models were used to estimate odds ratios and 95% confidence intervals for prehypertension in relation to pre- or postnatal noise exposure. Associations with NO_x exposure were estimated similarly to those for noise. Regression models adjusted for sex, age (continuous) and height (continuous) were considered as crude models. The fully adjusted models additionally included municipality at birth, maternal education, maternal smoking during pregnancy, maternal BMI in pregnancy, duration of exclusive breastfeeding, physical activity and nicotine use at 16 years of age. The selection of covariates in the full models was based on associations with prehypertension in our cohort (Table 1) as well as on previous literature. BMI of the study subject was not included in the fully adjusted model, although it was related to prehypertension, since it is associated with traffic noise exposure in the BAMSE cohort (Wallas et al., 2018a) and may be an intermediate factor in the causal pathway. Regression coefficients and odds ratios were expressed in relation to increments of 10 dB L_{den} for road traffic noise and 10 μg/m^3 for NO_X. This was close to the interquartile ranges of 10.4 dB L_{den} and 9.4 μg/m^3 for noise and NO_X, respectively.

Interactions were assessed between pre-selected risk factors and residential road traffic noise exposure in relation to the risk of prehypertension. Wald tests were used to calculate p-values for interaction. Sensitivity analyses were also performed including traffic NO_x in the fully adjusted models as well as by excluding individuals who reported use of nicotine products such as snuff or cigarettes prior to the blood pressure measurement. Statistical significance levels were set at 5%, except for the interaction analyses where 10% was used.

All statistical analyses were performed with Stata (release 14; Stata Corp., College Station, TX, USA).

The baseline and follow-up surveys were approved by the Regional Ethical Review Board, Karolinska Institutet, Stockholm, Sweden, and the participants as well as their parents provided informed consent.

3. Results

Blood pressure was close to normally distributed at 16 years of age, with systolic blood pressure levels of 83.5–164 mmHg (median 120.5) and diastolic levels of 44.5–91.5 mmHg (median 67) (Fig. S1). More than twice as many boys as girls were prehypertensive (22% and 8%, respectively) (Table 1). Prehypertensive individuals were significantly older and taller, and had less often reached post-pubertal stage. More individuals with prehypertension were overweight or obese (BMI ≥25) and the proportion of prehypertensives working out < 5 h/week was higher. The individuals of the study population lived mostly in residences exposed to road traffic noise levels < 55 dB L_{den}, regardless of whether they had normal blood pressure levels or were prehypertensive (Fig. S2). In the total study group 19.9% had time-weighted-average road traffic noise levels from birth to adolescence of 55 dB L_{den} or higher. The corresponding figure among those with prehypertension was 18.1%.

Table S1 compares information on covariates for the participants at the 16 year follow-up, shown in Table 1, with corresponding data

Table 1

Prehypertension in relation to selected covariates in 16-year olds in the BAMSE cohort from Stockholm.

Variable	Total	Prehypertension		
	n	n	%	p-value
Sex				< 0.01
Male	1256	272	21.7	
Females	1339	110	8.2	
Age (years)				0.02
≤16.5	964	121	12.6	
> 16.5	1631	261	16.0	
Height (m)				< 0.01
≤1.70	1332	154	11.6	
> 1.70	1263	228	18.1	
Gestational age (weeks)				0.55
< 37	146	24	16.4	
≥37	2449	358	14.6	
Birth weight (g)				0.77
≤2500	95	13	13.7	
> 2500	2500	369	14.8	
Puberty stage at 16 years				0.07
Late	1960	292	14.9	
Post	331	37	11.2	
BMI at 16 years				< 0.01
Normal	2156	253	11.7	
Overweight/Obese	435	128	29.4	
Exclusive breastfeeding				0.23
< 4 months	492	80	16.3	
≥4 months	2051	290	14.1	
Maternal BMI in pregnancy				0.02
Normal	1797	245	13.6	
Overweight/Obese	798	137	17.2	
Municipality at birth				0.17
Stockholm	814	105	12.9	
Järfälla	743	124	16.7	
Solna	668	102	15.3	
Sundbyberg	357	48	13.5	
Parental occupation				0.35
Blue collar worker	396	67	16.9	
White collar worker	2162	311	14.4	
Other	28	3	10.7	
Maternal education				0.58
University	1132	169	14.9	
> 2 years high school	1263	180	14.3	
Middle school or less	187	32	17.1	
Physical activity				0.01
< 5 h/week	1133	189	16.7	
≥5 h/week	1462	193	13.2	
Sleep duration				0.78
< 8 h/night	1113	165	14.8	
≥8 h/night	1462	211	14.4	
Maternal smoking during pregnancy				0.95
No	2299	338	14.7	
Yes	295	43	14.6	
Parental smoking during infancy				0.70
No	2063	305	14.8	
Yes	517	73	14.1	
Nicotine use at 16 years[a]				0.11
No	221	336	15.1	
Yes	361	43	11.9	
Maternal occupational noise exposure (dB L_{Aeq8h})				0.27
< 70	1970	281	14.3	
70–79	306	53	17.3	
≥80	203	34	16.8	
Maternal road traffic noise during pregnancy (dB L_{den})				0.18
< 45	402	66	16.4	
45–54	1179	182	15.4	
≥55	1006	132	13.1	
Road traffic noise from 0 to 16 years (dB L_{den})				0.06
< 45	731	125	17.1	
45–54	992	132	13.3	
≥55	430	57	13.3	
Road traffic noise at 16 years (dB L_{den})				0.61
< 45	831	128	15.4	

(*continued on next page*)

Table 1 (*continued*)

Variable	Total	Prehypertension		
	n	n	%	p-value
45–54	1017	141	13.9	
≥55	559	78	14.0	

[a] 315 individuals smoked cigarettes and/or 118 used snuff at 16 years of age, respectively.

available also for the non-participants obtained at recruitment. The table shows that the participants at 16 years of age include more women as well as individuals with higher education, white collar occupations and from Stockholm but fewer mothers smoking during pregnancy and with short breastfeeding period. None of these characteristics was related to prehypertension, except for sex (Table 1). However, sex was not related to noise exposure (data not shown). Table 2 shows results of analyses regarding systolic blood pressure at 16 years of age in relation to maternal noise exposure during pregnancy as well as road traffic noise exposure of the study subjects from birth to 16 years of age. In the crude model statistically significant inverse associations were generally seen in relation to road traffic noise exposure. However, these mostly did not remain significant in the fully adjusted model, nor did they show clear exposure-response relationships. On the other hand, children of mothers occupationally exposed to noise during pregnancy at 70 dB L_{Aeq8h} or higher tended to have an elevated systolic blood pressure. Findings for diastolic blood pressure resembled those for systolic blood pressure, but associations tended to be weaker (Table S2). Tendencies towards inverse associations with blood pressure were also seen for NO_x exposure (Table S3).

There was a significantly decreased odds ratio for prehypertension in relation to road traffic noise exposure from birth to adolescence in the crude model (Table 3). However, this did not remain significant in the fully adjusted model. Similar tendencies were observed for maternal

Table 3

Odds ratio for prehypertension at 16 years of age in relation to maternal occupational noise exposure and residential road traffic noise exposure during pregnancy as well as from birth to 16 years of age.

	Prehypertension				
	n	Crude model[a]		Fully adjusted model[b]	
		Odds Ratio	CI (95%)	Odds Ratio	CI (95%)
Maternal occupational exposure (dB L_{Aeq8h})					
< 70	281	Ref.	– –	Ref.	– –
70–79	53	1.32	0.95 1.84	1.33	0.95 1.88
≥80	34	1.23	0.82 1.83	1.19	0.79 1.80
Road traffic noise exposure, (dB L_{den})					
Maternal during pregnancy					
< 45	66	Ref.	– –	Ref.	– –
45–54	182	0.88	0.64 1.21	0.89	0.63 1.25
≥55	132	0.73	0.52 1.01	0.80	0.53 1.20
Continuous (per 10 dB)	380	0.85	0.74 0.97	0.90	0.75 1.08
Time-weighted average from birth to 16 years of age					
< 45	125	Ref.	– –	Ref.	– –
45–54	132	0.70	0.53 0.92	0.68	0.51 0.91
≥55	57	0.70	0.49 0.98	0.81	0.55 1.20
Continuous (per 10 dB)	314	0.83	0.71 0.97	0.87	0.72 1.04
At 16 years of age					
< 45	128	Ref.	– –	Ref.	– –
45–54	141	0.87	0.67 1.14	0.85	0.65 1.12
≥55	78	0.90	0.66 1.22	0.99	0.71 1.39
Continuous (per 10 dB)	347	0.90	0.79 1.04	0.94	0.81 1.08

[a] Adjusted for sex, age and height.
[b] Adjusted for sex, age, height, municipality at birth, duration of exclusive breastfeeding, maternal BMI in pregnancy, maternal education, maternal smoking during pregnancy, physical activity and nicotine use at 16 years of age.
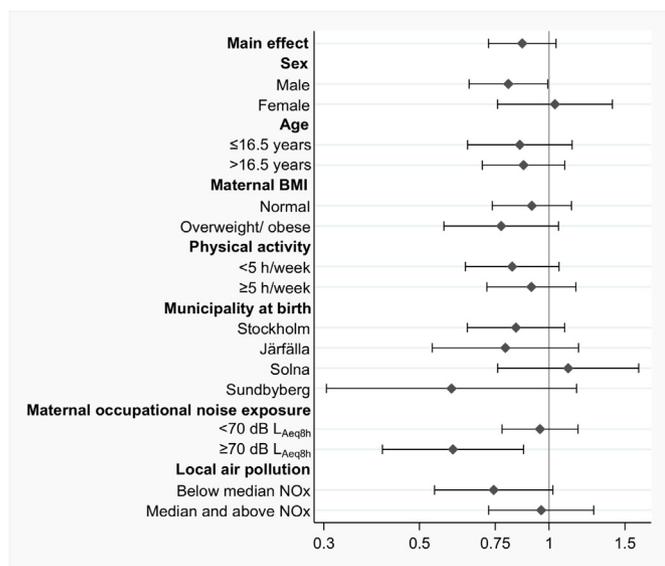
Table 2

Maternal occupational noise exposure and residential road traffic noise exposure during pregnancy as well as from birth to 16 years of age in relation to systolic blood pressure at 16 years of age.

	Systolic blood pressure (mmHg)					
	N (%)	Crude model[a]		Fully adjusted model[b]		
		β[c]	CI (95%)	β	CI (95%)	
Maternal occupational exposure during pregnancy (dB L_{Aeq8h})						
< 70	1970 (79.4)	Ref.	–	–	Ref.	– –
70–79	306 (12.4)	1.24	0.01	2.49	1.22	−0.02 2.48
≥80	203 (8.2)	0.97	−0.52	2.45	0.80	−0.72 2.31
Road traffic noise exposure, (dB L_{den})						
Maternal during pregnancy						
< 45	402 (15.5)	Ref.	-	-	Ref.	- -
45–54	1179 (45.6)	−1.32	−2.49	−0.16	−1.27	−2.52 −0.03
≥55	1006 (38.9)	−2.00	−3.19	−0.82	−1.27	−2.72 0.18
Continuous (per 10 dB)	2587	−0.91	−1.38	−0.43	−0.52	−1.16 0.13
Time-weighted average from birth to 16 years of age						
< 45	731 (34.0)	Ref.	-	-	Ref.	- -
45–54	992 (46.1)	−1.33	−2.31	−0.35	−1.34	−2.36 −0.32
≥55	430 (19.9)	−1.74	−2.96	−0.51	−0.55	−1.98 0.75
Continuous (per 10 dB)	2153	−0.86	−1.41	−0.30	−0.41	−1.04 0.21
At 16 years of age						
< 45	831 (34.5)	Ref.	–	–	Ref.	– –
45–54	1017 (42.2)	−0.83	−1.77	0.12	−0.85	−1.81 0.10
≥55	559 (23.3)	−1.04	−2.14	0.06	−0.33	−1.49 0.84
Continuous (per 10 dB)	2407	−0.52	−0.99	−0.04	−0.21	−0.72 0.29

[a] Adjusted for sex, age and height.
[b] Adjusted for sex, age, height, municipality at birth, duration of exclusive breastfeeding, maternal education, maternal BMI in pregnancy, maternal smoking during pregnancy, physical activity and nicotine use at 16 years of age.
[c] Regression coefficient based on linear models as well as 95% confidence interval.

Fig. 1. Odds ratio* for prehypertension at 16 years of age in relation to average time weighted residential road traffic noise exposure per 10 dB L_{den} increase (95% CI) from birth and onwards for different categories of risk factors.

* Adjusted for sex, age, height, municipality at birth, duration of exclusive breastfeeding, maternal BMI in pregnancy, maternal education, maternal smoking during pregnancy, physical activity and nicotine use at 16 years of age.

road traffic exposure during pregnancy as well as at 16 years of age. Just as for blood pressure the prevalence of prehypertension tended to be increased among children of mothers occupationally exposed to noise at levels above 70 dB(A) during pregnancy. Results for prehypertension related to NO_x exposure resembled those for road traffic noise exposure (Table S3).

Interactions between road traffic noise exposure from birth to adolescence and different risk factors in relation to prehypertension are explored in Fig. 1. Statistically significant decreases in the odds ratio were seen for males (OR 0.80 per 10 dB L_{den}, 95% CI 0.65–0.99) and in those with maternal occupational noise exposure ≥70 dB L_{Aeq8h} (OR 0.60 per 10 dB L_{den}, 95% CI 0.41–0.87). Only the interaction with maternal occupational was statistically significant (p = 0.03). No clear effect modification was seen for the other risk factors. In sensitivity analyses the odds ratio for prehypertension related to road traffic noise exposure from birth to adolescence remained virtually unchanged when those using nicotine products before the blood pressure measurement were excluded or when NO_x was included in the fully adjusted model (data not shown).

4. Discussion

In general, we did not find strong and consistent associations between pre- or postnatal exposure to road traffic noise and blood pressure at 16 years of age. However, there tended to be inverse relations between noise exposure and systolic blood pressure or prehypertension, in some subgroups reaching statistical significance. On the other hand, occupational noise exposure of the mother during pregnancy tended to be associated with increased systolic blood pressure and prehypertension risk among the adolescents. No associations were observed for air pollution exposure from local traffic, using NO_x as marker.

Our findings are in line with results from several studies on road traffic noise exposure and blood pressure, indicating inverse associations, primarily for systolic blood pressure (Belojevic and Evans, 2012; Clark et al., 2012; Paunovic et al., 2013; van Kempen et al., 2006). On the other hand, other studies have found positive associations between road traffic exposure and blood pressure in children (Belojevic et al., 2008), sometimes only for diastolic blood pressure (Bilenko et al., 2015;

Liu et al., 2013). Overall, the evidence on traffic noise exposure in relation to blood pressure in childhood and adolescence appears weak and inconsistent, and mostly based on cross-sectional analyses. There are major differences in the assessment methods of both exposure and outcome which may contribute to explaining the inconsistent findings (Dzhambov and Dimitrova, 2017). In adults, a recent meta-analysis of 26 cross-sectional studies showed a statistically significant association between exposure to road traffic noise and prevalence of hypertension (Kempen et al., 2018). To enable interpretations of causality it would be desirable with longitudinal outcome data to assess blood pressure development or incidence of hypertension in relation to environmental noise exposure. Unfortunately, we lacked information on incidence of hypertension since blood pressure measurements were not made before 16 years of age in the BAMSE cohort.

We did not find any association between traffic related exposure to NO_x and blood pressure levels at 16 years of age, although inverse associations were suggested. This is in line with the study by Liu et al. (2014). It has been speculated that decreased blood pressure may be related to vasodilation by inhalation of NO from motor vehicle emission (Sørensen et al., 2012). However, Bilenko et al. (2015) observed a statistically significant association between diastolic blood pressure and long-term exposure to NO_2 and $PM_{2.5}$, but they did not find corresponding associations with systolic blood pressure. In adults, there is diverging evidence on air pollution exposure and hypertension, some studies reporting inverse and others positive associations (Fuks et al., 2017; Harrabi et al., 2006; Ibald-Mulli et al., 2004; Sorensen et al., 2012). Since exposure to air pollution and noise from road traffic is correlated it may be difficult to disentangle effects of either exposure, even if both factors are included in the analyses.

In general, we did not see strong interactions between road traffic noise exposure and different risk factors in relation to the risk of prehypertension. However, there were statistically significant decreases in the odds ratio for prehypertension related to noise exposure for males as well as in those with mothers occupationally exposed to noise during pregnancy. Very limited information is available on interactions between noise exposure and other risk factors for prehypertension in children and adolescents (Kelishadi et al., 2011) or for hypertension in adults (Kempen et al., 2018). It is noteworthy that both groups where decreased odds ratios in relation to noise exposure were observed tended to have increased risks of prehypertension in our material. Obviously, the interpretation of interactions is influenced by the models used, e. g. additive versus multiplicative models, and this needs careful consideration. There is a need for more empirical data on potential effect modifiers of the association between noise exposure and blood pressure.

We did not find consistent associations between maternal exposure to noise during pregnancy and blood pressure at 16 years of age, although increased systolic blood pressure was suggested in relation to maternal noise exposure at work. Various adverse health effects such as hearing loss in children as well as prematurity and intrauterine growth retardation may be associated with maternal noise exposure during pregnancy (Etzel et al., 1997). Maternal exposure to aircraft noise during pregnancy has been shown to result in vasoconstriction leading to decreased utero-placental blood flow, and possibly increased fetal hypoxia (Schell, 1981). Furthermore, a decreased bodyweight in newborns and a significant decrease in height of 3-year-olds have been shown in relation to aircraft noise exposure during pregnancy (Viet et al., 2014). Further studies are needed to confirm our results on maternal occupational noise exposure during pregnancy and systolic blood pressure in their children.

We found factors such as BMI, physical activity and maternal BMI to be associated with prevalence of prehypertension at 16 years of age. This is in line with findings in other studies (Ewald and Haldeman, 2016). On the other hand, we did not find associations with smoking, which may be explained by low exposure in our study population. Furthermore, the blood pressure levels and prevalence of

prehypertension in our study population appear consistent with those reported among adolescents (Acosta et al., 2012; Flynn et al., 2017).

This study has several limitations. Our study group was exposed to relatively low noise levels from road traffic, and only about 20% of the individuals had residential road traffic noise levels of 55 dB L_{den} or higher. In an earlier study of the same cohort we found no clear association between road traffic noise exposure and saliva cortisol levels at 16 years of age (Wallas et al., 2018b). Another limitation is imprecision in the exposure assessment, and we did not have data on road traffic exposure near schools or day care facilities. More importantly, we lacked information on indoor noise levels. Furthermore, we lacked data on noise exposure from entertainment activities and the use of personal listening devices at 16 years of age and earlier. Imprecision in the outcome measurement may also have occurred since we only measured blood pressure on one occasion. Furthermore, residual confounding from socio-economic factors may have influenced the findings. In earlier studies of the cohort we found that municipality at birth was related to asthma and BMI (Gruzieva et al., 2013; Wallas et al., 2018a). We adjusted for municipality at birth, as well as for maternal education and a number of other factors, but we cannot exclude that some confounding may still be present. Our study sample showed differences in several covariates compared to the population at recruitment, however, only sex was related to prehypertension. On the other hand, sex was not related to noise exposure. Thus, we do not anticipate that selection bias would have a strong influence on our analyses of associations between noise exposure and prehypertension.

Comprehensive information on residential addresses is a strength of this study. The database enabled detailed estimation of long-term noise exposure based on individual assessment of noise levels from birth to adolescence. Furthermore, we were able to assess occupational noise exposure of the mother during pregnancy based on an extensive job-exposure matrix. Another strength of this study is the availability of extensive data on background characteristics as well as on environmental exposures and lifestyle factors for individuals of the BAMSE cohort. Although most of this information was based on questionnaire responses, we could confirm earlier evidence on a relation between maternal BMI as well as physical activity and prehypertension.

In conclusion, we did not find strong or consistent associations between exposure to noise or air pollution from road traffic and blood pressure in adolescents. However, pre- and postnatal exposure to road traffic noise exposure tended to be associated with lower systolic blood pressure and risk of prehypertension.

Acknowledgments

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijheh.2019.04.012.

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