ORIGINAL ARTICLE

Prenatal Alcohol Exposure and the Associated Risk of Elevated Blood Pressure: A Cross-sectional Analysis of 3- to 17-Year-Olds in Germany

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BACKGROUND
As the prevalence of obesity and high blood pressure increases among the population, early action is needed to reduce blood pressure. Certain lifestyles during pregnancy have negative effects resulting in high blood pressure for children and adolescents. Using data from the "German Health Interview and Examination Survey for Children and Adolescents" (KiGGS), this study analyzed: (i) the association between low-to-moderate prenatal alcohol exposure (PAE) and the risk of increased systolic and diastolic blood pressure and (ii) whether associations were modified by socioeconomic status (SES), prenatal smoke exposure (PSE), and body mass index (BMI) of the children and adolescents.

METHODS
We applied multivariate logistic regression analyses and stratified analyses by SES, PSE, and BMI with cross-sectional data from the KiGGS study (N = 14,253) to examine the association between PAE and prehypertension or hypertension in 3- to 17-year-olds.

RESULTS
Of the surveyed children and adolescents, 13.7% had a systolic prehypertension and 11.5% had a diastolic prehypertension. A further 7.5% were identified as having systolic hypertension and 6.0% diastolic hypertension. In the regression analyses, PAE resulted in a decreased risk of systolic prehypertension (odds ratio [OR]: 0.83, 95% confidence interval [CI]: 0.70, 0.99) and diastolic prehypertension (OR: 0.82, 95% CI: 0.68, 0.98). Risk reductions were not significant in surveyed children and adolescents with hypertension. Interactions between PAE and SES, PSE, and offspring BMI were not significant.

CONCLUSIONS
Contrary to our initial hypothesis, PAE reduces the risk of prehypertension. Animal studies suggest that vasodilation is induced by nitric oxide in small quantities of PAE.

Keywords: alcohol; blood pressure; diastolic; hypertension; pregnancy; prehypertension; systolic.

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The prevalence of pediatric hypertension (i.e., above 95th blood pressure percentile for pediatrics[1]) is about 5% worldwide.[1,2] Furthermore, hypertension prevalence increased during 1999–2008 for boys and girls, respectively, from 15.8% to 19.2% and 8.2% to 12.6% in an American study.[3] Elevated blood pressure values in children and adolescents are predictive of blood pressure values in later life.[1,4,5] This is associated with risk factors for hypertension, cardiovascular disease, stroke, kidney failure, blindness, and cognitive impairment.[4,6] To reduce morbidity and mortality rates and to improve quality of life, early screening and management of high blood pressure in children and adolescents is vital.[7]

A number of risk factors associated with a rise in blood pressure in children and adolescents including: lower socioeconomic status (SES), prenatal smoke exposure (PSE), body mass index (BMI), low birth weight, maternal BMI, or above-average maternal weight gain during pregnancy. Both increased maternal BMI and above-average weight gain during pregnancy raise the likelihood of increased BMI in the offspring, which in turn is related to increased blood pressure later in life.[8–12]

In particular, there are risks associated with prenatal alcohol exposure (PAE). Some studies have shown that in the adult population, moderate alcohol intake is associated with a reduced risk of hypertension.[13,14] One explanation that has been proposed is that the vasodilating effect of alcohol results from anti-inflammatory process and an increase in nitric oxide synthase expression.[13,14] At the prenatal stage, alcohol has been found to cause myocardial damage to the endothelium leading to later arteriosclerosis or increased blood pressure in children and adolescents. In addition, nephrons can be damaged prenatally, which can later result in high blood pressure.[12,15–17]

Such risk factors in mothers and their offspring may compound through their interactions. For example, stronger effects of PAE in offspring from women with low SES

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have been reported.[18] In addition, a large study from the United States (n = 129,153) has demonstrated an interaction between PAE and PSE in relation to congenital cardiac defects.[19] PSE creates a biological environment in which alcohol has a more disadvantageous effect due to toxic multiplier effects.[20–22] Furthermore, a high BMI was found to exacerbate the impact of alcohol on levels on blood pressure.[23] Amos-Kroohs et al.[18] described the mechanisms of the effect of PAE on the BMI of children and adolescents as disparate. Nevertheless, there are little direct data available on blood pressure effects.

Such findings from PAE research and their compounding risk factors prohibit the use of humans for practical and ethical reasons and are therefore based on animal models. For example, animal models show a glucose intolerance caused by PAE with the effect of an increased BMI.[24–26] However, major or nationwide epidemiological health monitoring surveys are generating large datasets that enable multiple variables to be explored and compared without the need for human experimentation. This is of particular advantage in PAE research.

The aims of this study were to investigate: (i) the association between low-to-moderate levels of PAE and the risk of increased systolic and diastolic blood pressure and (ii) whether the associations are modified by SES, PSE, and BMI in the early lifecourse of children and adolescents.

METHODS

KiGGS study

The "German Health Interview and Examination survey for Children and Adolescents" (KiGGS) is a nationwide German long-term health survey conducted by the Robert Koch Institute. In brief, the KiGGS study is a dynamic cohort study. The survey includes age-appropriate questionnaires to be filled out by parents and by the children and adolescents aged 11–17 years. The children and adolescents took part in physical examinations and a computer-assisted personal interview was performed by a physician. Data from the base-year survey from 2003 to 2006 were used as in subsequent survey waves, data on PAE and blood pressure were not collected. With a response rate of 66.6%, a total of 17,641 aged 0–17 participated. Data were collected from 167 study locations. The selection of the sample was determined using a multilevel sampling procedure. The KiGGS study was approved by the ethics committee of Charité University Hospital Berlin and the German Federal Office for the Protection of Data on 20 February 2003.[27]

Study population

In this study, participants aged 3–17 years were included if both information on PAE and blood pleasure were available. Data were missing for PAE (n = 454) and blood pressure (n = 133). No blood pressure measurement took place for participants under 3 years of age (n = 2,805), leaving a study population of N = 14,253 participants.

Measures

Outcomes. Blood pressure was measured in the KiGGS by an automatic blood pressure device (Datascope Accutorr Plus). The measurement was taken twice on the right upper arm after 5 minutes in a seated position. A second measurement was performed after 2 minutes. The mean value in mm Hg of the 2 measurements was calculated and used as the variable value.[28] Hypertension was defined as being above 95th blood pressure percentile for pediatrics and prehypertension as being between the 90th and the 95th blood pressure percentile for pediatrics,[29] leaving a total of 4 binary variables on prehypertension (yes/no) and hypertension (yes/no) for the systolic and diastolic area, respectively. Data from the KiGGS were used as reference values.[28]

Predictors. Within the KiGGS study, PAE was recorded retrospectively by parents using a questionnaire. There were 3 possible answers (regularly, moderately, and no) to the question "Did the mother of the child drink alcohol during pregnancy?" as only 25 disclosures for "regularly" were recorded. In addition, the information on "moderate" and "regular" PAE were based on self-reports and, thus, were influenced by the respondents' subjective evaluation of the quantities "moderate" and "regular." To avoid the difficulty of distinguishing between "moderate" and "regular" PAE, we created a dichotomous variable with the categories "yes" and "no." We have applied this categorization in previous studies.[30,31]

Covariates

Maternal factors were SES as measured by maternal education based on the Comparative Analysis of Social Mobility in Industrial Nations (CASMIN) classification (low/middle/high), PSE (self-reported: yes,regularly/yes,occasional/no), hypertension in pregnancy (yes/no), diabetes in pregnancy (yes/no/I do not know), weight gain in pregnancy (kg), and maternal age at childbirth (years). During questionnaire response, it was recommended that the maternity pass was referred to answer questions about pregnancy. Children factors were ethnicity (German, Slavic, Turkish, others), residential location (rural area, small town, medium-sized city, large city), birth weight (low: < 2,500 g/normal: 2,500–4,499 g/high: > 4,499 g) as well as BMI, which is age-dependent (severely underweight/underweight/normal weight/overweight/obesity). For the analyses, age, sex, and height were excluded because the blood pressure percentiles were corrected for age, sex, and height.

The CASMIN classification. The CASMIN classification is an international classification for educational level.[32,33] It is categorized into high, middle, and low educational status based on general and vocational education. The high education group (CASMIN categories: 3a and 3b) consists of mothers with a university degree or a university applied science degree. The middle education group has an intermediate education (CASMIN categories: 2a, 2c, 2c_gen, and 2c_voc), and the low education group (CASMIN categories:

1a, 1b, and 1c) has a secondary school certification or no general or no vocational education.[32,33]

Ethnicity. The country of birth of both parents was used for the approximation of ethnicity. If at least one parent was born abroad, the child was considered as non-German. The non-German group consisted of Slavic, Turkish, and other ethnic groups. For mixed origin, the mother's country of origin was used.[31]

Residential location. For urban–rural comparisons, the division into rural was made for locales with less than 5,000 inhabitants. Small towns were towns with a population of between 5,000 and 20,000 inhabitants. Towns with more than 20,000 and less than 100,000 inhabitants were referred to as medium-sized towns. Large cities were classified as having more than 100,000 inhabitants.[34]

Body mass index. In children, BMI is age and gender specific. The limit values were based on percentiles according to Kromeyer-Hausschild et al.[35] Severe underweight was under the 3rd percentile, underweight was located between the 3rd and 10th percentile, normal weight ranges from the 10th percentile to the 90th, overweight was above the 90th percentile, and finally obesity was beyond the 97th percentile.[35]

Statistical analyses

In the first step, descriptive analyses were performed including frequencies in percentages and means with SDs. The difference between drinkers and nondrinkers was measured by the chi-squared test and the *t*-test for mean differences between normally distributed variables. Subsequently, 4 hierarchal multivariate logistic regressions were performed to calculate the odds ratio (OR) and the 95% confidence interval (CI) to express the association of systolic and diastolic prehypertension and hypertension with levels of PAE (abstainers were used as reference). The model was adjusted stepwise for the mentioned covariates applying Wald statistics. In addition, the analyses of 3 interactions terms were undertaken in the full regression model. The interactions considered were between PAE and SES, PSE, and offspring BMI. Finally, subgroups were stratified according to SES, PSE, and offspring BMI.

By means of these terms, we assessed whether an interaction could be demonstrated with conventional levels of significance ($P < 0.05$). In addition, full regression models were fitted for subgroups of children and adolescents stratified according to SES, PSE, and offspring BMI. All statistical analyses were performed using SPSS Statistics, version 24.0.

RESULTS

Of the 14,253 participants, a total of 1,936 (13.6%) women consumed alcohol during pregnancy and 12,317 (86.4%) did not drink any alcohol. Descriptive information on the study population with respect to drinking status during pregnancy is shown in Table 1. Women who consumed alcohol during pregnancy had a statistically significant higher SES. Consumers of alcohol during pregnancy were more likely to smoke during pregnancy, compared to nondrinkers. Moreover, alcohol users during pregnancy were on average older than nondrinkers. The prevalence of obesity was higher in the offspring of nondrinkers compared to the offspring of drinkers. PAE was more prevalent in Germans than in other ethnic groups.

Table 2 shows the associations between PAE and the 4 outcome parameters of the 90th and 95th percentile of systolic and diastolic blood pressure in children and adolescents. Systolic prehypertension occurred more frequently in the offspring of nondrinkers compared to drinkers (14.0% vs. 11.6% ($P = 0.004$)). Also, the occurrence of diastolic prehypertension was higher in the offspring of nondrinkers compared to the offspring of drinkers (11.8% vs. 9.1% ($P = 0.001$)). For systolic and diastolic hypertension, the analyses did not reveal differences between the offspring of drinkers compared to nondrinkers. The overall prevalence of elevated systolic and diastolic blood pressure was 7.5% and 6.0%, respectively.

In addition, mean diastolic blood pressure was significantly lower in the group without PAE (64.15 mm Hg (SD ± 7.87)) compared to the group with PAE (64.76 mm Hg (SD ± 7.88; $P = 0.002$). The mean systolic blood pressure was also lower in the offspring of nondrinkers (106.82 mm Hg (SD ± 11.69)) compared to the offspring of drinkers (107.02 mm Hg (SD ± 11.58)), however, not significantly lower ($P = 0.124$).

In the multivariate regression analyses, PAE resulted in a risk reduction for prehypertension compared to no PAE. For systolic prehypertension, the OR for PAE was 0.83 (95% CI: 0.70, 0.99). For diastolic prehypertension, the OR for PAE was 0.82 (95% CI: 0.68, 0.98). For systolic and diastolic hypertension, PAE also showed a risk reduction, but this was not statistically significant (Table 3).

The interaction terms between PAE and the SES, PSE, and offspring BMI were not statistically significant. For systolic prehypertension, the *P* value ranged from 0.509 to 0.913. For diastolic prehypertension, the *P* value ranged from 0.305 to 0.592. For systolic hypertension, the *P* value ranged from 0.218 to 0.895 and for diastolic hypertension, the *P* value ranged from 0.613 to 0.874.

Stratified analyses showed that there was no difference in the effect of PAE on prehypertension and hypertension according to levels of SES. Testing effect modification by PSE showed that the positive association between alcohol intake during pregnancy and systolic and diastolic prehypertension was only detectable in children without PSE (systolic and diastolic prehypertension: OR: 0.80). In children with PSE, the positive effect of alcohol on prehypertension disappeared. Stratified analyses by BMI showed that the positive association between PAE and diastolic prehypertension was only detectable in children with normal weight. In children with obesity, the positive effect of alcohol on diastolic prehypertension disappeared. For systolic prehypertension, no statistically significant results were found (Table 4).

The ORs of the covariates for an increased risk of developing prehypertension or hypertension in children and adolescents are shown in Supplementary Table 1). The

Table 1. Sample characteristics (N = 14,253) according to levels of alcohol intake during pregnancy

Characteristics	Total n = 14,253	Abstainer n = 12,317 (86.4 %)	Drinker n = 1,936 (13.6 %)	P for trend
Maternal characteristics				
Prenatal smoke exposure				<0.001***
Yes, regular	4.4	4.0	7.3	
Yes, occasional	12.5	12.4	12.9	
No, never	83.1	83.6	79.8	
CASMIN classification				<0.001***
High	16.7	15.5	24.1	
Middle	60.6	60.7	60.3	
Low	22.7	23.8	15.6	
Maternal age at child birth (years)	28.5 (5.1)	28.3 (5.1)	29.58 (5.0)	<0.001***
Weight gain in pregnancy (kg)	13.7 (5.4)	13.7 (5.5)	13.4 (4.8)	0.019*
Hypertension during pregnancy				0.344
No hypertension	98.7	98.7	98.9	
Hypertension	1.3	1.3	1.1	
Pregnancy: diabetes				0.902
Yes	2.2	2.2	2.1	
No	96.3	96.3	96.3	
I do not know	1.5	1.5	1.6	
Child characteristics				
Ethnicity				<0.001***
German	80.5	79.4	87.6	
Slavic	5.6	5.9	4.0	
Turkish	6.2	6.9	1.6	
Others	7.7	7.8	6.8	
Residential location				0.923
Rural area	22.3	22.3	22.4	
Small town	26.4	26.5	25.9	
Medium-sized city	28.8	28.8	28.7	
Large city	22.5	22.4	23.0	
Birth weight				0.171
Low	5.7	5.8	4.8	
Normal	92.6	92.4	93.7	
High	1.7	1.8	1.5	
Body mass index				0.005**
Severely underweight (<P3)	1.9	1.9	2.0	
Underweight (P3–P10)	5.2	5.0	16.2	
Normal weight	77.7	77.5	79.2	
Overweight (<P90–P97)	9.0	9.2	7.9	
Obesity (>P97)	6.1	6.3	4.8	

Data were missing for prenatal smoke exposure (n = 85), Comparative Analysis of Social Mobility in Industrial Nations (CASMIN) classification (n = 938), maternal age at child birth (n = 161), weight gain in pregnancy (n = 1,509), pregnancy diabetes (n = 1,494), ethnicity (n = 171), birth weight (n = 399), body mass index (n = 61). Values are percentages for categorical factors, or means (with standard deviations) for continuous factors. *P < 0.05; **P < 0.01; ***P < 0.001.

Table 2. Frequencies (in %) of systolic and diastolic blood pressure prevalence categorized by levels of prenatal alcohol exposure

	Systolic prehypertension			Diastolic prehypertension		
	No	Yes		No	Yes	
	n = 12,299 (86.3%)	*n* = 1,954 (13.7%)	*P* value	*n* = 12,624 (88.6%)	*n* = 1,632 (11.5%)	*P* value
No prenatal alcohol exposure	86.0	14.0	0.004**	88.2	11.8	0.001**
Prenatal alcohol exposure	88.4	11.6		90.9	9.1	
	Systolic hypertension			Diastolic hypertension		
	n = 13,177 (92.5%)	*n* = 1,076 (7.5%)		*n* = 13,399 (94.0%)	*n* = 854 (6.0%)	
No prenatal alcohol exposure	92.4	7.6	0.397	93.9	6.1	0.064
Prenatal alcohol exposure	92.9	7.1		94.9	5.1	

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

Table 3. Odds ratios (95% confidence interval (CI)) of elevated blood pressure in relation to prenatal alcohol exposure (*n* = 11,199)

	Odds ratio	95% CI	*P* value	Nagelkerkes R^2	Cohen effect
Systolic prehypertension					
Crude model	0.79	0.67, 0.93	0.006**	0.001	0.03
Full model	0.83	0.70, 0.99	0.033*	0.060	0.25
Diastolic prehypertension					
Crude model	0.80	0.66, 0.96	0.016**	0.001	0.03
Full model	0.82	0.68, 0.98	0.033*	0.015	0.12
Systolic hypertension					
Crude model	0.88	0.71, 1.09	0.249	0.000	0.00
Full model	0.93	0.75, 1.16	0.539	0.057	0.25
Diastolic hypertension					
Crude model	0.88	0.69, 1.12	0.289	0.000	0.00
Full model	0.92	0.72, 1.17	0.494	0.024	0.16

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

Full model: adjusted for prenatal smoke exposure, maternal Comparative Analysis of Social Mobility in Industrial Nations classification, maternal age at child birth, weight gain in pregnancy, hypertension during pregnancy, diabetes in pregnancy, ethnicity, residential location, birth weight and body mass index.

No PAE is set to reference.

highest OR is seen for children's BMI. The risk of systolic hypertension is increased in children with obesity by OR: 5.09 (CI 95%: 4.15, 6.24).

DISCUSSION

Contrary to our initial hypothesis, this study showed that PAE was associated with reduced risks of systolic and diastolic prehypertension in children and adolescents. For hypertension, we did not find a significant association with PAE. The associations mentioned earlier did not differ according to levels of SES, PSE, or offspring BMI. Stratified analyses showed that the positive association between PAE and prehypertension disappears in children and adolescents with PSE and other weight categories than normal weight.

The unexpected result of an association between PAE and a reduced risk of prehypertension in children and adolescents has not yet been reported in other studies. A possible explanation can be drawn from some animal and human studies that describe how alcohol, by creating inflammatory responses in the body,[14,16] triggers increased expression of nitric oxide synthase[13] causing vasodilation as part of the inflammatory response, through which blood pressure values decrease. On the other hand, in the prenatal period, some animal studies have shown negative effects of PAE on the endothelium as well as on pulse wave velocity, which has been associated with increased blood pressure.[16,36] However, other animal studies have shown that PAE reduced blood pressure in offspring, especially with moderate levels of PAE.[14] Nevertheless, regular high prenatal alcohol consumption has been associated with vasoconstriction and corresponding vascular damage leading to hypertension in offspring.[14] This is also known as the J-shaped influence.[37] In contrast to this, some studies examining alcohol intake

Table 4. Odds ratios (95% confidence interval (CI)) of systolic/diastolic prehypertension/hypertension and PAE, categorized by SES, PSE and BMI

	No PAE *n* = 9,623	Systolic prehypertension			Diastolic prehypertension			Systolic hypertension			Diastolic hypertension		
		PAE *n* = 1,576											
		OR	95% CI	P-value	OR	95% CI	P-value	OR	95% CI	P-value	OR	95% CI	P-value
SES													
High SES	1.00	0.84	0.58, 1.21	0.360	0.68	0.45, 1.04	0.076	0.93	0.59, 1.48	0.763	0.95	0.57, 1.57	0.834
Middle SES	1.00	0.87	0.70, 1,08	0.190	0.84	0.66, 1.06	0.137	0.95	0.72, 1.26	0.735	0.89	0.65, 1.21	0.444
Low SES	1.00	0.70	0.45, 1.08	0.105	0.91	0.58, 1.42	0.679	0.95	0.56, 1.61	0.841	0.94	0.52, 1.70	0.837
PSE													
No prenatal smoke exposure	1.00	0.80	0.66, 0.97	0.024*	0.80	0.65, 0.99	0.037*	0.88	0.68, 1.12	0.295	0.96	0.74, 1.24	0.768
Prenatal smoke exposure (occasional)	1.00	1.03	0.66, 1.59	0.906	1.03	0.64, 1.65	0.899	1.36	0.80, 2.30	0.255	0.66	0.31, 1.40	0.281
Prenatal smoke exposure (regularly)	1.00	0.87	0.42, 1.77	0.693	0.60	0.25, 1.48	0.269	1.25	0.53, 2.96	0.607	0.77	0.26, 2.31	0.643
BMI													
Severely underweight	1.00	1.07	0.23, 5.08	0.930	0.90	0.19, 4.18	0.890	2.02	0.39, 10.49	0.402			
Underweight	1.00	0.53	0.18, 1.53	0.242	0.38	0.12, 1.26	0.114	0.82	0.23, 2.86	0.750	0.22	0.03, 1.68	0.146
Normal weight	1.00	0.85	0.70, 1.05	0.125	0.80	0.64, 0.99	0.038*	0.94	0.72, 1.22	0.634	0.96	0.73, 1.27	0.784
Overweight	1.00	0.90	0.56, 1.44	0.648	1.22	0.73, 2.06	0.446	0.93	0.51, 1.68	0.799	1.17	0.60, 2.28	0.652
Obesity	1.00	0.80	0.47, 1.37	0.417	0.84	0.44, 1.62	0.608	1.07	0.60, 1.91	0.823	0.76	0.34, 1.73	0.512

Abbreviations: BMI, body mass index; PAE, prenatal alcohol exposure; PSE, prenatal smoke exposure; SES, socioeconomic status,

Full model SES: adjusted for PSE, maternal age at child birth, weight gain in pregnancy, hypertension during pregnancy, diabetes in pregnancy, ethnicity, residential location, birth weight, and BMI.

Full model PSE: adjusted for maternal age at child birth, weight gain in pregnancy, hypertension during pregnancy, diabetes in pregnancy, ethnicity, residential location, birth weight, BMI, and SES.

Full model BMI: adjusted for PSE, maternal age at child birth, weight gain in pregnancy, hypertension during pregnancy, diabetes in pregnancy, ethnicity, residential location, birth weight, and SES.

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

and cardiovascular disease in adults have shown an artificial blood pressure lowering effect caused by other underlying diseases.[38]

Furthermore, Amos-Kroohs et al.[18] have suggested that, as a negative consequence of cognitive impairment in cases of FAS, there is a reduction in health-conscious behaviors in these children and adolescents, resulting in an increase in BMI and thus, increasing blood pressure levels.

Contrary to our initial hypothesis, there were no interactions between PAE and SES, PSE, or BMI. Other human studies have reported synergy effects, e.g., between PAE and PSE in relation to congenital heart diseases.[19] To the best of our knowledge, this is the first study on PAE and its effect on blood pressure in children and adolescents and therefore, we cannot compare our interaction analyses to other human studies.

Our subgroup analyses indicated that the positive effect of PAE only occurs when there is no PSE and when the offspring are of normal weight. This suggests that a vasodilatory effect is only present if no other risk factors prevail.

In addition, our study has shown that other lifestyle-related risk factors such as PSE increase the risk of prehypertension, especially in children and adolescents with elevated BMI, who have an increased risk of high blood pressure. This confirms results of previous studies.[3,8,39] More research is needed in this area to further investigate potential interaction effects, specifically related to PAE.

Strengths and limitations

Study findings on blood pressure parameters from this national health monitoring survey with a large dataset are representative for German children and adolescents. In addition, this is the first study investigating the influence of PAE on elevated blood pressure in a human population.

The study findings need to be interpreted with caution. One limitation is that the study has a potential recall bias as some pregnancies were up to 17 years before mothers' participation in the survey. In addition, results are based on data collected from participants that took place at one single survey point sometime during the years 2003–2006. This has relevance for the findings related to blood pressure parameters as, by definition, hypertension should be only diagnosed after testing at 3 different points in time.[2] Also, the prevalence of hypertension has been increasing since 2006 according to recent studies,[3] indicating the urgent necessity for collecting data on blood pressure in future surveys. Furthermore, the presented data analyses indicated a decreased risk of elevated blood pressure in association with PAE, but the dataset did not collect the exact levels and the timing of alcohol consumption during pregnancy. We assumed that predominantly low–to-moderate levels of alcohol were consumed during pregnancy, as only 25 women in our large sample reported regular alcohol consumption during pregnancy. It was not possible to perform risk calculations for this small group. Moreover, data analyses revealed a low Nagelkerkes R^2, suggesting that there are further factors that influence children's and adolescents' levels of blood pressure. These factors may be genetically determined or due to a lack of exercise or related to other unknown factors.[3]

Despite these limitations, findings from this study highlight the importance of blood pressure prevention and screening measures for children and adolescents addressing pre- and postnatal lifestyle-related risk factors.

Study findings on blood pressure parameters from this national health monitoring survey with a large dataset are representative for German children and adolescents.

PAE was associated with reduced risks of systolic and diastolic prehypertension in children and adolescents. Although there were no interactions between PAE and SES, PSE, or BMI, other lifestyle-related risk factors such as PSE increased the risk of prehypertension, especially in children and adolescents with elevated BMI.

SUPPLEMENTARY DATA

Supplementary data are available at *American Journal of Hypertension* online.

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DISCLOSURE

The authors declare that there are no conflicts of interest relating to this article.

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