Pollution Exposure, Child Health and Latent Factors: Evidence for Germany

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Abstract:

This paper examines the impact of outdoor pollution and parental smoking on children's health from birth until the age of three years in Germany. We use representative data from the German Socio-Economic Panel (SOEP), combined with five air pollution levels. These data were provided by the Federal Environment Agency and cover the years 2002 to 2007. Our work makes two important contributions. First, we use European data to replicate and extend an important US study by following the effects of pollution exposure and parental smoking on child health during the first four years of life. For infants, as well as for two- to three-year-olds, we are able to account for time-invariant and unobserved neighborhood and maternal characteristics. Second, instead of relying solely on mean pollution levels, we also calculate latent pollution measures. Our results suggest a significantly negative impact of some pollutants on infant health. High exposure to CO prior to birth causes, on average, a 289 gram lower birth weight. With respect to toddler health, we find that disorders such as bronchitis and respiratory illnesses are affected particularly by O_3 levels.

JEL: I12, Q53, J13

Keywords: pollution exposure, latent factors, child health, early childhood

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1 Introduction

Almost all Western industrialized countries have introduced measures for pollution abatement. These measures are often justified as promoting human health. Although there is still much to learn about pollution and the mechanisms underlying it, past research has argued that its impacts on adult health tend to be long-term and those on child health more short-term. To examine whether this latter assumption holds true, we study the effect of air pollution exposure on child health. The connection between them is of crucial interest due to children's high sensitivity to pollution. In children, the rate of metabolism is higher than in adults, which means that children need, relatively speaking, more energy and oxygen. Children also take in more food per kilogram of body weight and therefore more pollutants. Furthermore, they breathe more per kilogram of body weight, which means that the respiratory tract is more stressed by pollutants. Moreover, whereas adults may experience the onset of disease long after they were first exposed to pollutants, the connection between cause and effect is much closer temporally in children: in the case of infant death, it is immediate. In addition, there is increasing evidence of long-term effects of poor infant health on later health outcomes (Currie 2009; for more recent overview, see Currie 2011).

The economic literature has produced a number of studies focusing on air pollution and child health in the United States (see Section 2). However, there is little evidence from other industrialized countries on different measures of pollution abatement.[1] The German Federal Environment Agency ("Bundesumweltamt") is responsible for pollution measurement. Germany is covered by a network of stations that regularly measure pollution levels. Yet these data have seldom been used to analyze the relationship between pollution and child health. One exception is the German Environmental Survey for Children. In this survey, which is part of a larger study of child health in Germany (Kurth et al. 2008), a special module was added from 2003 to 2006 to measure the influence of environmental factors on child health. Exposure to chemical pollutants, mold spores, and noise was examined in a representative sample of 1,790 children between the ages of 3 and 14. With respect to one aspect of CO indoor pollution intensity, the survey shows that around 50% of the children were living in households with at least one smoker. However, the earlier years of life were not taken into account in this study. For a study focusing on earlier years in the German context, see Lüchinger (2009), who combined data from the Federal Environment Agency with data from birth statistics. However, given the data used, it was not possible to control for a broader set

[1] There are other studies focusing on environmental issues in developing countries. For example, see the study by Kim (2009) who analyzed the impact of rainfall on child health in the first five years of a child's life, also addressing the issue of child mortality.

of child and family characteristics. This was a limitation we have been able to overcome in the present study through the use of representative survey data.

In this study, we use data from the German Socio-Economic Panel (SOEP) combined with data from the German Federal Environment Agency. We make two important contributions to the literature. First, we take a similar approach to Currie et al. (2009), but expand the perspective from the US (New Jersey) to a European context. To enable comparison, we use similar health and pollution data from Germany. In contrast to Currie et al. (2009), we are able to follow the effects of pollution exposure on child health across the first four years of life. We observe infant health outcomes in the first year of life as well as health outcomes at the ages of two to three years. For infants as well as for two- to three-year-olds, we are able to account for time-invariant and unobserved neighborhood and mother-specific characteristics, which is in line with the methodology used by Currie et al. (2009). Second, we calculate different pollution intensity measures. Instead of relying solely on mean pollution levels, we also quantify latent pollution exposure. This allows us to summarize all pollution values into few meaningful values.

2 Background

Several past studies using US data have examined the link between air pollution and infant health. Not all of them focus on causal relationships. Yet the question of causality is of key importance, especially because many of the studies investigating the link between pollution and health have neglected to consider the possibility that pollution exposure is endogenously determined—for example, when individuals make choices to maximize their wellbeing and move to cleaner environments. Parents with high preferences for less polluted air are more likely to move to areas with better air quality and are also more likely to invest more in their child's health. Failing to appropriately account for such actions can yield misleading estimates of the causal effect of pollution on health. This has to be taken into consideration when summarizing the relevant studies.

Chay and Greenstone (2003) use historic data to analyze the implementation of the Clean Air Act of 1970 and the recession of the early 1980s. They take the 1981 to 1982 recession as a "quasi-experiment" to estimate the impact of total suspended particulates (TSPs) on infant mortality. They find that an one percent reduction in TSPs results in a 0.35 percent decline in the infant mortality rate at the county level, implying that 2,500 fewer infants died from 1980–1982 than would have died in the absence of the TSP reductions. Their estimates are stable across a variety of specifications.

Neidell (2004) and Currie and Neidell (2005), using data from California, address the endogeneity issue by assessing the within zip code variation in pollution levels rather than by exploiting quasi-natural experiments. They focus on infant health, including birth weight, gestational age, infant mortality, and asthma. Neidell (2004) estimates the effect of air pollution on child hospitalizations for asthma using naturally occurring seasonal variations in pollution within zip codes. He reports that the effect of pollution is greater for children of lower socio-economic status (SES), indicating that pollution is one potential mechanism by which SES affects health. However, all of these studies find mixed results on the effects of pollution on health at birth.

The study by Currie et al. (2009) for New Jersey makes two improvements on the two studies mentioned above. First, they determine the closest measuring stations to the households using the exact coordinates of the household address instead of the coordinates of the zip code center. Second, in addition to accounting for unobservable heterogeneity of the neighborhood, they also control for unobserved characteristics of the mother. The results confirm that CO has a significant effect on fetal health, birth weight, and infant mortality, even at low levels of pollution. The results are robust against different specifications. A recent study by Lleras-Muney (2010) based on data on US military families uses changes in location due to military personnel transfers (which are not matters of individual choice) to identify the causal impact of pollution on children's respiratory hospitalization. The study is unique in that it covers children from birth to the age of five. The results suggest that only ozone has an adverse effect on the health of military children, although not among infants.

A recent paper by Currie and Walker (2011) analyzes the effects of the introduction of electronic toll collection in two US states on infant health measures such as premature birth and low birth weight. They report that electronic toll collection reduced prematurity and low birth weight among mothers within two kilometers of a toll plaza by 10.8 percent and 11.8 percent, respectively, relative to mothers within two and ten kilometers of a toll plaza. There were no immediate changes in the characteristics of mothers or in housing prices near toll plazas that could explain these changes. The results are robust to many changes in specification and suggest that traffic congestion contributes significantly to poor health among infants. Another study by Currie et al. (2011) on the effect of site cleanups on infant health provides a relevant example of how policy measures affecting the quality of the immediate environment in turn affect infant health.

The only study to date on Germany is that of Lüchinger (2009) mentioned above. The study estimates the effect of sulfur dioxide pollution on infant mortality in Germany from 1985 to 2003. To avoid simultaneity problems, the author exploits the natural experiment created by the mandated desulfurization at power plants, with wind directions dividing counties into treatment and control groups. He found that the observed reduction in pollution implies an annual gain of 850 to 1,600 infant lives. Estimates are robust to controls for economic activity, climate, reunification effects, rural/urban trends, and total suspended particulate pollution and are comparable across subsamples.

In our study, we control for unobservable time-invariant characteristics of the neighborhood and the mother in line with the study by Currie et al. (2009). But contrary to Currie et al. (2009), we employ five different air pollution values, carbon monoxide (CO), ozone (O_3), particulate matter (PM_{10}), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2). For a short summary of how these pollutants could affect child health, see Appendix AI.

Given this rich set of pollution measures, it is questionable which value is the most appropriate to measure health effects. In the literature, it is not clear which pollution value is suitable for describing outliers or the duration of exposure in an appropriate manner. For instance, the study by Currie et al. (2009) finds that the exposure in the last trimester of pregnancy influences birth outcomes significantly negatively, at least for CO, but not in the first or second trimesters. However, the results indicate the multicollinearity of the three mean values (see also algebraic signs in Section 4). Therefore, the problem is how to make use of the variety of measurement values[2] in such a way that no important information is lost by aggregating the measurement values, and at the same time to ensure that the variety of (mean) values does not lead to multicollinearity in the results. For this reason, besides different mean value combinations, we also use latent factors that compress the variety of information into useful values.

3 Data

The main data source used in this study is the German Socio-Economic Panel (SOEP). It is a representative national longitudinal data set for Germany that annually surveys households and all household members aged 16 and above. The SOEP started in 1984 (Wagner et al. 2007).[3] It provides an informative database with a rich set of indicators of both parents' and children's characteristics. Since 2003, the SOEP has collected detailed information on child health through an additional questionnaire given to mothers of very young children. For our analysis, we use data on the

[2] The yearly mean pollution value consists of 17,520 = (2x24x365) single half-hourly pollution values.
[3] See http://www.diw.de/soep for more information on the SOEP.

2002 to 2007 birth cohorts. The SOEP sample design enables us to distinguish children in their first year of life (infants) from children at two to three years of age (toddlers). The sample size for the newborns varies between 1,154 and 1,268, and for the two- to three-year-olds between 629 and 775. The information provided in the SOEP allows us to use the following health measures: weight and length at birth, fetal growth, and any disorders a child develops later (e.g., motor or visual impairments).

In the SOEP data, the child characteristics can be linked with maternal and family characteristics. Here, we observe maternal age, education, and family characteristics. Moreover, we match the paternal information and household-related characteristics (household net income, municipality size, and migration background) to child-related variables.[4] The data also allow us to identify siblings born within a household.

The SOEP data furthermore provide information on the smoking behavior of both parents. We use this as an approximation for CO exposure within the household. However, there are also other sources of indoor air pollution, including pollution from various combustion sources, building materials and furnishings, and products for household cleaning and maintenance. This should be taken into account in the interpretation of our results.

For our analysis, we link the SOEP data with data from the Federal Environment Agency by matching SOEP households with the pollutant measures from the nearest measuring station. Since we know the exact coordinates of these measuring stations as well as the exact coordinates of the center of the zip code area for each SOEP household, it was possible to identify the nearest station (short distance principle) for each year[5] and each household. The distance between the station and the household is often less than a kilometer. In urban areas, the mean distance between the household and the monitor is less than 3 kilometers. In rural areas, the maximum distance between the household and the measuring station is 30 kilometers. However, we make use of the fact that pollution levels in rural areas do not change as much over large distances as they do in urban areas. The regional distribution of the SOEP households and the measuring stations is presented in Fig. A1, Appendix.[6]

[4] This is a dummy variable, which takes the value 1 if the mother or father or both parents have an immigration background and 0 otherwise.
[5] This approach, which we had to use for data security reasons, is not as precise as using the exact "household coordinates."
[6] Since not all the measuring stations in Germany measure all five air pollutants, there are households that have to be assigned to two measuring stations.

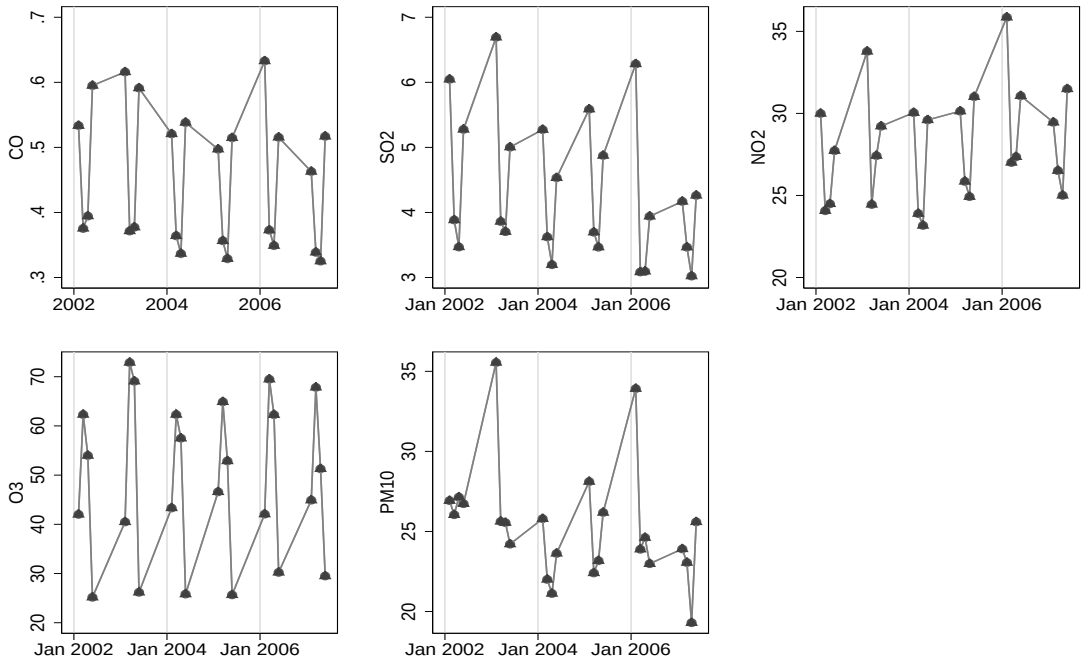
The detailed *data on air pollution levels* cover the years 2002 to 2007. The data are measured at monitors. In Germany, each state has between 11 (Bremen) and 268 (North Rhine-Westphalia) monitors. Altogether, around 500 monitors in the 11 states measure air quality in Germany. The Federal Environment Agency compiles the measurements in a data base and provides information on pollution in Germany, broken down by pollutant source. Most of the measuring stations do not measure all five of the pollutants used in our analysis. In many cases, CO, NO_2, and PM_{10} are measured at the same station, especially in traffic zones.[7] Which stations measure which pollutants depends significantly on the location and the specific problems affecting it. For instance, sites with high traffic are equipped with devices measuring the pollutants typical of this area only, such as PM_{10}, NO_2, and CO. On the other hand, O_3 is not a problem in traffic zones, but it is in urban, sub-urban, and rural areas. For CO, NO_2, SO_2, and O_3, half-hourly values are measured for every station and every day, and hourly values for PM_{10}. In our analysis, we use monthly means for the individual pollutants, which are all calculated according to the guidelines for calculating and analyzing emission data on the basis of the official EU guidelines.[8] Mean values for a week, decade, month, and year are measured on the basis of (half-)hourly means. The EoI (Exchange of Information) guidelines stipulate that hourly means may only be calculated when 75 percent of the data is available, that is, when both half-hourly means are provided. Based on the hourly means, daily means may only be calculated when at least 13 hourly means are available and when, at the same time, no more than six successive hourly means are missing.

The calculated average seasonal variation for the five pollutants for the years 2002 to 2007 can be seen in Fig. 1. The figure shows that for each air pollutant, considerable variation is observable not only within months but also over years.

Fig. 1: Seasonal variation in air pollution (2002-2007)

[7] Detailed information on which stations in Germany measure which pollutants can be found at http://www.env-it.de/stationen/public/open.do (accessed August 10, 2010).

[8] See Council Decision of 27 January 1997 establishing a reciprocal exchange of information and data from networks and individual stations measuring ambient air pollution within the Member States (97/101/EC) (short term: Exchange on Information decision on Air - EoI on Air) and changes made by the Council on 17 October 2011 (2001/752/EC).



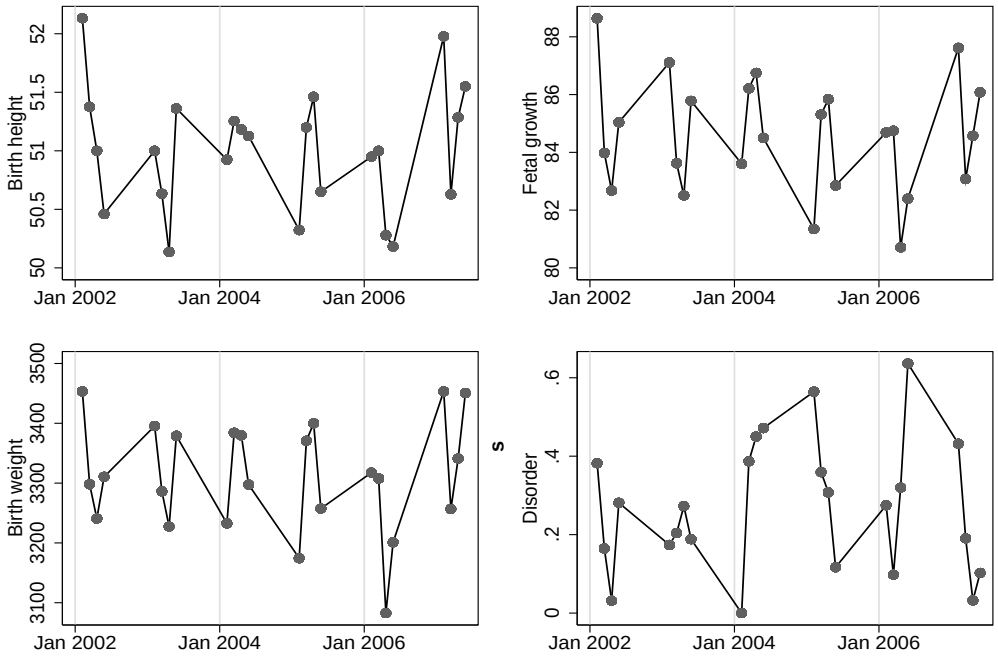
Source: Federal Environment Agency (2002-2007): own calculations.

Having detailed data on pollution levels collected at different points in time could theoretically offer advantages. However, as has been shown by Kvedaras and Rackauskas (2010), variables of different frequencies can cause a problem. In some cases, standard aggregation no longer works. This argument has been widely ignored in the previous literature. We argue that the method of data orthogonalization described above provides a possible means of dealing with this. For more details see section 4.

With respect to *child health*, our data set offers various options. Finding the appropriate measure of child health status is a challenge (see also Case et al. 2002). Health has many dimensions, including mental and physical health, chronic conditions, environmental conditions, nutrition, and injuries. Studies on Western industrialized countries often use low birth weight (LBW) as an indicator of poor health at birth (e.g., Oreopoulos et al., 2008). Alternative measures of child health are bed days and hospitalization episodes. Until a global definition of child health is found, we argue that it is useful to employ a variety of measures as they become available. This is a crucial advantage of the data used here: the SOEP allows us to observe different types of child health measures (for other SOEP-based studies using similar child health measures, see, e.g., Dunkelberg and Spiess, 2009; Cawley and Spiess, 2008; and Coneus and Spiess, 2011). For all age cohorts, we used anthropometric (health) measures such as weight and length of the child at birth. Anthropometric health

measures have the advantage that they are easy to administer and that potential measurement errors are more likely to be random. Weight and length at birth are reported by the mother.

Fig. 2: Seasonal variation in child health outcomes, first year of life (2002-2007)



Source: SOEP 2002-2007: own calculations.

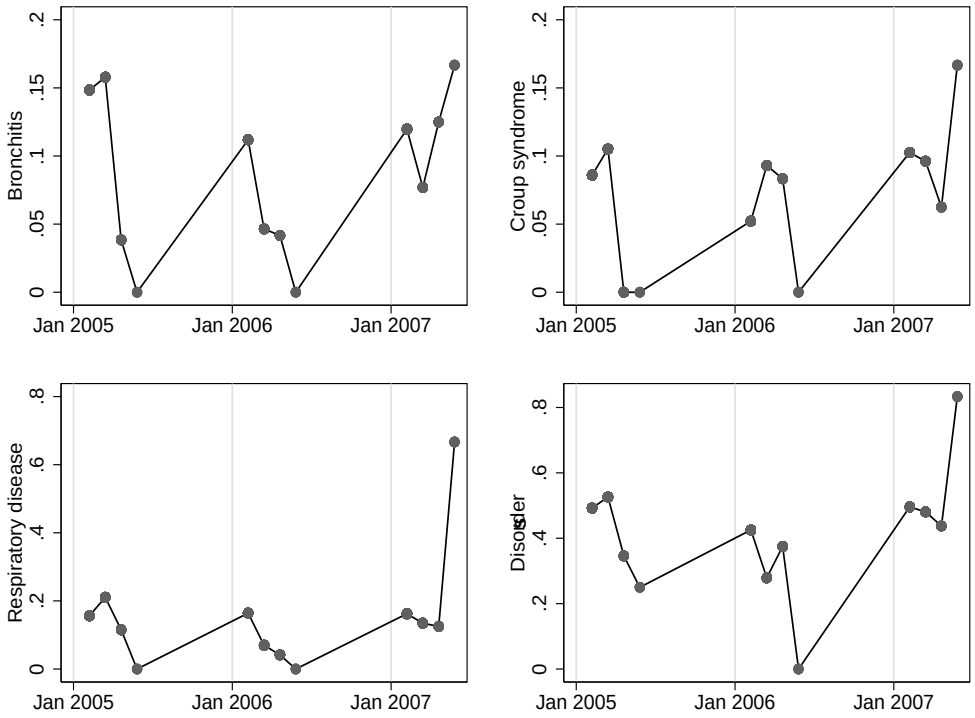
In Germany, preventive medical check-ups are offered to children on a regular basis from birth up to the age of five. They are free of charge. The weight and length of the child at birth (and later on) are measured by experts at each check-up and documented in a medical record booklet that is kept by the family. Ninety-eight percent of SOEP children have had such regular check-ups. In our data, the average weight (length) at birth is 3,327.23 grams (51.15 cm) (see Table A1, Appendix). These results are very similar to the child growth standards of the World Health Organization (WHO).[9] In our analysis, we calculate fetal growth, which is the birth weight divided by gestational age. In the SOEP survey, the mothers are also asked about any disorders (chronic illnesses, neurological disorders, physical disabilities, or other impairments) their child may have at the time of the interview, which takes place in the first year of a child's life. Six percent of the mothers in our sample report a disorder. In Fig. 2, we present the distribution of the child health variables over time. The results show variation between months as well as over time. Moreover, for our younger sample of children, we use other health outcome variables as well, such as preterm birth, gestational week,

[9] For these standards, see http://www.who.int/childgrowth/standards/en/ (October 2010).

head circumference, and LBW. However, we do not find any significant results for the health measures and thus do not discuss or use them further.

Two years after their first interview, mothers are asked again about any disorders their child may have, for example, motor impairments or asthma. We compute a dummy variable for having bronchitis, croup syndrome, respiratory diseases, or other disorders. In the case of toddlers, 46 percent of the mothers reported a disorder. This percentage, which is higher than that for infants, might be related to the fact that different disorders are measured in the two age groups. See Fig. 3 for a distribution of child health at two to three years of age. All health measures show seasonal as well as yearly variation.

Fig. 3: Seasonal variation in child health outcomes at two to three years of age (2005-2007)



Source: SOEP 2002-2007: own calculations.

Table A1 presents summary statistics for infant health, pollution measures, and control variables at birth, in the first year of life, and at the age of two to three years. The mean values for the various air pollutants lie very close together. This applies to children in their first year of life as well as to the two- to three-year-olds. For air pollution caused by smoking in households in which the number of cigarettes smoked was measured, there are barely any differences between the very young children and the two- to three-year-olds. However, the number of mothers and fathers who smoke

seems to be slightly higher among the two- to three-year-olds. Significant variations in the control variables between both samples only occur for the share of single parents. The share for infants is 7 percent; it has more than doubled two to three years later.

4 Conceptual Framework

For both age groups, we estimate the effect of ambient pollutants and parental smoking on child health. It has to be taken into account that the extent of pollution exposure is not endogenous. The decision to live in a less polluted area depends on family-related background variables such as education, immigration background, and income, because living in a better neighborhood often implies higher housing prices. Parents who choose a better neighborhood can also be expected to invest more money in their children's health. As a result, pollution exposure can be expected to be higher where individuals are poorer, and poorer individuals are likely to invest less money in child health. Additionally, an individual's pollution exposure might be correlated with avoidance behavior (for a recent empirical study, see Moretti and Neidell, 2011). Individuals may react to pollution alerts by decreasing the duration and time of day spent outside or by reducing stressful activities such as jogging or other types of sports. If these variables are correlated with a child's pollution exposure, omitting them would lead to biased estimates. Whether the bias is upward or downward is driven by two confounding effects. On the one hand, families with high preferences for cleaner air are more likely to invest in health, which leads to an overestimation of the true impact. On the other hand, pollution levels in urban areas are higher. Frequently, more highly educated individuals live in such areas, and the infrastructure is normally better, which might lead to underestimation of the true impact of pollution on health. However, the variation in pollution exposure in urban areas is quite large, so if highly educated parents decide to live in urban areas, it is likely that they will choose districts with a high quality of living. This might moderate the underestimation of the true impact.

Models for infants. We estimate the impact of pollution exposure on child health in the first year of life using the following health measures: length and weight at birth, fetal growth, and a dummy for a disorder in the first year of life.

Estimation equation for outdoor pollution:

(1a) $$health_{zytij} = \beta_0 P_{zytij} + \beta_1 X_{zytij} + Y_t + u_{zytij}$$

In equation (1a), health denotes our health outcomes in county *z*, in year *y* in the quarter of year *t* of the individual *i* in family *j*. The vector *X* includes observable characteristics of the child, the

mother, the father, and the household (for a detailed description, see below). The coefficient β_0 is our main parameter of interest and measures the impact of air pollution *P* in county *z*, in year *y* in the quarter of year *t* of the individual *i* in family *j* on a child's health *i*. We calculate four different pollution values *P* to estimate the impact of pollution exposure on child health around birth:

a) mean pollution exposure for each pollutant just before birth
b) mean pollution exposure for each pollutant during pregnancy
c) latent mean pollution level (mean by trimester during pregnancy)
d) latent maximum pollution level (maximum by trimester during pregnancy)

The mean pollution exposure just before birth (a) is the average of all (half-)hourly values multiplied by 24 (hours) and 30/31 (the number of days in the relevant period). Since our data allow us to specify the child's exact birth month, we computed the mean value for each of the five pollutants that capture the last monthly pollution intensity during pregnancy. For example, if a child was born in mid-August, our mean value contains all pollution values from mid-July to mid-August. In contrast, the mean pollution exposure during pregnancy is the average of all (half-)hourly values multiplied by 24 (hours per day) and 30.5 (days per month), and 9 months (normal duration of a pregnancy). In (c) and (d), we calculate the mean (maximum) pollution level on the basis of the respective measures by trimester. In doing so, we determine the number of latent factors required to reflect the data. In a factor analysis, we compute the number of eigenvalues of the correlation matrix that are greater than 1 (Kaiser, 1960), which reflects the number of latent factors. In each case, for c) and for d), we identify one latent factor. For further details see Appendix AII.
Altogether, we used four different outdoor pollution measures for each pollutant, (a)-(d), to estimate the impact of pollution exposure on infant health. We do so for several reasons: First, the problem is how to summarize a large number of values without losing important information. Means are one way to cope with this; latent factors are another. Second, given the high correlation of pollution values over time, it might be difficult to include a variety of pollution measure in *one* regression. Thus we use various measures in different regressions. Nevertheless, measures (a) and (b) have the advantage that the coefficients can be interpreted as a change in a natural unit. Measures (c) and (d) are latent factors and thus can be interpreted in the sense that a change in them affects child health: A higher latent factor means more pollution. We use them as an alternative measure of pollution combining various measures into one.

Estimation equation for outdoor pollution and parental smoking exposure:

(1b) $$health_{zytij} = \beta_0 P_{zytij} + \beta_1 X_{zytij} + \beta_2 I_{zyij} + Y_t + u_{zytij}$$

Further we control for parental smoking behavior. We do this as parental smoking exposure is one factor affecting indoor pollution and thus affects the overall air quality of a child (for such an approach see Currie et al. 2009). Equation (1b) includes one latent factor for outdoor pollution *P* during pregnancy and one latent factor for parental smoking exposure *I*, using cigarette smoking as a proxy for CO exposure.

In the estimation equations (1a) and (1b), the different pollution levels are calculated using the nearest monitor to the household in county *z*. β_0 measures the effect of a change in mean pollution levels within *t* while x_{zytil} captures observable characteristics of the child, mother, father, and household, which might be correlated with both pollution exposure and health. These characteristics include the child's sex and birth order; the mother's education, age, immigration background, and single parenthood; and the household's income and municipality. Finally, Y_t includes controls for seasonal changes because these are highly correlated with pollution levels. It includes all quarter and year dummies for our whole sample period.

As mentioned above, this estimation strategy suffers from the fact that unobserved time-invariant characteristics of the area are not taken into account but are potentially correlated with pollution and health. Ignoring this issue would prevent us from capturing the "biological" effect of pollution exposure on child health. To overcome this problem, we estimated the following models:

(2a) $$health_{zytij} = \beta_0 P_{zytij} + \beta_1 X_{zytij} + Y_t + \alpha_{zy} + u_{zytij}$$

(2b) $$health_{zytij} = \beta_0 P_{zytij} + \beta_1 X_{zytij} + \beta_2 I_{zyij} + Y_t + \alpha_{zy} + u_{zytij}$$

Estimation equations (2a) and (2b) include α_{zy}, which is a fixed effect for each year at the county level. Accounting for fixed effects at the county level will capture a large share of potentially unobserved omitted and time-invariant average characteristics of the neighborhood within one season. In this model, we estimate infant health of children living in close proximity to each other and who were born in the same period *t*. Given that parents who are also more likely to invest more resources in the health of their children might adjust their behavior based on pollution alerts by choosing to spend more time indoors or to engage in different outdoor activities, the model presented in equations (2a) and (b) might be still biased. To remove the influences of potentially con-

founding factors resulting from unobserved characteristics (behavior) from the mother, we include a mother fixed effect in model (3a) - (3b).

(3a) $health_{zytij} = \beta_0 P_{zytij} + \beta_1 X_{zytij} + Y_t + \alpha_{zy} + \lambda_j + u_{zytij}$

(3b) $health_{zytij} = \beta_0 P_{zytij} + \beta_1 X_{zytij} + \beta_2 I_{zyij} + Y_t + \alpha_{zy} + \lambda_j + u_{zytij}$

Models (3a) and (3b) control for unobserved time-invariant characteristics of both the neighborhood and the mother. Here, the effect of air pollution on child health in the first year is identified by variation in pollution between siblings in a particular area. A prerequisite for identifying this is that the unobservable fixed effects of the mother do not differ systematically with regard to the children. This assumption may be violated if, for instance, parents systematically reduce the amount of time one child spends outdoors due to a smog alert but do not make any such changes for another child.

Models for toddlers. We also estimate all models presented above for the two- to three-year-olds. As health outcomes, we observe whether the child had bronchitis, croup syndrome, respiratory disease, or other disorders. The age of the children varies between 26-47 months, so we control for age in months in all models. In order to better approximate the consequences of air pollution on the child's health during the first few years of life, we calculate pollution intensities during the entire period from birth (pregnancy) up to age two to three. Overall, β_0 measures five different pollution intensities:

a) mean pollution exposure for each pollutant during the last year
b) mean (monthly) pollution exposure for each pollutant during the interview month
c) three-year mean for each pollutant
d) latent pollution exposure factor during the last year
e) latent pollution exposure factor during the last month

Again, we use several measures. The first three, (a) to (c), are means measured in natural units. The other two, (d) and (e), are latent factors.

For both age groups and each pollutant, we estimate three different models in accordance with the models for infants. The first is an ordinary least squares model, the second includes a fixed effect for the county-year, and the third includes a fixed effect for the county-year and for the family. The

latter is restricted to mothers with at least two children. The standard errors are clustered on the county level.[10]

Moreover, pollution may be systematically related to climate or weather conditions. Only a few US studies, such as Currie and Neidell (2005) and Currie et al. (2009), control for weather data (e.g., temperature). However, as their results show no systematic pattern in this respect, we did not use weather data in our regression.

5 Results

Results for infants. Table 1 presents the estimation results for the first age group for all five pollutants and all specifications. All three models include the variables described in Table A1, but only the various effects of the five air pollution measures on birth length, birth weight, fetal growth, and later disorders are shown.

Table 1 about here.

As indicated in Table 1, CO exposure during pregnancy and just before birth has a significantly negative impact on fetal growth and birth weight in Model 3 (equation 3a). Hence, it becomes apparent that CO impairs the ability of the blood to transport oxygen and thus to supply oxygen to the fetus. An increase in the average CO exposure during the month before birth causes, on average, a 289 gram lower birth weight. Here, the impact on birth weight and fetal growth towards the end of pregnancy appears to be significantly higher than at earlier stages. Taking into consideration the mean value of CO exposure during pregnancy the total impact is less than 200 grams. The effect is significant independent of the pollutant measure; it applies for the means and the latent factors. This outcome is in line with the results reported by Currie et al. (2009), which show that pollution in the last trimester is most important for infant health. For O_3, the effect of exposure appears to be negative throughout the entire pregnancy, not only at the end. This holds for birth length, birth weight, and fetal growth. However, some of the effects are only significant on the 10% level, and not robust with respect to the pollutant measures used. Once the latent measures are used, only the effect on fetal growth stays significant. For a higher exposure to NO_2 and SO_2, we find a negative impact on birth length. Moreover, the higher the amount of SO_2, the higher the probability that the child will have disorders in the first year. Overall, the negative impact of SO_2 on birth length is greater than that of NO_2. Moreover, the results on these two polluntant measures are sensitive to the

[10] For all models with latent factors, the standard errors were bootstrapped with 500 replications.

pollutant measures used. For PM_{10}, we find no impact in most models and specifications that account for both unobservable neighborhood effects and unobservable effects within the family. However, we find a positive mean effect of fine particles at birth on fetal growth and birth weight. The same implausible effect is reported in Currie et al. (2009). A possible explanation for these mixed results could be that fine particles tend to cause long-term respiratory illnesses (cancer, pneumoconiosis), which is certainly harmful for fetuses but cannot easily be identified due to the variation in our model, which is designed to cover the short term. Moreover, there are some correlations in the models that do not control for unobservable effects within the family.

Table 2 about here.

Table 2 shows how the overall air pollution outside of the household impacts child health in the first year when parental smoking is controlled for. In most models, a pattern of ambient air pollution from Table 1 emerges where no impact of parental smoking behavior is observable—with the exception of the negative effects for the PM_{10} models—when controlling for the unobservable neighborhood and family effects. This effect may result from the smaller variation within the family with regard to parental smoking behavior. To obtain further insights, we estimate models covering parental smoking exposure only.[11] Almost all these models show that the mother's smoking has a negative impact on birth outcomes, whereas the father's smoking and the resulting passive smoking by the mother during pregnancy do not seem to be so harmful. However, the smoking intensity and, consequently, one aspect of the air quality in the home also impair fetal growth and reduce the birth weight. But obviously, this effect does not appear in the models with the outdoor pollutants.

In light of these results, it is interesting to see whether outdoor pollution interacts with parental smoking behavior (see, e.g., Currie et al., 2009). Thus we also estimate models including an interaction term. Mostly the interaction terms are not significant. Nevertheless, almost all significant coefficients have the expected sign, which means, for instance, that a higher level of $PM_{10,}$ which accompanies parental smoking behavior, reduces fetal growth or increases the probability of having any disorder (see Table A2).

Results for toddlers. The effects of the five air pollutants on selected health indicators for toddlers are depicted in Table 3. Analogously to the models for infants, in model (1) we present OLS results; in model (2) we control for county fixed effects; and in model (3) we also take family fixed effects

[11] Available from the authors upon request.

into account. In comparison to the results for the younger children, it has to be considered that this sample consists of around 300 observations fewer and that the temporal variation (2005 to 2007) and variation within the family is significantly smaller. For this reason, identifying air pollution effects is particularly difficult in Models 2 and 3 and we therefore do discuss the results of Model 1 as well. In most specifications, O_3 exposure leads to an increased probability of falling ill with bronchitis or respiratory diseases, or of having some impairment. This view is confirmed for some specifications and is partly robust when an area fixed effect and a family fixed effect are taken into account. The latter effect remains significant for the probability of having bronchitis. While the correlations are hardly sensitive to changes in the pollutant measures in the OLS models, they are sensitive, to some extent, to changes in the models controlling for county and family fixed effect. PM_{10} also increases the probability of contracting bronchitis or respiratory diseases. The latter effect even occurs in the models that account for area and family fixed effects and can be measured for latent and non-latent pollutant measures. In contrast, we find mixed results for the effects of CO, SO_2, and NO_2 on toddler health

Table 3 about here.

Table 4 shows the impact of the overall air pollution on child health at the age of two to three years, also controlling for parental smoking behavior. Consistent with the results from Table 3, higher O_3 exposure leads to an increased likelihood of respiratory diseases, bronchitis, or other impairments. But the results are not statistically significant when we control for county and family fixed effects. The models in Table 4 show no effect of parental smoking. If we again only regress on mothers` and fathers` smoking behavior and the control variables the results[12] suggest that parental smoking does not have a significant impact on the health measures at this age, with the exception that the probability of having bronchitis or a disorder increases if the father smokes.

Table 4 about here.

In line with the models for infants, we also estimate models with an interaction effect between outdoor pollution and parental smoking behavior (Table A3). Again, the results do not change significantly. For some pollutants, the interaction effects are significant. For example, a high level of PM_{10} in combination with parental smoking increases the probability of croup syndrome and other disorders of toddlers.

[12] Available from the authors upon request.

6 Conclusion

It has become a generally accepted fact that air pollution should be reduced for numerous reasons, including human health. The health of children is of particular importance here, both because of children's higher short-term vulnerability to pollutants and because early exposure has longer-term impacts on their mental and physical development and skill formation. In recent years, economists have begun to analyze the impacts of air pollution on child health, primarily in the US. This poses various challenges, first and foremost that of finding appropriate health and air pollution measures that make it possible to estimate the causal impact of air pollution accurately. Two further obstacles are the presence of confounding factors brought about through residential sorting and the lack of health measures that capture the range of morbidities purportedly related to pollution.

We cope with the aforementioned problems by utilizing representative German data to analyze the effect of air pollution on child health using county and family fixed effects. We employ different health measures such as anthropometric measures and the occurrence of impairments such as bronchitis that are known to have some correlation with air pollution. A further advantage of our study is the wide range of pollution measures used, including accurate measures for five different pollutants (CO, NO_2, SO_2, O_3, and PM_{10}) collected on a (half-) hourly basis.

Our analysis covers two age groups, infants and toddlers, to provide indications as to which age group shows the more pronounced effects. Moreover, our approach allows us to analyze the effects of different pollutants. It therefore gives further evidence as to which pollutants matter most for child health and which are of minor importance. Apart from the study by Lüchinger (2009), this is the only study to date focusing on a potential causal relationship between pollution and child health in Europe. In general, air pollution is less of a problem in Germany than in the United States, although it remains a major concern in densely populated urban areas.

Our estimation results show that air pollution matters, particularly at birth. CO levels affect fetal growth and birth weight. As traffic is the main cause of CO air pollution, policies and measures making cars more environmentally friendly may therefore lead to improvements in child health. The extreme vulnerability of infants and young children to high CO levels makes such efforts especially important, as even the smallest concentration of this pollutant may damage fetal brain cells.

Furthermore, our estimations show evidence that O_3 levels affect the probability of children having some type of disorder. O_3 is generally a major toxic agent in summer smog. Therefore, infants and toddlers are affected much more than adults by increased O_3 levels because their breathing frequency and thus their demand for oxygen is higher. Furthermore, since the infant immune system is not yet fully developed, areas with a high risk of summer smog may be dangerous for very young children. Similar results are found for the SO_2 level. Oxidation processes of SO_2 lead to acid rain. Again, areas with elevated SO_2 levels pose a health risk for infants. With our older group of children, the two- to three-year-olds, we mainly find effects for O_3 levels, which increase the probability of having bronchitis or respiratory disease. Thus, summer smog might be one cause of these types of impairments.

From a policy perspective, on the one hand, our results confirm the importance of efforts to raise awareness among parents of infants of the negative consequences of smoking on the health and development of their child. Potential activities include public campaigns and consultations with pediatricians and experts. On the other hand, our results underscore the need for further efforts on the regional and national level to reduce CO and O_3 levels in particular. Since these pollutants are higher in cities, environmental policies should focus on reducing these pollutants in these areas in order to improve child health. Here, the potential child health benefits of further reductions in air pollution appear particularly high.

Nevertheless, our study could benefit from further research using even more precise pollution measures—for example, personal air quality monitors that can be worn by study participants. Until such data become available for large representative data sets, however, the findings obtained should be interpreted as conservative estimates that may underestimate the actual effects (see Currie et al. 2009).

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Table 1: Effects of outdoor pollution on child's health at birth and in the first year of life (various model specifications)

	CO			O_3			NO_2			SO_2			PM_{10}		
	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]
Length															
Mean pollution just before birth	-0.47	0.13	-0.09	0.0001	0.01	-0.00	-0.01	-0.01	-0.01	-0.05	-0.03	-0.13***	0.03*	0.03*	0.01
	(0.48)	(0.63)	(0.55)	(0.009)	(0.014)	(0.01)	(0.006)	(0.01)	(0.01)	(0.05)	(0.065)	(0.05)	(0.01)	(0.02)	(0.02)
Mean pollution during pregnancy	-0.38	-0.24	0.17	0.001	0.01	-0.02**	-0.001	-0.01	-0.02**	-0.03	-0.04	-0.11**	0.05***	0.07***	0.02
	(0.43)	(0.61)	(0.51)	(0.011)	(0.01)	(0.01)	(0.007)	(0.01)	(0.01)	(0.037)	(0.05)	(0.04)	(0.018)	(0.027)	(0.02)
latent mean pollution level	-0.06	0.01	0.12	0.06	0.38	-0.16	-0.01	-0.17	-0.26**	-0.08	-0.10	-0.17	0.38**	0.36*	0.01
(basis trimester means)	(0.11)	(0.16)	(0.14)	(0.246)	(0.32)	(0.24)	(0.11)	(0.18)	(0.13)	(0.089)	(0.135)	(0.16)	(0.15)	(0.23)	(0.20)
latent maximum pollution	-0.07	0.01	0.06	0.13	0.38*	-0.10	0.01	-0.16	-0.26*	-0.04	-0.08	-0.17	0.42**	0.49*	0.27
(basis trimester maximum)	(0.11)	(0.16)	(0.14)	(0.21)	(0.29)	(0.21)	(0.11)	(0.18)	(0.13)	(0.11)	(0.149)	(0.18)	(0.18)	(0.28)	(0.23)
Fetal Growth															
Mean pollution just before birth	-0.02	2.72	-7.27***	-0.07**	-0.03	-0.06	0.003	0.002	0.03	-0.20	-0.14	-0.34	0.05	0.09	0.18**
	(1.99)	(3.08)	(2.78)	(0.034)	(0.04)	(0.04)	(0.024)	(0.04)	(0.04)	(0.20)	(0.27)	(0.24)	(0.06)	(0.09)	(0.08)
Mean pollution during pregnancy	0.004	1.31	-4.57*	-0.07*	-0.03	-0.08*	0.009	-0.004	0.01	-0.11	-0.16	-0.18	0.07	0.14	-0.01
	(1.97)	(2.61)	(2.62)	(0.04)	(0.046)	(0.05)	(0.024)	(0.04)	(0.04)	(0.176)	(0.22)	(0.23)	(0.07)	(0.11)	(0.09)
latent mean pollution level	0.32	0.55	-1.34*	-1.73**	-0.65	-2.15*	0.23	0.05	-0.22	-0.07	-0.26	0.16	0.23	0.53	0.63
(basis trimester means)	(0.49)	(0.70)	(0.73)	(0.93)	(1.06)	(1.19)	(0.40)	(0.69)	(0.66)	(0.42)	(0.65)	(0.81)	(0.58)	(0.97)	(0.91)
latent maximum pollution	0.26	0.42	-1.44*	-1.20*	-0.15	-1.92*	0.28	0.11	-0.17	0.02	-0.18	0.14	0.10	0.73	1.38
(basis trimester maximum)	(0.50)	(0.71)	(0.74)	(0.79)	(0.93)	(1.02)	(0.409)	(0.70)	(0.67)	(0.48)	(0.69)	(0.90)	(0.69)	(1.15)	(1.00)
Weight															
Mean pollution just before birth	5.14	103.45	-289.25**	-2.28	-0.20	-2.57	-0.37	-0.49	0.39	-9.24	-4.81	-15.87	2.52	4.85	8.06**
	(84.47)	(117.51)	(112.81)	(1.64)	(1.96)	(1.86)	(1.05)	(1.59)	(1.48)	(9.34)	(11.82)	(10.04)	(2.72)	(3.87)	(3.33)
Mean pollution during pregnancy	-11.72	17.12	-190.11*	-2.25	-0.50	-3.26*	-0.11	-0.59	-0.71	-4.39	-4.82	-5.38	4.28	7.13*	2.64
	(85.53)	(113.01)	(106.31)	(2.04)	(2.14)	(1.91)	(1.09)	(1.65)	(1.57)	(7.88)	(10.15)	(9.48)	(3.28)	(4.73)	(3.94)
latent mean pollution level	8.94	13.42	-54.47*	-57.93	-2.08	-68.78	1.27	-10.12	-23.94	-8.83	-4.09	19.33	23.06	29.98	28.61
(basis trimester means)	(21.43)	(30.59)	(29.53)	(43.62)	(50.20)	(49.67)	(18.09)	(28.16)	(27.02)	(19.69)	(29.68)	(33.81)	(26.34)	(45.11)	(38.01)
latent maximum pollution	8.37	10.23	-62.12**	-34.13	19.92	-53.54	3.63	-8.02	-22.51	-5.42	-1.24	14.51	15.06	35.74	52.48
(basis trimester maximum)	(22.25)	(31.05)	(29.88)	(37.89)	(44.76)	(42.87)	(18.29)	(28.32)	(27.70)	(23.0)	(31.83)	(37.50)	(31.63)	(54.45)	(42.08)
Disorder[1]															
Mean pollution just before birth	2.50	0.40	4.00	- 0.1	-0.1	0.1*	- 0.10	0.10	0.10	0.10	-0.30	-0.30	-0.10	0.10	0.10
	(2.80)	(0.601)	(4.15)	(0.1)	(0.08)	(0.07)	(0.04)	(0.07)	(0.05)	(0.32)	(0.38)	(0.32)	(0.11)	(0.18)	(0.12)
Mean pollution during pregnancy	3.80	3.90	3.90	-0.2***	-0.10**	-0.10**	- 0.10	0.01	0.10	-0.10	-0.20	-0.20	0.10	0.10	0.10
	(2.99)	(5.36)	(4.23)	(0.06)	(0.08)	(0.07)	(0.04)	(0.07)	(0.87)	(0.24)	(0.31)	(0.33)	(0.12)	(0.17)	(0.14)
latent mean pollution level	1.40*	1.50	1.50	-3.1**	-3.2*	-3.20*	-0.10	0.7	0.90	0.10	-0.70	4.0**	0.10	0.8	0.08
(basis trimester means)	(0.77)	(1.34)	(1.07)	(1.29)	(1.87)	(1.73)	(0.68)	(1.29)	(0.88)	(0.64)	(0.91)	(1.70)	(0.93)	(1.25)	(1.18)
latent maximum pollution	1.50*	1.70	1.70	-3.2**	-3.2*	-2.8*	-0.10	0.90	0.90	0.50	-0.30	5.7***	0.3	0.30	0.30
(basis trimester maximum)	(0.78)	(1.35)	(1.09)	(1.15)	(1.70)	(1.47)	(0.69)	(1.30)	(0.88)	(0.79)	(1.06)	(1.91)	(0.10)	(1.44)	(1.36)

Source: SOEP 2002-2007: own calculations.

Notes: All coefficients are from different regressions. Standard errors are in parentheses. *indicates statistical significance at the 10% level, ** at the 5% level, *** at the 1% level. All regressions include indicators of mother's education, age, income, municipality size, immigration background, sex of the child, birth order, month and year dummies. Model [1] OLS, refers to equation (1a), model [2] Area fixed effect, refers to equation (2a) and model [3] Area and family fixed effect, refers to equation (3a). Sample size varies between 1,154 and 1,268 observations. [1]Coefficient and standard errors are multiply with 100.

Table 2: Effects of outdoor pollution and parental smoking on child's health at birth and in the first year of life (various model specifications)

	^{i}CO			$^{i}O_3$			$^{i}NO_2$			$^{i}SO_2$			$^{i}PM_{10}$		
	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]
Length															
outdoor (latent)^{i}	-0.04	0.08	-0.10	-0.30**	-0.18	-0.49	-0.07	-0.25	-0.26*	-0.17	-0.14	-0.34**	0.36**	0.29	-0.25
	(0.12)	(0.18)	(0.15)	(0.148)	(0.19)	(0.30)	(0.11)	(0.18)	(0.14)	(0.11)	(0.16)	(0.16)	(0.16)	(0.24)	(0.21)
smoking (latent)	-0.39**	-0.37*	0.20	0.19	0.40	-0.05	-0.33**	-0.32*	-0.11	-0.31**	-0.21	-0.03	-0.46**	-0.59**	-1.11***
	(0.16)	(0.23)	(0.35)	(0.24)	(0.33)	(0.25)	(0.146)	(0.20)	(0.33)	(0.16)	(0.22)	(0.31)	(0.18)	(0.29)	(0.33)
Fetal growth															
outdoor (latent)^{i}	0.52	0.85	-1.65**	-1.64***	-1.05	-2.16	0.03	-0.15	-0.29	-0.21	-0.42	-0.20	0.02	0.40	0.65
	(0.42)	(0.72)	(0.79)	(0.59)	(0.80)	(1.59)	(0.42)	(0.75)	(0.72)	(0.42)	(0.75)	(0.87)	(0.58)	(0.96)	(0.96)
smoking (latent)	-1.81***	-1.25	-0.46	-1.03	-0.53	-1.82	-1.49**	-1.19	0.40	-1.38**	-0.64	-0.09	-2.34***	-2.24**	-3.82**
	(0.67)	(0.97)	(1.91)	(0.96)	(1.07)	(1.28)	(0.60)	(0.83)	(1.77)	(0.66)	(0.93)	(1.72)	(0.78)	(1.19)	(1.55)
Birth weight															
outdoor (latent)^{i}	19.86	25.08	-77.92**	-68.60***	-46.15	-62.17	-10.21	-19.22	-22.04	-14.65	-6.61	-8.35	11.20	11.17	10.27
	(19.35)	(33.38)	(32.81)	(24.58)	(33.27)	(65.50)	(18.59)	(30.74)	(30.20)	(19.615)	(32.27)	(37.04)	(26.37)	(43.86)	(40.92)
smoking (latent)	-77.13***	-62.54*	23.30	-29.45	12.56	-51.43	-67.86***	-62.37*	51.02	-57.15**	-33.79	35.73	-101.86***	-114.40**	-206.77***
	(27.28)	(40.56)	(77.23)	(44.97)	(52.79)	(53.77)	(24.99)	(34.38)	(72.45)	(28.12)	(38.70)	(71.80)	(31.33)	(49.76)	(65.97)
Disorder[1]															
outdoor (latent)^{i}	1.50*	1.20	1.20	-0.5	-0.1	-1.0	-0.10	1.10	1.1*	0.40	0.10	0.10	0.20	0.60	0.60
	(0.81)	(1.52)	(1.09)	(1.14)	(1.45)	(1.07)	(0.73)	(1.39)	(0.90)	(0.71)	(0.098)	(1.03)	(0.99)	(1.40)	(1.22)
smoking (latent)	0.10	1.40	1.40	-3.20**	-3.40*	-3.40*	0.50	0.90	0.90	0.40	1.1	1.10	0.10	0.31	0.30
	(1.08)	(1.57)	(1.16)	(1.33)	(1.94)	(1.78)	(1.19)	(1.54)	(1.08)	(1.32)	(1.79)	(1.21)	(1.47)	(1.96)	(1.30)

Source: SOEP 2002-2007: own calculations.

Notes: All coefficients are from different regressions. Standard errors are in parentheses. *indicates statistical significance at the 10% level, ** at the 5% level, *** at the 1% level. All regressions include indicators of mother's education, age, income, municipality size, immigration background, sex of the child, birth order, month and year dummies. Model [1] OLS, refers to equation (1b), model [2] Area fixed effect, refers to equation (2b) and model [3] Area and family fixed effect, refers to equation (3b). Sample size varies between 1,154 and 1,268 observations.[1]Coefficients and standard errors are multiply with 100.

Table 3: Effects of outdoor pollution on child's health at two to three years of age (various model specifications, coefficients and standard errors multiplied by 100)

	CO			O_3			NO_2			SO_2			PM_{10}		
	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]
Bronchitis															
mean year pollution	-5.40	-5.80	-8.0	0.20**	0.10	0.30	-0.10	0.10	-0.20	-0.20	-0.50	1.0	-0.40*	-0.40	0.50
	(6.62)	(11.98)	(13.8)	(0.07)	(0.11)	(0.2)	(0.06)	(0.13)	(0.15)	(0.52)	(1.12)	(1.6)	(0.23)	(0.52)	(0.4)
mean pollution at interview	-2.10	-7.60	-5.9	0.1*	0.10	0.20	-0.10	0.10	-0.20	0.10	0.40	0.90	-0.10	-0.10	0.20
	(6.11)	(11.09)	(1.5)	(0.08)	(0.14)	(0.2)	(0.06)	(0.13)	(0.2)	(0.46)	(1.11)	(1.4)	(0.17)	(0.41)	(0.3)
three-year mean	-7.70	-17.4	-21.1	0.20**	0.20	0.70**	0.10	-0.10	-0.30	0.10	0.10	1.40	-0.20	-0.30	-0.10
	(5.96)	(10.72)	(20.0)	(0.11)	(0.27)	(0.3)	(0.08)	(0.19)	(0.2)	(0.08)	(1.27)	(1.6)	(0.26)	(0.34)	(0.4)
latent pollution intensity	-0.80	-1.50	-2.7	2.20**	1.60	1.80	-0.40	0.30	-3.50	-0.50	-0.60	1.80	-2.30	-1.70	5.40
	(1.13)	(2.15)	(2.8)	(1.06)	(1.83)	(2.8)	(1.00)	(2.27)	(2.6)	(1.03)	(2.29)	(3.5)	(1.55)	(3.25)	(3.3)
Latent pollution intensity at interview	-1.10	-1.50	-2.8	2.0**	0.90	2.90	-0.50	0.30	-3.0	-1.10	-1.10	2.70	-2.60*	-2.0	3.80
	(1.23)	(2.17)	(2.5)	(0.9)	(1.51)	(2.8)	(1.00)	(0.28)	(2.7)	(0.96)	(2.31)	(3.3)	(1.52)	(3.17)	(3.1)
Croup syndrome															
mean year pollution	6.50	5.50	-26.0	-0.01	0.10	0.30	-0.10	-0.10	-0.20	-0.10	-0.40	-2.70	-0.10	-0.30	-0.50
	(6.92)	(9.68)	(18.4)	(0.07)	(0.17)	(0.3)	(0.06)	(0.15)	(0.2)	(0.52)	(1.18)	(2.2)	(0.27)	(0.54)	(1.1)
mean pollution at interview	4.50	3.60	-21.0	0.10	0.10	-0.10	0.10	-0.10	-0.20	0.10	0.30	-1.60	-0.20	-0.20	0.70
	(5.66)	(7.79)	(18.0)	(0.98)	(0.13)	(0.3)	(0.06)	(0.13)	(0.2)	(0.49)	(1.07)	(1.9)	(0.22)	(0.41)	(0.8)
three-years mean	4.20	-3.10	-25.0	0.10	0.30	0.30	0.10	-0.10	0.20	0.10	1.70	1.70	-0.20	-0.50	0.80
	(5.59)	(12.19)	(25.0)	(0.08)	(0.31)	(0.4)	(0.10)	(0.22)	(0.3)	(0.08)	(1.43)	(2.2)	(0.23)	(0.53)	(0.8)
latent pollution intensity	6.0***	0.60	-5.20	0.40	1.60	0.10	0.20	-1.60	-2.50	-0.30	-1.30	-10.0*	-1.60	0.60	7.0
	(1.16)	(1.79)	(3.8)	(1.09)	(2.0)	(3.6)	(1.14)	(2.44)	(3.7)	(1.27)	(2.77)	(5.0)	(1.58)	(2.72)	(9.0)
latent pollution intensity at interview	1.00	0.80	-5.0	-0.10	0.30	2.70	-0.20	-1.20	-2.60	-0.30	-1.0	-6.10	-1.10	-0.20	6.0
	(1.33)	(1.69)	(3.5)	(1.0)	(1.51)	(3.7)	(1.11)	(2.58)	(3.8)	(1.09)	(2.50)	(4.8)	(1.73)	(3.14)	(7.3)
Respiratory disease															
mean year pollution	-6.90	-8.0	-3.30	0.30***	0.20	0.20	-0.10	0.10	-0.10	-1.10*	-0.90	-0.40	-0.50	-0.40	1.50*
	(8.51)	(13.91)	(1.9)	(0.10)	(0.18)	(0.3)	(0.07)	(0.15)	(0.2)	(0.63)	(1.20)	(2.2)	(0.31)	(0.57)	(0.8)
mean pollution at interview	-3.10	-12.0	0.017	0.29*	0.18	0.10	-0.10	0.10	-0.10	-0.70	0.10	-0.10	-0.20	-0.10	0.80
	(8.44)	(13.63)	(0.20)	(0.14)	(0.24)	(0.3)	(0.07)	(0.15)	(0.2)	(0.53)	(1.22)	(2.0)	(0.21)	(0.40)	(0.6)
three-year mean	-11.20	-29.0**	-19.1	0.30**	0.20	0.50	-0.10	0.10	-0.20	-0.10	0.40	2.60	-0.20	-0.30	0.30
	(8.19)	(13.68)	(27.0)	(0.15)	(0.35)	(4.4)	(0.09)	(0.21)	(2.6)	(0.09)	(1.56)	(2.2)	(0.33)	(0.72)	(0.7)
latent pollution intensity	-1.20	-3.40	-0.03	3.6***	3.10	-0.30	-0.50	1.50	-2.20	-3.10**	-2.5	-3.70	-2.50	-1.10	0.18***
	(1.73)	(2.83)	(0.039)	(1.38)	(2.38)	(4.1)	(1.20)	(2.62)	(3.8)	(1.27)	(2.69)	(5.0)	(1.94)	(3.57)	(0.06)
latent pollution intensity at interview	-1.40	-2.3	-0.021	3.1**	3.10	2.30	-0.60	1.40	-1.80	-3.0**	-1.70	-0.50	-2.80	-2.0	9.00
	(1.62)	(2.48)	(0.035)	(1.40)	(2.38)	(4.1)	(1.22)	(2.63)	(3.9)	(1.21)	(2.47)	(4.7)	(1.99)	(3.48)	(5.7)
Disorder															
mean year pollution	3.80	-9.70	-16.7	0.40**	0.3	0.7*	0.1	0.20	-0.2	-0.4	-0.7	-0.044	0.50	-0.30	-0.80

	(11.73)	(21.84)	(25.0)	(0.16)	(0.28)	(0.4)	(0.11)	(0.19)	(0.28)	(1.09)	(1.98)	(0.028)	(0.43)	(0.64)	(1.0)
mean pollution at interview	3.0	-14.0	-11.0	0.5***	0.30	0.30	0.10	0.30	-0.10	-1.0	-0.80	0.017	0.20	-0.30	-1.0
	(11.56)	(21.64)	(27.0)	(0.162)	(0.28)	(0.4)	(0.10)	(0.18)	(0.3)	(0.93)	(1.80)	(0.025)	(0.36)	(0.54)	(0.8)
three-year mean	0.10	-25.40	-15.0	0.40*	0.40	0.60	0.10	0.10	0.70**	0.10	0.40	0.006	0.30	-0.30	2.0**
	(13.23)	(23.0)	(36.0)	(0.2)	(0.4)	(0.6)	(0.14)	(0.26)	(0.4)	(0.14)	(2.36)	(0.03)	(0.38)	(0.70)	(0.8)
latent pollution intensity	1.80	-0.2	-0.6	6.6***	6.20*	6.0	1.50	4.0	-0.4	-1.0	-2.2	0.085	0.60	-2.0	-12.0
	(2.29)	(4.69)	(5.7)	(1.97)	(3.46)	(5.5)	(1.82)	(3.38)	(4.7)	(2.32)	(4.47)	(0.06)	(2.63)	(4.05)	(8.5)
latent pollution intensity at interview	1.60	-0.70	-2.0	4.90**	2.20	8.0	1.70	4.40	-2.0	-1.0	-1.50	0.08	2.40	-0.90	0.80
	(2.33)	(4.38)	(5.0)	(2.14)	(3.68)	(5.3)	(1.88)	(3.41)	(4.6)	(2.30)	(4.17)	(0.059)	(2.70)	(3.90)	(6.8)

Source: SOEP 2002-2007: own calculations.

Notes: All coefficients are from different regressions. Standard errors are in parentheses. *indicates statistical significance at the 10% level, ** at the 5% level, *** at the 1% level. All regressions include indicators for mother's education, age, income, municipality size, immigration background, sex of the child, birth order, birth weight, child's age in months, month and year dummies. Model [1] OLS, refers to equation (1a), model [2] Area fixed effect, refers to equation (2a) and model [3] Area and family fixed effect, refers to equation (3a). Sample size varies between 629 and 775 observations.

Table 4: Effects of outdoor pollution and parental smoking on child's health at two to three years of age (various model specifications, coefficients and standard errors multiplied by 100)

	CO			O_3			NO_2			SO_2			PM_{10}		
	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]
Bronchitis															
outdoor (latent)	-0.60	-1.00	-0.40	2.30**	1.50	1.40	-0.20	0.60	-3.20	-0.20	-0.30	4.80	-2.70	-2.8	6.0*
	(1.13)	(2.16)	(2.48)	(1.08)	(1.86)	(2.93)	(1.04)	(2.31)	(2.51)	(1.08)	(2.00)	(3.17)	(1.7)	(3.63)	(3.56)
smoking (latent)	-0.50	0.90	-2.0	0.30	-2.20	-1.80	0.30	-2.0	-1.40	-0.30	-2.0	-1.90	1.70	-2.0	12.0
	(2.12)	(3.63)	(9.22)	(1.82)	(3.12)	(7.97)	(1.92)	(3.32)	(7.28)	(2.06)	(3.46)	(10.41)	(2.81)	(5.23)	(8.15)
Croup syndrome															
outdoor (latent)	0.50	1.20	-4.60	0.50	2.0	-2.0	-0.20	-1.60	-2.10	-0.50	-1.40	-10.0**	-2.20	-0.40	5.60
	(1.15)	(1.83)	(4.00)	(1.18)	(1.96)	(3.65)	(1.11)	(2.56)	(3.74)	(1.02)	(2.52)	(4.61)	(1.60)	(2.61)	(9.37)
smoking (latent)	-0.50	0.80	-11.7	0.50	0.10	-0.70	1.10	-0.10	-4.20	0.60	-0.20	-9.0	-0.20	1.50	-5.70
	(1.67)	(2.59)	(14.84)	(1.63)	(2.14)	(1.00)	(1.71)	(2.42)	(10.0)	(1.73)	(2.56)	(15.16)	(2.23)	(3.50)	(21.49)
Respiratory disease															
outdoor (latent)	-1.10	-3.10	-1.30	3.5**	2.40	-0.60	-0.20	1.60	-1.70	-2.70**	-2.0	-0.50	-3.0	-2.0	19.0***
	(1.71)	(3.02)	(3.79)	(1.42)	(2.42)	(4.27)	(1.22)	(2.67)	(3.78)	(1.30)	(2.55)	(4.71)	(2.01)	(4.01)	(6.75)
smoking (latent)	-1.20	-0.30	-3.80	-0.90	-2.30	-4.70	-0.60	-1.70	-5.70	-1.80	-2.60	-0.90	0.10	-0.70	1.80
	(2.23)	(4.17)	(14.07)	(1.39)	(3.21)	(11.60)	(2.08)	(3.65)	(10.97)	(2.24)	(3.95)	(15.49)	(3.02)	(5.32)	(15.48)
Disorder															
outdoor (latent)	1.80	-0.10	-0.60	7.0***	6.0*	2.00	1.20	3.50	-2.10	-1.10	-1.90	9.0	0.20	-3.0	-1.10
	(2.28)	(4.86)	(6.0)	(2.03)	(3.59)	(5.59)	(1.87)	(3.51)	(4.89)	(2.23)	(4.25)	(5.98)	(2.73)	(4.14)	(9.21)
smoking (latent)	-1.10	-0.10	0.90	-1.20	0.30	19.0	-0.10	1.80	2.10	0.40	2.40	6.0	4.30	12.0*	14.0
	(3.16)	(5.58)	(22.65)	(2.90)	(5.18)	(15.15)	(1.99)	(5.11)	(14.19)	(3.03)	(5.33)	(19.68)	(4.68)	(6.38)	(21.10)

Source: SOEP 2002-2007: own calculations.

Notes: All coefficients are from different regressions. Standard errors are in parentheses. *indicates statistical significance at the 10% level, ** at the 5% level, *** at the 1% level. All regressions include indicators of mother's education, age, income, municipality size, immigration background, sex of the child, birth order, birth weight, child's age in months, month and year dummies. Model [1] OLS, refers to equation (1b), model [2] Area fixed effect, refers to equation (2b) and model [3] Area and family fixed effect, refers to equation (3b). Sample size varies between 629 and 775 observations.

Appendix

A I. Mechanisms by which pollutants affect child health

CO is a colorless, odorless, and flavorless gas. It is contained in the fumes of motor vehicles and emerges when heating water, heating with coal, and smoking cigarettes. Automobile traffic is the main cause of CO pollution. CO impairs the intake of oxygen and even in small quantities has impacts on the central nervous system. CO is transferred from the lungs to the blood and attaches itself to the hemoglobin of the erythrocytes or red blood cells. The attachment of CO to these cells is 200 times stronger than that of oxygen. Consequently, CO paralyzes the erythrocytes and prevents them from taking in oxygen, which is crucial for the functioning of the organs, particularly the heart, brain, and muscles. The performance of the heart decreases as the risk to blood vessels increases. This impairs the oxygen supply during pregnancy. Infants and children are particularly at risk, as even the smallest concentration may damage fetal brain cells.[13] The risks are especially high for children whose mothers smoke. As of January 1, 2005, the target value for CO levels is 10 mg/m^3 on eight-hour average.

The colorless and poisonous gas **O_3** is one of the most important trace gases in the atmosphere. It is generated from precursor pollutants (nitrogen oxides and volatile organic compounds) with intensive isolation via photochemical processes. O_3 is considered to be the toxic substance of summer smog. Increased O_3 concentration can lead to impairments of the lung function or lung diseases in humans because it penetrates the respiratory tract. Ten percent of humans are oversensitive to O_3, among them children, allergy sufferers, and asthmatics. High O_3 levels damage the mucous membranes of the airways in particular. Therefore, children and toddlers are affected by raised O_3 levels to a much greater extent because their breathing frequency is higher and they have a higher demand for oxygen than adults. Furthermore, the infantile immune system is not yet fully developed. For O_3 concentration, there is an information threshold of 180 $\mu g/m^3$ and an alarm threshold of 240 $\mu g/m^3$. For the protection of human health, a maximum eight-hour value of 120 $\mu g/m^3$ has been determined as a target value for 2010. It may only exceed this value on 25 days a calendar year at the most.

The main reasons for the formation of **NO_2**, as with CO and PM_{10}, are processes of combustions in industry and power generation plants and traffic. In combination with hydrocarbons, nitrogen oxides are responsible for the aestival formation of O_3. In the winter half of the year,

[13] See http://www.jameda.de/blog/hebammen/kohlenmonoxid-laesst-saeuglinge-und-kinder-bereits-beigeringster-konzentration-empfindlicher-fuer-krankheiten-werden/ (accessed August 12, 2010).

ammonium nitrate particles emerge and contribute to the fine particle pollution. In the outside air, NO_2 always occurs in combination with other substances. The effect is probably not solely caused by NO_2 but by its persistent co-occurrence with other substances, especially fine particles from traffic. It harms the mucous membranes of the airway and impairs the respiratory function. The one-hour threshold value for NO_2 has been set to 200 µg/m^3 (by 2010, plus an annually decreasing tolerance margin), which must not exceed this value more than 18 times during a calendar year. The threshold value for a year amounts to 40 µg/m^3 (by 2010, also with an annually decreasing tolerance margin).[14]

SO_2 is a colorless gas with a pungent smell; it is water-soluble and highly toxic. It emerges especially when burning fossil energy carriers: coal and oil. Oxidation processes lead to acid rain. Sulfate particles, emerging in the atmosphere from SO_2, contribute to the pollution with fine particles (PM_{10}). Even a small concentration of 0.04% may lead to severe poison phenomena such as corneal haze, breathlessness, and inflammations of the respiratory tract; higher concentrations may be fatal. In humans, SO_2 causes impairments of the respiratory tract, especially in combination with dust: it irritates the mucous membranes, which may lead to tissue mutation of the upper respiratory tract and higher infection sensitivity. The one-hour threshold value was set to 350 µg/m^3 on January 1, 2005, and must not exceed this value more than 24 times a year. The daily threshold value of 125 µg/m^3 may not be exceeded more than three times a calendar year.[15]

PM_{10} describes the mass of all particles included in the total of dust, the aerodynamic diameter of which is smaller than 10 µm. It may be of natural origin (for example, as a result of soil erosion) or be caused by human action. It emerges from energy supply or industry plants and also in metal and steel processing. In conurbations, traffic is the main reason for fine particles. Airflow transports these fine particles over long distances. Fine particles are, in contrast to all other pollutants, from the smallest unit on, always harmful to health, whereas for other pollutants, certain threshold values have to be passed in order to induce an impact dangerous to health. When there is a high concentration of fine particles, respiratory illnesses or cardiovascular diseases as well as illnesses of the immune system are likely to increase. Individuals with pre-existing diseases are especially prone. Studies have shown a measurable decrease in life expectancy. The health risk is dependent in particular on how deep the particles enter the respiratory tract and how long they remain there. Smaller particles are even more harmful as

[14] To assess the mass concentration of NO_2 and nitrogen oxide, the chemiluminescence procedure is applied.
[15] To assess the concentration of SO_2, the UV fluorescence procedure is applied.

they can enter the bloodstream. Heavy metals or carcinogenic hydrocarbons (PAK) may lie on the surface. Increased stress during pregnancy may lead to alterations in the breathing frequency of newborns and lead to respiratory inflammations. New threshold values for fine particles (PM_{10}) were introduced on January 1, 2005. The daily threshold value is set at 50 $\mu g/m^3$ and must not exceed this value more than 35 times a year. As there is less air exchange in the wintertime, values exceeding the threshold occur more frequently then.

A II. The calculation of latent pollution factors

We apply factor analysis to each pollutant as one means of utilizing a broad set of measures at different points in time, and to overcome the problem of multicollinearity. In doing so, we reduce the heterogeneity of variables to a minimum number of factors. Such an approach has been used in other fields with high frequency data; for one application with temperature data, see Trendafilov and Unkel (2011).

First of all, we need to determine the number of latent factors required to reflect the data. It is obvious that too few factors may not be able to capture the all information in the pollution data, while too many factors may be highly correlated with each other and not solve the problem of multicollinearity. To identity the optimal number of latent factors, we compute the number of eigenvalues of the correlation matrix that are greater than one (Kaiser, 1960).

We apply this method to the models for infants and toddlers:

For *infants*, we estimate health models with two different latent variables:

a) latent mean pollution level (mean by trimester of pregnancy)
b) latent maximum pollution level (maximum by trimester of pregnancy)

For a) and b) we calculate the mean (maximum) pollution value for each trimester. For all three means, we apply factor analysis and determine the number of eigenvalues greater than one. In this case, the number is one. We include the latent factor in the estimation equation and bootstrap the standard errors for the latent factor. We apply this method in a similar way to the latent maximum pollution level. In both cases, we do not have to employ the orthomax rotation, because the number of latent factor for each model is one. We calculate various factor models, but in most cases we have only one latent factor.

For *toddlers*, we calculate two different latent variables as well:

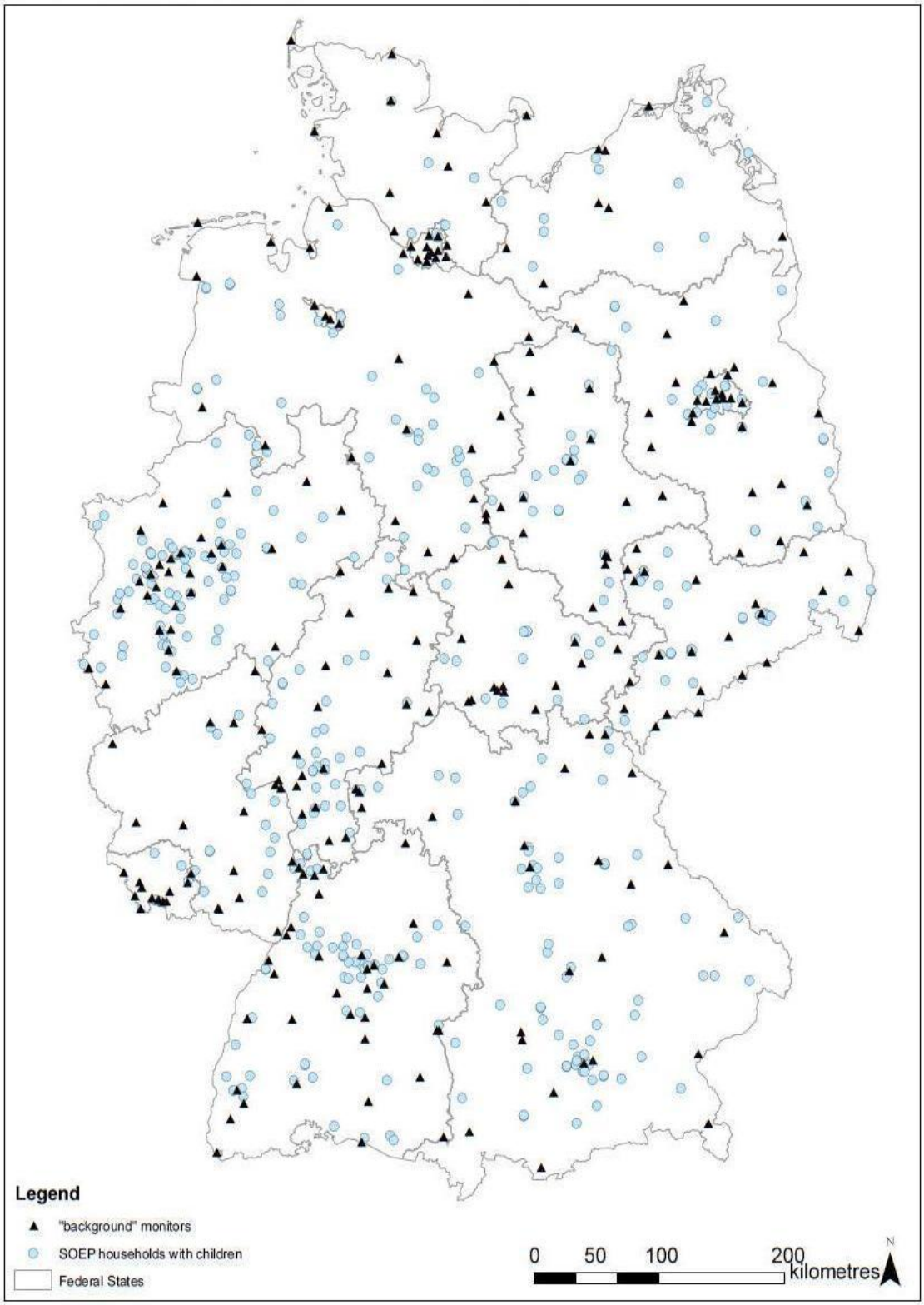
a) latent pollution exposure factor during the last year
b) latent pollution exposure factor during the last month

In a), we use all 12 monthly mean values for each pollutant separately during the last year. Since the resulting number of eigenvalues is one, we have identified one latent factor. For model b) we use all weekly mean values of the last month to calculate the number of eigen-

values. Both factors a) and b) are included in our estimation regression and standard errors are bootstrapped.

A III. Other figures and tables

Fig. A1: Distance of SOEP households with children to "background" monitors



Note: Location of air monitors in Germany: own calculations.

Table A1: Summary statistics

	Infants up to the age of one			Two- to three-year-olds	
	mean	std.dev.		mean	std.dev.
Child health outcomes					
Birth length (in cm)	51.15	(3.06)	Bronchitis (yes=1)	0.10	(0.29)
Fetal growth (grams/week)	84.98	(12.98)	Croup syndrome (yes=1)	0.08	(0.28)
Birth weight (in grams)	3327.23	(579.97)	Respiratory disease (yes=1)	0.14	(0.35)
Disorder (yes=1)	0.06	(0.24)	Disorder (yes=1)	0.46	(0.50)
Pollutants (outdoor) (in $\mu g/m^{3)2}$					
CO (daily mean during birth month)	0.46	(0.25)	CO (daily mean during interview month)	0.48	(0.21)
O_3 (daily mean during birth month)	45.97	(20.17)	O_3 (daily mean during interview month)	52.81	(16.24)
NO_2 (daily mean during birth month)	31.72	(16.69)	NO_2 (daily mean during interview month)	30.43	(17.98)
SO_2 (daily mean during birth month)	4.70	(2.85)	SO_2 (daily mean during interview month)	4.61	(2.31)
PM_{10} (daily mean during birth month)	26.84	(9.25)	PM_{10} (daily mean during interview month)	27.77	(7.80)
CO (monthly means last year before birth)	0.46	(0.22)	CO (monthly mean last year before interview)	0.46	(0.18)
O_3 (monthly means last year before birth)	46.17	(12.62)	O_3 (monthly mean last year before interview)	48.47	(13.08)
NO_2 (monthly means last year before birth)	32.06	(15.23)	NO_2 (monthly mean last year before interview)	29.19	(16.80)
SO_2 (monthly means last year before birth)	4.82	(2.41)	SO_2 (monthly mean last year before interview)	4.27	(1.86)
PM_{10} (monthly last year before birth)	27.04	(6.35)	PM_{10} (monthly mean last year before interview)	25.39	(5.65)
			CO (yearly mean last 3 years before interview)	0.49	(0.19)
			O_3 (yearly mean last 3 years before interview)	52.54	(13.90)
			NO_2 (yearly mean last 3 years before interview)	31.14	(13.29)
			SO_2 (yearly mean last 3 years before interview)	4.92	(1.93)
			PM_{10} (yearly mean last 3 years before interview)	29.56	(7.11)
Parental smoking exposure					
Mother smokes (yes=1)	0.21	(0.41)	Mother smokes (yes=1)	0.26	(0.44)
Father smokes (yes=1)	0.31	(0.46)	Father smokes (yes=1)	0.49	(0.50)
Number of cigarettes smoked	6.39	(11.47)	Number of cigarettes smoked	6.20	(9.36)
Control variables					
Mother's education (low level)	0.17	(0.38)	Mother's education (low level)	0.14	(0.35)
Mother's education (medium level)	0.61	(0.49)	Mother's education (medium level)	0.63	(0.48)
Mother's education (high level)	0.22	(0.42)	Mother's education (high level)	0.23	(0.42)
Mother's age (in years)	31.20	(5.44)	Mother's age (in years)	33.34	(3.95)
Immigration background (yes=1)	0.14	(0.35)	Immigration background (yes=1)	0.12	(0.32)
Net monthly household income (1,000 euros)	2.39	(1.69)	Net monthly household income (1,000 euros)	2.50	(1.91)
Single household (yes=1)	0.07	(0.25)	Single household (yes=1)	0.15	(0.36)
Municipality size[1]	3.79	(1.95)	Municipality size [1]	3.74	(0.32)
Firstborn (yes=1)	0.43	(0.50)	Firstborn (yes=1)	0.45	(0.50)
Girl (yes=1)	0.51	(0.50)	Girl (yes=1)	0.51	(0.50)
Observations (siblings)	1, 155- 1,268			629-775	

Source: SOEP 2002-2007: own calculations.
Note: [1]Municipality size varies between 1 < 2000; 2 = 2000-5000; 3= 5000-20000; 4= 20000-50000; 5=50000-100000; 6 = 1000000-500000; 7>500000. [2]All mean pollution values are calculated according to the EoL.

Table A2: Effects of outdoor pollution and parental smoking on child's health in the first year of life -including interaction terms-

	^{i}CO			$^{i}O_3$			$^{i}NO_2$			$^{i}SO_2$			$^{i}PM_{10}$		
	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]	[1]	[2]	[3]
Height															
Outdoor (latent)[i]	-0.03	0.09	0.043	-0.33**	-0.20	-0.73***	-0.07	-0.24	-0.31**	-0.16	-0.14	-0.31*	0.37**	0.30	-0.41**
	(0.12)	(0.19)	(0.16)	(0.14)	(0.20)	(0.024)	(0.01)	(0.18)	(0.14)	(0.11)	(0.17)	(0.17)	(0.16)	(0.24)	(0.18)
smoking (latent)	-0.37**	-0.36	-0.73**	0.25	0.45	0.44*	-0.33**	-0.31	-0.69***	-0.32**	-0.22	-0.52*	-0.46**	-0.57**	-0.81***
	(0.15)	(0.22)	(0.29)	(0.24)	(0.33)	(0.255)	(0.14)	(0.20)	(0.23)	(0.16)	(0.24)	(0.28)	(0.18)	(0.28)	(0.24)
interaction term	-0.09	-0.09	0.136	-0.18	-0.16	-0.04	0.02	-0.05	0.445***	0.04	0.03	0.20	-0.09	-0.14	0.24
	(0.18)	(0.28)	(0.20)	(0.15)	(0.23)	(0.26)	(0.16)	(0.23)	(0.12)	(0.086)	(0.11)	(0.18)	(0.20)	(0.29)	(0.17)
Fetal growth															
Outdoor (latent)[i]	0.62*	1.04	-1.45*	-1.67***	-1.20	-3.50***	0.03	-0.14	0.20	-0.18	-0.38	0.174	0.06	0.45	-1.01
	(0.43)	(0.78)	(0.78)	(0.57)	(0.80)	(1.176)	(0.42)	(0.75)	(0.71)	(0.43)	(0.76)	(0.82)	(0.57)	(0.96)	(0.89)
smoking (latent)	-1.71***	-1.19	-2.69*	-0.96	-0.29	-0.98	-1.49**	-1.18	-3.40***	-1.44**	-0.72	-3.08**	-2.30***	-2.17*	-4.65***
	(0.66)	(0.95)	(1.45)	(1.00)	(1.13)	(1.22)	(0.60)	(0.81)	(1.18)	(0.68)	(0.97)	(1.41)	(0.73)	(1.17)	(1.17)
interaction term	-1.22**	-1.42	-0.007	-0.21	-0.77	1.24	-0.03	-0.07	0.39	0.22	0.22	0.12	-0.50	-0.61	-1.54*
	(0.62)	(0.96)	(0.96)	(0.68)	(0.97)	(1.25)	(0.65)	(0.95)	(0.64)	(0.36)	(0.48)	(0.88)	(0.83)	(1.28)	(0.83)
Birth weight															
Outdoor (latent)[i]	23.63	33.37	-68.56**	-73.45***	-53.33	-142.1***	-10.08	-18.02	-6.65	-13.52	-4.77	-2.65	12.85	14.16	-44.95
	(19.55)	(35.76)	(32.72)	(23.39)	(33.37)	(49.78)	(18.75)	(31.40)	(30.02)	(19.58)	(32.79)	(34.97)	(25.75)	(43.56)	(37.62)
smoking (latent)	-73.51***	-60.57	-99.47	-20.32	24.78	-4.34	-67.64***	-60.75*	-134.8***	-58.90**	-36.98	-110.69*	-99.47***	-109.97**	-233.16***
	(26.49)	(39.32)	(60.34)	(46.45)	(53.62)	(51.83)	(24.93)	(33.59)	(49.51)	(29.04)	(40.72)	(59.77)	(30.91)	(48.21)	(49.70)
interaction term	-45.18*	-60.27	-15.42	-28.98	-38.83	39.56	-10.46	-22.85	5.55	7.11	9.33	22.63	-24.91	-38.09	-55.01
	(29.12)	(45.08)	(40.20)	(29.34)	(40.92)	(53.10)	(27.41)	(40.40)	(26.78)	(15.69)	(20.05)	(37.66)	(39.45)	(58.07)	(34.84)
Disorder[1]															
Outdoor (latent)[i]	1.41*	0.84	1.72	-0.20	0.37	6.5***	-0.08	1.30	2.45	0.42	0.18	2.61	0.31	0.54	-0.1
	(0.82)	(1.52)	(1.64)	(1.1)	(1.54)	(2.51)	(0.7)	(1.40)	(1.50)	(0.70)	(1.2)	(1.79)	(0.16)	(1.2)	(2.4)
smoking (latent)	-2.1	1.40	2.40	-4.21***	-4.4**	-0.60	0.51	0.81	6.7***	0.41	1.0	1.92	0.10	0.10	4.0
	(1.10)	(1.61)	(3.1)	(1.50)	(2.1)	(2.62)	(1.1)	(1.52)	(2.0)	(1.0)	(2.5)	(3.03)	(2.10)	(1.97)	(2.61)
interaction term	1.41	2.85	3.20	2.0*	2.34	1.21	2.2	1.78	-1.66	1.00	-0.9	-2.71	-0.60	1.68	3.39*
	(1.2)	(2.1)	(2.0)	(1.6)	(1.67)	(2.73)	(1.34)	(1.79)	(1.30)	(0.70)	(1.0)	(2.4)	(2.8)	(1.71)	(1.82)

Source: SOEP 2002-2007: own calculations.

Notes: All coefficients are from different regressions. Standard errors are in parenthesis. *indicates statistical significance at the 10% level, ** at the 5% level, *** at the 1% level. All regressions include indicators of mother's education, age, income, municipality size, immigration background, sex of the child, birth order, months and year dummies. Model [1] OLS, refers to equation (1b), model [2]. Area fixed effect, refers to equation (2b) and model [3] Area and family fixed effect, refers to equation (3b). Sample size varies between 1,154 and 1,268 observations. [1]Coefficient and standard errors are multiply with 100.

Table A3: Effects of outdoor pollution and parental smoking on child's health at two to three years of age -including interaction terms (various model specifications, coefficients and standard errors multiplied by 100).

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	CO			O_3			NO_2			SO_2			PM_{10}		
	[1]	**[2]**	**[3]**	**[1]**	**[2]**	**[3]**	**[1]**	**[2]**	**[3]**	**[1]**	**[2]**	**[3]**	**[1]**	**[2]**	**[3]**
Bronchitis															
outdoor (latent)	-0.61	-1.29	-1.0	2.4**	1.1	1.10	-0.31	0.81	0.81	-0.10	-0.13	-0.11	-3.10	-2.81	-2.81
	(1.20)	(2.5)	(2.1)	(1.1)	(2.0)	(1.91)	(1.5)	(2.33)	(2.3)	(1.4)	(2.6)	(1.9)	(2.01)	(3.61)	(2.62)
smoking (latent)	-0.20	0.91	0.94	0.82	-1.6	-1.61	0.72	-1.5	-1.57	0.22	-1.50	-1.5	2.3	-1.52	-1.50
	(2.3)	(3.69)	(2.31)	(2.04)	(3.6)	(2.14)	(2.2)	(3.8)	(2.21)	(2.0)	(3.51)	(2.32)	(3.41)	(5.3)	(3.12)
Interaction term	0.14	-1.87	-1.83	-2.0	-3.2	-3.2	0.30	-1.4	-1.1	1.30	1.32	1.30	2.34	1.11	1.3
	(2.0)	(3.10)	(2.4)	(2.0)	(2.40)	(2.7)	(2.4)	(3.21)	(2.5)	(1.60)	(2.60)	(2.28)	(2.0)	(3.2)	(2.6)
Croup syndrome															
outdoor (latent)	0.54	1.11	1.1	0.20	1.7	1.71	-0.30	-1.72	-1.77	-0.53	-1.42	-1.47	-2.41	-0.011	-1.11
	(1.21)	(2.0)	(1.92)	(1.26)	(1.8)	(1.61)	(1.9)	(2.61)	(1.62)	(1.20)	(2.63)	(1.77)	1.61)	(0.025)	(2.41)
smoking (latent)	-0.63	0.60	0.63	0.62	0.1	0.1	1.22	0.11	0.10	0.61	-0.11	-0.11	-0.41	0.014	1.41
	(1.62)	(2.54)	(2.0)	(1.61)	(2.30)	(1.9)	(1.78)	(2.31)	(2.05)	(1.71)	(2.51)	(2.13)	(2.2)	(0.034)	(2.8)
Interaction term	-0.23	-1.41	-1.41	-3.2**	-3.5*	-3.5*	1.36	0.50	0.57	-0.11	1.29	1.26	2.34	0.041	4.11*
	(1.61)	(4.3)	(1.91)	(1.4)	(2.0)	(1.9)	(1.91)	(2.85)	(1.78)	(1.8)	(2.91)	(1.98)	(1.5)	(0.025)	(2.30)
Respiratory disease															
outdoor (latent)	-1.0	-3.43	-3.41	3.41**	2.2	2.3	-0.38	2.0	2.4	-2.6**	-1.60	-1.62	-3.0	-2.11	-2.11
	(1.71)	(2.92)	(2.4)	(1.4)	(2.6)	(2).0	(1.2)	(2.71)	(2.11)	(1.18)	(2.6)	(2.22)	(2.11)	(4.3)	(3.2)
smoking (latent)	-0.60	-0.11	-0.13	-0.10	-1.4	-1.41	0.11	-0.98	-0.96	-1.0	-1.7	-1.79	0.61	0.10	0.10
	(2.0)	(4.3)	(2.71)	(2.3)	(3.4)	(2.51)	(2.21)	(3.6)	(2.51)	(2.32)	(4.02)	(2.63)	(3.1)	(5.4)	(3.5)
Interaction term	-1.65	-3.61	-3.62	-0.71	-0.70	-0.71	0.13	-0.70	-0.73	1.51	1.31	10.3	2.91	2.1	2.1
	(1.88)	(3.32)	(2.37)	(2.3)	(4.1)	(2.4)	(2.3)	(4.1)	(2.21)	(1.57)	(2.7)	(1.25)	(2.11)	(3.2)	(2.9)
Disorder															
outdoor (latent)	1.70	0.001	0.001	7.1***	6.1*	6.4**	1.01	3.81	3.81	-1.12	-1.67	-1.60	-0.40	-4.81	-4.81
	(2.30)	(0.05)	(0.035)	(2.4)	(4.0)	(3.3)	(2.0)	(3.62)	(2.91)	(2.20)	(4.34)	(3.02)	(2.6)	(3.7)	(3.80)
smoking (latent)	-0.91	0.002	0.002	-0.40	0.81	0.81	0.72	2.42	2.42	1.0	3.24	3.10	4.1	1.11*	1.05**
	(3.0)	(0.055)	(0.035)	(3.1)	(5.1)	(3.45)	(3.0)	(4.90)	(3.57)	(3.9)	(5.60)	(3.70)	(3.7)	(6.0)	(4.51)
Interaction term	2.64	0.03	0.002	-2.7	-3.61	-3.6	1.51	0.81	0.81	3.0	1.42	1.40	8.1***	11.1***	11.0***
	(2.60)	(0.05)	(0.039)	(2.8)	(4.3)	(3.4)	(2.31)	(4.6)	(3.12)	(2.71)	(5.45)	(3.41)	(3.2)	(3.4)	(3.70)

Source: SOEP 2002-2007: own calculations.

Notes: All coefficients are from different regressions. Standard errors are in parenthesis. *indicates statistical significance at the 10% level, ** at the 5% level, *** at the 1% level. All regressions include indicators of mother's education, age, income, municipality size, immigration background, sex of the child, birth order, child's age in months, month and year dummies. Model [1] OLS, refers to equation (1b), model [2] Area fixed effect, refers to equation (2b) and model [3] Area and family fixed effect, refers to equation (3b). Sample size varies between 629 and 775 observations. Equation (1c) and model [2] refer to equation (3c). Sample size varies between 629 and 775 observations.