

Author's response to reviews

Title: Does traffic exhaust contribute to the development of asthma and allergic sensitization in children? Findings from recent cohort studies

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Author's response to reviews: see over

Response to the referees:

Referee 1 (Hanns Moshhammer).

1. The sentence has been changed according to the comments from the referee:

Other common indicators of traffic exhaust are NO₂ and nitrogen oxides (NO_x), where the fraction emitted as NO₂ tends to increase due to new diesel engines with oxidative catalysts.

2. We agree that our statement is rather weak. The words “could play a role” have been replaced by “**contributes to**” both in the abstract and on page 12.

3. We have excluded the wording “and the findings were not consistent” on page 12 (2nd paragraph, 1st sentence).

We have also excluded the sentence “Genetic differences between populations may explain inconsistent findings in studies carried out in different countries.” (Page 13, 2nd paragraph)

Referee 2 (Francesco Forastiere)

1. Exposure assessment methods have been characterized and summarized in the first paragraph of the results:

Several different types of exposure variables are used; contrasts in community level of pollutants [25,30,31], measurements outside homes [26], distance to a large road or traffic flow [18,19,27] concentrations outside homes according to atmospheric dispersion models with emission data [22,23,24] and concentrations estimated with statistical models (land use regression models) including monitoring data. Also for a specific type of exposure variable there are differences in how exposure is described.

The differences in exposure measurements are also commented in the first paragraph of the discussion:

Different types of exposure assessment have been used in these studies; traffic proximity (GIS), dispersion modelling, pollution measurements and regression models. Some studies seem to have a very high spatial resolution in exposure data, but it is difficult to compare these different approaches. However, internal validations show for example that the dispersion model in the Norwegian study [24] seems to perform less well than the model in the similar Swedish study [22-23]. Such differences may contribute to inconsistent findings.

The third and fourth paragraphs on page 2 in the introduction have been slightly changed.

2. We have now briefly described the difficulties of studying asthma and symptoms in young children in the introduction. We have also addressed sensitization as a specific outcome in the same paragraph:

Misclassification of the outcome is often a concern in questionnaire-based studies of respiratory disease, particularly in young children with less distinctive symptoms. Lower respiratory illness and wheezing symptoms are frequent in early childhood but very few of these children develop asthma. Moreover, childhood asthma is not a homogeneous disorder and different phenotypes of wheeze and asthma have been identified based on prognosis, atopic status and lung function [15]. Sensitization is assessed by objective measurements and misclassification is therefore a minor problem. Sensitization to inhalant allergens predominate after infancy [16, 17]. Allergic sensitization is common in asthma and early sensitization predicts persisting symptoms [18].

3. The difficulties of doing a quantitative meta-analysis of the data are now briefly commented in a sentence in the first paragraph of the discussion:

With the limited number of studies with both the same exposure measure and the same definition of the health outcome, we found it too early to now perform a formal meta-analysis.

The Tables have been slightly changed

4. The abstract has been slightly changed according to the comments from the reviewer (line 2, 5-6).