A common functional variant on the pro-inflammatory Interleukin-6 gene may modify the association between long-term PM10 exposure and diabetes

JournalArticle (Originalarbeit in einer wissenschaftlichen Zeitschrift)

ID 3446736
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Year 2016
Title A common functional variant on the pro-inflammatory Interleukin-6 gene may modify the association between long-term PM10 exposure and diabetes
Journal Environmental health
Volume 15
Pages / **Article-Number** 39
Air pollutants have been linked to type 2 diabetes (T2D), hypothesized to act through inflammatory pathways and may induce interleukin-6 gene (IL6) in the airway epithelium. The cytokine interleukin-6 may impact on glucose homeostasis. Recent meta-analyses showed the common polymorphisms, IL6 -572G >and IL6 -174G >to be associated with T2D risk. These IL6 variants also influence circulatory interleukin-6 levels. We hypothesize that these common functional variants may modify the association between air pollutants and T2D.; We cross-sectionally studied 4410 first follow-up participants of the Swiss Cohort Study on Air Pollution and Lung and Heart Diseases (SAPALDIA), aged 29 to 73ăyears who had complete data on genotypes, diabetes status and covariates. We defined diabetes as self-reported physician-diagnosed, or use of diabetes medication or non-fasting glucose >11.1ămmol/L or HbA1c > 0.065. Air pollution exposure was 10-year mean particulate matter <10ăμm in diameter (PM10) assigned to participants' residences using a combination of dispersion modelling, annual trends at monitoring stations and residential history. We derived interaction terms between PM10 and genotypes, and applied mixed logistic models to explore genetic interactions by IL6 polymorphisms on the odds of diabetes.; There were 252 diabetes cases. Respective minor allele frequencies of IL6 -572G >and IL6 -174G >were 7 and 39ă%. Mean exposure to PM10 was 22ăμg/m(3). Both variants were not associated with diabetes in our study. We observed a significant positive association between PM10 and diabetes among homozygous carriers of the pro-inflammatory major G-allele of IL6 -572G >[Odds ratio: 1.53; 95ă% confidence interval (1.22, 1.92); P interaction (additive) = 0.003 and P interaction (recessive) = 0.006]. Carriers of the major G-allele of IL6 -174G >also had significantly increased odds of diabetes, but interactions were statistically non-significant.; Our results on the interaction of PM10 with functionally well described polymorphisms in an important pro-inflammatory candidate gene are consistent with the hypothesis that air pollutants impact on T2D through inflammatory pathways. Our findings, if confirmed, are of high public health relevance considering the ubiquity of the major G allele, which puts a substantial proportion of the population at risk for the development of diabetes as a result of long-term exposure to air pollution.
Publisher BioMed Central
ISSN/ISBN 1476-069X

Full Text on edoc Available;
Digital Object Identifier DOI 10.1186/s12940-016-0120-5
PubMed ID http://www.ncbi.nlm.nih.gov/pubmed/26911440
ISI-Number WOS:000370930700001
Document type (ISI) Article