Erratum to: Genome-wide association and HLA fine-mapping studies identify risk loci and genetic pathways underlying allergic rhinitis (Nature Genetics, (2018), 50, 8, (1072-1080), 10.1038/s41588-018-0157-1)

In the version of this article initially published, in Fig. 3, the y-axis numbering did not match the log scale indicated in the axis label. The error has been corrected in the HTML and PDF version of the article.

General information
State: Published
Organisations: Department of Bio and Health Informatics, National Veterinary Institute, Immunoinformatics and Machine Learning, Department of Mechanical Engineering, Department of Applied Engineering Design and Production, University of Amsterdam, Imperial College London, Pompeu Fabra University, University of Greifswald, APH Amsterdam Public Health, Technical University of Denmark, Icahn School of Medicine at Mount Sinai (ISMMS), University of California at San Francisco, University of Southern California, Erasmus University Rotterdam, University of Melbourne, University of Liege, Novo Nordisk Foundation Center for Basic Metabolic Research, Telethon Kids Institute, University of Oulu, University Children’s Hospital Regensburg (KUNO), University of Copenhagen, Helmholtz Zentrum Muenchen German Research Center for Environmental Health, University of Manchester, 23andMe Inc., University of Chicago, Hospital Universitario Nuestra Senora de Candelaria, CIBER - Center for Biomedical Research Network, Vrije Universiteit Amsterdam, Barcelona Institute for Global Health Foundation (ISGlobal), Kiel University, University of Arizona, Polytechnic University of Catalonia, University of Basel, Swiss Tropical Institute, Ludwig Maximilian University of Munich, University of Bristol, Henry Ford Health System, University of La Laguna, deCODE Genetics, University of Iceland
Number of pages: 1
Pages: 1343
Publication date: 1 Sep 2018
Peer-reviewed: Yes

Publication information
Journal: Nature Genetics
Volume: 50
Issue number: 9
ISSN (Print): 1061-4036
Ratings:
BFI (2018): BFI-level 3
Web of Science (2018): Indexed yes
BFI (2017): BFI-level 2
Scopus rating (2017): CiteScore 21.12 SJR 22.243 SNIP 5.867
Web of Science (2017): Impact factor 27.125
Web of Science (2017): Indexed yes
BFI (2016): BFI-level 2
Investigating the causal effect of smoking on hay fever and asthma: a Mendelian randomization meta-analysis in the CARTA consortium

Observational studies on smoking and risk of hay fever and asthma have shown inconsistent results. However, observational studies may be biased by confounding and reverse causation. Mendelian randomization uses genetic variants as markers of exposures to examine causal effects. We examined the causal effect of smoking on hay fever and asthma by using the smoking-associated single nucleotide polymorphism (SNP) rs1696968/rs1051730. We included 231,020 participants from 22 population-based studies. Observational analyses showed that current vs never smokers had lower risk of hay fever (odds ratio (OR) = 0.68, 95% confidence interval (CI): 0.61, 0.76; P < 0.001) and allergic sensitization (OR = 0.74, 95% CI: 0.64, 0.86; P < 0.001), but similar asthma risk (OR = 1.00, 95% CI: 0.91, 1.09; P = 0.967). Mendelian randomization analyses in current smokers showed a slightly lower risk of hay fever (OR = 0.958, 95% CI: 0.920, 0.998; P = 0.041), a lower risk of allergic sensitization (OR = 0.92, 95% CI: 0.84, 1.02; P = 0.117), but higher risk of asthma (OR = 1.06, 95% CI: 1.01, 1.11; P = 0.020) per smoking-increasing allele. Our results suggest that smoking may be causally related to a higher risk of asthma and a slightly lower risk of hay fever. However, the adverse events associated with smoking limit its clinical significance.

General information

State: Published
Organisations: Research Centre for Prevention and Health, University of Bristol, University of Copenhagen, Bispebjerg-Frederiksberg Hospitals, University of South Australia, MRC Unit for Lifelong Health and Ageing, Norwegian University of Science and Technology, St. Olav's University Hospital, Helmholtz Zentrum München, National Institute for Health and Welfare, University of Glasgow, Leiden University Medical Center, University Hospital Essen, Helmsley Medical Centre, University of Southern Denmark, University College London, University of Essex, University of Greifswald, University of Helsinki
Number of pages: 9
Publication date: 2017
Peer-reviewed: Yes

Publication information
Journal: Scientific Reports
Volume: 7
Issue number: 1
Article number: 2224
ISSN (Print): 2045-2322
Ratings:
BFI (2018): BFI-level 1
Web of Science (2018): Indexed yes
BFI (2017): BFI-level 1
Scopus rating (2017): CiteScore 4.36 SJR 1.533 SNIP 1.245
Web of Science (2017): Impact factor 4.122
Web of Science (2017): Indexed yes
BFI (2016): BFI-level 1
Scopus rating (2016): CiteScore 4.63 SJR 1.692 SNIP 1.354
Web of Science (2016): Impact factor 4.259
Web of Science (2016): Indexed yes
BFI (2015): BFI-level 1
A genome-wide association study identifies CDHR3 as a susceptibility locus for early childhood asthma with severe exacerbations.

Asthma exacerbations are among the most frequent causes of hospitalization during childhood, but the underlying mechanisms are poorly understood. We performed a genome-wide association study of a specific asthma phenotype characterized by recurrent, severe exacerbations occurring between 2 and 6 years of age in a total of 1,173 cases and 2,522 controls. Cases were identified from national health registries of hospitalization, and DNA was obtained from the Danish Neonatal Screening Biobank. We identified five loci with genome-wide significant association. Four of these, GSDMB, IL33, RAD50 and IL1RL1, were previously reported as asthma susceptibility loci, but the effect sizes for these loci in our cohort were considerably larger than in the previous genome-wide association studies of asthma. We also obtained strong evidence for a new susceptibility gene, CDHR3 (encoding cadherin-related family member 3), which is highly expressed in airway epithelium. These results demonstrate the strength of applying specific phenotyping in the search for asthma susceptibility genes.