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.
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) rs16969968/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.

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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.