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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