Secondhand-smoke–exposed children who carried a TNF G→308A variant allele were at highest risk for respiratory illness–related school absence. A strong dose–response relationship in this group of patients was found for respiratory illness–related absence risk in relation to the number of household smokers and number of cigarettes smoked. The genetic susceptibility associated with the TNF G→308A allele is likely mediated by variation in inflammatory responses to secondhand smoke.

CONCLUSIONS. Secondhand-smoke–exposed children who carried a TNF G→308A variant allele were at highest risk for respiratory illness–related school absence. A strong dose–response relationship in this group of patients was found for respiratory illness–related absence risk in relation to the number of household smokers and number of cigarettes smoked. The genetic susceptibility associated with the TNF G→308A allele is likely mediated by variation in inflammatory responses to secondhand smoke.

METHODS. Schools provided daily absence summary information for the study subjects, and each absence was recorded as illness or nonillness. Telephone interviews regarding each illness-related absence were conducted with parents. School absences were classified as lower respiratory illnesses if wet cough, wheeze, or asthma was present. Secondhand-smoke exposure was determined by parent/guardian written responses on self-administered questionnaires and categorized as none, 1 to 29, or ≥30 cigarettes smoked per day inside the home. Secondhand-smoke exposure was markedly increased. Children with the variant A allele and secondhand-smoke exposure had a 75% increase in risk for illness-related absences compared with children with the TNF G→308GG genotype. However, absence rates in children with the variant A allele and secondhand-smoke exposure were markedly increased. Children with the variant A allele and secondhand-smoke exposure had a 75% increase in risk for illness-related absences compared with unexposed children with the GG genotype. In children with the variant A allele, illness-related absence risk increased as the number of smokers in the home increased. In addition, children with 1 variant A allele who were exposed to ≥30 cigarettes per day had a relative risk of 2.75 for respiratory illness–related school absence compared with unexposed children with the GG genotype. Restricting analysis to children without asthma did not substantially alter the findings.

RESULTS. There was a 51% greater risk of lower respiratory illness–related school absence among children with secondhand-smoke exposure compared with those who were unexposed. Children with the TNF GG genotype exhibited similar absence rates regardless of whether they were exposed or unexposed to secondhand smoke. Unexposed children with at least 1 copy of the TNF G→308A allele had similar absence rates for lower respiratory illnesses compared with children with the TNF G→308GG genotype. However, absence rates in children with the variant A allele and secondhand-smoke exposure were markedly increased. Children with the variant A allele and secondhand-smoke exposure had a 75% increase in risk for illness-related absences compared with unexposed children with the GG genotype. In children with the variant A allele, illness-related absence risk increased as the number of smokers in the home increased. In addition, children with 1 variant A allele who were exposed to ≥30 cigarettes per day had a relative risk of 2.75 for respiratory illness–related school absence compared with unexposed children with the GG genotype. Restricting analysis to children without asthma did not substantially alter the findings.

CONCLUSIONS. Secondhand-smoke–exposed children who carried a TNF G→308A variant allele were at highest risk for respiratory illness–related school absence. A strong dose–response relationship in this group of patients was found for respiratory illness–related absence risk in relation to the number of household smokers and number of cigarettes smoked. The genetic susceptibility associated with the TNF G→308A allele is likely mediated by variation in inflammatory responses to secondhand smoke.
Patterns of Global Tobacco Use in Young People and Implications for Future Chronic Disease Burden in Adults

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