Population-Based Impact of Smoking, Drinking, and Genetic Factors on HDL-Cholesterol Levels in J-MICC Study Participants

著者	NINDITA Yora
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論 文 要 旨

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（J-MICC 研究参加者の HLD コレステロール値における
喫煙、飲酒および遺伝的要因の
人口ベースの影響に関する研究）

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Background: Environmental and genetic factors are suggested to exhibit factor-based association with HDL-cholesterol (HDL-C) levels. However, the population-based effects of environmental and genetic factors have not been compared clearly. We conducted a cross-sectional study using data from the Japan Multi-Institutional Collaborative Cohort (J-MICC) Study to evaluate the population-based impact of smoking, drinking, and genetic factors on low HDL-C.

Methods: Data from 11,498 men and women aged 35-69 years were collected for a genome-wide association study (GWAS). Sixty-five HDL-C-related SNPs with genome-wide significance ($P < 5 \times 10\text{-}8$) were selected from the GWAS catalog, and seven representative SNPs were defined, and the population-based impact was estimated using population attributable fraction (PAF).

Results: We found that smoking, drinking, daily activity, habitual exercise, egg intake, BMI, age, sex and the SNPs CETP rs3764261, APOA5 rs662799, LIPC rs1800588, LPL rs328, ABCA1 rs2575876, LIPG rs3786247, and APOE rs429358 were associated with HDL-C levels. The gene-environmental interactions on smoking and drinking were not statistically significant. The PAF for low HDL-C was the highest in men (63.2%) and in rs3764261 (31.5%) of the genetic factors, and the PAFs of smoking and drinking were 23.1% and 41.8%, respectively.

Conclusions: The present study showed that the population-based impact of genomic factor CETP rs3764261 for low HDL-C was higher than that of smoking and lower than that of drinking.