HIGH-DENSITY LIPOPROTEIN CHOLESTEROL AND CANCER INCIDENCE: DATA FROM THE FRAMINGHAM HEART STUDY

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Introduction: Epidemiologic data demonstrate a strong inverse correlation between high-density lipoprotein cholesterol (HDL-C) and cardiovascular disease risk. We recently extended this observation to include an association of low HDL-C with incident cancer in a systemic analysis of lipid-altering trials. The role of HDL-C in relation to cancer risk, however, remains unclear. At issue is a potential contribution of underlying processes that simultaneously affect HDL-C concentration and promote carcinogenesis, such as smoking, obesity, or hyperinsulinemia. Alternatively, low HDL-C levels may themselves manifest from active neoplastic processes (reverse causality). We examined these issues using data from the Framingham Heart Study Offspring Cohort to assess trends in HDL-C throughout time preceding cancer diagnosis.

Methods: Incident cancer cases and control subjects (propensity score matched for age, gender, diabetes, tobacco use, blood pressure, and body mass index) without history of lipid-lowering therapy, were followed for 4 time points prior to cancer diagnosis. Linear mixed model regression analyses delineated the relationship of HDL-C between cancer and cancer-free participants over time.

Results: 201 incident cancer cases and 402 matched controls were identified. HDL-C values were lower in cancer subjects than matched controls at each point of assessment throughout an average of 18.7 years prior to diagnosis (F = 7.76, p = 0.005). The trend for lower HDL-C in cancer patients compared with control subjects was consistent throughout the duration of the study (F = .18, p = .948 for differences between time points).

Conclusion: Our analysis utilizing propensity score matching to compare HDL-C in cancer patients to a closely matched control population over a prolonged course significantly mitigates the limitation of trial length and confounding inherent in previous observational studies. These findings suggest persistence of an inverse association between HDL-C and cancer incidence predating even subclinical malignancy. While unable to define an etiologic mechanism for HDL-C in carcinogenesis, these findings highlight the need for further research into this association.