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In Reply:

We appreciate the comments by Stefanidis et al. Their findings of the low sensitivity of hepatitis C virus (HCV) enzyme immunoassay (EIA) for the detection of HCV infection in patients on maintenance hemodialysis therapy are similar to ours[1] and highlight the limitations of such testing in these subjects. Unlike our findings, they report that HCV EIA-negative (EIA^-)/transcription-mediated amplification (TMA)-positive (TMA^+) test results, compared with EIA^+/TMA^+, were more likely to occur in patients with shorter hemodialysis therapy duration. These findings are of interest and in contrast to what has been described in patients with other serious chronic conditions in which HCV EIA false negativity in those with chronic infection tends to occur more frequently in advanced stages of the underlying disease. For example, human immunodeficiency virus–infected individuals with chronic HCV infection are more likely to be EIA^- with advanced immunosuppression.[2]

We also should note that the lack of difference in mortality between EIA^+/TMA^+ patients and other groups seen by Stefanidis et al might be caused by the cross-sectional design (ie, survival bias and/or type II error [lack of statistical power]). Furthermore, observations of lower transaminase levels in EIA^-/TMA^+ patients compared with those with EIA^+/TMA^+ results, as reported by both Stefanidis et al and us,[1] also are not straightforward. Design limitations of the investigations by us and Stefanidis et al underscore the need for longitudinal studies to better understand the significance of HCV infection in persons on maintenance hemodialysis therapy, including those with discordant diagnostic test results. The observed association between greater mortality and HCV infection in persons on maintenance hemodialysis therapy[3,4] further suggests that interventions to treat HCV in this population may have merit and be worthy of further investigation.

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ARE HOMOCYSTEINE AND MTHFR GENOTYPE POLYMORPHISM ASSOCIATED WITH ARTERIOVENOUS FISTULA PATENCY?

To the Editor:

In a recent article, Mallamaci et al[1] concluded that native arteriovenous fistula thrombosis in hemodialysis patients is associated with hyperhomocysteinemia. We appreciated this prospective cohort study controlling for access type; however, the methylenetetrahydrofolate reductase (MTHFR) genotype polymorphism failed to predict fistula outcome despite its correlation with serum homocysteine level. Although the investigators listed data for possible risk factors in all study subjects, differences in risk factors other than plasma homocysteine levels (such as prevalence of diabetes or supplemented dose of folic acid) were not shown among the 3 tertile groups. A recent meta-analysis by Den Heijer et al[2] reported that homocysteine levels are associated with risk for venous thrombosis, as is the *MTHFR* 677TT genotype; however, the 677TT genotype had no effect on venous thrombosis in patients in North America, probably resulting from the greater intake of folate and riboflavin there. Use of folate and other relevant vitamin supplements might decrease events of access thrombosis in patients harboring the