Figure 5. Associations between EBA175F2 immunoglobulin G responses and *Plasmodium falciparum* parasite densities. Kaplan-Meier curves are shown for the first polymerase chain reaction (PCR)–detectable reinfection (A; \( P = .979 \), by log-rank test), the first light microscopy (LM)–detected reinfection (B; \( P = .967 \), by log-rank test), the first reinfection with a parasite density \( >500 \) parasites/\( \mu L \) (C; \( P = .266 \), by log-rank test), and the first reinfection with an LM density \( >5000 \) parasites/\( \mu L \) (D; \( P = .020 \), by log-rank test). Antibody responses were divided into 3 equal response groups: high (light gray line), medium (dark gray line), and low (black line). Unadjusted data are shown.

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**APPENDIX A**

**ADDITIONAL DETAILS ON STATISTICAL ANALYSIS OF DATA**

Various factors were considered as potential confounders in the analysis of the association between antibodies and the risk of malaria and reinfection. Host age and location of residence were the only factors significantly associated with risk of malaria [32] and were therefore included as covariates in the Cox proportional hazards model. Although parasitemia by PCR at enrollment was associated with higher antibody levels, it was not associated with the risk of malaria, and including parasitemia in the model did not alter the HR. Additionally, erythrocyte polymorphisms (\( \alpha \)-thalassemia trait, South Asian Ovalocytosis, Gerbich, and CR1) were not associated with a risk of malaria.

The collinearity of antibody responses to the EBAs and other merozoite proteins meant that it was not possible to assess the effect of multiple antigens in the same model to quantify the effect of individual antibody responses to protective associations. Interaction terms were investigated between antibody tertile categories, but the highly correlated nature of the responses limited the value of this approach. Instead, we considered protective associations for broad antibody responses by calculating antibody response scores for the 3 EBAs combined; for MSP1, MSP2, and AMA1 combined; or for all antigens combined. Combined response scores were a summation of tertile responses (0, 1, or 2 for low, medium, and high responses, respectively) for each group. These scores were then used to create 3 groups reflecting high, intermediate, and low combined responses and used in the Cox proportional hazards model to calculate HRs.

**References**

3. Triglia T, Thompson JK, Cowman AF. An EBA175 homologue which