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ORIGINAL RESEARCH COMMUNICATION

Glutathione, glutathione peroxidase, and selenium status in HIV-positive and HIV-negative adolescents and young adults 1,2,3

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Background: Antioxidant nutrient deficiencies may hasten the progression of HIV disease by impairing antioxidant defenses.

Objective: The objective of the study was to determine whether HIV infection is associated with poor selenium status and low antioxidant protection by glutathione and glutathione peroxidase (GPX).

Design: In a cross-sectional study of 365 HIV-positive and HIV-negative adolescents and young adults, we examined the relation of plasma selenium, whole-blood glutathione, and whole-blood GPX to HIV status, disease severity, immune activation, and oxidative damage.

Results: Selenium deficiency (plasma selenium <0.070 µg/mL) was not seen in any subjects, and plasma selenium in 244 HIV-positive subjects (0.120 \pm 0.0013 µg/mL) did not differ significantly (P = 0.071) from that in 121 HIV-negative subjects (0.125 \pm 0.0020 µg/mL). However, multiple regression analysis after adjustment for covariates showed a significant (P = 0.002) negative association between HIV-associated immune activation (plasma neopterin) and plasma selenium concentrations. GPX activity was highest in HIV-positive subjects taking antiretroviral therapy (median: 14.2; 25th, 75th percentiles: 11.1, 18.7 U/mL; n = 130), intermediate in HIV-positive subjects (10.6; 8.6, 12.7 U/mL; n = 121; P < 0.05 for all comparisons). GPX was also positively associated with malondial dehyde, a marker of oxidative damage.

Conclusions: Subjects had adequate selenium status, although HIV-related immune activation was associated with lower plasma selenium concentrations. GPX activity appears to have been induced by the oxidative stress associated with HIV infection and use of antiretroviral therapy. Thus, young, well-nourished subjects can mount a compensatory antioxidant response to HIV infection.

Key Words: Glutathione • glutathione peroxidase • selenium • oxidative stress • HIV

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