Murine AIDS studies define antioxidant deficiency mechanisms

Dear Sir:

The exciting study of Allard et al (1) confirms apparently unrecognized studies of murine AIDS showing increased lipid peroxidation occurring concomitantly with reduced concentrations of antioxidant vitamins in tissue (2-4). Replacement of vitamin E reduced lipid peroxidation and immune dysfunction (2), which slowed cancer development (3), whereas vitamin A supplementation prolonged survival (5), in murine AIDS. However, ethanol consumption increased oxidative, accentuated immune dysfunction, stimulated cancer, and accelerated death during retrovirus infection (3, 6). Animal model studies (2, 3) have already answered a key question in Allard et al’s study: will supplementation with antioxidant vitamins reduce oxidative stress?

The authors queried, what are “the mechanisms underlying the increased oxidative stress in the HIV population?” Retrovirus oxidation increased production of T helper 2 cell cytokines by a subset of cells, highly activated by retroviral antigens (2, 4). In murine AIDS, suppression of these cytokines by injection with a T cell receptor peptide prevented excessive oxidation, loss of vitamin E, and retrovirus-induced immune dysfunction (4) while maintaining resistance to an opportunistic pathogen (7).

Murine retroviral oxidation, loss of antioxidant vitamins, and cytokine dysregulation have been prevented both by vitamin E (2) and antioxidant hormone (dehydroepiandrosterone) supplementation (8) and immunologically by direct suppression of the excessive cytokine secretion of T helper 2 cells with T cell receptor peptides (4, 7). Such knowledge from the murine AIDS model, supported by the current study in human AIDS (1), should be applied to human AIDS treatment (9). Over-the-counter antioxidant hormone or vitamin supplementation appears safe and efficacious in murine AIDS and in uninfected humans. Therefore, supplements of antioxidant hormones or vitamins should now be used to overcome antioxidant deficiencies in HIV-infected persons (9). This should help to prevent the loss of disease resistance and the resulting cytokine dysregulation due to retrovirus-induced antioxidant deficiencies, while we await confirmation of the benefits of such supplements in HIV-infected patients from clinical trials (10).

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REFERENCES

Reply to RR Watson

Dear Sir:

We thank Watson for his interesting letter. We knew that both animal (1, 2) and in vitro experiments (3) have shown that antioxidant vitamins reduce oxidative stress, improve immune function, and decrease HIV replication. These experiments were the basis for our study investigating oxidative stress and antioxidant defense status in HIV-infected humans (4). The key question of whether supplementation with antioxidant vitamins will reduce oxidative stress that Watson refers to concerns humans rather than animal subjects and relates not only to...
LETTERS TO THE EDITOR

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REFERENCES


Bioimpedance: 50 kHz parallel reactance and the prediction of body cell mass

Dear Sir:

Several important comments need to be made concerning the paper by Kotler et al (1). Although a rhetorical case was made for body cell mass (BCM) predicted by 50-kHz parallel reactance ($X_p$), no theoretical basis was provided. Kotler et al stated that a major uncertainty in the theory underlying bioelectrical impedance analysis is whether the body's ionic circuit is arranged as a series or parallel circuit. We can find no rigorous study in the literature supporting this statement or any single-frequency (SF) prediction of cell volume. It was suggested that the improved prediction of total body potassium (TBK) by $X_p$ compared with series reactance ($X_s$) was proof that $X_p$ was superior for predicting BCM. Impedance measured at any SF can be interpreted as a series or parallel circuit, with both resulting in 2 final elements, resistance ($R$) and reactance ($X$). The problem is that biological tissue consists of series of more than 2 elements ($R, C_{em}$) and so reactance ($X$) is meaningless because it is not used for anything and neither $X_p$ nor $X_s$ has any scientific basis.

It should be noted that the $X_p$ equation has a term to the model (exponent $\alpha$) in 1940 to represent the effect of the infinite number of series and parallel reactance ($X_s$ and $X_p$), which has been used since 1925 to interpret impedance measurements on individual biological cells (2). Cole added an important fourth term to the model (exponent $\alpha$) in 1940 to represent the effect of the infinite number of parallel reactance ($X_p$) in parallel with a parallel cell membrane capacitance ($C_{m}$) and resistance intracellular water ($R_i$), which has been used since 1925 to interpret impedance measurements on individual biological cells (2). Cole added an important fourth term to the model (exponent $\alpha$) in 1940 to represent the effect of the infinite number of series and parallel reactance ($X_s$ and $X_p$), which has been used since 1925 to interpret impedance measurements on individual biological cells (2). Cole added an important fourth term to the model (exponent $\alpha$) in 1940 to represent the effect of the infinite number of series and parallel reactance ($X_s$ and $X_p$), which has been used since 1925 to interpret impedance measurements on individual biological cells (2). Cole added an important fourth term to the model (exponent $\alpha$) in 1940 to represent the effect of the infinite number of series and parallel reactance ($X_s$ and $X_p$), which has been used since 1925 to interpret impedance measurements on individual biological cells (2). Cole added an important fourth term to the model (exponent $\alpha$) in 1940 to represent the effect of the infinite number of series and parallel reactance ($X_s$ and $X_p$), which has been used since 1925 to interpret impedance measurements on individual biological cells (2). Cole added an important fourth term to the model (exponent $\alpha$) in 1940 to represent the effect of the infinite number of series and parallel reactance ($X_s$ and $X_p$), which has been used since 1925 to interpret impedance measurements on individual biological cells (2).