tured supernatant, determining whether NO derived from Arg plays a role in modulating cytokine production or augments the function of macrophages requires further investigation.

The specific antibody against PEIF did not differ and there were no differences in the percentages of CD4 or CD8 T cells or the CD4/CD8 T cell ratio between the 2 groups. This finding suggests that the humoral and T cell populations were not enhanced by Arg supplementation. A previous study carried out by our laboratory showed that antioxidant enzyme activities and lipid peroxides in tissues tended to be lower in the Arg group than in the control group after a burn[13] Since a burn injury is a trauma with high oxidative stress,[35] it is possible that the reported beneficial effects of Arg supplementation on burn injury is due to its attenuation of oxidative stress instead of the mediating of humoral immunity and the T cell response.

In conclusion, Arg supplementation demonstrated no appreciable benefit in the production of a specific antibody against *P. aeruginosa* nor in plasma NO concentration in vaccinated burned rats. Because the major defense mechanism against bacterial infection is the production of specific antibodies which may neutralize bacteria toxins and opsonize the bacteria for phagocytosis,[19] the above results may explain why Arg supplementation did not improve the survival rate of vaccinated burned rats complicated with *P. aeruginosa* infection. Determining Arg supplementation has favorable effects on reducing in vivo proinflammatory cytokine secretion or the leukocyte oxidative burst requires further investigation.

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