Aldosterone, Inflammation, and Preeclampsia

To the Editor:

We read with great interest the recent article published by Freeman et al in the November issue of Hypertension, which focuses on a possible systemic inflammatory response in patients with preeclampsia. The authors suggest that the susceptibility to inflammation might be a common underlying risk factor for preeclampsia and conclude that the mechanism for this inflammatory susceptibility is unknown and could be attributable to a generalized upregulation of several inflammatory processes.

We would like to suggest that aldosterone could play an important role in the genesis of this increased susceptibility of inflammatory process in preeclampsia. This contention comes from a number of our studies that provide rationale for this possibility. We have demonstrated that in preeclampsia, plasma aldosterone and plasma progesterone are as high as in uncomplicated pregnancy, whereas the rectal subtractive potential difference, which is an index of biological effect of aldosterone, is increased only in preeclampsia to a similar extent as in primary aldosteronism. These data are consistent also with the measurement of aldosterone receptors in mononuclear leukocytes, whose number was normal in uncomplicated pregnancy and downregulated in preeclampsia, which, again, matched the changes observed in primary aldosteronism. We therefore suggested that uncomplicated pregnancy is characterized by a reduced response to the action of aldosterone. In addition, we have recently demonstrated that the coincubation of human mononuclear leukocytes from healthy subjects with high concentrations of aldosterone induces the expression of 2 inflammation and oxidative stress-related proteins, plasminogen activator inhibitor-1 and p22phox, as shown by Western blot analysis. In the same study, we have also shown that these effects of aldosterone were blocked by the aldosterone receptor antagonist canrenone.

Weber has recently stressed the importance of mononuclear leukocytes in the induction of the aldosterone-mediated fibrosis in human tissues through the demonstration in an animal model that all the processes of inflammation and fibrosis mediated by aldosterone are preceded by an invasion of monocytes, therefore relating aldosterone excess with invasion of mononuclear cells and inflammation. Therefore, in considerations of the inflammatory role of aldosterone, the report of Freeman et al and the results of our studies, it could be rational to hypothesize that in the long-term, a higher susceptibility to the inflammatory activity of aldosterone might be involved in women in whom preeclampsia develops. The increased risk of cardiovascular complications in these subjects may be caused, in part, by aldosterone-mediated inflammation.

Decio Armanini
Lorenzo A. Calo
Department of Medical and Surgical Sciences
and Department of Clinical and Experimental Medicine
University of Padua, Italy.

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Decio Armanini and Lorenzo A. Calò

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