Sodium and Chloride in Salt-Sensitive Hypertension

To the Editor:

In a recent letter to the editor, Dr. M. B. Blaustein refers to several reports (including two from our laboratory) demonstrating that dietary sodium loading, without chloride loading, fails to produce hypertension in two salt-sensitive models — the Dahl salt-sensitive rat and the uninephrectomized deoxycorticosterone acetate (DOCA)-treated rat. Blaustein suggests that an attempt to dissociate salt-sensitive hypertension from dietary sodium intake may be misleading. We agree; however, we would like to clarify several points addressed in that letter.

In the Dahl salt-sensitive rat, we have recently found that selective dietary chloride loading (without sodium loading) also fails to produce hypertension. In our studies, selective sodium loading and selective chloride loading were associated with markedly positive sodium and chloride balances, respectively. Hypertension developed only when both sodium and chloride balances were increased, and this was achieved only with dietary sodium chloride loading. Thus, hypertension in this model is dependent on the concomitant administration of high dietary intakes of both sodium and chloride. The terms sodium-dependent hypertension and chloride-dependent hypertension are equally misleading; sodium chloride-dependent hypertension would be more appropriate.

In his letter, Dr. Blaustein states that restriction of chloride may cause a hypochloremic, hypokalemic alkalosis and that sodium will not expand the extracellular fluid (ECF) volume because of the chloride deficiency. In both the Dahl-salt-sensitive rat and the DOCA-salt model, animals selectively fed high sodium were on "normal" chloride intakes, not chloride-restricted diets. These animals were not hypochloremic, hypokalemic, or alkalotic. Consequently, failure of animals fed this diet to become hypertensive was not related to a deficiency of dietary chloride.

Dr. Blaustein concludes his letter by stating that "ECF volume expansion appears to be a key factor in the pathophysiology of hypertension..." In the DOCA-salt model, we found that hypertension was associated with an expanded ECF volume. Therefore, our observations are consistent with the hypothesis that the development of hypertension in these salt-sensitive models is related to expansion of the ECF.

References


Shirley A. Whitescarver, B.G.S.
Michael Tachman, M.D.
Theodore A. Kotchen, M.D.
Departments of Physiology and Medicine
University of Kentucky
School of Medicine
Lexington, Kentucky 40536

(Hypertension 8: 552, 1986)
Sodium and chloride in salt-sensitive hypertension.
S A Whitescarver, C E Ott, M Tachman and T A Kotchen

Hypertension. 1986;8:552
doi: 10.1161/01.HYP.8.6.552

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1986 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/8/6/552.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org//subscriptions/