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Sodium Chloride-Dependent Hypertension

TO THE EDITOR:

In a letter recently published in Hypertension, Dr. Mordecai P. Blaustein comments that “two recent studies . . . have been interpreted as showing that salt-dependent hypertension in some animal models can be ‘dissociated from the intake of sodium’ and may, at least in part, be attributable to Cl-.” This sentence might suggest that we, the authors of one of the studies cited,2 hold that salt-dependent hypertension can be dissociated from the intake of sodium. We do not hold this position. Rather, we concluded that “it seems prudent to speak of ‘sodium chloride-depemt’ hypertension rather than ‘sodium-dependent’ hypertension.”2 This conclusion was based in large part on our finding that in rats given deoxycorticosterone, provision of dietary sodium as sodium chloride induced hypertension whereas provision of dietary sodium without chloride did not. As recently reiterated, we have not contended that chloride is the pressor component of sodium chloride2 nor have we proposed use of the term chloride-dependent hypertension.

Dr. Blaustein states that “the decision as to whether to use the term Na-dependent hypertension or Cl-dependent hypertension might appear to be semantic. Since Na+ is the actively transported ion and is the primary ion affected by mineralocorticoids, it seems most appropriate to define mineralocorticoid hypertension as Na+ dependent on the basis of the underlying physiology. . . .” We believe that neither “sodium-dependent” hypertension nor “chloride-dependent” hypertension is the optimal term. We prefer to speak of mineralocorticoid hypertension as “sodium chloride-dependent” since induction of the hypertension, whatever its mechanism, appears to depend on the provision of both sodium and chloride.2,4 We have recently found that supplementing the dietary sodium intake of rats given deoxycorticosterone with sodium bromide and sodium iodide combined can induce increases in blood pressure.5 Thus, the deoxycorticosterone model might also be referred to as “sodium-halide-dependent hypertension.” In this regard, it seems relevant that in 1928 Addison6 reported that oral loading with either sodium chloride or sodium bromide induced blood pressure increases in five hypertensive patients.

Dr. Blaustein states that “when an appropriate an-

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AUTHOR’S RESPONSE:
In my recent letter, I tried very carefully to indicate that I believe McCarron et al. have misinterpreted the reports of Kurtz and Morris and of Whitescarver et al. by suggesting that “salt-dependent hypertension in some animal models can be dissociated from the intake of sodium.” I did not mean to imply that the authors of these articles hold such views; indeed, I am pleased that Kurtz and Morris also take issue with McCarron’s interpretation of their data.

Current views regarding the mechanism of action of mineralocorticoids on salt reabsorption are briefly summarized in my letter and need not be repeated here. The evidence supplied by Kurtz and Morris that other halide ions can substitute for chloride in enabling the mineralocorticoid-dependent retention of sodium and generation of hypertension does not conflict with my view that sodium retention is required for the development of mineralocorticoid-salt hypertension.

Although there may be disagreement about the relationship between extracellular fluid volume expansion and hypertension, there seems to be general agreement that 1) mineralocorticoids act on the renal sodium transport systems and induce salt (NaCl) and water retention (the attendant volume expansion is primarily extracellular) and 2) mineralocorticoids apparently do not induce hypertension when salt intake is restricted. The question of whether the hypertension is due to the volume expansion per se or can be better correlated with the natriuretic (and diuretic) escape from the salt and water retention requires further study.

The foregoing considerations, and those mentioned by Kurtz and Morris appear to reinforce my main conclusion “that it may be misleading to infer pathophysiological mechanisms in the development of hypertension on the basis of dietary studies alone.”

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