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ESPS Peer-review Report
Name of Journal: World Journal of Cardiology
ESPS Manuscript NO: 9178
Title: African Americans, Hypertension and the Renin Angiotensin System
Reviewer code: 00608229
Science editor: Ling-Ling Wen
Date sent for review: 2014-01-24 22:44
Date reviewed: 2014-02-16 02:35

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[Y] Grade A: Priority Publishing	Google Search:	[ ] Accept
[Y] Grade B (Very good)	[ ] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[ ] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	[ ]Rejection
[ ] Grade D (Fair)	[ ] Grade D: rejected	BPG Search:	[Y] Minor revision
[ ] Grade E (Poor)		[ ] Existed	[ ] Major revision
		[ ] No records	

COMMENTS TO AUTHORS

This is a carefully conducted review that discloses interesting information with clinically important implications. The conclusions are persuasively supported by data presentation and interpretation. However, there are certain points of concern that should be addressed, so that the message conveyed becomes clearer. 1. Reference should be made to the most scathing rebuttal against the slave hypothesis of hypertension in blacks. This came from Philip Curtin, an historian of the slave trade on whose work Grim had drawn heavily. Curtin denied any historical validity to the proposition that Africa had traditionally been salt-scarce, and asserted that his own work had been misunderstood or misquoted on this point. He also disputed the mortality estimates cited by Grim, noting that these figures were not only incorrect or outdated, but cited so poorly that their original source could not be identified. Indeed, he disparaged Diamond's impressive statistics as "numbers of unknown provenance." Further, Curtin argued that Grim's proposition that a majority of deaths were due to diarrheal disease was equally baseless. He concluded that the Slavery Hypothesis not only lacked supporting evidence, but that what little evidence did exist directly contradicted the theory. http://ajph.aphapublications.org/doi/pdf/10.2105/AJPH.82.12.1681 2. Although there are conflicting data, a number of studies have indicated that the sympathetic nervous system may play a role in mediating salt sensitivity. This is based on the findings that many salt-sensitive subjects have higher levels of norepinephrine and decreased levels of dopamine. Norepinephrine is associated with sodium retention and dopamine promotes increased sodium excretion. Both plasma norepinephrine concentrations and urinary sodium excretion are higher among salt-sensitive than salt-resistant subjects. African American subjects seem to be especially sensitive to the role of dopamine in salt

sensitivity. Dopamine is a vasodilator with a natriuretic effect on the kidney. However, when salt-sensitive African Americans are faced with an increased sodium load, they do not have a corresponding increase of dopamine, resulting in increased sodium retention. A similar deficiency of another renal natriuretic/diuretic peptide hormone, kallikrein, has been documented in salt-sensitive individuals. Moreover, although African Americans have a lower level of urinary kallikrein than Caucasians, salt-sensitive whites also have reduced urinary kallikrein excretion. References Weinberger M. Salt sensitivity of blood pressure in humans. Hypertension. 1996;27(part 2):481-490. Luft F, Weinberger M. Heterogeneous responses to changes in dietary salt intake: The salt sensitivity paradigm. Am J Clin Nutr. 1997;65(suppl):612S-617S. Weinberger M. Hypertension in Africa-Americans: The role of sodium chloride and extracelluar fluid volume. Semin Nephrol. 1996;16(2):110-116. Ely D. Overview of dietary sodium effects on and interactions with cardiovascular and neuroendocrine functions. Am J Clin Nutr. 1997:65(suppl):594S-605S. Gill J, Gorssman E, Goldstein D. High urinary dopa and low urinary dopamine-to-dopa ratio in salt-sensitive hypertension. Hypertension. 1991:18:614-621. 3. The authors should also comment on excess adiposity emerging as a natural candidate to explain the higher prevalence of hypertension in blacks, who have a 51% greater prevalence of obesity than whites. Flávio D. Fuchs. Why Do Black Americans Have Higher Prevalence of Hypertension? An Enigma Still Unsolved. Hypertension 2011. 4. Differences between blacks and whites regarding adherence to treatment should also be addressed. Turner BJ, Hollenbeak C, Weiner MG, Ten Have T, Roberts C. Barriers to adherence and hypertension control in a racially diverse representative sample of elderly primary care patients. Pharmacoepidemiol Drug Saf. 2009 Aug;18(8):672-81. doi: 10.1002/pds.1766. 5. As the panel of JNC 8 st

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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[Y] Grade A (Excellent)	[Y] Grade A: Priority Publishing	Google Search:	[Y] Accept
[ ] Grade B (Very good)	[ ] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[ ] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	[ ]Rejection
[ ] Grade D (Fair)	[ ] Grade D: rejected	BPG Search:	[ ] Minor revision
[ ] Grade E (Poor)		[ ] Existed	[ ] Major revision
		[ ] No records	

COMMENTS TO AUTHORS

GENERAL COMMENTS In this review the authors collect the findings that discuss the relation of RAS with ethnic background, particularly African Americans which can be important during clinical decisions about therapeutic strategy. Finally based on the present data they concluded that the inclusion of RAS blockade agents in combinational therapy for African Americans with hypertension is critical. The authors provide the latest review about this important issue which may have high clinical implication. SPECIFIC COMMENTS Although the manuscript was well-written in total, there are some minor points to be corrected; For example; Osteopontin, Angiotensin II amd RAS abbreviated several times.

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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
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COMMENTS TO AUTHORS

I can enjoy your review. I have few questions. 1) Is there any mechanism of bradykinin in African American? 2) Page 2; Is there any possibility that African American take higher dose of salt than white American ? 3) Page 7; Is there any same mechanism between Darl rat model and African American especially in ACE polymorphism? 4) In clinical situation, what is most important organ failure that contributes to high mortality of African American? I hear that importance of cardiovascular factor especially myocardial infarction.