Critique on Racial (Black–White) Divergence in the Association Between Adiponectin and Arterial Stiffness in Asymptomatic Young Adults: The Bogalusa Heart Study

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Adiponectin has emerged as an exciting adipocytokine that modulates insulin resistance and possesses anti-inflammatory and antiatherogenic properties. We know there are significant interethnic differences in the prevalence of both diabetes and vascular disease with evidence for increased arterial stiffness and reduced adiponectin levels in blacks. Although low plasma levels of adiponectin are associated with increased arterial stiffness, there are no data on the impact of race on the relationship between arterial stiffness and adiponectin. In this issue of *American Journal of Hypertension*, Nguyen *et al.* present data on the relationship between arterial stiffness and adiponectin in a young biracial community of Bogalusa, as part of a long-term cohort follow-up study. The authors conclude that plasma adiponectin levels predict arterial stiffness in blacks but not in whites.

The study is novel, has considerable power due to its magnitude, and is well presented. However, there are certain limitations. One major concern is the possibility of selection bias in the study. The Bogalusa study population consists of 1,203 subjects, 35% black, of whom only 991 nondiabetic subjects were considered eligible for entry in the study. The reason put forward by the investigators is unavailability of complete data on the remaining 212 subjects, which makes it difficult for us to judge if certain subjects may have been lost to follow-up (e.g., overweight, smokers, alcoholics).

Another area that requires detail is the smoking status of the study subjects as we have no information on those who may have quit smoking. A recent study showed a linear relationship between smoking status and pulse wave velocity (PWV), with intermediate values observed in former smokers and related to the duration of smoking cessation. In this study, smoking was an independent determinant of PWV, preceded only by age and blood pressure, highlighting the importance of this variable, particularly in blacks where 50% were smokers. However, as the nonsmoker group probably included former smokers, this may have led to falsely higher levels of PWV observed in “nonsmokers”. In fact, it is quite possible that if the smoking data had been more precise, adiponectin may not have emerged as a predictor of PWV, when adjusted for other variables.

Another area of concern in this study is the surprisingly poor correlation between PWV and age and blood pressure, also reflected in the low magnitude of the explained variance of PWV in the regression model, despite both age and blood pressure being included in the model. This is at odds with published literature and raises methodological concerns regarding the technique of PWV measurement in this study. On a related note, the investigators did not observe any significant difference in arterial stiffness between the two races in this study, which contradicts their previous published work. This may be due to the use of different techniques to measure arterial stiffness in the same population, reinforcing the need for a consensus on the assessment of arterial stiffness.

Finally, measurement of the different adiponectin isoforms in the study could have improved the strength of the relationship between PWV and adiponectin levels. The high molecular weight (HMW) form of adiponectin has recently been shown to be an independent predictor of endothelial function, suggesting that HMW adiponectin may be a more useful marker of endothelial dysfunction than total adiponectin.

Despite the caveats, this is the first study to explore the complex relationship between arterial stiffness, adiponectin, and race. We, however, need further studies to confirm these findings in different ethnic groups and expand these observations into diseased populations where arterial stiffness and adiponectin both assume greater biological importance.

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