Response to Type 2 Deiodinase Thr92Ala Polymorphism Is Not Associated With Arterial Hypertension in Type 2 Diabetes Mellitus Patients

Canani et al reported that a type 2 iodothyronine deiodinase (Dio2) nonsynonymous polymorphism, threonine 92 to alanine (Thr92Ala), was not associated with arterial hypertension in 315 patients with type 2 diabetes mellitus. We have recently reported that among euthyroid adults not on thyroid hormone replacement therapy the common Ala92 Dio2 allele approximately doubles the risk for the development of hypertension in 372 hypertensive and normotensive subjects.

We agree with Canani and colleagues that the discrepant results of the 2 studies may be explained by the differences in populations studied: patients with type 2 diabetes mellitus versus carefully selected patients with mild to moderate essential hypertension without any major comorbidities.

Essential hypertension is a complex heterogeneous trait caused by interplay between genetic and environmental factors, resulting in individual hypertensive patients having unique combinations of genes contributing to their blood pressure elevation. However, within the heterogeneous population of hypertensive patients, more genetically homogeneous subgroups (intermediate phenotypes) can be identified, defined by a common physiological or phenotypic trait that is used to focus genetic investigation. Type 2 diabetes mellitus is also a complex genetic disorder with a variety of genetic and environmental factors contributing to the common phenotype of hyperglycemia. It is, therefore, likely that pathogenesis and genetic determinants of hypertension in patients with type 2 diabetes mellitus differ from those in patients developing essential hypertension in the absence of type 2 diabetes.

Results of our study support an important role for genetic variation in the hypothalamic-pituitary-thyroid pathway in influencing susceptibility to hypertension. Further work is needed to uncover the mechanisms by which Dio2 may affect hypertension susceptibility, and to define subgroups of hypertensive patients where the effect of Dio2 is most relevant.

Disclosures

None.

Olga Gumieniak
Gordon H. Williams

Endocrinology, Diabetes, and Hypertension Division
Department of Medicine, Brigham and Women’s Hospital
Harvard Medical School
Boston, Mass

Response to Type 2 Deiodinase Thr92Ala Polymorphism Is Not Associated With Arterial Hypertension in Type 2 Diabetes Mellitus Patients
Olga Gumieniak and Gordon H. Williams

Hypertension. 2007;49:e48; originally published online April 9, 2007; doi: 10.1161/HYPERTENSIONAHA.107.088559
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2007 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/49/6/e48

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/