

## Reconsider Hypertension Phenotypes and Osteoporosis

### To the Editor:

The explosion of biomedical sciences and new molecular techniques of defining certain genes' polymorphisms was an important step in research of genetic influence of many conditions such as hypertension (HTN). Orias and colleagues<sup>1</sup> have proposed a quite interesting approach in clarifying the genetic background of HTN. Although epigenetic mechanisms such as DNA methylation, and gene-environment interactions are involved in the genetic determination of hypertension, there is a great need for separate analysis of patients according to the type of HTN (diastolic, systolic, isolated, or predominantly). Orias and colleagues<sup>1</sup> also referred in their aforementioned paper to the need for searching more specific hypertension phenotypes linked with apparently unrelated associations of HTN, including osteoporosis.

Although osteoporosis seems to be an unrelated condition with HTN, they often coexist as two major age-related disorders. Except the paradigm of familial linkage (*LRP6* gene) for coronary artery disease, metabolic syndrome, and osteoporosis described by Mani and colleagues,<sup>2</sup> there have been several reports recently indicating the link between hypertension and osteoporosis.<sup>3-6</sup> Among them, a recent study by Asaba and colleagues<sup>7</sup> proposed that blocking the synthesis of angiotensin II may be an effective treatment of osteoporosis and hypertension, especially for patients afflicted with both conditions. Taking into account the hypothesis of Orias and colleagues<sup>1</sup> that younger patients have potentially less environmental influence on their blood pressure (and are therefore better suited for genetic and/or mechanistic protocols), an intensified attempt at defining phenotypes in this age group is essential. In addition, we propose the need for examining not only common genetic polymorphism affecting HTN and bone metabolism but also bone metabolism biomarkers in this age group according to several subtypes, which could also be useful in the early identification of individuals who may develop osteoporosis later in their life. An example

of a biomarker that might join both conditions could be vitamin D (and its receptors), which was recently intensively investigated concerning its correlation not only to bone metabolism<sup>8,9</sup> but mainly to metabolic syndrome and its components (eg, arterial HTN).<sup>10-15</sup>

In conclusion, we believe that age-related diseases such as HTN and osteoporosis may share a common genetic and pathophysiologic background. The paper by Orias and colleagues may be an important paper for further discussion on genetic research in both HTN and even apparently unrelated conditions such as osteoporosis. Therefore, the need for approaching the onset of both diseases is essential in order to have better therapeutic anti-hypertensive and antiosteoporotic interventions.

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