Does Arginase Activity In Vitro Represent That In Vivo?

To the Editor:

In the recent study by White et al,1 NO synthase (NOS) and arginase activities in rat aortic vessels were measured by standard methods, resulting in the conclusion that arginase plays a significant role in endothelial dysfunction by reducing endothelial NO bioavailability. As with all in vitro enzyme activity studies, assays are performed under the conditions that are not exactly the same as those in vivo, usually under optimal conditions, which is particularly the case with the assay for arginase activity. Lack of urea formation and lack of $[^{14}C]$ ornithine formation from $[^{14}C]$ arginine in cultured endothelial cells suggested that arginase is either not present or not active.2 The conclusion that arginase overexpression is responsible for aging induced endothelial dysfunction is also contradictory to the “arginine paradox,”3 which states that extracellular arginine is principally used by endothelial NOS to produce NO. This finding is explained by the fact that CAT1 and endothelial NOS are functionally and morphologically associated.4 In conclusion, modification of arginase activity and, thus, “cytosolic” intracellular arginine concentration in endothelial cells may not be relevant to NO production; further studies using transgenic, knockout, and selective arginase inhibitors are required to define the role of arginase in disease pathophysiology.

Disclosures

None.

Wei-Zheng Zhang
David M. Kaye
Wynn Department of Metabolic Cardiology
Baker Heart Research Institute
Melbourne, Australia

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Wei-Zheng Zhang and David M. Kaye

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