Response to Polycystic Ovary Syndrome: Implications of Corticotropin in the Regulation of Blood Pressure, Aldosterone, and Androgen Secretion

We thank Armanini et al1 for their interests and comments on our work2 regarding the association between hyperandrogenemia and elevated systolic and diastolic blood pressures in young women with polycystic ovary syndrome.

Armanini et al1 propose that the chronic corticotropin drive could activate aldosterone synthesis and adrenal androgen secretion and, therefore, increase the blood pressure. In our study, we did not find a positive correlation between dehydroepiandrosterone sulfate and blood pressure. Another recent study3 reported that aldosterone was independently related to the insulin resistance and, therefore, was related to the blood pressure level in women with polycystic ovary syndrome. Excess androgen and aldosterone could both be associated with insulin resistance. Therefore, further investigation is required to verify where there is an association between androgen and aldosterone that results in the increase of blood pressures. It could also be possible that the increase of blood pressures caused by either androgen or aldosterone might be through a common pathway, that is, insulin resistance.

Because of the close association between hyperandrogenism and blood pressures, we totally agree with Armanini et al4 that the antiandrogen therapy such as spironolactone and licorice might be beneficial in treating hypertension in women with polycystic ovary syndrome. However, further clinical trials for larger populations and longer follow-up periods are necessary to confirm this effect.

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Disclosures
None.

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