Angiotensin II Type 1 Receptor Autoantibodies (AT1AA):

Cause or Consequence of Human Primary Aldosteronism?

Maria Piazza, Teresa M. Seccia, Brasilina Caroccia, Giacomo Rossitto, Riccardo Scarpa,

Perla Persichitti, Daniela Basso and Gian Paolo Rossi

Department of Medicine-DIMED, University of Padua, Italy

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Correspondence:

Gian Paolo Rossi, MD, FACC, FAHA

Clinica dell’Ipertensione Arteriosa

Department of Medicine-DIMED

University Hospital

Via Giustiniani, 2

35128 Padova, Italy

Phone: +39-049-821-7821

gianpaolo.rossi@unipd.it

**Supplemental Methods**

Deming regression was used to look for systematic differences between methods.

The null hypothesis of identity (i.e. that Y = X) was tested by individual tests: the 95% confidence interval for the intercept to test the hypothesis that A=0. This hypothesis is accepted if the confidence interval for A contains the value 0, and rejected if A is significantly different from 0 and both methods differ at least by a constant amount. The 95% confidence interval for the slope was used to test the hypothesis that B=1; this hypothesis is accepted if the confidence interval for B contains the value 1. If the hypothesis is rejected, then it is concluded that B is significantly different from 1 and there is at least a proportional difference between the two methods.

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**Supplemental Figure 1**. Panel A. Dose-response curve of Angiotensin II (10-10M-10-5M) in engineered AT1R‐transfected Chinese hamster ovary (CHO) cells, assessed as chemiluminescent response. Engineered cells exposed to sera or purified IgG from APA patients provided low chemiluminescent responses that fell in the shaded area, corresponding to 5% equivalent bioactivity of the maximal response elicited by Angiotensin II. Panel B. Bioactivity measured in CHO cells exposed to sera or purified IgG from APA patients did not significantly differ from that of healthy normotensive subjects (HD).