Online Supplement

Estrous cycle dependent neurovascular dysfunction induced by angiotensin II in the mouse neocortex

Carmen Capone[1], Josef Anrather[1], Teresa A. Milner[1,2], and Costantino Iadecola[1]

[1]Division of Neurobiology
Department of Neurology and Neuroscience
Weill Cornell Medical College
1300 York Avenue
New York, NY 10021, USA

[2]Harold and Margaret Milliken Hatch Laboratory of Neuroendocrinology
The Rockefeller University
New York, NY 10065

Running head: Estrus cycle and cerebrovascular effects of AngII

Correspondence:
Costantino Iadecola, M.D.
Division of Neurobiology
Weill Cornell Medical College
407 East 61st Street
New York, NY 10065
Phone: 646/962-8279
Email: coi2001@med.cornell.edu

Supplemental table S1

Arterial blood gases and pH in the mice in which CBF was studied

Cycle phase	Treatment	n	pCO_2 (mmHg)	pO_2 (mmHg)	pH
Proestrus	saline	6	36.6±1.2	129.4±3.8	7.37±0.05
	AngII		35.8±1.1	132.2±3.7	7.37±0.04
Estrus	saline	6	35.9±1.7	128.8±4.6	7.35±0.06
	AngII		37.1±1.8	133.1±2.5	7.36±0.06
Diestrus	saline	6	36.9±1.5	131.3±4.2	7.35±0.07
	AngII		36.1±2.1	129.8±2.9	7.37±0.06
Proestrus+ ICI182,780	saline	5	36.9±1.4	130.1±4.8	7.36±0.05
	AngII		37.3±1.3	130.9±4.3	7.37±0.07

Online figures

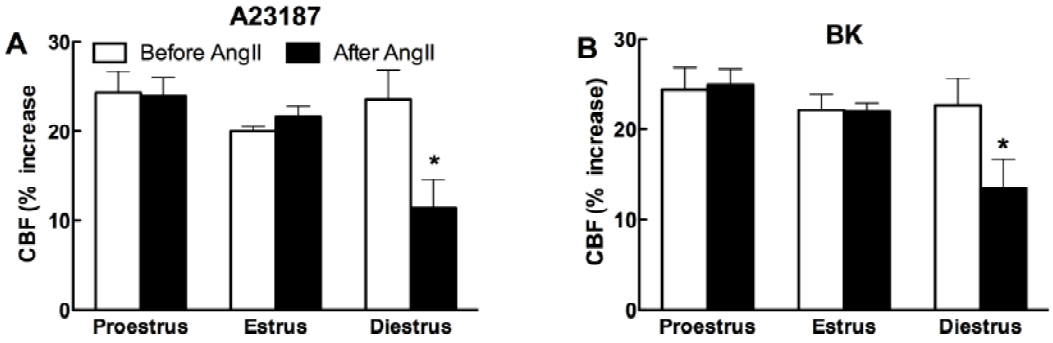

Figure S1

Figure S1: Cerebrovascular effect of acute i.v. administration of AngII in cycling female mice. Effect of AngII on CBF increase produced by A23187 (A) or BK (B). *p<0.05 from respective control before AngII; analysis of variance and Tukey's test; n=6/group.

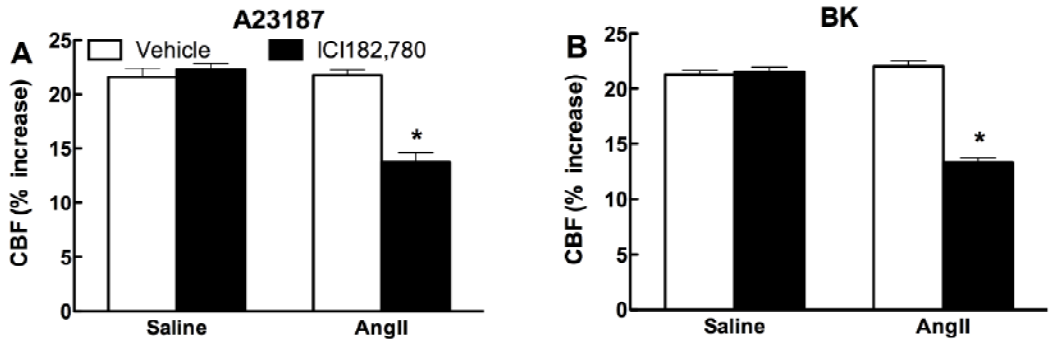

Figure S2

Figure S2: Cerebrovascular effect of AngII in proestrus mice treated with the estrogen receptor inhibitor ICI182,780 (10μM; neocortical application). Effect of AngII on the CBF increase produced by A23187 (A) or BK (B) *p<0.05 from vehicle; analysis of variance and Tukey's test; n=5/group.