European Society of Gynecology
Société Européenne de Gynécologie

10 Congress of the European Society of Gynecology
Congrès de la Société Européenne de Gynécologie

SQUARE-BRUSSELS MEETING CENTRE 18/21 Sept 2013

Certificate of Attendance

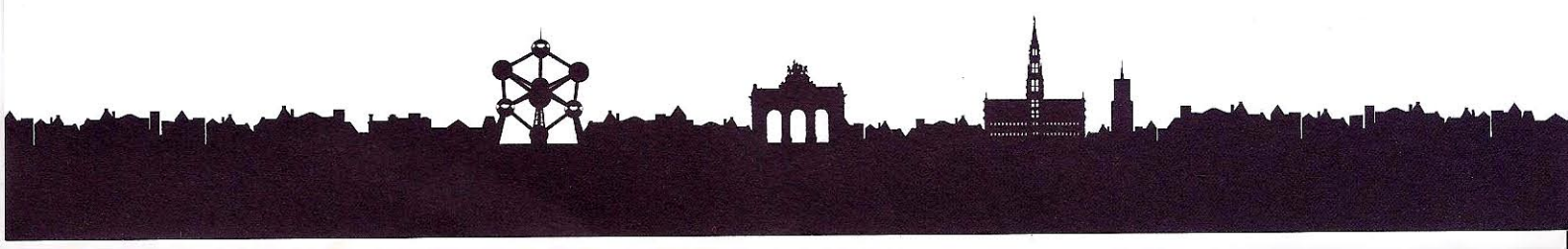
Certificat de participation

Dr. Adrián Esteban Campelo

has attended the 10th Congress of the European Society of Gynecology held in Brussels, Belgium, on September 18/21 2013.

a participé au 10ème Congrès de la Société Européenne de Gynécologie, tenu à Bruxelles, Belgique, du 18 au 21 septembre 2013

Jean Michel Foidart
Chairman of the congress



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Square Meeting Centre, Bruxelles 18/21 Sept 2013

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Kharkov medical academy of postgradual study

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[3]Centre de référence et registre des maladies trophoblastiques gestationnelles, Liège, Belgique

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"Iuliu Hatieganu" University of Medicine and Pharmacy Cluj-Napoca

31. Atypical hyperplasia and incidence of endometrial carcinoma
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Cellular and molecular actions of testosterone in vascular system

A. Campelo, M. Montt, T. Simoncini, V. Massheimer

Vascular function is regulated by various agonists including the sex steroid hormones estrogen, progesterone and androgens. In order to contribute to the knowledge of the role of androgens on vascular function, in this work we investigated the cellular and molecular actions of testosterone (T) on the regulation of cellular events involved in vascular physiology such as nitric oxide (NO) production, proliferation and migration of rat aortic endothelial cells (EC).

We provide evidence that T (10-7 a 10-11M) induces an acute stimulation of NO production in EC (15 to 125% above/control, $p<0.01$), in a gene transcription independent manner and independently of aromatase action. The mechanism of action of the steroid involves the participation of the androgen receptor (82 vs 2% s/control, T vs T+Flutamide respectively, $p<0.05$), and is dependent on MAPK (4.24±1.11 vs 7.80±0.93; 4.51±0.73 vs 4.40±0.72 nmol NO/mg prot, C vs T; C+PD98059 vs T+PD98059,$p<0.001$) , PLC/PKC (4.25±1.10 vs 7.84±1.27; 4.62±0.87 vs 4.95±0.89 nmol NO/mg prot, C vs T; C+Chelerythrine vs T+Chelerythrine,$p<0.001$) and PI3K/Akt (4.30±0.83 vs 7.52±0.98; 4,82±0.77 vs 4.31±0.52 nmol NO/mg prot, C vs T -/+ LY294002) signaling pathways activation and dependent of the influx of extracellular calcium (82.6; 29.6; 23.2 % above/control, T; T+EGTA; T+Verapamil).

Related to the cellular processes involved in vascular repair, we demonstrate that T promotes EC proliferation (3[H]-timidine incorporation) (17.3±1.4 vs 25.4±4.4, C vs T, $p<0.05$). The mitogenic action exhibited by the steroid is dependent on endothelial NO production since the proliferative effect is significantly reduced by the presence of the NOS inhibitor L-NAME. By means of wound healing essays it was also determined that T stimulates also EC migration. These results provide knowledge about vascular actions of T and the mechanisms of action involved.

Cellular and Molecular Actions of Testosterone in Vascular System

Adrián Campelo; Magdalena Montt-Guevara; Tommaso Simoncini; Virginia Massheimer

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Departamento de Biología, Bioquímica y Farmacia, Universidad Nacional del Sur, Bahía Blanca, Argentina - CONICET

INTRODUCTION

Vascular function is regulated by various agonists including the sex steroid hormones estrogen, progesterone and androgens. In order to contribute to the knowledge of the role of androgens on vascular function, in this work we investigated the cellular and molecular actions of testosterone on the regulation of cellular events involved in vascular physiology such as nitric oxide (NO) production, proliferation and migration of rat aortic endothelial cells (EC).

METHODS

Endothelial cells cultures: EC cultures were obtained from rat aortic rings explants isolated from young Wistar female rats (3-5 weeks old) and were identified by positive immunocytochemistry reactivity to CD34 and VEGFR2.
Nitric oxide production: was measured by Griess reaction. ECs were allowed to grow to 90% of confluence and immediately after hormonal treatment nitrites were measured in the incubation media as a stable and non-volatile breakdown product of the NO released using a microplate reader.
Thymidine incorporation assay: ECs were allowed to grow to 60-70% confluence and exposed to different hormonal treatments. The cells were pulsed with 1 µCi/mL of [^{3}H]-thymidine during the last 2 hours of treatment. Radioactivity was measured by liquid scintillation and the protein concentration was determined by Lowry method.
Cell migration assays: ECs were grown to 90% confluence and in order to evaluate EC migration a wound was made by means of tip, removing the cells along a straight line. After hormonal treatment cells were fixed and stained with Giemsa and migration was quantified by counting the number of cells present in the scratched area in ten different microscopic fields.
Statistical analysis: Each experimental condition was reproduced in at least three independent experiments performed by quadruplicate. All data are presented as mean ± SD. Comparisons between two means were made using Student's *t*-test, and multiple comparisons with one or two ways ANOVA followed by Fisher LSD. Differences at $p<0.05$ were considered significant. (*$p<0.05$; **$p<0.01$).

RESULTS

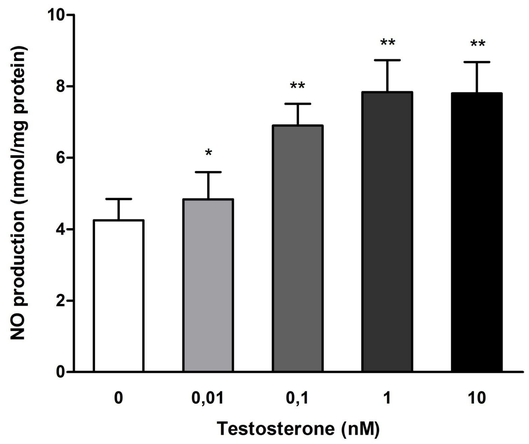


Figure 1. Dose–response profile of testosterone stimulation of endothelial NO production. ECs were cultured in the presence of FBS 1% (v/v) and were incubated with testosterone at the indicated concentrations for 5 min. NO production was measured as described in methods

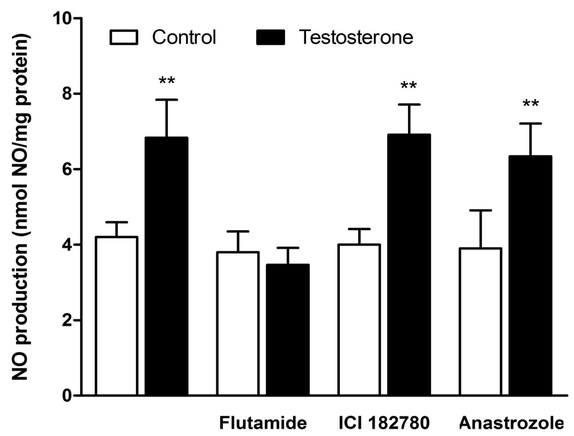


Figure 2. Participation of the AR, ER and aromatase on NO production induced by testosterone. ECs were preincubated in absence or presence of 50nM flutamide (AR antagonist), 5µM ICI 182780 (ER antagonist) or 50nM anastrozole (aromatase inhibitor) for 60 min, and then exposed to 1nM testosterone for 5 min.

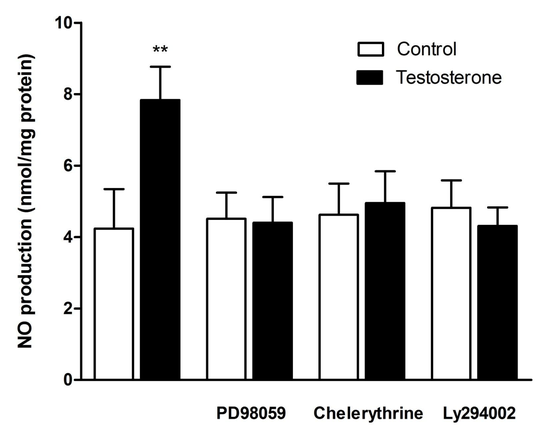


Figure 3. Characterization of the rapid action of testosterone on endothelial NO production. ECs were preincubated with 1µM PD98059 , 1µM chelerythrine or 0,5µM Ly294002 for 1 hour and then treated with 1nM testosterone for 5 min.

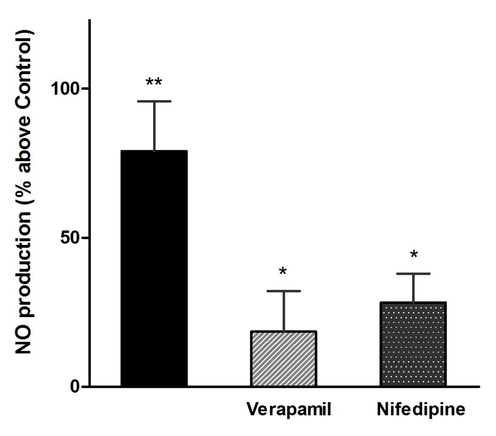


Figure 4. Effect of extracellular calcium on testosterone-induced NO synthesis. ECs were preincubated in the presence or absence of a calcium channel blocker verapamil (50µM) or nifedipine (1µM) for 1 hour and then exposed to 1nM testosterone for 5 min.

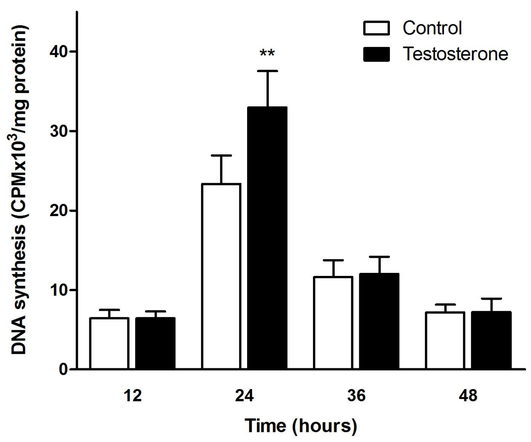


Figure 5. Effect of testosterone on [^{3}H]-thymidine incorporation. Subconfluent ECs were treated with 1nM testosterone at the indicated times. 1mCi/ml of [^{3}H]-thymidine was added during the last 2 h of treatment and [^{3}H]-thymidine incorporation was then measured by standard technique and expressed as counts per minute per milligram of protein.

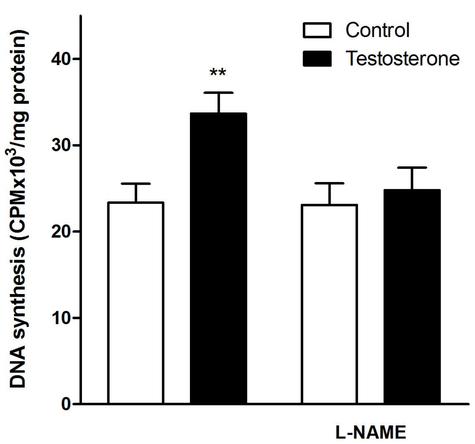
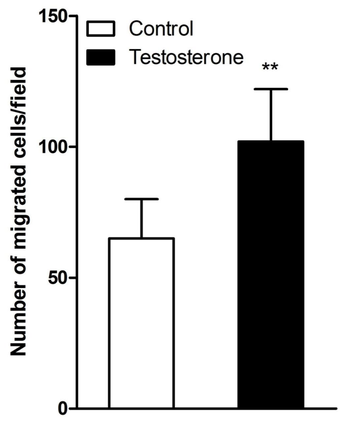


Figure 6. Effect of L-NAME on DNA synthesis induced by testosterone. Subconfluent Ecs were preincubated for 1 hour with or without L-NAME (1mM), and then treated with testosterone for additional 24 h. [^{3}H]-thymidine incorporation was measured as described in methods.



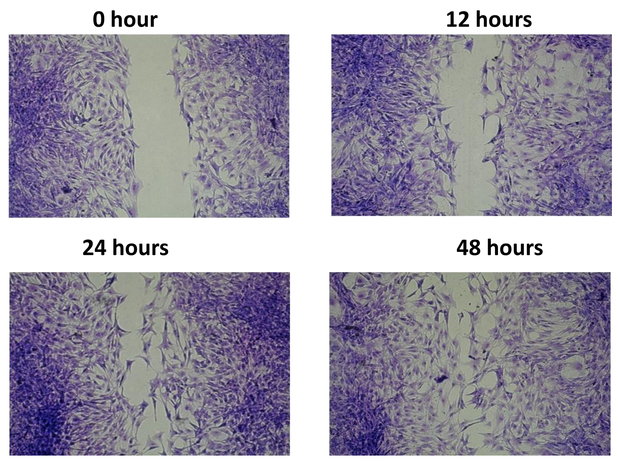


Figure 7. Effect of testosterone on EC migration. Confluent ECs cultures were serum starved for 24 h and cells were removed by making a straight wound with a pipette tip. Detached cells were washed with PBS and the remaining monolayer was treated for 24 h with 1nM testosterone or vehicle. Images of representative fields after Giemsa staining .

CONCLUSION

These results suggest that testosterone stimulates NO production in endothelial cells in a non genomic manner independently of ER participation and in a AR dependent manner. The androgen also stimulates the genomic effects of endothelial cell proliferation and migration. It was demonstrated that the proliferative action of testosterone depends on NO generation suggesting this a possible two step mechanism of the androgen in the regulation of ECs growth.

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