Endocrine Abstracts

May 2017 Volume 49
ISSN 1479-6848 (online)

19th European Congress of Endocrinology 2017

20–23 May 2017, Lisbon, Portugal

ECE 2017
19th European Congress of Endocrinology

published by
bioscientifica

Online version available at
www.endocrine-abstracts.org

Endocrine Abstracts

Volume 49
May 2017

19th European Congress of Endocrinology 2017
20–23 May 2017

EDITORS

The abstracts were marked by the Abstract marking Panel selected by the Programme Organising Committee

Programme Organising Committee

Bulent Yildiz (Turkey) Chair
Guillaume Assié (France) Co-chair
Riccarda Granata (Italy) Co-chair
João Jácome de Castro (Portugal)
Carlo Acerini (UK)
Richard Bergman (USA)
Charlotte Bevan (UK)
Heike Biebermann (Germany)
Evi Diamanti-Kandarakis (Greece)
Andrea Giustina (Italy)
Ilpo Huhtaniemi (Finland/UK)
Irena Ilovayskaya (Russia)
Tatjana isailovic (Serbia)
Gregory Kaltsas (Greece)
Beata Kos-Kudla (Poland)
Cesar Luiz Boguszewski (Brazil)
Djuro Macut (Serbia)
Rod Mitchell (UK)
Barbara Obermayer-Pietsch (Austria)
Pedro Oliveira (Portugal)
Robin Peeters (The Netherlands)
Duarte Pignatelli (Portugal)
Matti Poutanen (Finland)
Manuel Puig (Spain)

Local Organising Committee (LOC)

João de Castro (Lisbon, Portugal) (Chair)
Duarte Pignatelli (Porto, Portugal)
Jorge Dores (Porto, Portugal)
José Silva Nunes (Lisbon, Portugal)
Leonor Gomes (Coimbra, Portugal)
Mafalda Marcelino (Lisbon, Portugal)
Maria João Oliveira (Porto, Portugal)
Mário Mascarenha (Lisbon, Portugal)
Miguel Melo (Coimbra, Portugal)

Abstract Marking Panel

T Battelino Slovenia
K Chatterjee UK
D Cuthbertson UK
E de Koning The Netherlands
M Dentice Italy
D Fuhrer Germany
R Gärtner Germany
J Jacome de Castro Portugal
C Jazdzewski Poland
C Kanaka-Gantenbein Greece
S Pearce UK
G Roman Romania
E Stener-Victorin Sweden
E Visser The Netherlands
M-C Zennaro Spain
C Acerini UK
M Alevizaki Greece
W Arlt UK
G Assié France
S Aylwin UK
S Babajko France
K Badenhoop Germany
P Beck-Peccoz Italy
R Bergman USA
J Bertherat France
F Beuschlein Germany
C Bevan UK
H Biebermann Germany
M Bluher Germany
K Boelaert UK
A Boelen The Netherlands
R Bouillon Belgium
J-P Bourguignon Belgium
G Brabant Germany
M L Brandi Italy
K Briot France
G Brunetti Italy
C Buchanan UK
P Burman Sweden
S Cannavo Italy
J Cap Czech Republic
G Carmeliet Belgium
J Castano Spain
P Chanson France
F Chiarelli Italy
B Chini Italy
J Chowen Spain
C Daousi UK
M Dattani UK
C Dayan UK
W de Herder Netherlands
W Dhillo UK
G Di Dalmazi Germany
E Diamanti-Kandarakis Greece
F Dotta Italy
J Drouin Canada
L Duntas Greece
G Eisenhofer Germany
F Fallo Italy
P Farahani Canada
S Farooqi UK
M Fassnacht Germany
R Feelders The Netherlands
U Feldt-Rasmussen Denmark
E Fliers The Netherlands
C Flueck Switzerland
C Follin Sweden
S Franks UK
W Fraser UK
J Frystyk Denmark
L Fugazzolla Italy
F Gabalec Czech Republic
A Gimenez-Roqueplo France
F Giorgino Italy
A Giustina Italy
J Gomez-Ambrosi Spain
R Granata Italy
A Grossman UK
R Hampl Czech Republic
M Heikinheimo Finland
C Hoefig Sweden
A Hoeflich Germany
W Hoegler UK
L Hofland Netherlands
I Huhtaniemi UK
E Husebye Norway
P Igaz Hungary
I Ilovayskaya Russia
S Inzucchi USA
T Isailovic Serbia
E R Isenovic Serbia
M Jaffrain-Rea Italy
B Jarzab Poland
D Jezova Slovakia
A Kalsbeek Netherlands
G Kaltsas Greece
A Karlsson Sweden
M Keil USA
F Kelestimur Turkey
R Kineman USA
M Korbonits UK
B Kos-Kudla Poland
N Krone UK
H Krude Germany
M Laan Germany
E Lalli France
J Laven The Netherlands
G Lavery UK
J Leger France
T Links The Netherlands
P Lips Netherlands
S Llahana UK
A Luger Austria
C Luiz Boguszewski Brazil
R M Luque Spain
M Luster Germany
D Macut Serbia
M Maggi Italy
J Magre France
M Mannelli Italy
F Mantero Italy
D Marks USA
G Mastorakos Greece
C McCabe UK
R Mitchell UK
J Mittag Germany
L Morin-Papunen Finland
N Morton UK
A Mukherjee UK
E Nagy Hungary
J Newell-Price UK
B Obermayer-Pietsch Austria
P Oliveira Portugal
U Pagotto Italy
J J Palvimo Finland
R Peeters The Netherlands
L Persani Italy
T Pieber Austria
V Pirags Latvia
N Pitteloud Switzerland
M Poutanen Finland
D Power Portugal
V Prevot France
S Radian UK
N Rahman Finland
B Rainey USA
E Rajpert-De Meyts Denmark
G Raverot France
M Reincke Germany
S Rice UK
M Robledo Spain
P Rodien France
H Romijn The Netherlands
C Ronchi Italy
R Ross UK
G P Rossi Italy
M Ruchala Poland
E Rutten Belgium
D Salvatore Italy
S Sanlioglu Turkey
P Saunders UK
S Schmid Germany
J Schopohl Germany
D Schulte Germany
M Sherlock Ireland
M Simoni Italy
J Smit The Netherlands
A Spada Italy
G Stalla Germany
C Stratakis USA
T Tankova Bulgaria
M Tena-Sempere Spain
M Terzolo Italy
M Theodoropoulou Germany
C Thompson Ireland
H Timmers The Netherlands
J Toppari Finland
M Toth Hungary
S Tsagarakis Greece
M Tzanela Greece
E Valassi Spain
E van den Akker Netherlands
A van der Klaauw UK
A J van der Lelij Netherlands
J van Eck The Netherlands
W van Hul Belgium
J Visser The Netherlands
V Volke Estonia
R Voutilainen Finland
I Wilkinson UK
Z Wu Germany
P Yeoh UK
B Yildiz Turkey
J Young France
M Zatelli Italy

SPONSORS

The ESE would like to thank its Corporate Members and the ECE 2017 sponsors

ECE Corporate Members

Aegerion
Chiasma
Ipsen
Laboratoire HRA Pharma
Novartis Pharmaceuticals
Novo Nordisk
Pfizer
Sandoz International Gmbh
Shire Services BVBA
Strongbridge Biopharma

Gold Sponsors

Ipsen
Novartis Pharmaceuticals
Pfizer

ESE Office
Euro House
22 Apex Court
Woodlands
Bradley Stoke
Bristol BS32 4JT, UK

Contact: Andrea Davis
Tel: +44 (0)1454 642247
Fax: +44 (0)1454 642222
E-mail: info@euro-endo.org
Web site: www.ese-hormones.org

ECE 2016 Secretariat
Bioscientifica Ltd
Euro House, 22 Apex Court
Woodlands
Bradley Stoke
Bristol BS32 4JT, UK

Contact: Niki Cripps
Tel: +44 (0)1454 640467
Fax: +44 (0)1454 642222
E-mail: ece2016@bioscientifica.com
Website: http://www.bioscientifica.com

CONTENTS

19th European Congress of Endocrinology 2017

PRIZE LECTURES AND BIOGRAPHICAL NOTES

PLENARY LECTURES

SYMPOSIA

EPOSTER PRESENTATIONS: INTERDISCIPLINARY ENDOCRINOLOGY

EPOSTER PRESENTATIONS: PITUITARY AND NEUROENDOCRINOLOGY

EPOSTER PRESENTATIONS: REPRODUCTIVE ENDOCRINOLOGY

Eposter Presentations: Reproductive Endocrinology

Conclusion and relevance
Men with early-onset AGA and at least one among the following parameters BMI >25 kg/m^2, IR, SHBG <25 nmol/l had a borderline-low left TV and a impaired gonadal steroidogenesis. Hence, they might have a greater risk to develop a gonadal dysfunction later in life. These criteria may be used to suspect the male PCOS-equivalent.

DOI: 10.1530/endoabs.49.EP1163

EP1164

Improved conception of the normative values of testosterone in men with type 2 diabetes

Irina Khripun[1], Sergey Vorobyev[1], Elizaveta Dzantieva[1], Igor Belousov[1], Michail Kogan[1] & Michael Zitzmann[2]
[1]Rostov State Medical University, Rostov-on-Don, Russia; [2]University Clinics Muenster, Muenster, Germany.

Background
Men with type 2 diabetes mellitus (T2DM) have significantly lower levels of testosterone (T) than nondiabetic controls. But androgen deficit symptoms may be detected in males with normal T levels.
Aim
To analyze parameters of lipid, carbohydrate metabolism and endothelial function in diabetic males with low normal and middle-to-high normative T levels.
Patients and methods
We examined 86 men with T2DM and serum T levels higher than 12.1 nmol/l. Parameters of total T, lipid metabolism, HbA1c, biochemical markers of endothelial function – NO, endothelial NO synthase type 3 (NOS3) were analyzed. The patients were divided into two groups: 1–23 men with T levels 12.1–14.9 nmol/l, 2–63 patients with T levels $\geqq 15.0$ nmol/l. Statistic analysis was performed using Mann-Whitney U-test and Spearmen rank correlation method.
Results
The HbA1c value was 8.7% (6.7; 9.8) in 1st group and 7.7% (6.4; 9.2) in 2nd group ($P>0.39$), but the proportion of patients with HbA1c level $>7\%$ was higher in group 1 (71% vs 58.6%) as compared to group 2 ($P=0.002$). In patients with T levels lower than 15.0 nmol/l, higher levels of serum cholesterol (38.1% vs 27.6%), triglycerides (40.0% vs 24.1%) and low density lipoproteins (26.3% vs 17.2%) as compared to the 2nd group were found ($P<0.01$). There was a significant correlation in the T concentrations with NOS3 levels $|r|=0.350$ in 1st and $|r|=0.266$, ($P<0.05$) in 2nd group. Vascular endothelial dysfunction as assessed by ultrasonographic measurement of the dilatation of the brachial artery was more frequent in patients of group 1 compared to group 2 (55.6% vs 23.8%, $P=0.02$).
Conclusion
Males with T2DM and low-normal T level are at higher risks of dyslipidemia, endothelial dysfunction and progression of T2DM as compared to men with serum T higher than >15 nmol/l.
Acknowledgments
Supported by RSF, grant No. 14-25-00052.

DOI: 10.1530/endoabs.49.EP1164

EP1165

Endothelial dysfunction, inflammation and insulin resistance in patients with Klinefelter Syndrome

Cem Haymana[1], Aydogan Aydogdu[1], Ibrahim Demirci[1], Mustafa Dinc[1], Orhan Demir[1], Deniz Torun[2], Fatih Yesildal[3], Coskun Meric[1], Yalcin Basaran[1], Alper Sonmez[1] & Omer Azal[1]
[1]Gulhane Training and Research Hospital, Department of Endocrinology and Metabolism, Ankara, Turkey; [2]Gulhane Training and Research Hospital, Department of Medical Genetics, Ankara, Turkey; [3]Selahaddin Eyyubi Public Hospital, Department of Biochemistry, Diyarbakir, Turkey.

Introduction
The prevalence of cardiometabolic disorders such as type 2 diabetes mellitus, dysipidemia and metabolic syndrome is increased in patients with Klinefelter syndrome (KS). The mechanism by which cardiometabolic risk increases in patients with KS remains to be completely elucidated. We investigated the presence of inflammation, insulin resistance and endothelial dysfunction in an unconfounded population of KS.
Methods
A total of 31 patients with KS (mean age 21.59 ± 1.66 years) and 33 healthy control subjects (mean age: 22.15 ± 1.03 years) were enrolled. The demographic parameters, Asymmetric dimethylarginine (ADMA), high sensitive C reactive protein (hs-CRP) and homeostatic model assessment of insulin resistance (HOMA-IR) levels were measured in patients and controls.
Results
The patients had higher insulin, HOMA-IR and ADMA levels ($P<0.001$ for all) and lower HDL-C ($P=0.002$) and total testosterone ($P<0.001$) levels, compared to the healthy controls. There were significant negative correlations between the total testosterone levels and ADMA ($r=-0.479$, $P<0,001$), hsCRP ($r=-0.291$, $P=0.034$), and significant positive correlation with HDL-C ($r=0.429$, $P=0.001$) levels. The multivariate analysis has shown that total testosterone ($\beta=-0.412$, $P=0.001$) and TG ($\beta=0.332$, $P=0.009$) levels were the significant independent determinants of the plasma ADMA levels.
Conclusion
The results of the present study show that endothelial dysfunction and insulin resistance are prevalent even in the very young subjects with KS, who have no metabolic or cardiac problems at present.

DOI: 10.1530/endoabs.49.EP1165

EP1166

Testosterone is poorly related to erectile dysfunction in young/middle aged human immunodeficiency virus-infected men

Sara De Vincentis[1], Daniele Santi[1], Maria Chiara Decaroli[1], Flaminia Fanelli[2,3], Marco Mezzullo[2,3], Alessia Fazzini[2,3], Anna Ansaloni[1], Uberto Pagotto[2,3], Giovanni Guaraldi[4] & Vincenzo Rochira[1]
[1]Unit of Endocrinology, University of Modena and Reggio Emilia, Modena, Italy; [2]Unit of Endocrinology, University of Bologna, Bologna, Italy; [3]Center for Applied Biomedical Research, University of Bologna, Bologna, Italy; [4]Unit of Infectious Diseases, University of Modena and Reggio Emilia, Modena, Italy.

Background
human immunodeficiency virus (HIV)-infection is strongly associated to erectile dysfunction (ED) in men. Preliminary data suggests that ED is poorly associated with serum T in HIV+ men.
Aim
To investigate in HIV-infected men the relationship between sexual function as assessed by the validated International Index of Erectile Function (IIEF-15) and T deficiency using Liquid Chromatography-tandem Mass Spectrometry (LC-MS/MS).
Methodology
Prospective, cross-sectional, observational study on HIV-infected male patients with ongoing Highly Active Antiretroviral Therapy (HAART), attending the Clinic of Infectious Diseases. IIEF-15 and IIEF-5 questionnaires were used to define ED, while LC-MS/MS was used for hormonal assays.
Results
233 consecutive HIV-infected patients were enrolled (mean age 45.29 ± 5.33 years). Eight patients (3.4%) had total T <300 ng/dl, while 142 patients (61.5%) had ED (score ≤ 25). Age, hormonal data and duration of HIV-infection and HAART did not differ among groups of patients according to the degree of ED. The direct comparison of each ED cluster showed that months of infection were significantly higher in men with severe ED compared to mild ED ($P=0.037$). The erectile function domain at IIEF-15 was directly correlated with IIEF-5 score (0.778, $P<0.001$), as expected. Moreover, the IIEF-15 score was inversely related to months of infection (-0.147, $P=0.026$), but not to months of HAART therapy (-0.121, $P=0.071$).
Conclusions
To the best of our knowledge, this is the first, properly-designed prospective study aiming to investigate the relationship between erectile function and serum T, assessed by LC-MS/MS in HIV-infected men. In our cohort, i) IIEF-5 is reliable as IIEF-15 for ED diagnosis, ii) ED is not associated with serum T, iii) erectile

function is not influenced by T and HAART, but only by HIV-infection duration. In conclusion, several specific factors, such as the duration of HIV infection, are involved in erectile function in HIV-infected men and should be carefully considered in this setting, while hormonal status seems to be less important.

DOI: 10.1530/endoabs.49.EP1166

EP1167

Early arterial stiffness and chronic inflammation in male equivalents of polycystic ovary syndrome

Mehmet Sozen[1], Suleyman Baldane[2], Sedat Abusoglu[3], Levent Kebapcilar[2], Aysegul Kebapcilar[4], Fatma Akyurek[5], Ali Unlu[3] & Suleyman Hilmi Ipekci[2]
[1]Deparment of Internal Medicine, Selcuk University Faculty of Medicine, Konya, Turkey; [2]Division of Endocrinology and Metabolism, Selcuk University Faculty of Medicine, Konya, Turkey; [3]Department of Biochemistry, Selcuk University Faculty of Medicine, Konya, Turkey; [4]Department of Obstetrics and Gynecology, Selcuk University Faculty of Medicine, Konya, Turkey; [5]Department of Dermatology, Selcuk University Faculty of Medicine, Konya, Turkey.

Introduction
There is some evidence that a rise in the androgen hormone levels increases the risk for the development of a cardiovascular disease, obtained from the studies conducted on women with polycystic ovary and on men with androgenic alopecia. Inflammatory substances tend to increase in polycystic ovary syndrome and in androgenic alopecia. On this basis, we conducted a study that aimed to evaluate the early cardiovascular and metabolic effects in male patients with androgenic alopecia.
Methods
A total of 81 people, of whom 41 were patients with androgenic alopecia and 40 were healthy controls, were included in the study. Aged from 25 to 45, none of them had any cardiovascular risk factors, malignancy, any active infections and any liver or kidney diseases. Ambulatory blood pressure was measured for 24 h and sensitive CRP and galectin-3 were studied so as to assess the cardiovascular and metabolic risk.
Results
There were no differences between the patient and control groups in terms of ambulatory blood pressure of 24 h, sensitive CRP and galectin-3. A positive correlation was found in the patient group between sensitive CRP and waist and neck circumferences. While there was a positive correlation in the patient group between galektin-3 and HOMA-IR, waist and neck circumferences, a negative correlation was seen with free testosterone. Alopecia level correlated positively with daytime pulse wave velocity and night time reflection magnitude.
Conclusion
We did not find any difference in our study in terms of arterial stiffness and chronic inflammation in the early period when the control and androgenic alopecia groups were compared; however, a positive correlation between alopecia level and daytime pulse wave velocity and night time reflection magnitude may be considered as an early signal for atherosclerosis.

DOI: 10.1530/endoabs.49.EP1167

EP1168

Abstract withdrawn.

EP1169

Can salivary testosterone be used in the monitoring of men using transdermal testosterone replacement therapy?

Tomas Ahern[1], Jo Adaway[2], Phillip Monaghan[1], Peter Trainer[1] & Brian Keevil[2]
[1]The Christie NHS Foundation Trust, Manchester, UK; [2]University Hospital of South Manchester, Manchester, UK.

In community-dwelling men, salivary testosterone (Sal-T) concentrations are thought to represent tissue hormone levels and correlate strongly with serum free-T levels. Measurement of salivary glucocorticoid concentrations is a non-invasive and objective means of assessing cortisol exposure in users and non-users of hydrocortisone therapy. We assessed relationships of Sal-T with transdermal testosterone replacement therapy (TD-TRT) and with markers of testosterone exposure. In 40 men aged 50.7 ($\pm$13.9) years who were attending a university hospital endocrinology clinic, we measured serum and salivary androgen concentrations by immunoassay and liquid chromatography tandem mass spectrophotometry (LC-MS/MS) respectively. In our unit, TD-TRT (Tostran 2% Gel at an initial dose of 30–60 mg of testosterone once daily) is offered to men with sexual symptoms and low fasting serum testosterone (Ser-T) concentrations on at least two consecutive occasions. Ser-T concentrations did not differ between users ($n=23$) and non-users ($n=17$) of TD-TRT (16.6$\pm$10.2 vs 11.4$\pm$4.4 nmol/l, $P=0.131$). Sal-T concentrations, however, differed greatly (17.14$\pm$15.25 vs 0.23$\pm$0.15 nmol/l, $P<0.001$) as did salivary androstenedione (Sal-A4) concentrations (2.57$\pm$4.50 vs 0.17$\pm$0.04 nmol/l, $P<.001$) and Sal-T/Sal-A4 (16.26$\pm$14.25 vs 1.45$\pm$0.94, $P<0.001$). Haematocrit and serum prostate specific antigen concentrations (PSA) did not differ significantly between the two groups (0.44$\pm$0.05 vs 0.43$\pm$0.05 l/l, $P=0.563$ and 1.06$\pm$0.66 vs 0.79$\pm$0.53 ng/ml, $P=0.170$ respectively). With TD-TRT, there was a rise in blood testosterone (4.7$\pm$4.2 to 7.9$\pm$5.7 nmol/l, $P=0.162$), haematocrit (0.42$\pm$0.05 to 0.44$\pm$0.04 l/l, $P=0.049$) and PSA (0.68$\pm$0.33 to 1.06$\pm$0.76 ng/ml, $P=0.021$) levels. Two hours after a dose of TD-TRT, Ser-T rose modestly (10.5$\pm$13.2 to 16.6$\pm$11.0 nmol/l, $P=0.003$) and Sal-T rose tremendously (7.7$\pm$8.1 to 17.0$\pm$16.9 nmol/l, $P=0.004$). Despite normal Ser-T, haematocrit and PSA concentrations, Sal-T concentrations are 75-fold greater than normal in men using TD-TRT. This is unlikely due to contamination (Sal-A4 concentrations are also high) and may be due to a conduit between skin, the lymphatic system and salivary ducts. Measurement of Sal-T is unlikely to be useful in the monitoring of men using TD-TRT.

DOI: 10.1530/endoabs.49.EP1169

EP1170

Changes of dihydrotestosterone within the life in men

Hana Pospíšilová, Michaela Dušková, Martin Hill & Luboslav Stárka
Institute of Endocrinology, Prague, Czech Republic.

Abstract
Dihydrotestosterone (DHT) is the most powerful naturally occurring androgen with three to six times higher biopotency than testosterone. Time onset of benign prostatic hyperplasia and alopecia in middle life could be the result of an imbalance between androgens. The decline of testosterone during lifespan is well known, controversial data can be found concerning the age dependence of DHT levels. We analysed the levels of testosterone, dihydrotestosterone and SHBG from 3076 men and we observed changes of their relationship and the ratio of total and free fraction of T and DHT, depending on age in men. We found that the DHT/T and fDHT/fT ratios during the life of adult males are constant, and that there is no evidence of a reversal in ratios of these hormones after puberty. Given that the ratio DHT/T remains constant during the age, the role in the development of androgenetic alopecia and benign prostatic hyperplasia is rather unlikely. The question remains, however, local status in androgen-dependent tissues, which would change the expression of enzyme, it could be caused just by local change in this ratio. This study was supported by the project MZCR for conceptual development of research organization 00023761 Institute of Endocrinology and grant 17-28692A.

DOI: 10.1530/endoabs.49.EP1170

Author Index