Endocrine Abstracts

April 2013 Volume 32
ISSN 1479-6848 (online)

15th European Congress of Endocrinology

27 April – 1 May 2013, Copehagen, Denmark

ECE2013
27 April - 1 May 2013
Copenhagen, Denmark

European Society of Endocrinology

published by
bioscientifica

Online version available at
www.endocrine-abstracts.org

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Volume 32
April 2013

15th European Congress of Endocrinology

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The European Journal of Endocrinology Prize Winner

The *European Journal of Endocrinology* Prize is a awarded to a candidate who has contributed significantly to the advancement of the knowledge in the field of endocrinology through publication. Further inforamtion on the prize can be found on http://www.ese-hormones.org/prizes/eje.aspx. This year's recipient is Professor Felix Beuschlein. The prize will be presented as part of the ECE 2013 opening ceremony where Prof. Beuschlein will deliver his lecture. Prof. Beuschlein has also written a review article based on this lecture that has been accepted for publication in the *European Journal of Endocrinology* and can be accessed at DOI:10.1530/EJE-13-0263.

Felix Beuschlein, Germany

Felix Beuschlein was born in 1969 and went to medical school at the University of Würzburg. For his doctoral thesis he worked on projects related to adrenal tumorigenesis. This field of expertise was further intensified during a postdoctoral fellowship under the mentorship of Gary Hammer at the University of Michigan where he utilized a number of mouse models to elucidate mechanisms of adrenal growth and steroidogenesis. Upon his return to Germany at the University Hospital in Freiburg he established his own group on adrenal stem cell research and completed his medical training. In 2006, he became a full professor as the head of Endocrine Research at the University in Munich. Prof Beuschlein has received a number of awards including the Marius-Tausk and Schöller-Junkmann award from the German Endocrine Society as well as the Merck Senior Fellow Award by the Endocrine Society. He serves as the vice-president of the German Endocrine Society, as a board member of the Research Affairs Core Committee of the Endocrine Society and the executive committee of Pressor. He is the chairman of the European Network for the Study of Adrenal Tumors (ENSAT) and coordinator of an FP7 consortium.

The *European Journal of Endocrinology* Prize Lecture

Regulation of aldosterone secretion: from physiology to disease

Felix Beuschlein, University Clinic Munich, Munich, Germany

Arterial hypertension is a major cardiovascular risk factor that affects between 10 and 40% of the population in industrialized countries. Primary aldosteronism (PA) is the most common form of secondary hypertension with an estimated prevalence of ~10% in referred patients and 4% in primary care. Despite its high prevalence until recently the underlying genetic and molecular basis of this common disease has remained largely obscure with the exception of the small subgroup of patients with familial hyperaldosteronism type I.

Over the past decade a number of insights have been achieved that rely on *in vitro* cellular systems, wild-type and genetically modified *in vivo* models as well as pre-clinical and clinical studies in well-characterized patient populations. This progress has been made possible by a number of independent technical developments including that of specific hormone assays that allow measurement in small sample volumes as well as genetic techniques that enable high throughput sequencing of a large number of samples. Furthermore, animal models have provided important insights in the physiology of aldosterone regulation that have served as a starting point for investigation of mechanisms involved in autonomous aldosterone secretion. Finally, national and international networks that have built up registries and biobanks have been instrumental to foster translational research endeavors in PA.

Recently, a number of approaches including genome wide association studies, exome sequencing and mutagenesis screens have been applied in patient cohorts and *in vivo* models of PA. Thereby, it is to be expected that in the near future further pathophysiological mechanisms that result in autonomous aldosterone secretion will be unraveled.

The Geoffrey Harris Prize Winner

Professor Clarke graduated from Massey University, New Zealand (B. Agric. Sci., 1971) followed by M. Agric. Sci. (1st Class) in reproductive physiology in 1973). He then obtained his PhD at Edinburgh University, Scotland in 1976, studying endocrinology and behaviour. He has since moved to Australia and became Senior Research Fellow of NHMRC in 1986, rising to Principal Research Fellow in 1991. He became Chairman, Department of Physiology, Monash University in 2007.
Prof. Clarke's seminal studies on the measurement of hypothalamic secretion of GnRH in sentient animals were published in 1982 and he has contributed extensively to the field of reproductive neuro endocrinology for 35 years. His laboratory currently focuses on Central regulation of reproduction by kisspeptin and gonadotropin inhibitory hormone; estrogen signalling in neuroendocrine systems; control of food intake and energy expenditure by leptin and novel regulatory factors; melanocortins and reproduction; central regulation of energy expenditure.
Prof. Clarke has published 430 research papers and has received The Woodward Prize for Excellence in Research in Neuroscience (1992), a Senior Fulbright Award (1997), the Asia and Oceania Medal of Society for Endocrinology (UK) in 2001 and the TransPacific Lecturership, Endocrine Society (USA) in 2004. Most recently (2009), he was the recipient of The Geoffrey Harris Memorial Award of the International Federation of Neuroendocrinology (2009).

The Geoffrey Harris Prize Lecture

Speakers, cross-talk and chatter in reproductive neuroendocrinology

Iain Clarke, Department of Physiology, Monash University, Victoria, Melbourne, Australia

The brain controls reproduction through the secretion of GnRH, but a series of higher brain centres control the secretion of GnRH into the hypophysial portal system. Whereas GnRH might be considered the 'speaker' for the brain in the reproductive axis, there is significant 'cross-talk and chatter' relating to the higher brain centres of control. Most prominently, kisspeptin mediates the feedback effect of sex steroids on GnRH cells, which may be effected at the level of the GnRH cell bodies or the secretory terminals in the median eminence. Gonadotropin inhibitory hormone (GnIH) has also emerged as a major negative regulator of GnRH secretion and action, acting on the GnRH neurons but also being secreted into the hypophysial portal system to act on the pituitary gonadotropes. This provides 'cross-talk' and data will be reviewed for GnIH function in mammals. The 'chatter' within this system involves input from centres within the brain that respond to altered nutritional status/metabolic condition, season and stress. Alterations in energy balance change the activity of appetite regulating peptide neurons in the hypothalamus. These neurons interact with kisspeptin cells, leading to changes in GnRH secretion. Season also involves changes in kisspeptin function as well as GnIH secretion. Stress has a negative impact on the reproductive neuroendocrine system and very recent data show that this involves upregulation of GnIH gene expression. In essence, GnRH neurons are controlled by serial and converging neuronal inputs from various brain centres. The same appears to be true for GnIH neurons, so the combined output of GnRH and GnIH dictates reproductive function.

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Corticotroph adenoma and fertility

Lila Brakni
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Introduction
The occurrence of a pregnancy in a woman with a corticotroph adenoma is rare, <25 observations have been reported.
Maternal complications are especially hypertension (60–75%) gestational diabetes (25%) and preeclampsia in 10%.
Fetal complications are spontaneous miscarriage, preterm birth and intrauterine growth retardation.
Observation
We report a case of a 30 years old female followed for primary subfertility. The diagnosis of Cushing's disease is withheld to: obesity, amenorrhea, hypertension and osteoporosis

- Cortisol at midnight = 712 nmol/l
- Cortisol at 0800 h = 809.4 nmol/l ACTH = 9 pg/ml
- Low braking: negative
- Strong braking: positive
- Pituitary MRI: microadenoma 6.4 mm.

Under anti-cortisol arrested 2 weeks later for liver toxicity. The resection of microadenoma was delayed because he was 6 weeks' pregnant.
Noting a gestational diabetes than the 24^{e} week. Delivery term.
After 2 years of remission (post resection)a 2nd spontaneous pregnancy with abortion at 12 weeks.
Conclusion
The impact of secretions ACTH of pregnancy requires a multidisciplinary support.

DOI: 10.1530/endoabs.32.P964

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Central hypothyroidism and adjusted thyroxine dose study (CHATS): impact of increasing free thyroxine levels in patients with hypopituitarism

Anna-Elisabeth H Minder[1], Andreas Jostel[2], Claire E Higham[1], W David J Ryder[3], Peter J Trainer[1] & Stephen M Shalet[1]
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Introduction
Patients with pituitary deficiencies suffer from impaired quality of life regardless of substitution therapy with hydrocortisone, thyroxine (T_4), sex hormones or GH. Central hypothyroidism (CH) is difficult to diagnose and treat because symptoms are non-specific and TSH-levels cannot be used for assessment. There is no consensus for the fT_4-goal of thyroxine-replacement in patients with CH.
Aim
To determine the impact of increased fT_4 on quality of life in patients with hypopituitarism.
Methods
Randomized, double-blind, placebo-controlled trial of additional T_4-supplementation. 40 patients (age 20–70 years) with hypopituitarism and fT_4-levels in the lowest third of normal reference range were included. Patients received placebo or T_4-titration aiming for fT_4 levels in the upper third of reference range, irrespective of TSH values. Total study duration 42 weeks (24 weeks dose adjustment, 18 weeks stable dose). Quality of life assessments (QoL) using four questionnaires (SF-36v2, PGWBS, EQ-5D, AGHDA) at baseline and end of study. Statistics were performed using an analysis of covariance.
Results
The increase in fT_4-values in the treatment group did not translate into significant changes in vitality score as assessed by SF-36v2 (estimated treatment effect 4.65 (95% CI -7.86, 17.15) or general health score (estimated treatment effect is 1.57 (-8.19, 11.33)), nor in any of the other questionnaires (PGWBS -1.11 (-8.80, 6.58); AGHD 0.88 (-2.77, 4.53); EQ5D-VAS -4.40 (-13.45, 4.65)).
Conclusion
The increase of fT_4 to the upper third of normal range did not significantly change the vitality score, general health or quality of life in hypopituitary patients and therefore does not provide support for the commonly used strategy of thyroxine-supplementation to the upper limit of normal. With 40 patients, however, the study may be underpowered to detect small effects. Other explanations for lack of effect include an inappropriately high or low fT_4 goal and further research is required.

DOI: 10.1530/endoabs.32.P965

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A retrospective-prospective study of the effect of octreotide LAR in acromegalic patients

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University Clinic of Endocrinology, Skopje, Macedonia.

Objective
Surgery is considered first-line treatment for pituitary GH secreting macroadenoma. Since surgical removal of the pituitary tumor is subtotal, medical treatment has become the mainstay of acromegaly. The aim of our study was to assess the effect of octreotide LAR (OCT-LAR) therapy in patients with acromegaly.
Patients and methods
Ten acromegalic patients were treated with OCT-LAR 20 mg/28 days. The effect was evaluated after 12 months of treatment. Eight patients received OCT-LAR as adjunctive therapy after surgical debulking, and two as primary treatment. Tumor size was assessed with pituitary magnetic resonance imaging (MRI) before the treatment and after 12 months. Biochemical evaluation was performed at baseline, 6 and 12 months after beginning OCT-LAR. Normalization of IGF1 was considered as biochemical remission of active acromegaly.
Results
Among the evaluated patients five were males and five females, mean age 40.2 ± 8.04 years (age range: 30–50 years). After 12 months of treatment biochemical remission (normalization of IGF1 levels) of acromegaly was achieved in nine patients. Values of IGF1 after 6 and 12 months showed a mean decrease of 55.69 and 71.53% respectively. Average tumor size before treatment and after 12 months was 17.7 and 10.42 mm respectively with an average decrease of 48.18% in size. Also, GH levels were evaluated and showed a mean decrease of 74.57% after 12 months. Along the treatment significant improvements in headache, arthralgia, sweating and soft tissue swelling was attained.
Conclusions
Treatment with OCT-LAR can be considered as effective therapy in achieving biochemical remission and tumor shrinkage in active acromegaly. Given its efficacy and safety it is a treatment of option as primary therapy in patients who are at risk or refuse operative treatment.

Keywords
Acromegaly, octreotide LAR, GH, IGF.
DOI: 10.1530/endoabs.32.P966

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Craneopahrygioma: a false enemy

Jaime Lorenzo, Ruth Boente, Eloisa Santos & Manuel Sas
Hospital povisa, Vigo, Spain.

Background
Craneopharygioma is a rare solid or mixed tumor, that arise from remnants of Rathke's pounch. Usually they are in the suprasellar region and very few of them arise from the sella. Bimodal (5–14 years old and 50–75) and slow growth are typical characteristics as well.
Clinical case
A 20 years old boy was refered to endocrine clinic by hypothyroidism, hypotension, and sexual dysfunction. Central hypothyroidism, hypogonadotropic hypogonadism and secundary adrenal insufficiency due to sellar mass was diagnosed. Sellar and suprasellar mixed mass with solid and cystic changes were found and due to its stability for 5 years and because of the clinical presentation was interpreted as a macroadenoma. Sustitutive hormone treatment is prescribed and regular clinical and radiologic follow up is made. For 5 years the mass experimented no changes but in the next evaluation mass enlargament with significant suprasellar extension was seen. Pterional craneotomy are made and a cranepharygioma is diagnosed. Treatment is completed with stereotactic radiotherapy and the patient has a good response so far.
Discussion
Usually calcification in the suprasellar region is seen up to 80% and cyst are present very often (up to 75%). If calcification is not seen, suspection is more