CONTENTS

e-ECE 2020
22nd European Congress of Endocrinology

PRIZE LECTURES AND BIOGRAPHICAL NOTES
The Geoffrey Harris Prize Lecture ................................................................. AP1
The European Journal of Endocrinology Prize Lecture ................................ AP2
European Hormone Medal Lecture ............................................................ AP3
Clinical Endocrinology Trust Lecture ......................................................... AP4

PLENARY LECTURES
Exercise as medicine – a translational perspective ....................................... PL1
Glucocorticoids in cancer: a new paradigm ................................................. PL2
Harnessing the microbiome in metabolic disease ....................................... PL3
Mechanisms for SARS-CoV-2 cell entry ................................................... PL4
Maternal thyroid hormone and child brain development ........................... PL5
It takes thyroid hormone to make sense .................................................. PL6
Effects of EDCs on neuro-endocrine systems and behaviour ..................... PL7

SYMPOSIA
New horizons in phaeochromocytoma and paraganglioma ....................... S1.1–S1.3
Osteoporosis and fracture prediction ......................................................... S2.1–S2.3
Controversial issues in bariatric surgery ................................................... S3.1–S3.3
Unveiling signatures in pituitary neuroendocrine tumours ......................... S4.1–S4.3
Hyperthyroidism across the lifespan ......................................................... S5.1–S5.3
Adrenocortical carcinoma ........................................................................ S6.1–S6.3
Endocrine disruptors, just a hype or not? ............................................... S7.1–S7.3
PCOS: from Genetics to Treatment ........................................................ S8.1–S8.3

COVID-19 SESSION
Endocrine targets related to COVID infection ........................................... CS1.1
Managing the Cytokine storm ................................................................. CS1.2
How strong is obesity as a risk factor for COVID-19 patients .................... CS1.3

ORAL COMMUNICATIONS
Adrenal and Cardiovascular Endocrinology .............................................. OC1.1–OC1.7
Bone and Calcium ................................................................................ OC2.1–OC2.7
Diabetes, Obesity, Metabolism and Nutrition ........................................ OC3.1–OC3.7
Pituitary and Neuroendocrinology ........................................................ OC4.1–OC4.7
Thyroid .................................................................................................. OC5.1–OC5.7
Hot Topics (including COVID -19) ......................................................... OC6.1–OC6.7
Endocrine-related Cancer ........................................................................ OC7.1–OC7.7
Environmental Endocrinology ............................................................... OC8.1–OC8.6
Reproductive and Developmental Endocrinology .................................... OC9.1–OC9.7
Young Investigators .............................................................................. YI1–YI12
AUDI EPOSTER PRESENTATIONS

Adrenal and Cardiovascular Endocrinology ................................................................. AEP1–AEP121
Bone and Calcium ................................................................. AEP122–AEP242
Diabetes, Obesity, Metabolism and Nutrition ........................................ AEP243–AEP527
Endocrine-related Cancer ......................................................... AEP528–AEP540, AEP655
Environmental Endocrinology ............................................................. AEP541–AEP542
General Endocrinology .......................................................... AEP543–AEP559
Pituitary and Neuroendocrinology ................................................. AEP560–AEP777
Reproductive and Developmental Endocrinology ........................... AEP778–AEP856
Thyroid .......................................................... AEP857–AEP1000
Hot topics (including COVID-19) .................................................. AEP1001–AEP1110

EPOSTER PRESENTATIONS

Adrenal and Cardiovascular Endocrinology ................................................................. EP1–EP58
Bone and Calcium ................................................................. EP59–EP123
Diabetes, Obesity, Metabolism and Nutrition ........................................ EP124–EP265
Endocrine-related Cancer ......................................................... EP266–EP270
Environmental Endocrinology ............................................................. EP271
Reproductive and Developmental Endocrinology ........................... EP374–EP410
Thyroid .......................................................... EP411–EP532
Hot topics (including COVID-19) .................................................. EP533–EP589

AUTHOR INDEX
AEP730

Global methylation-demethylation status in pituitary neuroendocrine tumors as potential therapeutic target

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Background
The altered DNA methylation of certain genes in Pituitary Neuroendocrine Tumors (PitNETs) are well known. However little information is available regarding global methylation changes and the process of demethylation in these tumors. In addition, influencing global methylation-demethylation could be a potential new therapeutic option especially in clinically non-functional PitNETs.

Material and methods
Overall, 44 fresh frozen pituitary adenoma tissues (29 gonadotroph, 12 somatotroph, 3 corticotroph) were collected and characterized according to the 2017 WHO classification. Decitabine was used to alter global methylation-demethylation status in vitro GH3 and RC-4B/B cell lines. In tissue samples 5-hydroxymethylcytosine (5 hmC), UHRF1-2 protein and Ki-67 were assessed by immunohistochemistry; gene expression of DNA Methyltransferase (DNMT1), methyl-cytosine dioxygenases (TET1-3) and ubiquitin-like with PHD and ring finger domain (UHRF1-2) were investigated by RT-qPCR. 5-methylcytosine (5 mC) and 5 hmC level were determined by HPLC-MS/MS method.

Results
Decitabine decreased 5-methylcytosine (5 mC) and increased 5 hmC levels in vitro in both pituitary cell lines. Parallel, cell proliferation and viability were decreased significantly. UHRF-1 were also altered upon decitabine treatment in vitro. Interestingly, in PitNET tissue samples 5 hmC was gradually decreased in samples with higher Ki-67 index. In samples with different histology UHRF2 showed different expression, while UHRF1 showed gradual increase in adenoma samples with higher Ki-67 index. Additionally, UHRF2 positively correlated with 5 hmC level in pitNET tissues and both UHRF1 and UHRF2 showed significant positive correlation with DNMT1 and TET1-3 expression.

Conclusion
Our results showed that methylation-demethylation process (5 hmC, DNMT1, TET1-3 and UHRF1-2) is closely linked to proliferative behaviour of PitNETs. Altering global 5 mC and 5 hmC level can be a potential, new therapeutic target in therapy resistant pituitary tumors.

Grants and financial support
This work has been funded by the National Program of Bionics (Program Medical Bionics lead by Attila Patócs) and Semmelweis Innovation Found (STIA, 19) to Henriett Butz. Henriett Butz is a recipient of Bolyai Research Fellowship of Hungarian Academy of Sciences and ÚNKP-18-4-SE-8 New National Excellence Program of The Ministry of Human Capacities.

DOI: 10.1530/endoabs.70.AEP730

AEP731

The expression of oxytocin receptor (OXTR) in metastatic pancreatic neuroendocrine tumors (PNETs)

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Introduction
Pancreatic neuroendocrine tumors (PNETs) are rare malignant neoplasms which incidence is continually increasing. They are characterized by diverse biological behaviour and impact on the patients’ prognosis, ranging from clinically indolent to very aggressive. Oxytocin receptor (OXTR) is a member of the family of G-protein receptors and is present on the cell-surface of the gastrointestinal organs. Unfortunately, the impact of OXTR signaling on the development of PNETs and its underlying molecular mechanisms involved in gastrointestinal oncogenesis remains insufficiently researched.

The aim of our study was to assess the expression of OXTR in a group of patients diagnosed with metastatic PNETs.

Material and methods
Metastatic PNETs (liver metastases) specimens (n=24) matched control (normal) tissue were surgically collected and mRNA expression was determined by Real-time polymerase chain reaction (Real-time-PCR). OXTR expression for tumor and control tissue was additionally analysed by immunohistochemistry.

Results
Compared to normal tissue, the OXTR showed significant overexpression in metastatic PNETs. Moreover, significant overexpression of OXTR in tumor tissue was confirmed by immunohistochemistry.

Conclusion
Our findings highlight the possibility of making OXTR a promising novel molecular target for imaging and different therapeutic approach in patients diagnosed with metastatic PNETs.

DOI: 10.1530/endoabs.70.AEP731

AEP732

Metastatic insulinoma managed with lutetium (177Lu) and somatostatin analog

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Background
Insulinoma is a rare tumour representing 1–2% of all pancreatic neoplasms and it is malignant in only 10% of cases. Locoregional invasion or metastases define malignancy, whereas dimension (> 2 cm), CK19 status, tumor staging and grading (Ki67 >2%), and age of onset (> 50years) can be considered elements of suspect.

Case presentation
We report a case of malignant insulinoma in a 80 year old woman presenting symptoms compatible with hypoglycemia. Low blood glucose levels (< 40 mg/dl) were documented during of these episodes. Symptoms regressed with food intake and intravenous glucose administration. No abnormality was detected in the biochemical evaluation. Prolong fasting test was performed, and the patient underwent symptomatic hypoglycemia at the 5th hour. Plasma glucose level was 39 mg/dl, insulin level 36.4 μIU/mL and C-peptide 9.28ng/ml. Glucagon response was measured 10 min. intervals, 85mg/dl, 95mg/dl and 113 mg/dl respectively. These results were suggestive of endogenous hyperinsulinemia. Magnetic resonance imaging revealed a massive invasive mass in the pancreatic tail location – 63 x 40 mm in size and multiple metastatic nodules in the the liver. Ga–68 DOTATATE PET-CT, which showed a lesion located in the pancreatic tail location and multiple metastatic lesion in the liver, with a high somatostatin receptor density. Tru-cut biopsies made from liver lesions revealed the insulinoma tumour metastasis. Synaptophysin, pancytoceratine and chromogranin were positive. The histopathological diagnosis was suggestive of a neuroendocrine, grade-2 tumour (mitotic rate 1/10 HPF, Ki-67 proliferative index 15%). Lanreotide 120 mg IM was started an every 28 days basis. The patient received 2 infusions of radionabeled somatostatin analog lutetium (177Lu) 8 weeks apart and denied any hypoglycemia. After the second administration of the lutetium, IM was started an every 28 days basis. The patient received 2 infusions of lanreotide 120 mg IM was started an every 28 days basis. The patient received 2 infusions of lanreotide 120 mg IM was started an every 28 days basis. The patient received 2 infusions of lanreotide 120 mg IM was started an every 28 days basis. The patient received 2 infusions of lanreotide 120 mg IM was started an every 28 days basis. The patient received 2 infusions of lanreotide 120 mg IM was started an every 28 days basis.

Conclusions
We report a case of metastatic insulinoma treatment with somatostatin analog and Lutetium. Due to previous glycemie control reports and objective responses in unresectable cases, we decided to use Lutetium together with lanreotide. The patient’s hypoglycaemia improved immediately after treatment. Unresectable metastatic insulinomas may present as a major therapeutic challenge for the physician. Treatment with lanreotide and Lutetium