comprising genome of 2504 unselected individuals collected worldwide. The combination of 184 SNPs associated with PTC was used to group individuals in different risk-clusters according to their genetic structure, calculated by Bayesian statistics, as previously performed for polycystic ovary syndrome [2]. Individuals were distributed among 7 groups worldwide, indicating different degree of genetic predisposition to PTC. We then considered genetic data from about 1200 individuals (697 PTC *versus* 497 healthy controls) of Central/South Italian origin registered in a GWAS, specific for PTC [3]. This first analysis was refined using the 33 SNPs reasonably most causative of genetic clustering (26 with $p<0.05$ at trend test in GWAS and 7 with $p<0.05$ in the model of recessive inheritance). At multivariate logistic regression analysis, PTC and healthy controls resulted genetically different (ODDS RATIO 188.6, 95%CI 64.35-552.8), revealing diverse predisposition to develop cancer. Afterwards, these results have been confirmed in an independent cohort of Italian subjects (234 PTC and 100 controls). Then, the genetic structure of each subject was indicated as a percentage of affinity to each risk-cluster and re-analyzed together with other risk factors: sex, body-mass index, area of origin and familiarity (quantified in a growing score as the degree of kinship increases). These data were analyzed together by principal component analysis and clustering of the two groups was even more pronounced. The most contributive factors to the diversity between PTC and healthy controls were genetics and familiarity.

CONCLUSION. We demonstrated that PTC affected subjects are genetically different from healthy controls, and that the difference is identifiable in a peculiar combination of genetic variants.

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Neuroendocrinology and Pituitary

PITUITARY TUMORS II

AIP Gene Germline Mutations in Non-Selected Patients with Apparently Sporadic Pituitary Macrodenomas

Malgorzata Trofimiuk-Muldner, MD,PHD[1], Bartosz Domagała, MD[2], Grzegorz Sokolowski, MD, PhD[1], Anna Skalniak, PhD[1], Jakub Piatkowski, Msc[3], Alicja Hubalewska-Dydejczyk, MD, PhD[1].

[1]Department of Endocrinology, Jagiellonian University Medical College, Krakow, Poland, [2]Department of Endocrinology, University Hospital in Krakow, Krakow, Poland, [3]Department of Endcrinology, University Hospital in Krakow, Krakow, Poland.

MON-300

Up to 5% of all pituitary tumors are hereditary (e.g. due to menin or AIP genes germline mutations). AIP gene mutations are more common in subjects with acromegaly, less than 30 years old at the onset of disease, and with FIPA family history.

The study was aimed at the assessment of the frequency and characteristics of AIP-mutation related tumors in non-selected patients with pituitary macroadenomas.

Material and methods. The study included subsequent 131 patients (57 males, 74 females; median age 42 years (IQR 25 years) diagnosed with pituitary macroadenomas, and with a negative family history of FIPA or MEN1 syndromes. The following tumors were identified: 11 ACTH-secreting, 49 GH-secreting (including 7 pluri-hormonal ones), 6 gonadotropinomas, 23 prolactinomas, 1 TSH-oma, and 43 non-secreting adenomas. Sanger sequencing was used for the assessment of AIP gene variants. The study was approved by the Ethics Board of JUMC.

Results. An AIP mutation was identified in five of 131 included subjects (3.8%): one diagnosed with Cushing's disease, two with acromegaly, and two with non-secreting adenomas. In two patients, the identified mutation usually predisposes to ACTH-secreting adenomas, in two patients - mutations of unknown clinical significance were found (usually connected with pituitary adenomas), and the mutation detected in one patient was described as benign. Patients harboring hereditary AIP gene variations did not differ from the rest of the study group in median age at diagnosis (41 vs. 42.5 years, $p=0.8$), median largest tumor diameter (25 vs. 24 mm, $p=0.6$), gender distribution (60% of females vs. 56.3%, $p=0.8$), secreting tumor frequency (60% vs. 67.5%, $p=0.7$), or acromegaly diagnosis frequency (40% vs.37.3%, $p=0.9$). 2 of the 5 patients with identified AIP gene mutations agreed for their families to be offered AIP genetic testing: (1) An AIP mutation was found in the asymptomatic mother of one acromegalic female patient. (2) The AIP mutation of unknown clinical significance was detected in the son of a male acromegalic patient with acromegaly, clinically unscreened yet.

Conclusions. In our series of apparently sporadic pituitary macroadenomas, AIP gene mutation carriers did not differ substantially from patients with negative genetic testing. A risk factor-centered approach to AIP genetic screening may result in missing germline mutations, therefore, there is a need to establish if such a situation negatively impacts a patient's and his/her family outcomes.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL GLUCOSE METABOLISM AND DIABETES

Frequency and Associated Factors with Multidrug-Resistant Organism Infection in Diabetic Foot Ulcers in a Peruvian Public Hospital

Marlon Augusto Yovera-Aldana, MD, MSc, Liset Paola Sifuentes, MD, Delia Cruz-Estacio, MD, Diana Consuelo Flores, MD, Lucy Nelly Damas-Casani, MD, MSc.

María Auxiliadora Hospital, Lima, Peru.

MON-626

Objective: To determine the frequency and associated factors with multidrug-resistant organism (MDRO) infection among patients with diabetic foot ulcers in a Peruvian Public Hospital.

Materials and methods. Cross-sectional survey was conducted from January 2017 -December 2018 at National