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Aims

• Elucidate the effect of wildtype and polymorphic AHR on GH3 cell proliferation and on AHR-transcriptional response in the presence and absence of TCDD.

• Determine the allele frequency of the most common AHR SNP; the Arg554Lys in PA patients and in a small cohort of the Maltese population.

Method

The two missense mutations were introduced within the AHR-expressing vector and transfected in GH3 cells by magnetofection, followed by the exposure to TCDD. Cell viability of GH3 transfected cells was measured using the MTT assay. Functional analysis of GH3 transfected cells treated with TCDD was carried out using luciferase assay and real-time PCR to detect and quantify the AHR-transcriptional activity. Genotyping of the Arg554Lys was performed on PA patients and neonatal controls using allele specific PCR. The Mann-Whitney test was used to compare two groups and Kruskall-Wallis test was used to compare three groups or more.

Results

In the absence and presence of low TCDD concentrations (1 and 10 nM), over-expression of wildtype AHR (wtAHR) did not affect GH3 cell proliferation. GH3 cells transfected with the AHR mutants did not exhibit any significant differences in their proliferative ability when compared with the wtAHR, both in the presence and absence of TCDD. Luciferase reporter analysis showed that there was a significant difference between the treated and untreated wtAHR (P = 0.016), however this difference was not observed between the treated and untreated AHR mutants. Statistically significant difference in Cyp1a1 gene expression analysis was detected between the treated and untreated wtAHR (P = 0.021), Arg554Lys (P = 0.005) and Val570Ile (P = 0.054). Genotyping of the Arg554Lys in patients with PA gave a minor allele frequency (MAF) of 3% vs 0% in neonatal controls.

Conclusion

Gene expression and quantification analyses of AHR-target genes suggests that these AHR mutants might interfere with AHR target gene expression. Genotyping results suggested that this mutation is quite rare and may be similar to the frequencies of other European populations.

DOI: 10.1530/endoabs.56.P777

P779

Next generation sequencing for characterization of mitochondrial genome in pituitary adenomas

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Introduction

Disrupted mitochondrial functions and genetic variations of mitochondrial DNA (mtDNA) have been observed in different tumors. Regarding pituitary adenomas mtDNA was evaluated only in oncocytic type using PCR based methods and it showed high prevalence of Complex I variants. Next generation sequencing (NGS) allows high throughput sequencing and it is useful for accurate identification of heteroplasmy of mitochondrial genome as well.

Aim

We aimed to investigate the entire mitochondrial genome in different adenoma types.

Material and methods

We collected 22 gonadotroph (GO), 11 GH producing (GH) and 11 null-cell (NC) adenoma specimens from samples removed by transphenoidal surgery. From fresh frozen tissues DNA extraction was performed using QIAamp Fast DNA Tissue Kit. For library preparation VariantPro Amplicon Mitochondrion Panel kit was used. The total mtDNA (16569 bp) was sequenced on Illumina MiSeq Instrument. Following complex bioinformatic analysis Revised Cambridge Reference Sequence (rCRS) of the human mitochondrial DNA was used as reference. Heteroplasmy was determined using 3% cutoff.

Results

The whole mitochondrial genome were covered by 630±370 (avg ± S.E.) reads per base. 496 variants were identified in adenomas compared to reference sequence. Overall a low (7.22%) heteroplasmy prevalence was found. Based on mitochondrial sequence variants by hierarchical cluster analysis we could not discriminate different adenoma types. No association between Ki-67 index or recurrent-nonrecurrent status of adenomas and mitochondrial variants were detected. Four variants appeared more often in null-cell adenomas compared to gonadotroph adenomas (chrM_188: 18% vs 0%, chrM_16093: 18% vs 0%, chrM_185: 27% vs 0% and chrM_14798: 36% vs 5%; Padj = 0.01542 and 0.0246, respectively). Of these variants chrM_14798, chrM_4216 and chrM_15452 are non-synonymous polymorphisms leading to amino acid change in MT-CYB (mitochondrially encoded cytochrome b) and in MT-ND1 (mitochondrially encoded NADH dehydrogenase 1) genes. We identified chrM_16189 variant (non-protein coding variant) in 40% (6/15) of nonrecurrent adenomas compared to recurrent ones where this variant was not present (9/11) (P = 0.0209).

Conclusions

Next-generation sequencing is a reliable method for investigating mitochondrial genome and heteroplasmy in pituitary adenomas. In pituitary adenomas the prevalence of heteroplasmy of mitochondrial genome is low suggesting that these alterations may not influence mitochondrial function considerably. Of pituitary
tumours only null cell adenomas possess alterations of mitochondrial genome with potential functional consequences suggesting that during the development of this subtype of pituitary tumours mitochondrial function-associated mechanisms may have role.

DOI: 10.1530/endoabs.56.P779

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Pituitary cell activation and recruitment in hypothyroidism

Fernando Oroz, Montserrat García-Lavandera, Sihara Pérez-Romero, Ángela García-Rendueles & Clara V Alvarez

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SOM230 exerts anti-proliferative actions by reducing phospho-ERK/1/2 levels in ACTH-secreting pituitary tumour cells

Donatella Treppiedi, Erika Peverelli, Elena Giardino, Rosa Catalano, Federica Mangili, Maura Arosio & Giovanna Mantovani

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Currently, the multi-ligand somatostatin (SS) analogue pasireotide (SOM230) is the only pituitary-targeted drug used to treat patients with Cushings disease. SOM230 displays the highest affinity to somatostatin receptor type 5 (SSTR5) and compared to octreotide resulted more effective in reducing ACTH release. Despite its anti-secretory role, SOM230 has been associated with tumor shrinkage in patients subjected to long term treatment, although to date the key factors involved are poor elucidated. The present work aimed to investigate the molecular mechanisms implicated in SOM230-induced cytostatic and cytotoxic effects in ACTH-secreting primary tumour cultures and murine corticophob tumour cells.

First, by western blot we found SSTR5 expressed at comparable levels in 17 different ACTH-secreting pituitary samples, whereas SSTR2 was detectable in 15 out of 17 tissues. SSTR5 and SSTR2 were expressed in ACT-20 cells. Then, we tested the effect of 96h stimulation with 1 μM SOM230 on cell proliferation in 6 different ACTH-secreting tumours by measuring 5-bromo-20-deoxyuridine incorporation during DNA synthesis. We found a significant in vitro suppression of cell growth in half of the analyzed samples (12.1±4.3%, P<0.01). Accordingly, SOM230 significantly inhibited cell growth in a dose-dependent manner in ACT-20 cells (−10.5±7.7% at 10 μM, P<0.05; −3.9±10.9% at 100nM, P<0.05; −26.8±4.9% at 1 μM, P<0.01), whilst octreotide was effective only at 1 μM (−13.3±9.1%, P<0.05). To investigate whether direct antiproliferative actions SOM230-mediated might involve MAPK and cyclins pathways, we evaluated the expression level of phospho-ERK1/2 and CDK in ACTH-secreting primary cultures exposed to 1 μM of SOM230. SOM230 reduced phospho-ERK1/2 levels in 5 of 8 tumours tested (−36.4±20.5%, P<0.01), whereas no significant difference was found in CDK expression levels in 3 tumours. These data were further confirmed in ACT-20 cells, where octreotide did not have any effect. Furthermore, we found that 48h incubation with 1 μM SOM230 was able to induce a significant increase of caspase 3/7 activity in 2 of 4 ACTH-secreting primary cultures (17.2±3.6%, P<0.05). Altogether these data suggest a downstream implication of phospho-ERK1/2 inhibition in ACTH-secreting pituitary tumour cells by SOM230 resulting in cell proliferation suppression and indicating that broader-spectrum SS analogues may play a crucial role in the treatment of tumours where the MAPK pathway is overactivated. Moreover, we describe a pro-apoptotic effect of SOM230. Ongoing experiments are aimed to discriminate the specificity effects played by SSTR5 and SSTR2.

DOI: 10.1530/endoabs.56.P781

P782

Abstract withdrawn.

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Corticotroph pituitary adenomas: the functioning vs the silent: a gene expression study comparing differentially expressed genes in the regulation of POMC

Kjersti Ringvoll Normann1,2,3, Arvind Sundaram4, Kristin Astrid Berland Øystese1,2, Tove Løvka1, Alexander Eieland1, Jens Bøllerslev1,2 & Nicoleta Cristina Olărescu1,3

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The exact mechanism behind the hypersecretion of ACTH and lack of negative cortisol feedback on POMC regulation in functional corticotroph adenomas (FCA) is unknown. Silent corticotroph adenomas (SCA) express, but do not secrete functional ACTH and have lower POMC expression. Using RT-qPCR and immunohistochemistry, previous studies have identified some POMC-transcription factors, regulators and processing enzymes to be differentially expressed.