ERRATUM

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In the published abstract supplement, there were two abstracts under Oral presentations S31 and Poster presentations S52 that were not included.

The additional abstracts were given below.

Oral Presentation

NGS and ddPCR as an Innovative Approach for Personalized Therapy in Cancer Diagnostics and Treatment.

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When it comes to cancer diagnosis, speed and accuracy makes all the difference. Thanks to our cancer predisposition panels and somatic mutation tests, the diagnosis is faster and more effective than ever before. We are moving towards assessing

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molecular biomarkers in the peripheral blood or FFPE samples to detect specific germline or somatic mutations, select the best targeted therapy and monitor disease progression, recurrence and stability. All of these can be achieved by using a broad range of the newest technologies.

There is a great potential for integration of NGS and digital PCR in terms of targeted resequencing, the development and usage of individualized biomarkers and monitoring the response to chemotherapy. Thanks to our ONCO panels based on NGS technology, we are able to screen patients towards a wide range of “hot spot” mutations in 56 tumor suppressor genes and oncogenes annotated in the COSMIC database. The result allows for selection of the best targeted therapy for an individual patient, indicating the most suitable clinical studies that the patient may be eligible for.

Detection of a somatic mutation by NGS, allows us to assess the frequency of the mutant allele by digital PCR. With this technique we can precisely quantify the mutant allele frequency of rare tumorigenic mutations in a high background of “normal” cells, routinely with a sensitivity down to 0.01 % and often further. The usage of very sensitive screens for specific mutations with no information on the mutant allele frequency may be misleading, and may result in targeting of a minor subclone. Therefore, it is plausible that two important parameters – the percentage of cancer cells that carry a specific druggable mutation and whether a specific allele is amplified as well as mutated – may have a major impact on the response to specific therapies.

Additionally, we encourage to monitor cancer regression by checking the number of circulating tumor cells (CTC) in patient blood. Enumeration of CTCs provides us with an information whether the cells are successfully reduced by the therapy.

Targeted cancer therapies give medical oncologists a better way to customize cancer prevention and treatment. The pipeline that we suggest will shorten the time of diagnosis, help to choose an individualized treatment that potentially causes less harm to a patient and fewer side effects, and in longer term will improve quality of life of a patient.

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Poster Presentation

The Effect of Forced Expression of Mutated KRAS Gene on Gastrointestinal Cancer Cells and IGF-1R Targeting

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Background: Mutation in KRAS plays important roles in both the progression and the resistance to anti-EGFR therapy in gastrointestinal tumors. Insulin-like growth factor-1 receptor (IGF-1R) signaling is required for carcinogenicity and progression of many cancers, including gastrointestinal carcinomas. We have

previously shown successful therapy for gastrointestinal cancer cell lines with KRAS mutation using recombinant adenoviruses expressing dominant negative IGF-1R (IGF-1R/dn) and an anti-IGF-1R monoclonal antibody.

Study Aims: In this study, we sought to evaluate the effect of accumulation of mutated genes and forced expression of mutated KRAS gene on gastrointestinal cancer cell lines and IGF-1R targeting therapies for those cells.

Material and Methods: We made stable transfectants of mutated KRAS in gastrointestinal cancer cell lines. We assessed the effect of forced expression of mutated KRAS on proliferation, apoptosis induction, migration, invasion, and signal transductions in gastrointestinal cancer cell lines. Then we assessed the anti-tumor effect of IGF-1R/dn on mutated KRAS transfectants.

Results: Overexpression of mutated KRAS generated more aggressive phenotypes of gastrointestinal cancer cell lines, characterized by higher proliferative rate, anti-apoptotic features, higher motility and invasiveness. IGF-1R/dn inhibited colony formation, migration, and invasion, but induced apoptosis of mutated KRAS transfectants. The transfectants showed stronger inhibition of the Akt-pathway than of the ERK-pathway by IGF-1R/dn.

Conclusions: Mutated KRAS gene might be important for the progressive phenotype in gastrointestinal cancers. IGF-1R might be a candidate for a molecular therapeutic target for gastrointestinal cancers even if KRAS is mutated.