Aims and objectives of this presentation

Various mechanisms of resistance have been proposed for different anti-androgens. Specific mutations have been discovered in patients treated with hydroxyflutamide, bicalutamide, or more recently, enzalutamide. Importantly, appearance of truncated, constitutively active androgen receptors during therapy with enzalutamide or abiraterone and clinical implications will be discussed in this session.

10:30 - 10:35
Introduction The current status of AR research
Z. Culig, Innsbruck (AT)

10:35 - 10:50
State-of-the-art lecture Truncated androgen receptor and intracrine androgen synthesis in resistant prostate cancer
A. Gao, Sacramento (US)

10:50 - 11:05
State-of-the-art lecture Imaging of primary and secondary resistance to new AR pathways inhibitors
N. Tunariu, London (GB)

Aims and objectives of this presentation
The presentation aims to present a concise review of the emerging modern imaging techniques with emphasis on 1) improved assessment of response to therapy in bone metastases and 2) depiction of intra-patient heterogeneity as potential tools for a better understanding of the new AR pathways inhibitors resistance mechanisms.

11:05 - 11:20
State-of-the-art lecture Will new AR pathways inhibitors reshape the early prostate cancer landscape?
G. Kramer, Vienna (AT)

11:20 - 11:30
Late breaking news A Randomized Trial of Abiraterone Acetate (AA) Administered With 1 of 4 Glucocorticoid (GC) Regimens in Metastatic Castration-Resistant Prostate Cancer (mCRPC) Patients (its)
A.S. Merseburger, Lübeck (DE)

11:30 - 11:45
State-of-the-art lecture Abiraterone acetate resistance and plasma androgen receptor
D. Gasi Tandefelt, Sutton, Surrey (GB)

Aims and objectives of this presentation
By using next-generation sequencing on circulating tumor DNA obtained from plasma through a minimally invasive blood test, we have demonstrated the capacity to identify genomic aberrations.
that associate with resistance to abiraterone.

11:45 - 11:52

Late breaking news

LBA03

Trop-2 expression is driven by epithelial-to-mesenchymal transition in prostate cancer cells
By: Binó L.², Fedr R.², Kozubík A.³, Pernicová Z.², Remíjik J.¹, Šimečková S.¹, Souček K.¹
Institutes: Institute of Biophysics, Academy of Sciences, Dept. of Cytokinetics, Brno, Czech Republic, ²International Clinical Research Center, St. Anne’s University Hospital Brno, Center of Biomolecular and Cellular Engineering, Brno, Czech Republic, ³Masaryk University, Department of Experimental Biology, Faculty of Science, Brno, Czech Republic

11:53 - 12:00

Associated abstract presentation

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Periprostatic adipose tissue acts as a driving force for the local invasion of prostate cancer in obesity: Role of the CCR3/CCL7 axis
By: Roumiguie M.¹, Laurent V.², Toulet A.², Zaidi F.³, Valet P.⁴, Mazerolles C.³, Malavaud B.¹, Muller C.²
Institutes: Institut Universitaire Du Cancer, Dept. of Urology, Toulouse, France, ²Institut De Pharmacologie Et Biologie Structurale Du CNRS, Dept. of Oncology, Toulouse, France, ³Institut Universitaire Du Cancer, Dept. of Pathology, Toulouse, France, ⁴INSERM, U1048, Toulouse, France

Aims and objectives of this presentation
The prostate is surrounded by adipose tissue (PPAT), an active endocrine organ able to secrete chemokines, referred to as adipokines. Compared to benign epithelium, cancer cells overexpress receptors for adipokines suggesting a crosstalk between PPAT and cancer.
We hypothesized that this could be instrumental in the increased aggressiveness reported in obese cancer patients and in extracapsular disease.
The ability of PPAT to attract cancer cells away from the prostate gland is dependent on an original CCR3/CCL7 axis. Up-regulation of CCL7 secretion in obesity facilitates extra-prostatic extension and local dissemination, which is abrogated when the CCR3/CCL7 axis is inhibited.
Attention is driven towards CCR3 antagonists, which are being developed in other medical conditions.