GENERAL SESSION 6

Alle Vortragsfolien

[GS6-01] Oral Paclitaxel with Encequidar: The first orally administered paclitaxel shown to be superior to IV paclitaxel on confirmed response and survival with less neuropathy: a phase III clinical study in metastatic breast cancer

Umanzor G, Rugo H, Cutler DL, Barrios FJ, et al.

The authors conclude that: *Oral paclitaxel + encequidar is the first orally administered paclitaxel shown to be superior to IV paclitaxel for confirmed response, progression-free survival, and overall survival, with minimal clinically meaningful neuropathy.*

[GS6-02] Apobec and BRCA mutation signatures are mutually exclusive in breast cancer

Harris RS, Smid M, Wilting S, Roelofs P, et al.

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[GS6-03] Cisplatin versus doxorubicin/cyclophosphamide as neoadjuvant treatment ingermline BRCA mutation carriers (BRCA carriers) with HER2-negative breast cancer:Results from the INFORM trial (TBCRC 031)

Tung N, Arun B, Hofstatter E, Hacker MR, et al.

The authors conclude that: While CDDP has single-agent activity in ***BRCA*** **carriers with HER2-negative breast cancer, the pCR rate with CDDP is not higher than**

with standard AC chemotherapy. CDDP activity was notably lower in *BRCA* carriers with hormone-receptor positive breast cancer, though the sample size was small. Study-collected tumor and blood samples are being analyzed for biomarkers of response.

[GS6-04] Co-occurring gain-of-function mutations in HER2 and HER3 modulate HER2/HER3 activation, breast cancer progression, and HER2 inhibitor sensitivity

Hanker AB, Sekar Jayanthan H, Ye D, Lin C, et al.

The authors conclude that: Co-expression of mutant HER2 and mutant HER3 promotes ligand-independent HER2/HER association, HER3 phosphorylation, and cancer cell invasion via enhanced activation of the PI3K pathway; this enhanced signaling output is incompletely blocked by neratinib. Therefore, breast cancers expressing co-occurring HER2 and HER3 mutations may require the addition of a PI3Kα inhibitor to a HER2 TKI.

[GS6-05] A joint atlas of single-cell and bulk RNA-seq in metastatic breast cancer allows inference of oncogenic and drug-resistant transcriptional programs

in malignant cells and the tumor microenvironment

Cohen O, Abravanel D, Slyper M, Klughammer J, et al.

The authors conclude that: To the best of our knowledge these data represent the first integration of single cell and bulk RNA-seq data in MBC, resulting in a comprehensive single-cell-resolution transcriptional atlas, and a catalog of drug-resistance oncogenic progs with implications for immunotherapy and precision-oncology.

[GS6-06] A neoadjuvant trial with letrozole identifies *PRR11* in the 17q23 amplicon as a mechanism of resistance to endocrine therapy in ER-positive breast cancer

Lee K, Guerrero-Zotano A, Hanker A, Servetto A, et al.

The authors conclude that: These data suggest that 1) *PRR11* is a mediator of resistance to antiestrogens via amplification of PI3K/AKT signaling, and 2) PI3Kα is a potential therapeutic target in ER+ BCs harboring *PRR11* amplification.

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San Antonio - Mosaic on a pillar of streetcar station

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