models. Moreover, in dMMR cases, a custom in-house Next-Generation Sequencing (NGS) (Ion Torrent S5) panel was performed, assessing mutational status of 26 tumor-genes (EGFR, MET, ALK, RET, KRAS, NRAS, HRAS, BRAF, KIT, PDGFR α/β, GNAQ, GNA11, PIK3CA, AKT1, MAP2K1, MAP2K2, TP53, ERBB2, SMAD4, PTEN, STK11, CTNNB1, NOTCH1, POLE, ESR1).

Results: We selected 76 out 90 patients, with a median age at diagnosis of 61 years. Fifty-nine patients received NAC, while 17 were treated with adjuvant chemotherapy alone. Overall, dMMR/MSI-H counted for 8% of cases, entirely consistent between endoscopic and surgical samples. Six percent of tumors were HER2 positive on endoscopic tumor assessment and 4% on surgical samples. Tumor downstaging was observed in 52.5% of the population, with 3 pCR (5.1%), none of them in MSI-H cancers. According to MSI status and pCR, EFS and OS were better for MSI-H patients and MSS achieving pCR compared to MSS without pCR [EFS NR vs NR vs 30.0 months (95% CI 16.8 — NR.), P = .08; OS NR vs NR vs 39.6 (95% CI 27.6 — NR) P = .10]. Considering the entire population, EFS and OS were analyzed according to MSI status with a better outcome for MSI-H patients [EFS NR vs 48.0 months (95% CI 25.2 — 229.4), P = .121; OS NR vs 62.4 (95% CI 28.8 — 229.4) P < .143]. The most common alteration in MSI-H cases was TP53 mutation (4/6), other mutations detected were KRAS, SMAD4, ERBB2, BRAF, PIK3CA, RET and PTEN.

Conclusions: Our work confirms the positive prognostic effect of MSI-H in the curative setting of LAGC, not correlated with the rate of pathologic tumor response to NAC. Prospective ad-hoc trial focused on dMMR/MSI-H and more accurate molecular profiling are strongly needed in resectable LAGC.

Legal entity responsible for the study: The authors.

Funding: Has not received any funding.

Disclosures: N. Fazio: Advisory / Consultancy: MSD; Research grant / Funding (institution): Local PI of trials for Astellas, Beigene, Research grants from MSD, Merck ; Non-remunerated activity/iesA: ESMO, GI Faculty member. All other authors have declared no conflicts of interest.

https://doi.org/10.1016/j.annonc.2022.04.136

P-47 Inhibition of phosphoprotein phosphatase 2A (PP2A) sensitizes pancreatic cancer to PARP inhibitors by modulation of homologous recombination repair

I. Elgenaidi, M. Lowery

Trinity College Dublin, Trinity College Dublin, Dublin, Ireland

Background: Pancreatic cancer remains one of the most difficult to treat cancers with a 5-years survival of less than 6%, representing one of the most lethal types of cancer, recent research has shown that PP2A inhibitors (LB-100) sensitize pancreatic cancer to chemotherapy and radiation. In addition, the finding from clinical trial study demonstrated that the effectiveness of single-agent Talazoparib for treatment of patients with and without germline BRCA1/2 mutation in ovarian, breast, small cell lung and Pancreatic cancers. The aim of this work was to I. investigate activity of LB-100 against pancreatic cancer cells as monotherapy and in combination with PARP inhibitions (Talazoparib). II. Investigate the mechanism by which LB-100 and Talazoparib effect pancreatic cancer cells by studying effect of drugs on cell cycle and modulation of cell cycle regulatory proteins and assess the role of treatment combination in control DSB and HR repair through modulation of ATM phosphorylation.

Methods: Human pancreatic cancer cell lines Panc-1, MIA-Pa-Ca-2 and BxPC-3 were obtained from European Collection of Authenticated Cell Culture (ECACC) and grown in either Dulbecco's modified Eagle medium (DMEM) (MiaPaCa-2 and Panc-1) or RPMI medium (BxPC-3) with 10% FBS. Cell cultures were maintained in an atmosphere of 5% CO2/95% air at 37 C. PP2A activity was measured by PP2A Immunoprecipitation phosphatase assay Kit (Millipore), cell cytotoxicity were measured by MTT assay, cell cycle determined by flow cytometry. Western blot techniques were used to assess levels of proteins associated with regulation of cell cycle (Cdc2, p-Cdc2, and Cdc25c), apoptosis (caspase3) and DNA damage (γ-H2AX). Data are expressed as the mean $\pm$ (SEM) and analysed by an analysis of variance (ANOVA).

Results: The results show that LB-100 decreased PP2A activity in all pancreatic cancer cells in dose dependent manner. LB-100 significantly decreased cell viability of Panc-1, MIA-Pa-Ca-2 and BxPC-3 with IC50 (3.94 µM, 6.86 µM, and 10.87 µM) respectively. Interestingly adding 25 nm of Talazoparib further decreased IC50 in all cells (1.88 µM, 5.24 µM and 5.83 µM). Treatment combination attenuated pancreatic cancer cells growth through caspase activation and G2/M cell-cycle arrest. Combination therapy impaired cellular repair and induced DNA double-strand breaks by inducing U-H2AX.

Conclusions: Our results suggest that treatment combination of LB100 and talazoparib in vitro inhibit cancer cells growth by modulation of the DNA damage response pathway and cell cycle checkpoint abrogation. The combination of PP2A inhibitor with PARP inhibitor has a synergistic effect in vitro. Further in vivo studies are needed to explore this combination as an effective option in the treatment of pancreatic cancer.

Legal entity responsible for the study: The authors.

Funding: Has not received any funding.

Disclosures: M. Lowery: Advisory / Consultancy: Astra zenica, Servier, Roche. The author has declared no conflicts of interest.

https://doi.org/10.1016/j.annonc.2022.04.137

P-48 Phase 2a study of NT-I7, a long-acting interleukin-7, plus pembrolizumab: Cohort of subjects with checkpoint inhibitor-naïve advanced pancreatic cancer

A. Naing[1], H. Mamdani[2], M. Barve[3], M. Johnson[4], R. Wolff[5], D. Kim[6], S. Yang[7], B. Lee[7], T. Adebanjo[7], R. Georgevitch[7], S. Ferrando-Martinez[7], C. Haymaker[1], M. Chaney[8], J. Fan[7], R. Kim[6], S. Pant[1]

[1]University of Texas MD Anderson Cancer Center, Houston, United States; [2]Karmanos Cancer Institute, Detroit, United States; [3]Mary Crowley Cancer Research, Dallas, United States; [4]Sarah Cannon Research Institute/Tennessee Oncology, Nashville, United States; [5]The University of Texas MD Anderson Cancer Center, Houston, United States; [6]Moffitt Cancer Center, Tampa, United States; [7]NeoImmuneTech, Inc., Rockville, United States; [8]Merck & Co., Inc., Kenilworth, United States

Background: Pancreatic cancer (PaC) is immune-quiescent and resistant to single-agent checkpoint inhibitor (CPI). NT-I7 (efineptakin alfa) is a long-acting IL-7 that can increase T-cell infiltration in the tumor microenvironment (TME) and may enhance tumor responsiveness to CPI therapy. We hypothesize that the combination of NT-I7 and pembrolizumab may result in enhanced efficacy in CPI-naïve advanced PaC.

Methods: This is an open-label, phase 2a study in subjects with relapsed/refractory (R/R) tumors, including CPI-naïve R/R PaC. The study follows Simon's 2-stage minimax design to enroll 17 in the first stage, and 8 additional subjects in the second stage. Subjects received NT-I7 intramuscularly at 1200 µg/kg every 6 weeks (Q6W) plus pembrolizumab 200 mg intravenously Q3W. Antitumor activity based on Overall Response Rate (ORR) was assessed by Response Evaluation Criteria in Solid Tumors (RECIST) v1.1 and iRECIST. Biomarker analyses of peripheral blood and tumor biopsies were performed.

Results: As of 14-March-2022, 32 subjects were enrolled in the CPI naïve R/R PaC cohort. Median age was 66 years [31-81], with ECOG PS 0 (31%), 1 (69%). Twenty-eight (90.3%) subjects had $\geq$ 2 prior therapies. All subjects had metastatic or locally advanced disease at enrollment. The median duration of follow-up was 3.7 months. Among 26 evaluable subjects, the ORR and disease control rate (DCR) were 4% (1/26) and 31% (8/26) by RECIST 1.1; 8% (2/26) and 35% (9/26) per iRECIST. In addition to 2 subjects with iPR, 9 subjects are still ongoing to follow up responders. It was observed that among subjects with $\leq$ 1 liver mets vs $\geq$ 2 liver mets, the ORR by iRECIST was 18.2% (2/11) vs 0; DCR was 63.7% (7/11) vs 13.3% (2/15) and PFS was 19.1 weeks vs 6.0 weeks. The ORR and DCR by iRECIST were 25% (1/4) and 75% (3/4) in 1L; 13% (1/8) and 38% (3/8) in 2L. All subjects with responses continue treatment. NT-I7 treatment-related adverse events (trAEs) occurred in 23 (71.9%) subjects, 18 (56.2%) G1-2, 3 (9.4%) G3; 2 (6.3%) G4; no G5 trAEs were reported. No subjects discontinued from treatment due to trAE. A cPR subject with available biopsy had enhanced T-cell infiltration in the TME at week 5.

Conclusions: The chemotherapy-free combination of NT-I7 + pembro was well tolerated in heavily pretreated subjects with CPI-naïve R/R PaC. The encouraging antitumor activity showed that subjects with with $\leq$ 1 liver mets achieved clinical benefit from the combination of NT-I7 and pembro therapy. Biomarker analyses demonstrated improved peripheral and intratumoral T cell responses. These results support continued evaluation of NT-I7 + pembrolizumab in subjects with CPI-naïve R/R PaC.

Clinical trial identification: NCT04332653.

Legal entity responsible for the study: NeoImmuneTech, Inc.

Funding: NeoImmuneTech, Inc.

Disclosures: A. Naing: Advisory / Consultancy: Novartis, CytomX Therapeutics, OncoSec, STCube Pharmaceuticals Inc, Kymab, Takeda (I), CSL Behring (I), Horizon Pharma (I), , Genome & Company, Immune-Onc, Deka Bioscience, Nouscom; Research grant / Funding (institution): NCI, EMD Serono, MedImmune, Atterocor, Amplimmune, ARMO BioSciences, Karyopharm Therapeutics, Incyte, Novartis, Regeneron, Merck, Bristol Myers Squibb, Pfizer, CytomX Therapeutics, Neon Therapeutics, Calithera Biosciences, TopAlliance BioSciences Inc, Healios, Lilly, Kymab, PsiOxus Therapeutics, Immune Deficiency Foundation (I), Arcus Biosciences, NeoImmuneTech, ImmuneOncia, Surface Oncology, Baxalta (I), Jeffrey Modell Foundation (I), Chao Physician-Scientist Awards (I); Travel / Accommodation / Expenses: ARMO BioSciences; Spouse / Financial dependant: Research funding: The Texas Medical Center Digestive Diseases Center, Jeffery Modell Foundation, Immune Deficiency Foundation, Baxalta US Inc, Chao Physician-Scientist Foundation., Consultant/Advisory board: Takeda, Pharming Healthcare Inc, and Horizon Therapeutics USA, Inc., Ad hoc consultancy speaker: Alfaisal University. M. Johnson: Advisory / Consultancy: Abbvie, Amgen, Astellas, AstraZeneca, Axelia Oncology, Black Diamond, Boehringer Ingelheim, Bristol Myers Squibb, Calithera Biosciences, Checkpoint Therapeutics, CytomX Therapeutics — ALL PAID TO INSTITUTION , Daiichi Sankyo, EcoR1, Editas Medicine, Eisai EMD Serono, G1 Therapeutics, Genentech/Roche, Genmab, Gritstone Oncology, Ideaya Biosciences, iTeos — ALL PAID TO INSTITUTION , Janssen. Lilly, Merck, Mirati Therapeutics, Oncorus, Regeneron Pharmaceuticals, Ribon Therapeutics, Sanofi-Aventis, Turning Point Therapeutics, WindMIL — ALL MONEY PAID TO INSTITUTION ; Research grant / Funding (institution): Abbvie, Acerta, Adaptimmune, Amgen, Apexigen, Arcus Biosciences, Array BioPharma, Artios Pharma, AstraZeneca, Atreca, BeiGene, BerGenBio, BioAtla, Boehringer Ingelheim, Calithera Biosciences, Checkpoint Therapeutics, Corvus Pharmaceuticals, Curis, CytomX, Daiichi Sankyo, Dracen Pharmaceuticals, Dynavax, Lilly, Elicio Therapeutics, EMD Serono, Erasca, Exelixis, Fate Therapeutics, Genentech/Roche, Genmab, Genocea Biosciences, GlaxoSmithKline, Gritstone Oncology, Guardant Health, Harpoon, Helsinn Healthcare, Hengrui Therapeutics, Hutchinson MediPharma, Ideaya Biosciences, IGM Biosciences, Immunocore, Incyte, Janssen, Jounce Therapeutics, Kadmon Pharmaceuticals, Loxo Oncology, Lycera, Memorial Sloan-Kettering, Merck, Merus, Mirati, NeoImmune Tech, Neovia Oncology, Novartis, Numab Therapeutics, Nuvalent, OncoMed Pharmaceuticals, Pfizer, PMV Pharmaceuticals, RasCal Therapeutics, Regeneron Pharmaceuticals, Relay Therapeutics, Revolution Medicines, Ribon Therapeutics, Rubius Therapeutics, Sanofi, Seven and Eight Biopharmaceuticals / Birdie Biopharmaceuticals, Shattuck Labs, Silicon Therapeutics, Stem CentRx, Syndax Pharmaceuticals, Takeda Pharmaceuticals, Tarveda, TCR2 Therapeutics, Tempest Therapeutics, Tizona Therapeutics, TMUNITY Therapeutics, Turning Point Therapeutics, University of Michigan, Vyriad, WindMIL, Y-mAbs Therapeutics. S. Yang: Full / Part-time employment:

NeoImmuneTech. B. Lee: Shareholder / Stockholder / Stock options: NeoImmuneTech Inc.; Full / Part-time employment: NeoImmuneTech Inc.. S. Ferrando-Martinez: Full / Part-time employment: NeoImmuneTech, Inc. M. Chaney: Full / Part-time employment: Merck and Co., Inc. J. Fan: Full / Part-time employment: NeoImmuneTech, Inc; Officer / Board of Directors: Chief Clinical Officer of NeoImmuneTech, Inc. R. Kim: Advisory / Consultancy: Incyte, Roche , Servier, eisai, Exelixis; Speaker Bureau / Expert testimony: incyte, Taiho. All other authors have declared no conflicts of interest.

https://doi.org/10.1016/j.annonc.2022.04.138

P-49 GOBLET: A phase 1/2 multiple indication signal finding and biomarker study in advanced gastrointestinal cancers treated with pelareorep and atezolizumab — safety and preliminary response results

M. Collienne[1], D. Arnold[1], A. Stein[2], E. Goekkurt[3], U. Martens[4], H. Loghmani[5], T. Heineman[5]

[1]Asklepios Tumorzentrum Hamburg AK Altona, Hamburg, Germany; [2]University Medical Center Hamburg-Eppendorf, Hamburg, Germany; [3]Hematology-Oncology Practice Eppendorf (HOPE) and University Cancer Center Hamburg (UCCH), Hamburg, Germany; [4]SLK-Kliniken Heilbronn GmbH, Heilbronn, Germany; [5]Oncolytics Biotech Inc., Calgary, Canada

Background: In GI cancers, checkpoint inhibitors are only effective in patients with high microsatellite instability (MSI-H) or deficient mismatch repair (dMMR) tumors. Oncolytic viruses may improve the susceptibility of microsatellite stable (MSS) tumors to immunotherapy by modifying the tumor microenvironment (TME). Pelareorep (pela) is an intravenously delivered, non-genetically modified, oncolytic reovirus that mediates cancer cell killing by activating innate and adaptive immune responses directed against the tumor as well as direct tumor oncolysis. Accordingly, treatment with pelareorep results in increased T cell infiltration and PD-L1 expression in tumors which primes the TME for responsiveness to checkpoint inhibitors. Pela has demonstrated activity in many cancers including colorectal and pancreatic cancer. The GOBLET study is designed to assess the efficacy of pela plus atezolizumab (atezo) with or without chemotherapy in multiple GI cancers.

Methods: GOBLET is a phase 1/2, open-label, non-randomized study in patients with advanced or metastatic GI cancers and utilizes a Simon two-stage design. In the first stage of the study, four treatment groups are being enrolled: Cohort 1 — First-line metastatic/advanced pancreatic cancer treated with pela plus atezo and chemotherapy (gemcitabine and nab-paclitaxel) (N=12); Cohort 2 — First-line MSI-H/dMMR metastatic CRC treated with pela plus atezo (N=19); Cohort 3 — Third-line metastatic CRC treated with pela plus atezo and chemotherapy (trifluridine/tipiracil) (N=14); and Cohort 4 — Second-line or later advanced squamous cell carcinoma of the anal canal treated with pela plus atezo (N=10). In Cohorts 1 and 3 (chemotherapy-containing cohorts) the first 3-6 patients enrolled comprise a safety run-in. The primary objectives of GOBLET are safety and efficacy measured by the objective response rate (ORR) at week 16. Based on pre-specified response thresholds, any cohort showing a positive ORR signal in Stage 1 may advance to the 2nd Stage and enroll additional patients.

Results: The three safety run-in patients in Cohorts 1 and 3 have been enrolled and their safety data reviewed by the independent Data Safety Monitoring Board (DSMB). The DSMB identified no safety signal and recommended that enrollment into these cohort continue without modification. Enrollment into Cohorts 2 and 4 is ongoing as these cohorts do not include safety run-ins. Tumor response results to date indicate that two of the three Cohort 1 patients had a partial response at week 8, the third Cohort 1 patient had a partial response at week 16.

Conclusions: No safety signal was observed in either the Cohort 1 (first-line pancreatic cancer) or Cohort 3 (third-line CRC) patients. This is consistent with the favorable safety profile observed in prior studies of pela in multiple cancer indications, and it supports the ability to safely treat patients with advanced GI cancers using pela in combination with checkpoint inhibitors and chemotherapy. Preliminary tumor responses to therapy in first-line pancreatic cancer patients are encouraging.

Clinical trial identification: Eudra-CT: 2020-003996-16.

Legal entity responsible for the study: The authors.

Funding: Oncolytics Biotech Inc.

Disclosures: M. Collienne: Research grant / Funding (self): Oncolytics BioTech Inc. D. Arnold: Honoraria (self): Merck, Sharp and Dome; Terumo, Merck Serono, Boston Scientific, Bristol-Meyer Squibb, Pierre Fabre Pharma, Servier, Astra Zeneca, Roche, GSK, Lilly, Sanofi (Genzyme); Honoraria (Institution): Merck, Sharp and Dome, Terumo, Merck Serono, Boston Scientific, Bristol, Meyer Squibb, Pierre Fabre Pharma, Servier, OncoLytics; Advisory / Consultancy: Merck, Shard and Dome, Terumo, Merck Serono, Boston Scientific, Bristol, Meyer Squibb, Pierre Fabre Pharma, Servier, Roche; Research grant / Funding (self): OncoLytics; Travel / Accommodation / Expenses: Boston Scientific. H. Loghmani: Full / Part-time employment: Oncolytics Biotech, Oncolytics Biotech, Oncolytics Biotech. T. Heineman: Shareholder / Stockholder / Stock options: Oncolytics Biotech, Oncolytics Biotech, Oncolytics Biotech; Full / Part-time employment: Oncolytics Biotech, Oncolytics Biotech, Oncolytics Biotech; Officer / Board of Directors: Oncolytics Biotech, Oncolytics Biotech, Oncolytics Biotech. All other authors have declared no conflicts of interest.

https://doi.org/10.1016/j.annonc.2022.04.139

P-50 RETRO-TAS, a retrospective observational study of rifluridine/tipiracil in chemorefractory metastatic colorectal cancer

A. Koumarianou[1], A. Ntavatzikos[2], G. Vourli[2], D. Symeonidis[3], C. Vallilas[2], S. Xynogalos[3], I. Boukovinas[4], G. Papaxoinis[5], S. Demiri[5], K. Kampoli[2], G. Oikonomopoulos[6], M. Giannakakou[2], E. Samantas[6], E. Res[2], N. Androulakis[2], M. Karamouzis[2], J. Souglakos[7]

[1]Hematology Oncology Unit, Fourth Department of Internal Medicine, Attikon University Hospital, National and Kapodistrian University of Athens, Athens, Greece; [2]The National and Kapodistrian University of Athens, Athens, Greece; [3]Metaxa Anticancer Hospital, Piraeus, Greece; [4]BIOCLINIC, Thessaloniki, Greece; [5]Second Department of Oncology, Agios Savvas Anticancer Hospital, Athens, Greece; [6]Metropolitan Hospital, Athens, Greece; [7]University of Crete, Heraklion, Greece

Background: Trifluridine/tipiracil (TAS-102) is an oral combination of an antineoplastic thymidine-based nucleoside analogue, trifluridine, and the thymidine phosphorylase (TPase) inhibitor, tipiracil hydrochloride, indicated for patients (pts) with metastatic colorectal cancer (mCRC) as third line therapy. The approved dose of TAS-102 in adults is 35 mg/m2/dose administered orally twice daily on Days 1 to 5 and Days 8 to 12 of each 28-day cycle as long as benefit is observed or until unacceptable toxicity occurs. We conducted an investigator initiated retrospective analysis in patients with chemorefractory mCRC treated with TAS-102 to record clinical practice and to collect real world data on the clinical efficacy of TAS-102 in the Greek population.

Methods: Clinicopathologic characteristics of patients treated in 8 Cancer Centres were collected to assess physician's choice of treatment in chemoresistant mCRC with TAS-102 in third line and beyond. In addition, the clinicopathologic features related to mCRC (focus on molecular profile), duration of treatment, dose modification and toxicity were analysed. The PFS, the OS, the 6-/8-month PFS rate and the disease control rate were calculated. Prognostic factors were evaluated by Cox regression model and Kaplan-Meier curves, along with log-rank tests using Stata/MP 16.0 for Windows.

Results: From October 2018 to October 2021, 200 patients with a median age at diagnosis of 63.7 years (IQR 54.2, 72.1) and at TAS-102 treatment initiation was 67.0 (IQR 58.0, 75.0). At the time of the analysis the median follow-up time was 14 months (IQR 7, 23), 158 PDs and 106 deaths were recorded. Of all patients 42% were females and 58% were metastatic at diagnosis. Molecular analysis revealed mutations in KRAS (52%), NRAS (5%), HER2 (3.5%), BRAF (3.5%) and MSI (9%). Adjuvant chemotherapy and radical surgery was delivered in 39.5% and 51.5% respectively. TAS-102 was administered as a third (70.5%), fourth (17.0%) or fifth line (12.5%) of therapy. Serious adverse events reported were neutropenia (4pts), anemia (2pts), thrombocytopenia (1pt), diarrhea (1pt), nausea (1 pt) and fatigue (8 pts). Dose reduction, delay of initiation of the next cycle and shorter duration of therapy was reported in 25%, 31% and 14.5% of patients. Patients received TAS-102 as monotherapy (71.5%), in combination with bevacizumab (24.5%) or with an anti-EGFR agent (4.0%). The median duration of TAS-102 therapy was 119.5 days and 81% of patients discontinued therapy due to progressive disease. Objective responses during TAS-102 therapy included 0.5% CR, 25% PR, 20% SD and 47% PD, while 7.5% of patients were not evaluable. The median PFS time was 4.8 and the median OS was 11.4 months. The 6 and the 8-month PFS rate was 41.4% and 31.5% respectively. In the multivariable analysis PS>1 and metastatic disease in the liver and lung were adversely associated with survival whereas tumor sidedness and mutational status were not.

Conclusions: This real-world observational study confirms and adds on the findings of the RECOURSE phase III study in relation to the toxicity and the effectiveness of TAS-102 in all subgroups of patients with chemotherapy refractory mCRC, regardless of mutational status and sidedness.

Clinical trial identification: RETRO-TAS study NCT04965870.

Legal entity responsible for the study: The authors.

Funding: Hellenic Study Group of Psychoneuroimmunology in Cancer.

Disclosures: J. Souglakos: Honoraria (Institution): Servier; Advisory / Consultancy: Servier; Travel / Accommodation / Expenses: Servier. All other authors have declared no conflicts of interest.

https://doi.org/10.1016/j.annonc.2022.04.140

P-51 Non-invasive HER2 status diagnosis in gastric cancer using surrogate DNA methylation markers

X. Liu[1], G. Ye[2], X. Lei[2], H. Li[3], T. Yang[3], S. Chen[3], Y. Yu[2], X. Chen[2], G. Zhang[2], H. Sun[2], M. Bibikova[1], C. Cui[2], Z. Chen[3], J. Fan[2]

[1]AnchorDX INC., Fremont, United States; [2]Southern Medical University, Guangzhou, China; [3]AnchorDX Medical Co. Ltd, Guangzhou, China

Background: Gastric cancer (GC) is the fifth most common and fourth most lethal cancer worldwide. Unlike other cancer types, e.g., lung or breast cancer, very few targeted therapeutics have been developed for GC. HER2 (ERBB2) status is an essential biomarker for guiding the trastuzumab (Herceptin) therapy, which is the only molecularly targeted drug accepted as a first-line therapy, for the treatment of patients with advanced HER2-overexpressing GC. HER2 detection in GC often requires repeated testing to improve the accuracy of the result due to its high degree of