

REPORT ASCO 2022 HYBRID: RESPIRATORY ONCOLOGY 10 POINT HIGHLIGHTS

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Overall, at ASCO 2022 there were some refinements or longer follow-up data of previously reported practice changing trials, but no truly primary presentations of new trials that will change current practice.

1/ Resectable NSCLC – adjuvant immunotherapy

The ultimate goal of adjuvant therapy in resected NSCLC is improvement of cure rates, often measured by 5-year overall survival (OS). As this is the case with adjuvant cisplatin-based chemotherapy for completely resected stage II-IIIa NSCLC, this approach is standard in the current ESMO guidelines.

The *IMpower-010* study added to this, by documenting a significantly improved disease-free survival (DFS) with **adjuvant atezolizumab** in this particular group. The effect was dependent on the PD-L1 expression of the tumor, and most relevant for tumors with PD-L1 $\geq 50\%$. Consequently, EMA has approved adjuvant atezolizumab for stage II and IIIa non-oncogene-addicted NSCLC with PD-L1 expression $\geq 50\%$ after complete resection and adjuvant chemotherapy.

First results of the similar phase 3 trial *PEARLS/Keynote-091* with **adjuvant pembrolizumab** were presented at a recent ESMO virtual plenary. In the overall analysis, pembrolizumab significantly improved DFS (Hazard Ratio (HR) 0.76, 95%CI [0.63-0.91], $P=0.0014$). PD-L1 expression had a somewhat counterintuitive influence on efficacy: HR 0.78 [0.58-1.03] for PD-L1 $<1\%$, HR 0.67 [0.48-0.92] for 1-49%, and 0.82 [0.57-1.18] for PD-L1 $\geq 50\%$.

#8512 reported a subgroup analysis of this *PEARLS* trial with a median follow-up time of 35.6 months (mo). The most notable results were for nodal status (HR 1.00 for pN2, N=231), and for delivery of adjuvant chemotherapy (HR 1.25 for no chemotherapy, N=167). The author concluded that “pembrolizumab generally improved DFS regardless of all of these factors”. A quite debatable statement. First presenting a subanalysis with quite remarkable findings, and then concluding that the therapy is similarly good for all.

Based on the now reported phase 3 trials, we have data in favor of adjuvant immunotherapy only in patients who are able to receive adjuvant chemotherapy. It remains unclear how the controversial PD-L1 expression findings in the *PEARLS* trial need to be interpreted, as the *IMpower 010* study and most neoadjuvant trials showed a qualitative enrichment of the effect of immunotherapy with increasing PD-L1 levels.

2/ Potentially resectable NSCLC – neoadjuvant chemo-immunotherapy

For patients with potentially resectable NSCLC – especially those with stage IIIa-N2 – an approach of neoadjuvant chemotherapy followed by resection is often chosen. The Spanish Lung Cancer Group previously reported the single-arm phase 2 *NADIM* trial, with 3 cycles of neoadjuvant nivolumab plus chemotherapy for resectable stage IIIa-N2 patients, with very favorable results for pathologic complete response (pCR) and outcome, compared to historical controls.

#8501 now brought a first analysis of the follow-up phase 2 randomized *NADIM-2* trial comparing **nivolumab plus chemotherapy with chemotherapy alone**, followed by 6 months nivolumab postoperatively. The primary endpoint, pCR by blinded independent pathological review (BIPR), was significantly better in the combined arm: 36.2% vs 6.8%, $P = 0.0071$). Surgery occurred in 91% of the patients in the combination arm and 69% in the chemotherapy arm (difference mainly due to more progressive disease). The pCR rate rose across increasing categories of PD-L1: $< 1\%$ 14.3%; 1-49% 41.7%; $\geq 50\%$ 61.1% ($P = 0.008$).

The phase 3 *Checkmate-816* compared **3 cycles of neoadjuvant nivolumab plus chemotherapy vs. chemotherapy alone** in stage IB-IIIa (pTNM 7th) NSCLC patients eligible for surgery. At the recent AACR meeting, the first outcome data showed a significant improvement in event-free survival (EFS) with a HR 0.63 [0.43-0.91]. The effect was clearly PD-L1 dependent: HR 0.85 [0.54-1.32] for PD-L1 $<1\%$, HR 0.58 [0.30-

1.12] for 1-49%, and 0.24 [0.10-0.61] for $\geq 50\%$. Moreover, nearly all benefit came from stage IIIA (HR = 0.54 [0.37-0.80]), while there was no significant difference in stages IB/II (HR = 0.87 [0.48-1.56]). ASCO #LBA8511 reported a post-hoc analysis from *CheckMate-816* on the **association between pCR and EFS**. The 2-year EFS with nivolumab plus chemotherapy was 93% in patients with pCR (N=46) vs. 58% in those without pCR (N=95). Moreover, there was semi-quantitative correlation between depth of EFS and % residual viable tumor cells in the resection specimen: 2-year EFS was 90%, 60%, 57%, and 39% in case of 0–5%, >5–30%, >30–80%, and >80% residual viable tumor.

Of note, the discussant clearly stated that chemo-immunotherapy now is the best choice for induction in potentially resectable stage III-N2, but that this approach should not be used to transform unresectable stage III into surgical stage III. Assessment of resectability, and preference of neoadjuvant vs adjuvant systemic therapy, remains to be determined at the time of diagnosis in a dedicated multidisciplinary tumor board.

3/ Unresectable stage III NSCLC

Current ESMO guidelines recommend concurrent chemoradiotherapy (CRT) followed by 1 year of durvalumab consolidation in unresectable stage III NSCLC. The 2-year PFS rate was 45.0%, 2-year OS rate was 66.3%. In the recent 5-year follow-up report (J Clin Oncol 40:1301, 2022), the 5-year OS was 43%, with 33% of patients free of disease at 5 years. Ongoing clinical trials now explore if the concurrent administration of immunotherapy during CRT or the use of combination immunotherapy may give further improvement.

#8508 gave longer follow-up data (median 30.2/25.4 mo) of the *Keynote-799* phase 2 study looking at **pembrolizumab concurrently with CRT**. There were 2 cohorts according to the chemotherapy, either carboplatin-paclitaxel (pac, N=112) or cisplatin-pemetrexed (pem, non-squamous only, N=102). After CRT, 14 additional cycles of pembrolizumab 200 mg q3w were given. Primary endpoints were objective response rate (ORR) and incidence of grade ≥ 3 pneumonitis. ORR was 71.4% (pac) and 75.5% (pem). Grade ≥ 3 pneumonitis occurred in 16 patients (7.5% overall, 8.0% for pac, 6.9% for pem). 2-year PFS rates were 55.3% (pac) and 60.6% (pem). 2-year OS rates were 64.3% (pac) and 71.2% (pem). Treatment-related adverse events (TRAEs) grade ≥ 3 occurred in 64.3% (pac) and 51.0% (pem). Side effects were judged to be manageable.

#8509 reported on the *BTCRC LUN 16-081* phase 2 study with **consolidation immunotherapy with either nivolumab (nivo) or nivolumab-ipilimumab (nivo-ipi)**. Particular for this study was the duration of consolidation of 6 mo only. The primary endpoint was 18 mo PFS compared to historical controls. The percentage of patients completing the full therapy was 70.4% (nivo, N=54) and 56.9% (nivo-ipi, N=51). The 18 mo PFS was 62.3% (nivo) and 67% (nivo-ipi), and median PFS was 25.8 and 25.4 mo. The 2-year OS estimates were 76.6% (nivo) and 82.8% (nivo-ipi). Grade ≥ 3 TRAEs were seen in 38.9% (nivo) and 52.9% (nivo-ipi) of the patients. The rate of grade ≥ 2 pneumonitis was 22.2% (nivo) and 29.4% (nivo-ipi), with 9.3% and 15.7% grade ≥ 3 pneumonitis events.

As a whole, these two phase 2 trials show somewhat better 2-year PFS and OS rates than what has been seen in PACIFIC, but at the cost of increased toxicity, including pneumonitis, especially with nivo-ipi. Results of ongoing phase 3 trials are needed to judge if the balance between increased efficacy and increased toxicity will be worthwhile to replace the standard PACIFIC regimen.

4/ Advanced NSCLC with high tumor PD-L1 expression: 1L immunotherapy vs. chemo-immunotherapy

For non-oncogene driven advanced NSCLC with tumor PD-L1 expression $\geq 50\%$, we have both chemotherapy plus immunotherapy (chemo-IO) and immunotherapy alone (IO) reimbursed in 1L. However, we do not have a head-to-head comparison, and the choice between these two strategies in clinical practice is therefore at the discretion of the treating physician, based on patient characteristics and patient or physician preferences.

#9000 presented a retrospective **exploratory pooled FDA analysis of 12 RCTs** that supported FDA approval of IO-based 1L regimens. Data on 455 patients who received chemo-IO and 1,298 patients who

received IO in 1L were abstracted, all with tumor PD-L1 expression $\geq 50\%$ and without *EGFR* or *ALK* driver. The primary outcome measure – OS – was not significantly different between both regimens, with a median pooled OS of 25.0 mo with chemo-IO and 20.9 mo with IO (HR 0.82 [0.62-1.08]). PFS and ORR were, however, superior with chemo-IO vs IO regimens: PFS of 9.6 vs 7.1 mo (HR 0.69 [0.55-0.87]), and ORRs of 61% vs 43% (odds ratio 1.2 [1.1-1.3]). Data on subsequent therapies or toxicity were not presented. Likewise, data according to additional PD-L1 expression cut-off points ($\geq 60\%$, $\geq 70\%$, $\geq 80\%$, $\geq 90\%$, 100%) were not provided. Selected subgroup analyses confirmed that never-smokers are no good candidates for IO only regimens. In addition, outcomes in older patients (≥ 75 years, 11% of the study population) favored IO only, as assessed by OS (HR 1.68 [0.69-4.06]) and PFS (HR 1.22 [0.58, 2.57]). At this time, it is not clear whether the latter finding is related to more chemotherapy-related toxicity in older patients. However, these exploratory data underscore the need to always properly weigh the risks and benefits of adding chemotherapy to immunotherapy in patients with high PD-L1 expressing tumors.

5/ Advanced NSCLC: impact of *KRAS* mutation status on 1L treatment outcomes

#9001 further explored the potential impact of the *KRAS* mutation status on the efficacy of 1L therapy (chemo-IO, IO only, chemo only) in the pooled data of the same 12 FDA selected registrational trials, but now including all PD-L1 levels. In the mutation evaluable population (16%, N=1,430), 39% was *KRAS* mutant (*KRAS*-Mt) and 11% was positive for the G12C mutation. PD-L1 expression levels (<1%, 1-49%, $\geq 50\%$) were fairly evenly distributed among *KRAS*-Mt and *KRAS* wild-type (*KRAS*-wt) subgroups.

ORRs to first line treatment were comparable between *KRAS*-Mt and *KRAS*-wt tumors. Likewise, OS did not differ significantly according to *KRAS* mutation status: HR 1.12 [0.86-1.46] for chemo-IO, HR 1.01 [0.76-1.34] for IO only, and HR 1.02 [0.81-1.29] for chemo only, with a similar course of the Kaplan-Meier curves. In addition, no clear interaction between *KRAS* mutation status and PD-L1 expression on treatment efficacy was observed. Data on co-mutations (*KEAP1*, *STK11*, *TP53*) were not available. Analysis of response and outcome data of the *KRAS* G12C subgroup according to PD-L1 level is ongoing. So, to date, *KRAS* mutation status cannot guide first line treatment decisions.

6/ Advanced NSCLC: next line treatment after platinum-doublet chemotherapy and immunotherapy

Despite the survival benefit with immune checkpoint inhibition as 1L (or now rarely 2L treatment) for advanced NSCLC, only 20-30% of patients derive long-term clinical benefit. The currently available insights into the molecular mechanisms of acquired resistance have not yet led to a therapeutic break-through to address progressive disease on IO. The LUNG-MAP trial is a master protocol in the United States trying to address this issue, including sub-studies testing different strategies in previously IO-treated non-oncogene driven NSCLC.

#9004 presented data of the randomized phase 2 *LUNG-MAP S1800A* sub-study on the combination of **ramucirumab plus pembrolizumab (RP)** vs. standard of care (SoC) therapy regimens after progression on PD-(L)1 inhibition and platinum-doublet chemo (either concurrent or sequentially). Ramucirumab is a monoclonal antibody targeting the extracellular domain of the vascular endothelial growth factor receptor 2 (VEGFR2), which is involved in tumor angiogenesis and anti-tumor immunity. Importantly, 67% of the patients in the SoC arm also received ramucirumab in combination with docetaxel. The study was positive with a significant improvement in the primary endpoint of OS in the experimental arm of 14.5 mo vs 11.6 mo for SoC (HR 0.69). A survival benefit with RP was observed across all subgroups, including by PD-L1 expression. Squamous cell histology seemed to benefit more than the non-squamous subgroup. There was, however, no difference in ORR or PFS. The authors hypothesized that a survival benefit – in the absence of ORR or PFS improvement – is related to ramucirumab that renders the tumor sensitive to pembrolizumab again, and this favorable effect on tumor biology is then carried over to the post-progression period. It has indeed already been reported that prior IO may enhance the efficacy of subsequent therapy. The safety was consistent with known toxicity of both drugs: grade ≥ 3 TRAEs occurred less in the chemotherapy-free RP vs. SoC arm (42% vs 60%), but there were 31% grade ≥ 3 immune-related AEs in the RP arm. The survival benefit observed with RP in this rather

small (N=69 vs. 67) phase 2 trial now needs to be confirmed in a phase 3 trial before this would become practice changing. Moreover, more insights on mechanism of action of this combination after IO resistance and on predictive biomarkers are needed.

7/ Advanced NSCLC oncogene addiction: EGFR & overcoming osimertinib resistance

Disease progression on 1L osimertinib is based on the development of a broad range of resistance mechanisms: known on-target resistance (such as acquired kinase domain mutations e.g. C797X mutation, EGFR amplification), bypass alterations (such as MET amplification, HER2 amplification, PI3KCA mutation), histologic transformation, or yet unknown.

#9006 reported on updated results from *CHRYSALIS-2* cohort A, a phase 2 trial of **amivantamab 1050/1400 mg i.v. plus lazertinib 240 mg orally** for the treatment of osimertinib-relapsed and chemotherapy treated (as last line of therapy) *EGFR* (ex19 or ex21) mutant NSCLC. Avivantamab is a bispecific antibody targeting EGFR and MET, while lazertinib is a potent third generation EGFR-TKI. In 162 efficacy-evaluable patients (median FU time of 10.0 mo), an ORR of 33% and a median duration of response (DoR) of 9.6 mo were reported. The safety profile was tolerable with grade ≥ 3 TRAEs in 10% and TRAEs leading to discontinuation of both drugs in 7% of patients. Phase 3 *Mariposa-1* and *Mariposa-2* trials are now investigating amivantamab plus lazertinib as a first-line regimen, and amivantamab plus lazertinib in combination with chemotherapy after osimertinib failure as a second-line regimen, respectively.

#9013 was a phase 1/1b study of **osimertinib plus telisotuzumab-vedotin i.v. q2w** (an anti-c-MET antibody drug conjugate, ADC) for the treatment of osimertinib-relapsed *EGFR*-mutated NSCLC with c-MET overexpression (intermediate to high 3+ IHC staining). In 25 patients, an ORR of 58% shows an early sign of activity, with grade ≥ 3 AEs in 32% and no dose-limiting toxicities reported.

8/ Advanced NSCLC oncogene addiction: KRAS(G12C)

A somatic activating *KRAS* mutation is present in 30% of lung adenocarcinomas. The most common variant is *KRAS(G12C)* accounting for 13%. At AACR 2022, updated results on **sotorasib** 960 mg orally once daily in *KRAS(G12C)* mutant NSCLC (*CodeBreak100* trial; N=174, with median FU time of 25 mo) reported an ORR 41%, disease control rate (DCR) 84%, median DoR 12.3 mo, median OS 12.5 mo, 2-year OS rate 33%, grade ≥ 3 TRAEs in 21%, and TRAEs leading to treatment reduction in 22% and discontinuation in 6% of patients, respectively. Similar to sotorasib, adagrasib is a small molecule inhibitor that irreversibly and selectively binds to the cysteine residue of the mutant *KRAS* protein.

#9002 reported on the registrational phase 2 cohort of the *KRYSTAL-1* trial considering **adagrasib** 600 mg orally bid (fasted) in pretreated (platinum-based chemotherapy and anti-PD-1/L1 therapy) *KRAS(G12C)* mutant NSCLC. 116 patients were enrolled and treated with a median FU time of 12.9 mo. 112 patients were evaluable for efficacy: ORR by BIRC was 43%. Secondary endpoints DCR 80%, median DoR 8.5 mo, median PFS 6.5 mo, 1-year OS rate 51%, and median OS 12.6 mo. Grade ≥ 3 TRAEs were observed in 43%, and TRAEs led to dose reductions in 52% and to treatment discontinuation in 7%, respectively.

#LBA9009 reported on the activity of **adagrasib** 600 mg bid in pretreated *KRAS(G12C)* mutant NSCLC patients with active, asymptomatic, untreated CNS metastases (N=25; 19 had radiographically evaluable lesions). Intracranial (IC) response by mRANO-BM BICR was 32% and IC DCR was 84%. Concordance between systemic and IC disease control was 88%. At median FU time of 6.6 mo, median IC DoR was not reached and median IC PFS was 4.2 mo.

9/ Advanced NSCLC oncogene addiction: updates in EGFRex20ins/MET/ROS1/NTRK/NRG1

a. EGFR exon 20 insertion mutation (EGFRex20ins)

These mutations are present in about 2-3% of NSCLC and have a poorer outcome than more common *EGFR* mutations. Several targeted therapies are in clinical investigation of whom 2 have been recently FDA approved. **Amivantamab** (*CHRYSALIS* phase 1; N=81; median FU time 9.7 mo) demonstrated an ORR 40%, CBR

74%, median DoR 11.1 mo, and median PFS 8.3 mo. Grade ≥ 3 TRAEs occurred in 16% (of note rash in 4% and infusion related reaction in 3%), and TRAEs leading to discontinuation in 11% of patients. **Mobocertinib** 160 mg QD in platinum-pretreated patients (N=114; median FU time 14 mo; ASCO 2021 #9014) demonstrated an ORR 28%, DCR 78%, median DoR 17.5 mo, median PFS 7.3 mo. Grade ≥ 3 TRAEs occurred in 47% (of note grade 3-4 diarrhea in 21% and rash in 45%), and TRAEs led to treatment discontinuation in 17% of patients. Safer and more effective novel therapies to treat *EGFR* mutant NSCLC remains an unmet medical need.

#9007 presented results of a phase 1/2a study on **CLN-081**, a novel selective irreversible EGFR inhibitor for *EGFR* mutant NSCLC, in heavily pretreated patients (N=73; dose 30 to 150 mg BID). For the optimal dose of 100 mg BID, the ORR was 41%, median DoR >21 mo, and median PFS 12 mo. Of note, grade ≥ 3 TRAEs in only 5% of the patients (no grade ≥ 3 rash or diarrhea occurred at doses <150mg BID), and TRAEs led to treatment discontinuation in 8% of patients.

#9015 presented results of two ongoing *WU-KONG* phase 1/2 studies on **sunvozertinib** in platinum-pretreated *EGFR* mutant NSCLC (N=52; median follow-up time 10.5 mo): ORR 40%, DCR 85%, and median DoR 5.9 mo. In platinum-pretreated without versus with prior ICI, grade ≥ 3 TRAEs occurred in 39% versus 44% and TRAE led to treatment discontinuation in 3% versus 6% of patients, respectively.

b. MET exon 14 skipping mutation (METex14)

Two targeted therapies have been recently EMA approved within a relapse context. **Capmatinib** 400 mg bid (*GEOMETRY-mono-1* study) showed in a pretreated cohort (N=100) an ORR of 44%, median DoR of 9.7 mo, and median PFS of 5.5 mo. **Tepotinib** 500 mg qd (phase 2 *VISION* study) showed in a pretreated cohort (N=83) an ORR 45%, DCR 72%, median DoR of 11 mo, and median PFS 11 mo.

#9008 reported on an expansion cohort of the *CHRYSALIS* study, with **amivantamab** for relapsed primary *METex14* mutated NSCLC. So far, 55 patients (of whom 28 MET-TKI pretreated) were included. In 46 efficacy evaluable patients, ORR was 33% (55% in MET-TKI naive and 17% in MET-TKI pretreated), DCR 59%, median DoR not reached, and median PFS of 6.7 m. A tolerable safety profile was reported (of note, any grade hypoalbuminemia in 27%, any grade peripheral edema in 20%, any grade pneumonitis in 4%, grade ≥ 3 rash-related AEs in 4%, and grade ≥ 3 infusion-related reaction in 5%). TRAEs led to discontinuation in 5% of patients.

c. c-MET overexpressing EGFRwt NSCLC.

#9016 reported updates of *LUMINOSITY* phase 2 study on **telisotuzumab-vedotin** (an ADC joining anti-c-MET mAb to cytotoxic microtubule inhibitor payload) in pretreated c-MET overexpressing NSCLC (N=136). Criteria for futility were reached for the SQ-NSCLC and *EGFR* mutant NSQ-NSCLC cohorts. In evaluable *EGFR*-Wt NSQ-NSCLC patients (N=52), ORR was 37% (52% in c-Met high and 24% in c-Met intermediate) with a median DoR of 6.9 mo. Any grade TRAE occurred in 76% (of note pneumonitis in 7%), and TRAEs led to discontinuation in 13% of patients.

d. ROS1 gene fusion.

#LBA9023 showed the results of **entrectinib** 600 mg bid in untreated ROS1+ (on liquid biopsy) NSCLC patients enrolled in *BFAST* (N=55): ORR 82%, median DoR 13 mo, and median PFS 13 mo. The lower survival efficacy of entrectinib within *BFAST*, compared to tissue biopsy positive ROS1 NSCLC patients within *STARTRK* trials, can be explained by a lower likelihood of ROS1 detection by liquid biopsies in indolent cancers with low tumor burden, lower levels of ctDNA, and thus better prognosis.

e. NTRK gene fusion.

#9024 showed updates on **larotrectinib** 100 mg bid in 26 pretreated TRK+ (NTRK1 81% and NTRK3 19%) lung cancer patients. The ORR was 83%. At a median FU time of 12.9 mo, median DoR and median PFS were not reached, and 24-months OS is 72%. For 10 evaluable patients with CNS metastases, the ORR was 80%.

f. NRG1 gene fusion.

Neuregulin 1 (NRG1) rearrangements are oncogenic drivers and their frequency in NSCLC is 0.3% (enriched in lung invasive mucinous adenocarcinoma). NRG1 is a ligand that binds to HER3.

#105 Zenocutuzumab is a bispecific antibody that docks on HER2 and blocks NRG1 interaction with HER3. Updated results on zenocutuzumab 750 mg i.v. q2w within an ongoing phase 1/2 open label multi-tumor trial and early access program demonstrated an ORR of 35% with a median DoR 9.1 mo in 47 NSCLC patients. Zenocutuzumab was well tolerated with grade ≥ 3 TRAEs in only 6% and treatment discontinuation due to TRAEs in <1% of patients.

g. Oncogenic alteration other than EGFRex19/21.

#9017 reported preliminary data from a phase 1 trial of **patritumumab-deruxtecan** (HER3-DXd; an ADC composed of a HER3 directed mAb patritumab and a topoisomerase I inhibitor payload) in pretreated non-EGFRex19/21 mutant NSCLC. In 47 evaluable patients (median FU time of 19.7 mo) treated with HER3-DXd (5.6 mg/kg), an ORR of 29% and median PFS of 6 mo were observed. Efficacy was similar whether an oncogenic alteration was identified or not. Safety profile was manageable with of note treatment-related interstitial lung disease in 11%. Grade ≥ 3 TRAEs occurred in 51% and TRAEs led to discontinuation in 11% of patients.

10/ Metastatic SCLC

In the recently updated ESMO guidelines (Ann Oncol 32:839, 2021), the standard 1L therapy for stage IV SCLC is platinum-etoposide chemotherapy plus an anti-PD-L1 antibody, either atezolizumab or durvalumab. Just as in NSCLC, recent clinical trials try to improve the results further with combination immunotherapy.

#LBA8507 reported on the phase 3 study *Skyscraper-02* comparing 1L chemotherapy plus atezolizumab with **chemotherapy plus atezolizumab & tiragolumab** (anti-TIGIT immuno compound). TIGIT is a novel inhibitory immune checkpoint present on activated T cells and NK cells. Tiragolumab synergized with anti-PD-(L)1 inhibitors in preclinical data. 490 patients with untreated SCLC (asymptomatic treated or untreated brain metastases permitted) were randomized 1:1 to receive either the standard IMpower-133 regimen plus either Tiragolumab or placebo. In short, co-primary endpoints of investigator-assessed PFS and OS reproduced the IMpower-133 results, but were identical in both arms, no additional benefit whatsoever.

Full abstracts of this ASCO meeting can be found at: <https://meetings.asco.org/abstracts-presentations> .

For your calendar:

Respiratory Oncology Update 2022: Saturday 08/10/2021, in Dolce La Hulpe, follow updates on <https://www.update-respiratoryonco.be/>