Targeted agents in SCLC

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Head of thoracic tumour unit
Disclosure

• Receipt of honoraria or consultation fees: Roche, BMS, MSD, Novartis, Pfizer, AstraZeneca, BI, Abbvie, Guardant.

• Participation in a company sponsored speaker’s bureau: Roche, BMS, MSD, Novartis, Pfizer.
Outline

• Introduction
• Main therapeutic areas of interest:
  – DLL3
  – MYC
  – PARP1
  – IO
• Take home messages
Introduction

- It accounts for 15% of lung cancer:
  - It kills 250,000 people worldwide yearly.
- Almost all patients are current or former smokers
  - The incidence is increasing in LMIC.*
- Very different from other lung cancers: pathologically, molecularly, biologically and clinically.
- Since the approval of Topotecan in 1996, no important clinical advances.

LMIC: low- medium income countries
• Treatment and survival has not changed in more than 40 years.
• Development of durably effective drugs is needed desperately.
• A plethora of molecular targeted agents have been investigated and most have failed to demonstrate clinical benefit.
• By contrast with NSCLC, progress has been hampered by the scarcity of specific molecular targets.

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<th>Activity</th>
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<tbody>
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Introduction II

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SCLC, a recalcitrant disease

- Since the approval of Topotecan in 1996, no important clinical advances.

The US Congress declares that SCLC is a recalcitrant cancer requiring special resources and efforts.

The challenges for developing translational research in SCLC

• Scientific studies are hampered by a lack of tissue availability.
• Lack of resected tumours have increased the importance of preclinical models for understanding the biology of SCLC
• Resurgence of studies on SCLC:
  – Comprehensive molecular analysis
  – Development of relevant genetically engineered mouse model (GEMM models)
  – Establishment of patient-deriver xenografts (PDX models)
• Discovery of new potential vulnerabilities of SCLC
• Clinical problems: time, clinical trials, setting
Comprehensive genomic profiles

Nearly universal and biallelic loss

George et al. Nature 2015
Frequent Genomic Alterations in SCLC

- 883 SCLC cases reviewed assayed with comprehensive genomic profiling (CGP).
- Median number of genomic alterations: 5
- Most common altered genes: TP53 (90%), RB1 (69%), MLL2 (12.0%), LRP1B (10.9%), PTEN (8.5%), MYCL1 (8.0%), RICTOR (6.5%), and MYC (6.1%)
- MYCL1 amplification was found in 68 (7.8%) of cases.
- 7 cases (<1%) harbored MYCL1 Fusions, and 5 of these cases also had MYCL1 amplification.
SCLC is not an homogeneous disease

- Three distinct molecular subtypes determined by differential expression of transcription factors:\(^1\):
  - **High expression levels of ASCL1 (75%)**:
    - It is a master regulator and induces neuronal and neuroendocrine differentiation
    - ASCL1 + SCLC usually express the entire set of NE markers
    - ASCL1 expression is associated with DLL3 expression, which encodes an inhibitor of NOTCH signalling.
  - **High expression levels of NEUROD1 (15%)**:
    - Neuronal master regulator
    - Over-expression of MYC
  - **Lack of expression** of either of these neuroendocrine markers

Gazdar A. Nature Reviews 2017
SCLC is not an homogeneous disease (II)

• These subgroups are clearly different disease entities based on gene-expression profiling but the clinical implication of this molecular classification have not been defined yet.
  – The switch from ASCL1 to NEUROD1 expression is associated with a change from the typical to the ‘variant’ form of SCLC.
  – Loss of ASCL1 expression more frequent in relapsed tumours.

¹Savari J. Nature Reviews 2017
SCLC: Main therapeutic areas of interest

Sabari J, et al. Nat Reviews 2017
The Notch signaling pathway acts as a developmental pathway essential in the control of differentiation of neuroendocrine cells of the lung.

It is dysregulated in a variety of malignancies, and can behave as either an oncogene or a tumor suppressor depending upon cell context.

It is involved in signaling between contiguous cells and impacts multiple aspects of cancer biology.
Targeting DLL3
Rova-T: an antibody-drug conjugate (ADC)

- DLL3 is an atypical inhibitory Notch ligand induced by ASCL1.
- > 80% of SCLC and LCNEC express DLL3 on the cell surface but it is not expressed on normal tissues
- Rova-T is an ADC targeting DLL3
- It binds to DLL3-expressing cells and is internalized.
- Pyrrolobenzodiazepine toxin is released in the cytosol, enters the nucleus, and binds to the DNA, which causes DNA damage and eventual cell death.
Rova-T: first in class phase 1 study

- 84 patients (74 SCLC); Recommended dose: 0.3 mg/kg/6 wk, **2 cycles**
- **Unique toxicity profile:**
  - Trombocytopenia (12% Gr 3-4). Median onset 15 days. Median duration 22 days.
  - Serosal effusions (11% Gr 3-4). **Median onset 74 days.** Median duration 15 days
  - Skin reactions (8% Gr 3-4). Median onset 30 days. Median duration 21 days.
- **Activity** (65 p. assessable for activity):
  - RR 17%, SD 54%. MST 4.6 m. 1-y OS 18%
- **Exploratory analysis** (39 samples):
  - DLL3 high (29 p): RR 35%, DCR 90% MST 5.8 m, 1-y OS 32%
  - DLL3 low (10 p): RR 0%, DCR 60%, MST 2.7 m, 1 y OS 0%
  - Chemo-resistant DLL3 high RR 29%

Rudin C. Lancet Oncol 2016
Rova-T: Best Response, Time on Study and Duration of Response

Confirmed response rate
Overall: 11/60 (18%)
DLL3-high: 10/26 (38%)

Investigator and Central Data

<table>
<thead>
<tr>
<th>DLL3 Expression</th>
<th>DOR, mo (95% CI)</th>
<th>PFS, mo (95% CI)</th>
<th>DOR, mo (95% CI)</th>
<th>PFS, mo (95% CI)</th>
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</thead>
<tbody>
<tr>
<td>All</td>
<td>5.6 (2.5-8.3)</td>
<td>2.8 (2.5-4.0)</td>
<td>4.4 (2.2-6.5)</td>
<td>4.0 (2.6-4.8)</td>
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<tr>
<td>High</td>
<td>4.3 (2.2-15)</td>
<td>4.3 (2.8-5.6)</td>
<td>4.6 (2.2-6.9)</td>
<td>4.6 (4.0-5.7)</td>
</tr>
</tbody>
</table>

Rova-T: first in class phase 1 study summary

- First biomarker-directed therapy in SCLC (~67% DLL3^{Hi})
- Safety profile is emerging and appears manageable.
- Single-agent activity in recurrent / refractory SCLC
  - Comparable responses in both second- and third-line setting
  - Encouraging response and survival data prompting further development
- Results justify further clinical development.
### Overview of Ongoing Rova-T Studies

<table>
<thead>
<tr>
<th>Ongoing Studies</th>
<th>SCRX001-002 (TRINITY)(^1)</th>
<th>M16-289 (TAHOE)(^2)</th>
<th>M16-298 (MERU)(^3)</th>
<th>SCRX001-004 (Frontline)(^4)</th>
<th>M16-300 (IO Combo)(^5)</th>
<th>SCRX001-007 (QT)(^6)</th>
<th>SCRX001-006 (Basket)(^7)</th>
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</thead>
<tbody>
<tr>
<td>Phase</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>1a/1b</td>
<td>1/2</td>
<td>1</td>
<td>1</td>
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<tr>
<td>Study design</td>
<td>Multicenter, open-label, single arm</td>
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<td>Multicenter, double-blind</td>
<td>Multicenter, open-label</td>
<td>Multicenter, open-label</td>
<td>Multicenter, open-label, single arm</td>
<td>Multicenter, open-label</td>
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<td>N</td>
<td>154</td>
<td>411</td>
<td>740</td>
<td>94</td>
<td>90</td>
<td>50</td>
<td>378</td>
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<tr>
<td>DLL3 expression</td>
<td>Positive</td>
<td>High</td>
<td>All comers</td>
<td>High</td>
<td>NA</td>
<td>All comers</td>
<td>Positive</td>
</tr>
<tr>
<td>Rova-T dose schedule</td>
<td>0.3 mg/kg IV q6w x 2</td>
<td>0.3 mg/kg IV, q6 wk; omit every 3 cycles</td>
<td>0.1 or 0.3 mg/kg IV q6w x 2(^b)</td>
<td>0.3 mg/kg IV</td>
<td>0.3 mg/kg IV, q6 wk; omit every 3 cycles</td>
<td>0.3 mg/kg IV</td>
<td>0.2-0.4 mg/kg IV q6w</td>
</tr>
<tr>
<td>Primary endpoint</td>
<td>ORR and OS</td>
<td>ORR and OS</td>
<td>PFS and OS</td>
<td>PFS (Ph1b)</td>
<td>DLTs</td>
<td>Effect on QTcF interval</td>
<td>MTD and AEs</td>
</tr>
</tbody>
</table>

\(^a\) Cisplatin 80 mg/m\(^2\) day 1 of each cycle plus etoposide 100 mg/m\(^2\) on days 1-3 of each cycle. \(^b\) Cohort with concurrent CE will receive 0.1 mg/kg; all other cohorts will receive 0.3 mg/kg.
# Rova-T Clinical Development Program in SCLC

## 1L Therapy
- **SCRX001-004**  
  **Phase 1:** Rova-T ± cisplatin & etoposide vs cisplatin & etoposide in 1L DLL3+ SCLC

## Maintenance Therapy
- **MERU**  
  **Phase 3:** Rova-T versus placebo as maintenance following platinum-based therapy

## 2L Therapy
- **SCRX16-001**  
  **Phase 1/2:** Rova-T monotherapy in recurrent SCLC

## 3L+ Therapy
- **SCRX16-001**  
  **Phase 1/2:** Rova-T monotherapy in recurrent SCLC

## SCRX16-001  
**Phase 1/2:** Rova-T monotherapy in recurrent SCLC

## M16-300  
**Phase 1/2:** Rova-T + nivolumab ± ipilimumab in 2L+ SCLC

## TAHOE  
**Phase 3:** Rova-T versus topotecan in 2L DLL3 high SCLC

## TRINITY  
**Phase 2:** Rova-T monotherapy in 3L+ DLL3-positive R/R SCLC
Prevalence of DLL3 in SCLC

- Retrospective analysis (2012-2016) of 63 SCLC Japanese patients.
- Tissue availability: 49 biopsies and 14 surgical resections.
- Results:
  - 83% (52/63) DLL3 +
  - 32% (20/63) DLL3 high
- No significant association with age, smoking status, sex, disease stage or biopsy site.
- No significant differences in OS: DLL3 high 12.5 m and DLL3 low 15.7 months

Tanaka K. Lung Cancer 2018
Comprehensive genomic profiles

George et al. Nature 2015
Targeting MYC

- Genomic amplification in MYC have been identified in 6-25% of primary tumours and 30-50% of SCLC cell lines.
- Despite numerous efforts, MYC remains difficult to target with small molecules.
- MYC promotes a NE-low phenotype associate with high expression of NEUROD1.
- MYC driven SCLC may be less responsive to NE targeted agents.
- It can be targeted by Aurora Kinase A inhibitors

In the context of TP53/RB1 deletion, MYC:
• Accelerates oncogenesis and promotes metastasis
• Drives shift to NEUROD1-high, neuroendocrine-low subtype of SCLC
• Correlates with sensitivity to aurora kinase inhibition

Mollaoglu et al. Cancer Cell 2017
Alisertib doses and schema

- Alisertib is an investigational, orally-available, selective small-molecule AAK-inhibitor.
- Different schema have been tested
  - Phase I monotherapy
    - Recommended dose: 50 mg BID 7 days on/14 off
  - Alisertib in combination with CT (phase I and II)
    - Alisertib 20 mg BID 7/14 days + Docetaxel 75 mg/m2
    - Alisertib 60 mg 7/14 days + Irinotecan 50 mg/m2 + temozolomide100 mg d1-5 (pediatric population, neuroblastoma)
    - Alisertib 40 mg BID 3/4 days + Paclitaxel wk 60 mg/m2 (ph II ovarian cancer)
- Efficacy of single-agent alisertib demonstrated in a Phase 2 study of 249 patients, including 48 relapsed SCLC (ORR=21% (all PRs))

RANDOMIZED PHASE 2 STUDY OF
ALISERTIB + PACLITAXEL VERSUS PLACEBO + PACLITAXEL

Patients
- Age ≥18 years with histologically/cytologically confirmed SCLC
- Previously treated with one prior platinum-based chemotherapy regimen
- Relapse <180 days after last platinum dose (Amend 2)
- ECOG PS 0/1
- Measurable disease (RECIST v1.1)

Arm A – Alisertib + Paclitaxel
- Alisertib 40 mg BID PO, 3 days on/4 days off
- Paclitaxel 60 mg/m² IV, days 1, 8, 15

Arm B – Placebo + Paclitaxel
- Matched placebo BID PO, 3 days on/4 days off
- Paclitaxel 80 mg/m² IV, days 1, 8, 15

Primary endpoint: PFS
Key exploratory endpoint: Biomarkers including C-Myc

Owonikoko T. ESMO 2016
RANDOMIZED PHASE 2 STUDY OF ALISERTIB + PACLITAXEL VERSUS PLACEBO + PACLITAXEL

PRIMARY ENDPOINT: PFS (ITT POPULATION)

Median PFS: 101 days (3.32 months) vs 66 days (2.17 months)

PFS IN PATIENTS WITH RESISTANT/REFRACTORY RELAPSE (CORRECTED)

Median PFS: 87 days (2.86 months) vs 50 days (1.64 months)

Survival Probability vs Survival Time (days)
PFS IMPROVEMENT IN PATIENTS WITH C-MYC EXPRESSION*

*Archived tumor tissue available from 46 patients. Modal intensity for c-Myc positive = 1+, 2+, 3+ IHC score. Modal intensity for c-Myc negative = 0 IHC score.

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<th>Median PFS (months)</th>
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<td>Alisertib + Paclitaxel</td>
<td>17</td>
<td>4.64</td>
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<tr>
<td>Placebo + Paclitaxel</td>
<td>16</td>
<td>2.27</td>
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<tr>
<td>Hazard Ratio (95% CI)</td>
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<td>0.29 (0.12–0.72)</td>
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<td>3.32</td>
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<td>7</td>
<td>5.16</td>
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<td>Hazard Ratio (95% CI)</td>
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<td>11.8 (1.52–91.2)</td>
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DO CURRENT RESULTS WITH ALISERTIB JUSTIFY NEW STUDIES?

- The global results of Alisertib + wk paclitaxel in second-line patients are modest, both ITT and R/R population.
- Difficult interpretation of results (small samples size, post-hoc analysis)
- Results in c-Myc subpopulation are encouraging but need to be validated in a specific clinical trial where c-Myc status were assessed in all patients.
- Even positive results in molecular-driven population should be balanced with toxicity.
  - Drug-related Grade ≥3 AE 67% vs. 25%
  - Dose reduction due to AE 38% vs. 10%.
  - On study deaths: 15 patients (4 drug related deaths) vs. 12 patients

Garrido P. ESMO 2016
SCLC: Cell surface and intracellular targets

Sabari J, et al. Nat Reviews 2017
PARP1 is a protein that is mainly involved in DNA repair.

Cancer cells accumulate DNA lesions which cannot be repaired.

SCLC showed the highest expression of PARP among all histologic subtypes of lung cancer, suggesting a biologically relevant role for this protein in this disease.

PARP inhibitors may enhance cytotoxicity of DNA damaging chemotherapeutic agents and ionizing radiation.

Foy et al. Lung Cancer 2017

PARP 1 inhibitors + chemotherapy

- Modest single agent efficacy based on early phase studies but significantly potentiated cytotoxicity of chemotherapy.
- Combination therapy of a PARP inhibitor with TMZ is synergistic, well-tolerated and effective across multiple SCLC cell lines and PDXs¹.
  - Olaparib + Temozolamide phase I/II study (13 patients)²
    - O 200 mg BID and T 75 mg/m² QD was selected as the RP2D
    - 6 confirmed PR (ORR 46%). Median PFS 5.6 months and median duration of response 3.4 months.
  - Velaparib or placebo + Temozolamide (100 patients)³
    - RR 39% vs 14% in 93 evaluable patients
    - No difference in PFS (4 vs. 3.6 months) or MOS (8.2 vs. 7 months)
    - Increase G3-4 toxicity: thrombocytopenia 50%, neutropenia 31%
- No benefit in PFS or OS with Olaparib in maintenance (STOMP trial)

¹Lok B. Clin Cancer Res 2017; ²Farago A. AACR 2017; ³Pietanza A. ASCO 16; ⁴Owonikoko T. Lung Cancer 2015; ⁵Woll P. JTO 2017
PARP1 inhibitors in the first line setting

• Phase I Velaparib + cisplatin and etoposide (9 patients)\textsuperscript{1}
  – Recommended dose Veliparib at 100mg BID days 1-7 + standard dose of PE
  – Investigator-assessed efficacy outcome in 7 evaluable patients:
    • SD 2/7 (28.6%), PR 4/7 (57.1%) and CR 1/7 (14.3%) patients.
• Randomized study PE + Velaparib or placebo
  – PO: PFS
  – N: 128 patients
  – \textit{No significant differences in activity but increased toxicity}
    • Median PFS: 6.1 vs 5.5 months
    • MOS: 10.3 vs. 8.9
    • RR 71.9 vs 65.6

\textsuperscript{1}Owonikoko T. Lung Cancer 2015
\textsuperscript{2}Owonikoko T. ASCO 2017
Biomarkers enrichment strategy will be needed to optimize the benefit of PARP inhibition as a therapeutic strategy.
Biomarkers for PARP inhibitors

- BRCA1/2 and ATM mutations are response predictors associated with PARP inhibitor activity in breast, ovarian, and prostate cancers. No such predictive biomarker has been defined for SCLC.

- *Schlafen family member 11* (SLFN11), a DNA damage repair protein, is aberrantly expressed in SCLC.

- **SLFN11** has been reported as a critical determinant of PARP inhibitor sensitivity in SCLC.

- SLFN11 expression by IHC is associated with tumor response to talazoparib in multiple PDX models.

Immunotherapy in SCLC

Sabari J, et al. Nat Reviews 2017
Rationale for immunotherapy

- Occurrence of paraneoplastic disorders
  - Result from an immune response directed against specific antigens expressed on both SCLC and normal nerves cells
- Immune activity and prognosis
  - More CD45^T T cells infiltrating SCLC tumours predictive of better OS
  - More effective T cells in LD-SCLC as compared with ED-SCLC
- High mutational burden
  - High non-synonymous mutation rate 5.5-7.4/Mb (ADC 8.9, SqCC 8.1)
Few studies have been completed

- **Pembrolizumab**
  - Tested in PDL1 + patients
  - Results in two settings:
    - Pretreated patients: Multicohort Phase Ib (KN028)
    - Maintenance (phase II trial)
  - Ongoing ph. III in first line (KN604)

- **Nivolumab + Ipilimumab**
  - Tested regardless PDL1-expression status
  - Results in pretreated patients (CM 032): Nivo ó Nivo + Ipi
  - Ongoing ph3 in maintenance (CK451) and second line (CK331)
KN 028: Ph IB Multicohort in PDL1 +

- PDL1 screening (patients)
  - Tested 163
  - Positive 46
  - Treated 24
- **Progressive Disease 54.2%**
- RR 33%, **Median DOR 19.4 m**
- Median PFS 1.9 m
- Median OS 9.7 m
- 1-y SV 37.7%

*Response assessment: Every 8 weeks for the first 6 months; every 12 weeks thereafter
Primary end points: ORR per RECIST v1.1 (investigator assessed) and safety
Secondary end points: PFS, OS, duration of response
Phase II study of pembrolizumab as maintenance

- ED SCLC with CR, PR or SD after 4-6 cycles of standard first line CT
- Primary endpoint PFS
- N: 45. No PDL1 selection
- Efficacy:
  - Median PFS 1.4
  - Median PFS in PDL1 + (3 p/30 assessed): 4.8-9.8 m
  - Median OS 9.4; 1-y OS 30%
- **Pembrolizumab did not improve median PFS in this study**
Nivolumab/Ipilimumab in SCLC

• CK 032 ph 1/2 trial\(^1,2\):
  – Nivo (N 98): ORR 11%; Median DOR 17.9 m; MST: 4.1 m; 2-y OS 14%
  – Nivo + IPI (61): ORR 23%, Median DOR 14.2 m; MST 7.8 m; 2-y OS 26%

• CK 032 ph 1/2 trial randomized cohort\(^2\)
  – A randomized cohort was added to further evaluate Nivolumab + Ipilimumab in pretreated SCLC patients.
    – Nivo (N: 147): ORR 12%;
    – Nivo + IPI (N: 95): ORR 21%;

• Responses regardless platinum sensitivity, line of therapy or PD-L1 status
• Combo increases toxicity: 37% Gr 3-4 vs. 12%; 5 treatment related deaths (4:1)

\(^1\) Antonia S. Lancet Oncology 2016; Hellman M, WCLC 2017
Ongoing Phase 3 Studies With Nivolumab ± Ipilimumab in SCLC

**CheckMate 451: study design**

- **Currently enrolling patients**
  - Key eligibility criteria:
    - ED-SCLC
    - Ongoing SD/PR/CR after 4 cycles of 1L PLT-CT
    - No symptomatic CNS metastases
    - Toxicities from prior therapy resolved to grade ≤1
    - ECOG PS ≤1
  - Randomize 1:1:1

- **Primary outcome measures:**
  - OS, PFS

- **Secondary outcome measures:**
  - OS and PFS descriptive analyses: nivolumab vs nivolumab + ipilimumab

**CheckMate 331: study design**

- Key eligibility criteria:
  - SCLC
  - Recurrence/PD after 1L PLT-CT or CRT (≥4 cycles)
  - ECOG PS ≤1
  - No symptomatic CNS metastases

- Randomize 1:1

- **Primary outcome measures:**
  - OS

- **Secondary outcome measures:**
  - PFS, ORR

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1L = first-line; CT = chemotherapy; CRT = chemoradiation therapy; CTLA-4 = cytotoxic T lymphocyte antigen-4; PD-1 = programmed-death 1; PD-L2 = PD ligand 2
PLT = platinum-based; *Where locally approved

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Presented By Matthew Hellmann at 2017 ASCO Annual Meeting
STIMULI: A randomized Phase 2 trial of consolidation with nivolumab and ipilimumab in limited-stage SCLC after chemoradiotherapy

**Key Inclusion Criteria**
- ≥18 years of age
- Untreated LS-SCLC
- ECOG PS 0–1
- Non PD after completion of CRT, PCI
- Adequate hematological, renal, hepatic and lung function
- Recovery of all AEs to Grade ≤1*

**Induction:**
- Ipilimumab 3 mg/kg + Nivolumab 1 mg/kg Q3W X4

**Maintenance:**
- Nivolumab 240 mg Q2w

**Observation**

**Primary outcome measure:** OS, PFS
N: 260
Predictive biomarkers in SCLC

• **PD L1 > 1%**: Frequency lower than in NSCLC\(^1\)
  - KN 028: 31.6%; Median PFS in PDL1 + (3 p /30 assessed): 4.8-9.8 m
  - CM 032: PDL1 >1% and <5%: 8-16%.; No differences in results

• **PDL1 expression at the stromal interface\(^2\)**
  - Pembrolizumab (20 tumours)
  - Stromal + (8 patients): PFS 5.5 m and OS 10.1 m vs. stromal negative (12 patients): PFS 1.3 m and OS 7.2 m

\(^1\) Smit E. JCO 2017. \(^2\) Gadgeel S. ASCO 2017
Predictive biomarkers

Checkmate 032: PFS and OS by TMB

Nivolumab

<table>
<thead>
<tr>
<th>TMB Level</th>
<th>Median PFS (95% CI), mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>1.3 (1.2, 1.4)</td>
</tr>
<tr>
<td>Medium</td>
<td>1.3 (1.2, 1.4)</td>
</tr>
<tr>
<td>High</td>
<td>1.4 (1.3, 2.7)</td>
</tr>
</tbody>
</table>

1-y PFS = 21.2%
1-y PFS = NC
1-y PFS = 3.1%

Nivolumab + ipilimumab

<table>
<thead>
<tr>
<th>TMB Level</th>
<th>Median OS (95% CI), mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>3.1 (2.4, 6.8)</td>
</tr>
<tr>
<td>Medium</td>
<td>3.9 (2.4, 9.9)</td>
</tr>
<tr>
<td>High</td>
<td>5.4 (2.8, 8.0)</td>
</tr>
</tbody>
</table>

1-y OS = 35.2%
1-y OS = 26.0%
1-y OS = 22.1%

Take home messages

- This is a time of extraordinary change and opportunity in cancer care.
- Large scale genomic analyses have led to the identification of new druggable targets in SCLC.
- Targeted agents and IO are under active clinical investigation.
- Predictive biomarkers are crucial to identify the key patients to be treated.
Thanks!

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