Primary PFS and safety analyses of a randomised Phase III study of carboplatin + paclitaxel +/- bevacizumab, with or without atezolizumab in 1L non-squamous metastatic NSCLC (IMpower150)

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Disclosures

- Dr Martin Reck has the following to disclose:
  - Consulting or advisory role for AstraZeneca, Boehringer Ingelheim, Bristol-Myers Squibb, Celgene, Eli Lilly, F. Hoffmann-La Roche, MSD, Novartis and Pfizer
  - Speakers’ bureau for AstraZeneca, Boehringer Ingelheim, Bristol-Myers Squibb, Celgene, Eli Lilly, F. Hoffmann-La Roche, MSD, Novartis and Pfizer
- This study is sponsored by F. Hoffmann-La Roche, Ltd
Background: NSCLC landscape

- Standards of care for patients with advanced 1L NSCLC include\textsuperscript{1,2}:
  - Targeted therapies (patients with \textit{EGFR} mutation or \textit{ALK} rearrangement)
  - Pembrolizumab (anti–PD-1) in patients with PD-L1 expressing tumours with TPS ≥ 50% (≈ 25%-30% prevalence)
  - Platinum-based chemotherapy +/- bevacizumab\textsuperscript{3}

- Atezolizumab (anti–PD-L1) has demonstrated overall survival benefit\textsuperscript{4} and is approved in the US\textsuperscript{5} and EU\textsuperscript{6} for the treatment of 2L+ NSCLC regardless of PD-L1 expression

- Phase Ib data of atezolizumab + platinum-doublet chemotherapy in patients with 1L NSCLC demonstrated promising efficacy and tolerable safety\textsuperscript{7}

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Rationale for combining atezolizumab + bevacizumab

- In addition to its known anti-angiogenic effects\(^1\), bevacizumab’s inhibition of VEGF has immune modulatory effects

\begin{itemize}
  \item Atezolizumab’s T-cell mediated cancer cell killing may be enhanced through bevacizumab’s reversal of VEGF-mediated immunosuppression
\end{itemize}


Figure adapted from Chen DS, Mellman I. *Immunity*, 2013.
**IMpower150 study design**

- **Stage IV or recurrent metastatic non-squamous NSCLC Chemotherapy-naive**
- **Tumour tissue available for biomarker testing**
- **Any PD-L1 IHC status**

**Stratification factors:**
- **Sex**
- **PD-L1 IHC expression**
- **Liver metastases**

N = 1202

**Arm A**
- Atezolizumab + Carboplatin + Paclitaxel
- 4 or 6 cycles

**Arm B**
- Atezolizumab + Carboplatin + Paclitaxel + Bevacizumab
- 4 or 6 cycles

**Arm C (control)**
- Carboplatin + Paclitaxel + Bevacizumab
- 4 or 6 cycles

**Maintenance therapy (no crossover permitted)**

- Treated with atezolizumab until PD by RECIST v1.1 or loss of clinical benefit
- AND/OR

- Treated with bevacizumab until PD by RECIST v1.1

The principal question is to assess whether the addition of atezolizumab to Arm C provides clinical benefit

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*a* Patients with a sensitising *EGFR* mutation or *ALK* translocation must have disease progression or intolerance of treatment with one or more approved targeted therapies.

*b* Atezolizumab: 1200 mg IV q3w.

*c* Carboplatin: AUC 6 IV q3w.

*d* Paclitaxel: 200 mg/m² IV q3w.

*e* Bevacizumab: 15 mg/kg IV q3w.

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Reck M, et al. IMpower150 PFS analysis.
IMpower150 study populations and objectives

1. Co-primary objectives
   - Investigator-assessed PFS in ITT-WT

- **ITT**
  - All randomised patients

- **ITT-WT**
  - (87% of patients)
  - WT refers to patients without EGFR or ALK genetic alterations.

- **EGFR/ALK +**
  - (13% of patients)
The T-effector (Teff) gene signature is defined by expression of PD-L1, CXCL9 and IFNγ and is a surrogate of both PD-L1 IHC expression and pre-existing immunity (Kowanetz M, et al. WCLC, 2017).
IMpower150 study populations and objectives

**Co-primary objectives**
- Investigator-assessed PFS in ITT-WT
- Investigator-assessed PFS in Teff-high WT
- OS in ITT-WT

The T-effector (Teff) gene signature is defined by expression of PD-L1, CXCL9 and IFNγ and is a surrogate of both PD-L1 IHC expression and pre-existing immunity (Kowanetz M, et al. WCLC, 2017).
IMpower150 study populations and objectives

**Co-primary objectives**
- Investigator-assessed PFS in ITT-WT
- Investigator-assessed PFS in Teff-high WT
- OS in ITT-WT

**Key secondary objectives**
- Investigator-assessed PFS and OS in ITT
- Investigator-assessed PFS in PD-L1 IHC subgroups
- Independent review facility (IRF)-assessed PFS
- ORR and DOR per RECIST v1.1
- Safety in ITT

The T-effector (Teff) gene signature is defined by expression of PD-L1, CXCL9, and IFNγ and is a surrogate of both PD-L1 IHC expression and pre-existing immunity (Kowanetz M, et al. WCLC, 2017).
Biomarkers in IMpower150

- IMpower150 provided the opportunity to evaluate multiple strategies to enrich for PFS, including T-effector (Teff) gene signature expression and PD-L1 IHC.

- The Teff gene signature is defined by mRNA expression of 3 genes (PD-L1, CXCL9 and IFNγ) and is a surrogate for both PD-L1 expression and pre-existing immunity.
  - In the OAK study, the Teff gene signature appeared to be a more sensitive biomarker of PFS benefit for monotherapy atezolizumab vs docetaxel than PD-L1 IHC expression.

- PD-L1 expression was evaluated using the SP142 IHC assay, as defined in the Phase III OAK study of atezolizumab vs docetaxel.
Statistical testing plan for the co-primary endpoints in IMpower150

Arm A: atezo + CP
Arm B: atezo + bev + CP
Arm C: bev + CP (control)

November 2017

Arm B vs C
PFS in ITT-WT

Arm B vs C
PFS in Teff-high WT

Arm B vs C
OS in ITT-WT

Arm B vs C
OS in ITT-WT

Arm B vs C
PFS in Teff-high WT

Arm A vs C
PFS in ITT-WT and Teff-high WT

Arm A vs C
OS in ITT-WT

If OS is significant

1H 2018 (interim)

Reck M, et al. IMpower150 PFS analysis.
Statistical testing plan for the co-primary endpoints in IMpower150

Arm A: atezo + CP
Arm B: atezo + bev + CP
Arm C: bev + CP (control)

Arm B vs C
PFS in ITT-WT

Arm B vs C
PFS in Teff-high WT

Arm B vs C
OS in ITT-WT

Arm A vs C
PFS in ITT-WT and Teff-high WT

Arm A vs C
OS in ITT-WT

If OS is significant

November 2017

Arm B vs C
OS in ITT-WT

1H 2018 (interim)
## Baseline characteristics in ITT

<table>
<thead>
<tr>
<th>Baseline characteristics</th>
<th>Arm A: atezo + CP (N = 402)</th>
<th>Arm B: atezo + bev + CP (N = 400)</th>
<th>Arm C (control): bev + CP (N = 400)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age (range), years</td>
<td>63 (32-85)</td>
<td>63 (31-89)</td>
<td>63 (31-90)</td>
</tr>
<tr>
<td>Sex, male, n (%)</td>
<td>241 (60%)</td>
<td>240 (60%)</td>
<td>239 (60%)</td>
</tr>
<tr>
<td>ECOG PS, 0, n (%)</td>
<td>180 (45%)</td>
<td>159 (40%)</td>
<td>179 (45%)</td>
</tr>
<tr>
<td>Tobacco use history, n (%)</td>
<td>98 (24%)</td>
<td>227 (57%)</td>
<td>77 (19%)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>Previous smoker Never smoker</td>
<td>53 (13%)</td>
<td>53 (13%)</td>
</tr>
<tr>
<td>Liver metastases, yes, n (%)</td>
<td>53 (13%)</td>
<td>53 (13%)</td>
<td>57 (14%)</td>
</tr>
<tr>
<td>EGFR mutation, positive, n (%)</td>
<td>46 (11%)</td>
<td>35 (9%)</td>
<td>45 (11%)</td>
</tr>
<tr>
<td>ALK rearrangement, positive, n (%)</td>
<td>9 (2%)</td>
<td>13 (3%)</td>
<td>21 (5%)</td>
</tr>
<tr>
<td>Teff gene signature expression, high, n (%)</td>
<td>177 (44%)</td>
<td>166 (42%)</td>
<td>148 (37%)</td>
</tr>
<tr>
<td>Of those tested</td>
<td>36 (29%)</td>
<td>47 (44%)</td>
<td>38 (33%)</td>
</tr>
<tr>
<td>KRAS mutation, positive, n (%)</td>
<td>137 (34%)</td>
<td>140 (35%)</td>
<td>133 (33%)</td>
</tr>
<tr>
<td>PD-L1 expression, n (%)</td>
<td>TC2/3 or IC2/3</td>
<td>188 (47%)</td>
<td>195 (49%)</td>
</tr>
<tr>
<td>TC1/2/3 or IC1/2/3</td>
<td>213 (53%)</td>
<td>209 (52%)</td>
<td>205 (51%)</td>
</tr>
<tr>
<td>TC0 and IC0</td>
<td>267 (67%)</td>
<td>229 (57%)</td>
<td>229 (57%)</td>
</tr>
</tbody>
</table>

IC, tumour-infiltrating immune cells; TC, tumour cells.

- The Teff gene signature high cut-off ≥ -1.91 was used.
- 1 patient in Arm A had unknown PD-L1 IHC expression.

TC2/3 or IC2/3 = TC or IC ≥ 5% PD-L1+; TC1/2/3 or IC1/2/3 = TC or IC ≥ 1% PD-L1+; TC0 and IC0 = TC and IC < 1% PD-L1+.

Data cutoff: September 15, 2017

Reck M, et al. IMpower150 PFS analysis.
INV-assessed PFS in ITT-WT (Arm B vs Arm C)

Arm B: atezo + bev + CP
Arm C: bev + CP

HR, 0.617 (95% CI: 0.517, 0.737)  
$P < 0.0001$

Minimum follow-up: 9.5 mo  
Median follow-up: ~15 mo

No. at Risk
Atezo + Bev + CP 356 332 311 298 290 265 232 210 186 151 124 111 87 77 58 55 42 39 27 24 16 12 4 3 2 2 2
Bev + CP 336 321 292 261 243 215 179 147 125 91 69 55 32 21 18 12 9 7 6 3 2 1 1
Reck M, et al. IMpower150 PFS analysis.

INV-assessed PFS in ITT-WT (Arm B vs Arm C)

Arm B: atezo + bev + CP
Arm C: bev + CP

Minimum follow-up: 9.5 mo
Median follow-up: ~15 mo

INV, investigator.
Data cutoff: September 15, 2017

Reck M, et al. IMpower150 PFS analysis.
INV-assessed PFS in Teff-high WT (Arm B vs Arm C)

Landmark PFS, %

<table>
<thead>
<tr>
<th>Arm B: atezo + bev + CP</th>
<th>Arm C: bev + CP</th>
</tr>
</thead>
<tbody>
<tr>
<td>6-month</td>
<td>72%</td>
</tr>
<tr>
<td>12-month</td>
<td>46%</td>
</tr>
</tbody>
</table>

HR, 0.505 (95% CI: 0.377, 0.675)

$P < 0.0001$

Minimum follow-up: 9.5 mo

![Graph showing progression-free survival](Image)
PFS in subgroups of interest in ITT-WT

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>n (%)</th>
<th>HR&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Arm B</th>
<th>Arm C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>425 (61%)</td>
<td>0.55</td>
<td>8.4</td>
<td>6.8</td>
</tr>
<tr>
<td>Female</td>
<td>267 (39%)</td>
<td>0.73</td>
<td>8.2</td>
<td>6.8</td>
</tr>
<tr>
<td>&lt; 65 years</td>
<td>375 (54%)</td>
<td>0.65</td>
<td>8.0</td>
<td>6.8</td>
</tr>
<tr>
<td>65-74 years</td>
<td>248 (36%)</td>
<td>0.52</td>
<td>9.7</td>
<td>6.9</td>
</tr>
<tr>
<td>75-84 years</td>
<td>64 (9%)</td>
<td>0.78</td>
<td>9.7</td>
<td>6.8</td>
</tr>
<tr>
<td>ECOG PS 0</td>
<td>282 (41%)</td>
<td>0.55</td>
<td>11.1</td>
<td>8.0</td>
</tr>
<tr>
<td>ECOG PS 1</td>
<td>404 (58%)</td>
<td>0.64</td>
<td>7.2</td>
<td>6.0</td>
</tr>
<tr>
<td>Current/previous smoker</td>
<td>584 (84%)</td>
<td>0.58</td>
<td>8.3</td>
<td>6.8</td>
</tr>
<tr>
<td>Never smoker</td>
<td>108 (16%)</td>
<td>0.80</td>
<td>8.3</td>
<td>8.3</td>
</tr>
<tr>
<td>Liver metastases</td>
<td>94 (14%)</td>
<td>0.42</td>
<td>7.4</td>
<td>4.9</td>
</tr>
<tr>
<td>No liver metastases</td>
<td>598 (86%)</td>
<td>0.63</td>
<td>8.3</td>
<td>7.0</td>
</tr>
<tr>
<td>KRAS mutant</td>
<td>80 (12%)</td>
<td>0.50</td>
<td>8.1</td>
<td>5.8</td>
</tr>
<tr>
<td>KRAS wild type</td>
<td>124 (18%)</td>
<td>0.47</td>
<td>9.7</td>
<td>5.8</td>
</tr>
<tr>
<td>KRAS unknown</td>
<td>488 (71%)</td>
<td>0.67</td>
<td>8.3</td>
<td>7.1</td>
</tr>
</tbody>
</table>

| ITT-WT                            | 692 (100%)    | 0.62           | 8.3   | 6.8   |

<sup>a</sup> Stratified HRs for ITT-WT; unstratified HRs for all other subgroups.

Data cutoff: September 15, 2017
# PFS in key biomarker populations

<table>
<thead>
<tr>
<th>Population</th>
<th>n (%)^a</th>
<th>Arm B (mo)</th>
<th>Arm C (mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ITT (including EGFR/ALK mutant +)</td>
<td>800 (100%)</td>
<td>8.3</td>
<td>6.8</td>
</tr>
<tr>
<td><strong>EGFR/ALK mutant + only^b</strong></td>
<td>108 (14%)</td>
<td>9.7</td>
<td>6.1</td>
</tr>
<tr>
<td>ITT-WT</td>
<td>692 (87%)</td>
<td>8.3</td>
<td>6.8</td>
</tr>
<tr>
<td>Teff-high (WT)</td>
<td>284 (43%)</td>
<td>11.3</td>
<td>6.8</td>
</tr>
<tr>
<td>Teff-low (WT)</td>
<td>374 (57%)</td>
<td>7.3</td>
<td>7.0</td>
</tr>
<tr>
<td>PD-L1 IHC TC2/3 or IC2/3 (WT)</td>
<td>244 (35%)</td>
<td>11.1</td>
<td>6.8</td>
</tr>
<tr>
<td>PD-L1 IHC TC1/2/3 or IC1/2/3 (WT)</td>
<td>354 (51%)</td>
<td>11.0</td>
<td>6.8</td>
</tr>
<tr>
<td>PD-L1 IHC TC0 and IC0 (WT)</td>
<td>338 (49%)</td>
<td>7.1</td>
<td>6.9</td>
</tr>
<tr>
<td>PD-L1 IHC TC3 or IC3 (WT)</td>
<td>135 (20%)</td>
<td>12.6</td>
<td>6.8</td>
</tr>
<tr>
<td>PD-L1 IHC TC0/1/2 or IC0/1/2 (WT)</td>
<td>557 (80%)</td>
<td>8.0</td>
<td>6.8</td>
</tr>
</tbody>
</table>

^a ITT, EGFR/ALK mutants, and ITT-WT % prevalence out of ITT (n = 800); Teff % prevalence out those tested in ITT-WT (n = 658); PD-L1 IHC % prevalence out of ITT-WT (n = 692).

^b Patients with a sensitising EGFR mutation or ALK translocation must have disease progression or intolerance of treatment with one or more approved targeted therapies.

^c Stratified HRs for ITT, ITT-WT and Teff-high WT populations; unstratified HRs for all other subgroups.

Data cutoff: September 15, 2017
ORR\textsuperscript{a} and DOR in ITT-WT and Teff-high WT

**ITT-WT**

- **Arm B:** atezo + bev + CP
  - CR/PR: 4%/60%
  - ORR: 64%
  - Median DOR (range), mo: 9.0 (0.4-24.9\textsuperscript{b})

- **Arm C:** bev + CP
  - CR/PR: 1%/47%
  - ORR: 48%
  - Median DOR (range), mo: 5.7 (0.0\textsuperscript{b}-22.1)

**Teff-high WT**

- **Arm B:** atezo + bev + CP
  - CR/PR: 4%/65%
  - ORR: 69%
  - Median DOR (range), mo: 11.2 (0.5-24.9\textsuperscript{b})

- **Arm C:** bev + CP
  - CR/PR: 2%/51%
  - ORR: 54%
  - Median DOR (range), mo: 5.7 (0.0\textsuperscript{b}-22.1)

\textsuperscript{a} Investigator-assessed ORR.
\textsuperscript{b} Censored value.

Data cutoff: September 15, 2017

Reck M, et al. IMpower150 PFS analysis.
Preliminary OS in ITT-WT (Arm B vs Arm C)

- Promising preliminary OS benefit for Arm B vs Arm C was observed; next OS interim data are anticipated in 1H 2018
Preliminary efficacy in ITT-WT (Arm A vs Arm C)

| ITT-WT |
|------------------|------------------|
| Arm A:           | Arm C (control): |
| atezo + CP       | bev + CP         |
| (n = 348)        | (n = 336)        |

<table>
<thead>
<tr>
<th>PFS HR(^a) (95% CI)</th>
<th>0.936 (0.787, 1.112)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ORR, (^b) n (%)</td>
<td>171 (49%)</td>
</tr>
</tbody>
</table>

- Formal statistical testing for Arm A vs Arm C will be conducted only after the OS boundary for Arm B vs Arm C is crossed

\(^a\) Stratified HR.
\(^b\) \(n = 347\) (Arm A) and \(n = 331\) (Arm C).

Data cutoff: September 15, 2017
**Preliminary efficacy in ITT-WT (Arm A vs Arm C)**

<table>
<thead>
<tr>
<th></th>
<th>ITT-WT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Arm A: atezo + CP (n = 348)</td>
</tr>
</tbody>
</table>
| **PFS HR**  
  (95% CI)  | 0.936 (0.787, 1.112)                        |
| **ORR, n (%)** | 171 (49%)                                  | 159 (48%)                           |
| **OS HR**  
  (95% CI)  | 0.884 (0.709, 1.101)                       |

- Formal statistical testing for Arm A vs Arm C will be conducted only after the OS boundary for Arm B vs Arm C is crossed

---

a Stratified HR.

b n = 347 (Arm A) and n = 331 (Arm C).

Data cutoff: September 15, 2017
## Safety summary

<table>
<thead>
<tr>
<th></th>
<th>Arm A: atezo + CP (n = 400)</th>
<th>Arm B: atezo + bev + CP (n = 393)</th>
<th>Arm C (control): bev + CP (n = 394)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Median doses received (range), n</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atezolizumab</td>
<td>10 (1-37)</td>
<td>12 (1-38)</td>
<td>NA</td>
</tr>
<tr>
<td>Bevacizumab</td>
<td>NA</td>
<td>10 (1-38)</td>
<td>8 (1-33)</td>
</tr>
<tr>
<td><strong>All cause AE, n (%)</strong></td>
<td>389 (97%)</td>
<td>385 (98%)</td>
<td>390 (99%)</td>
</tr>
<tr>
<td>Grade 3-4</td>
<td>226 (57%)</td>
<td>242 (62%)</td>
<td>230 (58%)</td>
</tr>
<tr>
<td>Grade 5</td>
<td>10 (3%)</td>
<td>23 (6%)</td>
<td>21 (5%)</td>
</tr>
<tr>
<td><strong>Treatment-related AE, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 3-4</td>
<td>372 (93%)</td>
<td>371 (94%)</td>
<td>376 (95%)</td>
</tr>
<tr>
<td>Grade 5a</td>
<td>170 (43%)</td>
<td>219 (56%)</td>
<td>188 (48%)</td>
</tr>
<tr>
<td><strong>Serious AE, n (%)</strong></td>
<td>155 (39%)</td>
<td>165 (42%)</td>
<td>134 (34%)</td>
</tr>
<tr>
<td>Treatment-related serious AE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>77 (19%)</td>
<td>100 (25%)</td>
<td>76 (19%)</td>
<td></td>
</tr>
<tr>
<td><strong>AEs of special interest, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 3-4</td>
<td>184 (46%)</td>
<td>199 (51%)</td>
<td>108 (27%)</td>
</tr>
<tr>
<td>Grade 5</td>
<td>37 (9%)</td>
<td>45 (11%)</td>
<td>13 (3%)</td>
</tr>
<tr>
<td><strong>AE leading to withdrawal from any treatment</strong></td>
<td>56 (14%)</td>
<td>128 (33%)</td>
<td>98 (25%)</td>
</tr>
<tr>
<td><strong>AE leading to dose interruption or modification</strong></td>
<td>203 (51%)</td>
<td>235 (60%)</td>
<td>189 (48%)</td>
</tr>
</tbody>
</table>

a Including fatal haemorrhagic AEs: Arm C: haemoptysis n = 1, pulmonary haemorrhage n = 2; Arm B haemoptysis n = 3, pulmonary haemorrhage n = 2, haemorrhage intracranial n = 1; Arm A: haemoptysis n = 1, haemorrhage intracranial n = 1.

b Investigator text for AEs encoded using MedDRA v20.1.

Data cutoff: September 15, 2017
<table>
<thead>
<tr>
<th>AEs of special interest, n (%)</th>
<th>Arm A: atezo + CP (n = 400)</th>
<th></th>
<th>Arm B: atezo + bev + CP (n = 393)</th>
<th></th>
<th>Arm C (control): bev + CP (n = 394)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All grade</td>
<td>Grade 3-4</td>
<td>All grade</td>
<td>Grade 3-4</td>
<td>All grade</td>
<td>Grade 3-4</td>
</tr>
<tr>
<td>Rash</td>
<td>114 (29%)</td>
<td>14 (4%)</td>
<td>113 (29%)</td>
<td>9 (2%)</td>
<td>52 (13%)</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>Hepatitis Laboratory abnormalities</td>
<td>39 (10%)</td>
<td>12 (3%)</td>
<td>54 (14%)</td>
<td>19 (5%)</td>
<td>29 (7%)</td>
<td>3 (1%)</td>
</tr>
<tr>
<td></td>
<td>34 (9%)</td>
<td>10 (3%)</td>
<td>47 (12%)</td>
<td>16 (4%)</td>
<td>29 (7%)</td>
<td>3 (1%)</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>30 (8%)</td>
<td>1 (&lt;1%)</td>
<td>50 (13%)</td>
<td>1 (&lt;1%)</td>
<td>15 (4%)</td>
<td>0</td>
</tr>
<tr>
<td>Infusion-related reactions</td>
<td>16 (4%)</td>
<td>3 (1%)</td>
<td>13 (3%)</td>
<td>2 (1%)</td>
<td>11 (3%)</td>
<td>3 (1%)</td>
</tr>
<tr>
<td>Pneumonitis</td>
<td>21 (5%)</td>
<td>7 (2%)</td>
<td>11 (3%)</td>
<td>6 (2%)</td>
<td>5 (1%)</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td>11 (3%)</td>
<td>0</td>
<td>16 (4%)</td>
<td>1 (&lt;1%)</td>
<td>5 (1%)</td>
<td>0</td>
</tr>
<tr>
<td>Colitis</td>
<td>3 (1%)</td>
<td>2 (1%)</td>
<td>9 (2%)</td>
<td>5 (1%)</td>
<td>2 (1%)</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>Severe cutaneous reaction</td>
<td>3 (1%)</td>
<td>3 (1%)</td>
<td>4 (1%)</td>
<td>0</td>
<td>1 (&lt;1%)</td>
<td>0</td>
</tr>
<tr>
<td>Adrenal insufficiency</td>
<td>2 (1%)</td>
<td>0</td>
<td>2 (1%)</td>
<td>1 (&lt;1%)</td>
<td>3 (1%)</td>
<td>1 (&lt;1%)</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>2 (1%)</td>
<td>2 (1%)</td>
<td>5 (1%)</td>
<td>2 (1%)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Data cutoff: September 15, 2017

Immune-related AEs of special interest in ≥ 5 patients across arms

Reck M, et al. IMpower150 PFS analysis.
IMpower150 is the first phase III immunotherapy-based combination study to demonstrate a statistically significant and clinically meaningful improvement in PFS in all-comer 1L NSQ mNSCLC, providing a potential new standard of care for patients.

PFS benefit was demonstrated with the addition of atezolizumab to bevacizumab + CP (Arm B) vs bevacizumab + CP (Arm C) in all populations tested, including patients with sensitising EGFR or ALK genetic alterations, Teff-low tumours, PD-L1–negative tumours and liver metastases.

Atezolizumab in combination with chemotherapy ± bevacizumab appears to be well tolerated and its safety profile is consistent with known safety risks.

OS data, while not mature, are promising in Arm B vs Arm C; next interim analysis for all arms is anticipated in 1H 2018.
Acknowledgements

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- Participating study investigators and clinical sites
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