ESMO PRECEPTORSHIP ON NON-SMALL CELL LUNG CANCER

New Treatment Options for EGFR+ NSCLC

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Advisor to company:
AstraZeneca, Novartis, Chugai, Boehringer-Ingelheim, Pfizer, Roche, Synta, Clovis, MSD, Thermofisher, Roche diagnostics, Bristol Myers Squibb, Ono pharmaceuticals, Taiho, Takeda

Lecture fees:
AstraZeneca, Chugai, Boehringer-Ingelheim, Pfizer, Taiho, Eli-Lilly, Daiichi-Sankyo, Thermofisher, MSD, Bristol Myers Squibb, Ono pharmaceuticals

Research expenses:
AstraZeneca, Chugai, Boehringer-Ingelheim, Pfizer, Taiho, Ono, Daiichi-Sankyo, Eli-Lilly, Ethicon, Gritstone
Discovery of EGFR mutation

Lynch et al., NEJM, Paez et al., Science, Pao et al., PNAS

- Lung cancer specific
- Associated with a certain patient background
  - Adenocarcinoma, female, non-smoker, east Asian
- Tyrosine kinase domain

<table>
<thead>
<tr>
<th></th>
<th>740</th>
<th>750</th>
<th>760</th>
<th>860</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>‧</td>
<td>‧</td>
<td>‧</td>
<td>‧</td>
</tr>
<tr>
<td>deletion (exon 19) 50%</td>
<td>‧</td>
<td>‧</td>
<td>‧</td>
<td>‧</td>
</tr>
<tr>
<td>Point mutation (codon 858) 40%</td>
<td>‧</td>
<td>KIPVAIK-----TSPKANKEILD</td>
<td>‧</td>
<td>FGLAKLLG</td>
</tr>
<tr>
<td>Others (codons 709, 719, 768, 861, exon20 insertion…) 10%</td>
<td>‧</td>
<td>KIPVAIKRELATSPKANKEILD</td>
<td>‧</td>
<td>FGRAKLLG</td>
</tr>
</tbody>
</table>

- Associated with EGFR-TKI response
EGFR mutation and sensitivity to TKIs

Normal

- Ligand (EGF)
- Ligand-dependent EGFR phosphorylation
- Activation of downstream pathway
- Ordered proliferation

EGFR mutation

- Oncogene addiction
- Ligand-independent EGFR phosphorylation
- Cancer

EGFR-TKI treatment

- TKI
- Apoptosis
Phase III trials comparing EGF-TKI with platinum doublet for EGFR mu NSCLC

- gefitinib
- erlotinib
- afatinib

<table>
<thead>
<tr>
<th>Trial</th>
<th>gefitinib</th>
<th>erlotinib</th>
<th>afatinib</th>
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<tbody>
<tr>
<td>NEJ002</td>
<td>10.8</td>
<td>13.7</td>
<td>11.1</td>
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<tr>
<td>WJTOG3405</td>
<td>9.6</td>
<td>4.6</td>
<td>6.9</td>
</tr>
<tr>
<td>OPTIMAL</td>
<td></td>
<td>10.4</td>
<td>5.5</td>
</tr>
<tr>
<td>EURTAC</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>ENSURE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lux Lung 3</td>
<td>11.0</td>
<td></td>
<td>11.0</td>
</tr>
<tr>
<td>Lux Lung 6</td>
<td></td>
<td></td>
<td>5.6</td>
</tr>
</tbody>
</table>
Not all EGFR mutations are created equal

Kobayashi and Mitsudomi, Cancer Sci, 2016
EGFR-TKIs

1G
- gefitinib
  ![gefitinib](image1)
- erlotinib
  ![erlotinib](image2)

2G
- afatinib
  ![afatinib](image3)
- dacomitinib
  ![dacomitinib](image4)

3G
- osimertinib
  ![osimertinib](image5)
- pyrimidine
  ![pyrimidine](image6)
- quinazoline
  ![quinazoline](image7)
- acrylamide
  ![acrylamide](image8)
Not all EGFR mutations are created equal

Kobayashi and Mitsudomi, Cancer Sci, 2016
EGFR exon 20

**Poziotinib**

Prior therapy:
- P = AP32788
- S = ASP 8273
- E = Erlotinib
- A = Afatinib

**TAK-788**


Doebele, ASCO 2018
Gefitinib versus cisplatin plus docetaxel in patients with non-small-cell lung cancer harbouring mutations of the epidermal growth factor receptor (WJTOG3405): an open label, randomised phase 3 trial

Tetsuya Mitsudomi, Satoshi Morita, Yoshitaka Yatabe, Shinichi Nango, Isamu Okamoto, Junji Tsunetani, Takashi Seto, Masao Satouchi, Nihito Tsuda, Tomomi Hiroshima, Kazuhito Aoki, Motoko Kobayashi, Masumi Takada, Hiroshi Yoshida, Kazuhiro Shibata, Shinsuke Kodoh, Eiji Shimizu, Hiroshi Satoh, Shintichi Toyooka, Kazuhiro Nakagawa, Katsuhito Fukuoka, for the West Japan Oncology Group

Final overall survival results of WJTOG3405, a randomized phase III trial comparing gefitinib versus cisplatin with docetaxel as the first-line treatment for patients with stage IIIIB/IV or postoperative recurrent EGFR mutation-positive non-small-cell lung cancer


**Survival Analysis**

- **Median (95% CI) progression-free survival**
  - Gefitinib (n=86): 6.3 months (5.0–7.8)
  - Cisplatin and docetaxel (n=86): 9.2 months (8.0–13.0)

- **Overall survival**
  - Gefitinib (N=86): MST 34.9 months, P-value 0.2070
  - Cisplatin and docetaxel (N=86): MST 37.3 months

HR = 1.252 (95%CI: 0.883–1.775)
Co-occurring genetic alterations in EGFR mutated NSCLC

Blakely, et al., Nature Genetics 2017

Yu et al, Clin Cancer Res., 2018
Effects of co-occurring genetic alterations in EGFR targeted therapy

Yu et al, Clin Cancer Res., 2018
Blakely, et al., Nature Genetics 2017
Kim, et al., JTO 2019
Multi-region sequencing of EGFR mu adenocarcinoma of the lung

Nahar et al., Nature Comm, 2018
Newer strategies to treat EGFR+ patients

- **Newer generation EGFR-TKI**
  - 2G… afatinib, dacomitinib, 3G osimertinib (active for T790M, CNS penetrance)

- **Chemotherapy combination**
  - Early combo trails without selection failed (Intact1,2, Talent, Tribute)
  - Promising efficacy results with CBDCA/PEM/gefitinib combo (NEJ009, India study)

- **Other targeted therapy combination**
  - MEK-TKI, PI3K/mTORi, JAK/STAT-TKI, SRC-TKI, MET TKI, AXL TKI, HER3 Mab, EGFR Mab, HDACi,

- **Anti-angiogenesis agent combination**
  - Promising PFS with bevacizumab (JO NEJ026 ARTEMIS) and ramucirumab (RELAY)
  - May improve CNS penetrance, control of effusion, liver mets?

- **New antibodies**
  - EGFR-MET bispecific
  - HER3-ADC

- **Immunotherapy**
  - IMpower150
**FLAURA study**

Patients with locally advanced or metastatic NSCLC

- **Key inclusion criteria**
  - ≥18 years
  - WHO performance status 0 / 1
  - Exon 19 deletion / L858R (enrolment by local or central EGFR testing)
  - No prior systemic anti-cancer / EGFR-TKI therapy
  - Stable CNS metastases allowed

Endpoints
- **Primary endpoint:** PFS based on investigator assessment (according to RECIST 1.1)

**A Progression-free Survival in Full Analysis Set**

<table>
<thead>
<tr>
<th>Drug</th>
<th>No. of Patients</th>
<th>Median Progression-free Survival (95% CI)</th>
<th>mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osimertinib</td>
<td>279</td>
<td>18.9 (15.2–21.4)</td>
<td></td>
</tr>
<tr>
<td>Standard EGFR-TKI</td>
<td>277</td>
<td>10.2 (9.6–11.1)</td>
<td></td>
</tr>
</tbody>
</table>

Hazard ratio for disease progression or death, 0.46 (95% CI), 0.37–0.57

*P* < 0.001

**D Overall Survival**

<table>
<thead>
<tr>
<th>Drug</th>
<th>No. of Patients</th>
<th>Median Overall Survival (95% CI)</th>
<th>mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osimertinib</td>
<td>279</td>
<td>NC (NC–NC)</td>
<td></td>
</tr>
<tr>
<td>Standard EGFR-TKI</td>
<td>277</td>
<td>NC (NC–NC)</td>
<td></td>
</tr>
</tbody>
</table>

Hazard ratio for death, 0.63 (95% CI), 0.45–0.88

*P* = 0.007

Crossover was allowed for patients in the SoC arm, who could receive open-label osimertinib upon central confirmation of progression and T790M positivity

Soria et al., NEJM, 2017
FLAURA OS results

Ramalingam, et al., ESMO 2019
FLAURA OS by ethnicity

Asian patients

Non-Asian patients

Ramalingam, et al., ESMO 2019
FLAURA OS by ethnicity

Asian patients

Non-Asian patients

Ramalingam, et al., ESMO 2019
NEJ009

gefitinib vs. gefitinib+carboplatin+pemetrexed in EGFR mut NSCLC

Presented By Atsushi Nakamura at 2018 ASCO Annual Meeting
Gefitinib versus gefitinib-pemetrexed-carboplatin in \textit{EGFR} mutated lung cancer (Gef vs. Gef + C)

Vanita Noronha (VanitaNoronha)
On behalf of Kumar Prabhash, Vijay Patil, Amit Joshi, Nandini Menon; and
Department of Medical Oncology and Thoracic oncology disease management group, Tata Memorial Center (Mumbai, India)

ASCO 2019
Rationales to combine EGFR-TKI with anti-angiogenesis agents

Anti VEGF therapy

- Direct inhibition of tumor angiogenesis
- Improvement of drug delivery by normalizing tumor vasculature
- Direct action to tumor cells (inhibition of EMT, stemness, dedifferentiation)
- Enhancement of tumor immunity

JO25567
erlotinib vs erlotinib + bevacizumab in EGFR mu NSCLC

Updated PFS: investigator-assessed

<table>
<thead>
<tr>
<th></th>
<th>EB</th>
<th>E</th>
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<tbody>
<tr>
<td>Median (months)</td>
<td>16.4</td>
<td>9.8</td>
</tr>
<tr>
<td>HR</td>
<td>0.52 (95% CI: 0.35-0.76)</td>
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<tr>
<td>P value*</td>
<td>0.0005</td>
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</table>

*Log-rank test, two-sided

Final Overall survival

<table>
<thead>
<tr>
<th></th>
<th>EB</th>
<th>E</th>
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</thead>
<tbody>
<tr>
<td>Median (months)</td>
<td>47.0</td>
<td>47.4</td>
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<tr>
<td>HR</td>
<td>0.81 (95% CI: 0.53-1.23)</td>
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<tr>
<td>P value*</td>
<td>0.3267</td>
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</table>

*Log-rank test, two-sided

Presented By Noboru Yamamoto at 2018 ASCO Annual Meeting
Phase 3 trials comparing erlotinib with erlotinib plus bevacizumab

**NEJ026**

Saito et al., Lancet Oncol., 2019

**ARTEMIS (CTONG 1509)**

Zhou et al., ESMO., 2019
RELAY
erlotinib vs erlotinib ramucirumab in EGFR mu NSCLC

Consistent PFS benefit by blinded, independent central review
All patients: stratified HR 0.671 (95% CI: 0.518–0.869); East Asian patients: unstratified HR 0.692 (95% CI:0.522–0.918)

Nakagawa et al., ASCO 2019
Network metaanalysis for trials for EGFR mu+ NSCLC (Hazard ratios for PFS)

So, how to individualize?

<table>
<thead>
<tr>
<th></th>
<th>Pros</th>
<th>Cons</th>
</tr>
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<tbody>
<tr>
<td><strong>Osimertinib</strong>&lt;br&gt;(Flaura)</td>
<td>• Best HR&lt;br&gt;• Active for most frequent resistant mechanism, T790M&lt;br&gt;• High penetration to CNS&lt;br&gt;• Less toxicity (Any/G3≤ 98/32%, Flaura)&lt;br&gt;• Less expensive JPY 670,113 / 4w</td>
<td></td>
</tr>
<tr>
<td><strong>Erlotinib+ Ramucirumab</strong>&lt;br&gt;(RELAY)</td>
<td>• Requires second Bx (only 23.7% in REMEDY )&lt;br&gt;• Toxicity (Any/G3≤ 100/72%)&lt;br&gt;• Outpatient visit and DIV every 2w&lt;br&gt;• Expensive JPY 1,008,993/4w (1.5x osi)</td>
<td></td>
</tr>
</tbody>
</table>
Metastatic non-small cell lung cancer: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up®

Patients with a tumour with a sensitising EGFR mutation should receive first-line EGFR TKIs including erlotinib, gefitinib or afatinib [I, A], or dacomitinib [I, B; MCBS v1.1 score: 3]. None of the four EGFR TKIs is consensually considered as a preferred option [IV, C].

Erlotinib/afatinib represents a front-line treatment option in patients with EGFR-mutated tumours [II, B; ESMO-MCBS v1.1 score: 3]
Ramosertib with erlotinib is associated with longer PFS compared with erlotinib and placebo at the first interim analysis but data are still immature [II, B; not EMA-approved]
Addition of carboplatin and pemetrexed to gefitinib represents a first-line option in patients with EGFR-mutated tumours [I, B; not EMA-approved]
Stage IV lung carcinoma with EGFR-activating mutation

PS 0-2 [I, A]
PS 3-4 for all following options [II, A]

- Osimertinib [I, A; MCBS 4]\textsuperscript{a,b}
- Gefitinib [I, A]
- Erlotinib [I, A]
- +/- bevacizumab [II, B; MCBS 3]\textsuperscript{a,c}
- +/- ramucirumab [II, B]\textsuperscript{a}
- Afatinib [I, A]
- Dacomitinib [I, B; MCBS 3]\textsuperscript{a}
- Gefitinib/carboplatin/pemetrexed [I, B]\textsuperscript{a}

Disease progression

First-line osimertinib is now considered one of the options for patients with a tumour with sensitising EGFR mutations [I, A; MCBS score v1.1 score: 4]

Erlotinib/bevacizumab represents a front-line treatment option in patients with EGFR-mutated tumours [II, B; ESMO-MCBS v1.1 score: 3]

Ramucirumab with erlotinib is associated with longer PFS compared with erlotinib and placebo at the first interim analysis but data are still immature [II, B; not EMA-approved]

Addition of carboplatin and pemetrexed to gefitinib represents a first-line option in patients with EGFR-mutated tumours [I, B; not EMA-approved]

Exon 20 T790M mutation testing:
Mechanisms of resistance in the paired samples (N=38)

Yu et al, Clin Cancer Res., 2018
Randomised Phase III study of osimertinib vs platinum /pemetrexed for EGFR T790M+ advanced NSCLC (AURA3)

Papadimitrakopoulou et al., WCLC 2016
Mok et al., NEJM, 2016

Key eligibility criteria:
- ≥ 18 years (30 years in Japan)
- Locally advanced or metastatic NSCLC
- Evidence of disease progression following first-line EGFR-TKI therapy
- Documented EGFRm and central confirmation of tumour EGFR T790M mutation from a tissue biopsy taken after disease progression on first-line EGFR-TKI treatment
- WHO performance status of 0 or 1
- No more than one prior line of treatment for advanced NSCLC
- No prior neo-adjuvant or adjuvant chemotherapy treatment within 6 months prior to starting first EGFR-TKI treatment.

Endpoints:
- Primary:
  - PFS by investigator assessment (RECISTv1.1)
- Secondary and exploratory:
  - Overall survival
  - Objective response rate
  - Duration of response
  - Disease control rate
  - Tumour shrinkage
  - BCR-assessed PFS
  - Patient reported outcomes
  - Safety and tolerability

"Squash" asymptomatic CNS metastases allowed!

- Patients were stratified at randomisation based on ethnicity (Asian/Non-Asian)
- RECISTv1.1 assessments performed every 6 weeks until objective disease progression; patients could receive study treatment beyond RECISTv1.1 defined progression as long as they experienced clinical benefit.
- With 2:1 events of progression or death, the study would have 80% power to reject the null hypothesis of no significant difference in duration of PFS between the two treatment groups, assuming a treatment effect HR of 0.67 at 5%, two sided significance.

Graph showing PFS by investigator assessment

Analysis of PFS by BICR was consistent with the investigator-based analysis. HR 0.28 (95% CI 0.20, 0.38), p<0.001; median PFS 11.0 vs 4.2 months.
Signalling pathways driving resistance to EGFR TKIs in NSCLC
Select NSCLC trials evaluating combinations to address resistance mechanisms

<table>
<thead>
<tr>
<th>Drug regimen</th>
<th>Phase</th>
<th>Patient population</th>
<th>Results</th>
<th>Clinical trial identifier*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erlotinib + MEK inhibitor</td>
<td>I</td>
<td>EGFR+ns, prior EGFR TKI</td>
<td>Activity at preliminary analysis (absent)</td>
<td>NCT02143466</td>
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<tr>
<td>Gefitinib + erlotinib</td>
<td>I</td>
<td>EGFR+, prior EGFR TKI</td>
<td>Ongoing</td>
<td>NCT02035121</td>
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<tr>
<td>Erlotinib + gefitinib + XL185</td>
<td>I</td>
<td>EGF/erbB</td>
<td>Poorly tolerated</td>
<td>NCT00777009</td>
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<tr>
<td>Gefitinib + erlotinib</td>
<td>I</td>
<td>EGFR mutation</td>
<td>PF5, 14 months, 5% CR</td>
<td>NCT01570586</td>
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<tr>
<td>Gefitinib + erlotinib</td>
<td>III</td>
<td>Unselected</td>
<td>17% PR, 1% CR</td>
<td>NCT00090409</td>
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<tr>
<td>Erlotinib + gefitinib</td>
<td>II</td>
<td>EGFR+, no prior TKI</td>
<td>Ongoing</td>
<td>NCT03135470</td>
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<tr>
<td>Erlotinib + gefitinib</td>
<td>II</td>
<td>EGF/erbB</td>
<td>MCR</td>
<td>NCT03170301</td>
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<tr>
<td>Erlotinib + erlotinib</td>
<td>I</td>
<td>Molecular or clinical suggestion of EGFR+, prior EGFR TKI or EGFR-T790M</td>
<td>Ongoing</td>
<td>NCT01099995</td>
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<tr>
<td>Erlotinib + erlotinib</td>
<td>I</td>
<td>Molecularly selected</td>
<td>15% PR, 6% DCR</td>
<td>NCT03124537</td>
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<tr>
<td>Gefitinib + erlotinib</td>
<td>I</td>
<td>EGFR+, no prior TKI</td>
<td>5% PR</td>
<td>NCT03154460</td>
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<tr>
<td>Erlotinib + gefitinib + erlotinib</td>
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<td>EGF/erbB, no prior TKI</td>
<td>Ongoing</td>
<td>NCT03191703</td>
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<td>Erlotinib + gefitinib</td>
<td>II</td>
<td>EGF/erbB, no prior TKI</td>
<td>Ongoing</td>
<td>NCT00341015</td>
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<tr>
<td>Erlotinib + gefitinib</td>
<td>III</td>
<td>EGF/erbB, no prior TKI</td>
<td>Ongoing</td>
<td>NCT03835185</td>
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<tr>
<td>Gefitinib + erlotinib</td>
<td>II</td>
<td>EGF/erbB, no prior TKI</td>
<td>Ongoing</td>
<td>NCT00335944</td>
</tr>
<tr>
<td>Erlotinib + erlotinib</td>
<td>II</td>
<td>Wild-type EGFR</td>
<td>Ongoing</td>
<td>NCT01610336</td>
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<tr>
<td>Erlotinib + erlotinib</td>
<td>II</td>
<td>Unselected, no prior EGFR TKI</td>
<td>PR 10% (combination) versus 7% (erbB</td>
<td>NCT00777309</td>
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<tr>
<td>Erlotinib + erlotinib</td>
<td>II</td>
<td>Molecularly unselected</td>
<td>No effect in unselected patients</td>
<td>NCT00854308</td>
</tr>
</tbody>
</table>

Rotow and Bivona, Nature Rev Cancer 2017
Activities of MET-TKI plus EGFR-TKI for EGFR mu, TKI resistant NSCLC

- **capmatinib+gefitinib**
  
  \[\text{GCN} \geq 6 \ (N=36) \ \text{ORR} \ 47\%\]

- **savolitinib+osimertinib**
  
  \[\text{MET FISH}^+ \ (N=46) \ \text{ORR} \ 52\%\]

---

Wu, et al., ASCO 2019

Sequist, et al., ASCO 2019
The molecular and histologic landscape of resistance to osimertinib identified by tumor tissue analysis in EGFR+ lung cancers

**Conclusions**
- By evaluating tissue rather than plasma, we observed a high rate of histologic transformation, including squamous cell transformation.
- Early progressors and 1st line osimertinib may have different resistance patterns than treatment after prior TKIs, with off-target resistance emerging earlier and on-target resistance mutations later.

Schoenfeld, wt al., ASCO 2019
JNJ-61186372, an EGFR-cMET bispecific antibody

All patients with diverse EGFR mutations

32/108 (30%) patients with best response of PR across diverse EGFR mutations:
- Primary:
  - Exon 21 L858R
  - Exon 19 deletion
  - Exon 20 insertion

- Secondary:
  - Exon 20 T790M
  - Exon 18 G719A
  - cMet amplification
  - C797S

Post 3G-TKI patients

16/58 (28%) patients with best response of PR (8 confirmed):
- 8 patients with C797S
- 3 patients with cMet amplification (≥6 copies)
- 5 patients without identified EGFR- or cMet-based resistance

Exon 20ins

8/27 (30%) patients with best response of PR (6 confirmed)

Haura et al., ASCO 2019
U3-1402, a HER3-targeted ADC

Janne et al., ASCO 2019
Patients with a sensitizing EGFR mutation of ALK translocation must have disease progression or intolerance of treatment with one or more approved targeted therapies

Socinski et al., ASCO 2018
Addition of Bevacizumab to Atezolizumab and Chemotherapy Prolongs Survival of EGFR/ALK+ Patients

Presented By Mark Socinski at 2018 ASCO Annual Meeting

“国内未承認情報を含む”
Preventive combination therapy may induce T790M in lung cancer cells that otherwise become resistant using non-T790M mechanisms.

Suda, Mitsudomi et al., Clin Cancer Res., 2010

Sesumi, Mitsudomi et al., Lung cancer 2017
Longitudinal genomic analysis of tumor and cfDNA in a patient with EGFR-mutant lung cancer from diagnosis to death

Blakely, Watkins, Wu and Gini et al., Nature Genetics 2017
Tumor evolution as a therapeutic target
Strategies to target clonal evolution

- e.g. EGFR-TKI monotherapy
- Targeting trunk mutations
- e.g. EGFR-TKI+MET TKI
- Alternating therapy under the guidance of ctDNA analysis

Angelozzi, Swanton, and Bardelli, Cancer Discov., 2017
Take home messages

...New treatment options for EGFR+ NSCLC

- Not all EGFR mutations are created equal. Furthermore co-occurring driver mutations affect clinical outcome
- First-line treatment should be ideally individualized by genetic context. But how?
  - Patients with TP53/RB...chemo combo?
  - Patients with minor MET clone...MET TKI+EGFR TKI
- Virtually all patients acquire resistance...Treat according to the molecular context.
- Role of immunotherapy for EGFR mu patients is not fully understood
- How to cope with tumor heterogeneity
- Adaptive therapy according to ctDNA analysis is attractive
The 60th Annual Meeting of the Japan Lung Cancer Society

60th JLCS2019

Toward a World without Lung Cancer

CALL FOR ABSTRACTS
Due Date May 30

December 6-8, 2019  OSAKA, JAPAN

Visit our Website  http://conference.haigan.gr.jp/60/

General Information

Theme
Toward a World without Lung Cancer

Date
December 6 – December 8, 2019

Venue
Osaka International Convention Center
5-3-51, Nakanoshima,
530-0005 Japan

Submission Period
Tuesday, April 9 - Noo

Call for Papers for In!
Please be encouraged to present at the 60th Annu

This year, proffered p