Pipeline: Oncology

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# AstraZeneca Oncology

## Common vision
- Redefine cancer treatment paradigm
- Restore patients’ lives
- Eliminate cancer as cause of death

## Bold ambition
- By 2020, we will be a recognised leader in oncology, delivering 6 new medicines to patients

## 4 key MoA & platforms

## 4 core disease areas

<table>
<thead>
<tr>
<th>Combinations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Personalised healthcare</td>
</tr>
<tr>
<td>Smart development crucial to leadership</td>
</tr>
</tbody>
</table>
Oncology:
Combine therapies to change treatment paradigm

**Combination therapies may enhance efficacy by:**

- Targeting complementary pathways
- Establishing synergistic effects
- Overcoming resistance to monotherapy
- Improving risk / benefit profile
Oncology: Personalised healthcare as key driver

<table>
<thead>
<tr>
<th>Tissue-based assay</th>
<th>Single-gene mutation analysis</th>
<th>Circulating tumour DNA</th>
<th>Next-generation sequencing</th>
</tr>
</thead>
<tbody>
<tr>
<td>MEDI4736 (PD-L1)</td>
<td>Lynparza</td>
<td>Iressa / AZD9291</td>
<td>Oncology portfolio</td>
</tr>
</tbody>
</table>

- Tissue-based assay: MEDI4736 (PD-L1)
- Single-gene mutation analysis: Lynparza
- Circulating tumour DNA: Iressa / AZD9291
- Next-generation sequencing: Oncology portfolio

[Images of diagnostic tools and technologies related to genetic analysis and tumour DNA.]
### Oncology:
**Smart development crucial to leadership**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AZD9291</td>
<td>Potential filing less than 2½ years from first dose</td>
</tr>
<tr>
<td>MEDI4736 (PD-L1) PACIFIC study</td>
<td>Leapfrog competition into early line of therapy</td>
</tr>
<tr>
<td><em>Iressa / AZD9291</em></td>
<td>First in ctDNA diagnostic testing</td>
</tr>
<tr>
<td>MEDI4736 BRAF / MEK</td>
<td>Good combinability enables novel triplet combination</td>
</tr>
</tbody>
</table>
**Scientific leadership:**

Four key platforms

**Tumour drivers and resistance**
- AZD9291 (EGFRm)
- Selumetinib (MEK)
- AZD2014 (TORC1/2)
- AZD4547 (FGFR)
- AZD5363 (AKT)
- AZD6094 (cMET)
- AZD8186 (PI3Kβ)
- AZD8835 (PI3Kα)
- MEDI0639 (aDLL4)
- MEDI-573 (aIGF1/2)
- AZD9496 (SERD)
- AZD5312 (AR antisense)

**DNA damage repair**
- Olaparib (PARP)
- Cediranib (VEGF)**
- AZD1775 (Wee1)
- AZD6738 (ATR)
- AZD0156 (ATM)*
- AZD2811 (AKB)*

**Immunotherapy**
- MEDI4736 (PD-L1)
- Tremelimumab (CTLA-4)
- MEDI0680 (PD-1)
- MEDI6469 (murine OX40)
- MEDI0562 (OX40 humanised mAb)
- AZD9150 (STAT3)
- AZD5069 (CXCR2)

**Antibody drug conjugates**
- Moxetumomab (CD22)
- ADC-Spirogen*
- ADC-Bispecific*

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* Preclinical
** Combination with DDR

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Scientific leadership: Tumour drivers & resistance

- Highly altered signalling pathways in cancer
- Interconnected pathways allow resistance / escape mechanisms
- Multiple targets identified as potential tumour driver mutations
AZD9291: EGFR mutant selective inhibitor in lung cancer

- Potentially first irreversible selective inhibitor of double EGFR mutations
- Awarded FDA Breakthrough Therapy Designation
- FSI 1st line Phase III Q4 2014, data expected 2017
- Combinations with MEDI4736 (PD-L1), MET (cMET) and selumetinib (MEK) ongoing (FSI Q3 2014)

Strong evidence of monotherapy activity

Best percentage change from baseline in target lesion*

US NDA submission expected Q2 2015 in NSCLC 2L

* ESMO 2014
AZD9291: Early efficacy in 1st line EGFRm NSCLC

Clinical activity of AZD9291 in brain mets has been observed in a Phase I study in patients with acquired resistance to current EGFR-TKIs.

Overall disease control rate (CR+PR+SD) = 95%

FLAURA: Phase III NSCLC 1L EGFRm H2H vs 1st gen. TKI to start Q4 2014
**AZD2014:**
Dual TORC1/2 inhibitor

**Strong evidence of activity**

Dual TORC1/2 + paclitaxel*

- **Differentiated clinical activity**
  - Broad potential in breast, lung, ovarian cancer and lymphoma
  - Dual TORC1/2 and intermittent weekly dosing schedule to deliver better efficacy and tolerability
  - Potential accelerated Phase III investment decision in 2015

**Combination with Faslodex in ER+ breast cancer to be submitted for presentation in 2015**

*Basu et al ESMO 2014*
AZD6094:
Potent, selective cMET inhibitor of MET-driven tumours

Activity in MET-driven papillary renal cell cancer (PRCC)

- Active in MET amplified and MET-mutant settings
- First-in-class opportunity in papillary renal cell cancer (PRCC)
- Phase II trial in PRCC ongoing
- Phase II trial in MET-amplified gastric and lung cancer ongoing
- Combination with AZD9291 in 2nd line EGFR mutant lung cancer ongoing

Gan et al ASCO 2014
AZD9496: Oral selective estrogen receptor degrader (SERD)

Greater tumour inhibition than Faslodex

- Improved potency and bioavailability allows greater estrogen receptor (ER) knockdown
- Oral formulation
- Clinical development started Q4 2014
- Pharmacological data submitted for AACR 2015
DNA Damage Repair (DDR): Targeting the Achilles heel of cancer

- Highly altered pathways in cancer
- Traditional chemotherapies depend on DDR pathways
- Most extensive portfolio of DDR agents in the industry
Lynparza (olaparib): First-in-class PARP inhibitor

Clinical benefit in BRCAm ovarian cancer

<table>
<thead>
<tr>
<th></th>
<th>Olaparib, n=74</th>
<th>Placebo, n=62</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>11.2 mo</td>
<td>4.3 mo</td>
</tr>
<tr>
<td>HR</td>
<td>0.18</td>
<td>95% CI (0.10, 0.31); P&lt;0.00001</td>
</tr>
</tbody>
</table>

Ongoing Phase III programmes

- BRCAm ovarian cancer: SOLO-2 (2015*), SOLO-1 (2016*)
- BRCAm breast cancer: OlympiAD (2016*)
- Gastric cancer: GOLD (2017*)
- BRCAm pancreatic cancer: POLO (FSI Q4 2014)
- Promising activity in late-stage prostate cancer (10/30 RR, ESMO 2014)

EU: Positive CHMP opinion
US: PDUFA 3 January 2015

*Data available
**AZD1775:**
Wee1 inhibitor; encouraging data in ovarian cancer

**Wee1: Role in cell cycle progression and DNA damage checkpoints**

- **G1/S checkpoint**
- **G2/M checkpoint**
- **S phase checkpoint**

**Platinum-sensitive relapsed ovarian cancer**

- 11/14 RECIST responses
- PFS 10.8 months
- 13/14 GCIG responses (includes CA125 responses)
- Phase II study in ovarian; *Lynparza* combination due to start Q1 2015
- Phase I/II trials in NSCLC

**Phase III ovarian cancer investment decision expected 2015**
AZD2811: Aurora Kinase B inhibitor (AKBi): AML proof of concept

**Novel mechanism of action:**
- Regulates mitosis and chromosomal segregation
- Nanoparticle formulation in development*
- Potential in DLBCL and AML
- Plan to discuss further steps with regulators early in 2015

**Phase II: AKBi vs low-dose cytarabine (LDAC) in elderly unfit AML**

<table>
<thead>
<tr>
<th></th>
<th>AKBi vs LDAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>OCRR (+Cheson)</td>
<td>+30%</td>
</tr>
<tr>
<td>Overall Survival (HR)</td>
<td>0.88 (0.49-1.58)</td>
</tr>
</tbody>
</table>

Differentiated profile

- DNA damage repair

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Kantarjian et al Cancer 2013;119:2611-19
* In collaboration with BIND Therapeutics
Immuno-oncology (IO):
Changing the treatment paradigms for cancer

- An effective immune response is durable - possibly life-long
- Cancer hijacks many immune pathways to escape destruction
- Our robust pipeline allows identification of combinations that restore the immune response
Immuno-oncology (IO):
Three major components to cancer immune response

1. Antigen presentation
- HPV Vaccine*
- Radiotherapy
- Iressa
- NME-2
- AZD9291
- BRAF/MEK*
- NME-1
- NME-3

2. Optimizing T cell function and memory
- OX40
- PD-L1
- PD-1
- CTLA-4
- NME-4
- NME-5

3. Inhibition by microenvironment
- IDO*
- CCR4*
- CXCR2
- CCR4*
- STAT3
- NME-6
- NME-7
- NME-8

* Clinical collaborations

Clinical
Preclinical

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MEDI4736 (PD-L1): Triplet w/dabrafenib and trametinib in BRAFm melanoma

**BRAFi/MEKi treatment effect**

- Increase in CD8 Tumour Infiltrated Lymphocytes (TILs)
- Increase in PD-L1 expression

*Frederick et al, 2013*

**Potential for well-tolerated, durable benefit in BRAFm melanoma**

- Clinical data for BRAFi/MEKi provide rationale for “triplet” combination
- Potential for durable response in 1L BRAFm melanoma patients
- Phase I “triplet” combination well tolerated at full monotherapy doses; MTD not reached

Presentation of Phase I “triplet” combination planned for H1 2015

In collaboration with GSK
MEDI6383 (OX40): Pathway drives potent, durable anti-tumour T cell immunity

Murine OX40: Phase I evidence of activity

Best tumour assessment change from baseline (%)

- PD on day 56
- SD on day 56+

Stimulatory activity in periphery

3 unique OX40 molecules with distinctive biology

- Murine anti-human OX40 (active in monotherapy; in combination with MEDI4736 now)
- Human OX40L fusion protein (currently in dose escalation)
- OX40 (FSI Q1 2015)
AZD9150: STAT3 and roles in tumour microenvironment

STAT3 inhibition decreases immune tolerance in tumour microenvironment

Durable responses in Phase I monotherapy studies

Phase I oral presentation in plenary session at EORTC 19 Nov 2014
STAT3 + MEDI4736 Phase I study start H1 2015
AZD5069: CXCR2 affects myeloid-derived suppressor cell trafficking

- First-in-class CXCR2 antagonist in oncology
- Potential synergistic activity with MEDI4736 (PD-L1)
- Phase I combination study of CXCR2 + MEDI4736 (PD-L1) expected to start in H1 2015
### Immuno-oncology (IO): Combinations address multiple immune pathways

<table>
<thead>
<tr>
<th>Antigen presentation</th>
<th>PD-L1</th>
<th>EGFR</th>
<th>T-cell activation combined with increased tumour visibility</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PD-L1</td>
<td>MEK/BRAF</td>
<td>T-cell activation combined with increased antigen presentation</td>
</tr>
<tr>
<td></td>
<td>PD-L1</td>
<td>HPV Vaccine</td>
<td>T-cell activation combined with increased priming</td>
</tr>
<tr>
<td>CTLA-4</td>
<td>PD-L1</td>
<td></td>
<td>Increased T-cell activation through blocking multiple inhibitory pathways</td>
</tr>
<tr>
<td>PD-1</td>
<td>PD-L1</td>
<td></td>
<td>Increased T-cell activation through complete blockade of the PD-1/PD-L1 axis</td>
</tr>
<tr>
<td>PD-L1/CTLA-4</td>
<td>OX40</td>
<td></td>
<td>Increasing T cell number, function and memory</td>
</tr>
<tr>
<td>T-cell killing and memory</td>
<td>PD-L1</td>
<td>IDO</td>
<td>T-cell activation combined with removal of inhibition</td>
</tr>
<tr>
<td></td>
<td>PD-L1</td>
<td>CCR4</td>
<td>T-cell activation combined with T-reg depletion</td>
</tr>
<tr>
<td></td>
<td>PD-L1</td>
<td>CXCR2</td>
<td>T-cell activation combined with reduced MDSC suppression</td>
</tr>
<tr>
<td></td>
<td>PD-L1</td>
<td>STAT3</td>
<td>T-cell activation combined with myeloid reprogramming</td>
</tr>
</tbody>
</table>

Enables precise identification, location and relationship between multiple components of tumour microenvironment
Immuno-oncology (IO): Unique indications, novel combos, speed of execution

**1. Speed**
- Quickest path to approval

- **Monotherapy MEDI4736 (PD-L1)**
  - Late-stage NSCLC
  - 2L head/neck (SCCHN)

- **MEDI4736 (PD-L1) / tremelimumab combo**
  - Late-stage NSCLC
  - 2L head/neck (SCCHN)

**2. Differentiation**
- Rapid program expansion
- Adaptive decision-making
- Patient selection

- **Differentiated tumour types and biomarker subsets**

- **High value leapfrog indications:**
  - Stage 3 unresectable NSCLC
  - Adjuvant NSCLC

**3. Leadership**
- First or best-in-class agents

- **Combinations**
  - MEDI4736 (PD-L1) + tremelimumab
  - MEDI4736 (PD-L1) + AZD9291

- **Three OX-40 antibodies**
  - Murine, humanised, fusion
  - Monotherapy, combination
MEDI4736 (PD-L1): Monotherapy; early, durable activity in multiple tumours*

* Patients with baseline and ≥1 on-treatment scan; disease assessment at 6 weeks, 12 weeks, 16 weeks, and then every 8 weeks
Data cut-off: 21 August, 2014

Total study population (10 mg/kg q2w)

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ongoing responders</td>
<td>92% (33/36)</td>
</tr>
<tr>
<td>RECIST response</td>
<td></td>
</tr>
<tr>
<td>PD-L1+</td>
<td>22% (18/81)</td>
</tr>
<tr>
<td>PD-L1-</td>
<td>5% (12/233)</td>
</tr>
<tr>
<td>Total</td>
<td>10% (36/352)</td>
</tr>
<tr>
<td>Disease control rate at 12 weeks</td>
<td></td>
</tr>
<tr>
<td>PD-L1+</td>
<td>47% (38/81)</td>
</tr>
<tr>
<td>PD-L1-</td>
<td>28% (64/233)</td>
</tr>
<tr>
<td>Total</td>
<td>33% (115/352)</td>
</tr>
</tbody>
</table>
MEDI4736 (PD-L1) + tremelimumab:
Encouraging efficacy for combination in NSCLC

Tumour shrinkage by dose cohort (n=18)

Best change in tumour size by dose cohort (n=18)

<table>
<thead>
<tr>
<th>All patients</th>
<th>ORR</th>
<th>Stable disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>MEDI4736+tremelimumab</td>
<td>28% (5/18)</td>
<td>28% (5/18)</td>
</tr>
</tbody>
</table>
MEDI4736 (PD-L1) + tremelimumab: Potentially better response in PD-L1 negative tumours

**PD-L1 negative patients in NSCLC**

- **MEDI4736 (PD-L1)**
  - ORR: 10% (7/74), 95% CI (3.9%, 18.5%) SD≥12 weeks: 32.4% (24/74), 95% CI (22.0%, 44.3%)
- **MEDI4736 (PD-L1) + tremelimumab**
  - ORR: 30% (3/10), 95% CI (6.7%, 65.2%) SD≥12 weeks: 40% (4/10), 95% CI (12.2%, 73.8%)

* Mono: ORR 10% (7/74), 95% CI (3.9%, 18.5%) SD≥12 weeks: 32.4% (24/74), 95% CI (22.0%, 44.3%)
** Combination: ORR 30% (3/10), 95% CI (6.7%, 65.2%) SD≥12 weeks: 40% (4/10), 95% CI (12.2%, 73.8%)
## MEDI4736 (PD-L1): Development in NSCLC

**Fast-to-market monotherapy**

<table>
<thead>
<tr>
<th>Study</th>
<th>Phase</th>
<th>NSCLC</th>
<th>PD-L1 Status</th>
<th>Treatment</th>
<th>Design</th>
<th>Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATLANTIC</td>
<td>II</td>
<td>3L</td>
<td>positive</td>
<td>MEDI4736 (PD-L1) monotherapy</td>
<td>Single-arm Phase II</td>
<td>2015</td>
</tr>
<tr>
<td>ARCTIC</td>
<td>III</td>
<td>3L</td>
<td></td>
<td>MEDI4736 (PD-L1) monotherapy; tremelimumab combination</td>
<td>Randomised vs. SOC*</td>
<td>2017</td>
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**First-mover advantage Early NSCLC**

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<th>Data</th>
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<tbody>
<tr>
<td>PACIFIC</td>
<td>III</td>
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<td>MEDI4736 (PD-L1) monotherapy</td>
<td>Randomised vs. SOC*</td>
<td>2017</td>
</tr>
<tr>
<td>ADJUVANT</td>
<td>III</td>
<td></td>
<td></td>
<td>MEDI4736 (PD-L1) monotherapy</td>
<td>Randomised vs. SOC*</td>
<td>2020</td>
</tr>
</tbody>
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**Phase III MEDI4736 (PD-L1) + tremelimumab to start early 2015**

*SOC = standard of care
Non-small cell lung cancer (NSCLC): Focus on medical need in PD-L1 negative disease

- Largest segment of NSCLC
- Not addressed by marketed targeted therapies (EGFR, ALK)
- Significant unmet medical need remains

EGFRm 16%; KRASm 18%; ALKm 3%; EGFR/ALK/KRAS wt 63% (Kantar and AZ estimates 2020)
MEDI4736 (PD-L1): Head and neck cancer (SCCHN)

Before MEDI4736 (PD-L1) infusion

After two MEDI4736 (PD-L1) infusions (30 days)

96 year old patient who had progressed on cetuximab prior to study entry (HPV negative, PD-L1 positive)

SCCHN=Squamous cell carcinoma of head and neck
**MEDI4736 (PD-L1):**
Head and neck cancer development

<table>
<thead>
<tr>
<th></th>
<th>Fast-to-market monotherapy</th>
<th>First-mover advantage combination therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HAWK</strong></td>
<td>Phase II</td>
<td>Phase II</td>
</tr>
<tr>
<td></td>
<td>Platinum failures</td>
<td>Platinum failures</td>
</tr>
<tr>
<td></td>
<td>PD-L1 positive</td>
<td>PD-L1 negative</td>
</tr>
<tr>
<td>MEDI4736 (PD-L1)</td>
<td>monotherapy</td>
<td>MEDI4736 (PD-L1) + tremelimumab</td>
</tr>
<tr>
<td></td>
<td>Single-arm Phase II</td>
<td>Contribution of component study</td>
</tr>
<tr>
<td></td>
<td>Data: 2015</td>
<td>Data: 2016</td>
</tr>
</tbody>
</table>

**CONDOR**
Phase II
Platinum failures
PD-L1 negative
MEDI4736 (PD-L1) + tremelimumab
Contribution of component study
Data: 2016

**EAGLE**
Phase III
Platinum failures
Unselected
MEDI4736 (PD-L1) + tremelimumab
Randomised study vs. SOC
Data: 2017

Phase III monotherapy and combination with tremelimumab to start early 2015
### Ongoing studies

<table>
<thead>
<tr>
<th>Immuno-oncology Combination</th>
<th>Phase</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-L1</td>
<td>III</td>
<td>NSCLC</td>
</tr>
<tr>
<td>tremelimumab</td>
<td>III</td>
<td>Mesothelioma</td>
</tr>
<tr>
<td>Seq. AZD9291/selumetinib + docetaxel//Iressa/CTLA-4 &amp; PD-L1</td>
<td>II</td>
<td>NSCLC</td>
</tr>
<tr>
<td>PD-L1</td>
<td>II</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>PD-L1 + mOX40</td>
<td>II/II</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>PD-L1</td>
<td>II</td>
<td>MDS</td>
</tr>
<tr>
<td>PD-L1 + tremelimumab</td>
<td>I</td>
<td>NSCLC</td>
</tr>
<tr>
<td>PD-L1 + tremelimumab + BRAFi + MEKi</td>
<td>I</td>
<td>Melanoma</td>
</tr>
<tr>
<td>PD-L1 + Iressa</td>
<td>I</td>
<td>EGFRm NSCLC</td>
</tr>
<tr>
<td>PD-L1 + PD-1</td>
<td>I</td>
<td>Solid &amp; haems</td>
</tr>
<tr>
<td>PD-L1 + AZD9291</td>
<td>I</td>
<td>EGFR M+ NSCLC</td>
</tr>
<tr>
<td>tremelimumab + Iressa</td>
<td>I</td>
<td>EGFRm NSCLC</td>
</tr>
<tr>
<td>OX40 fusion protein</td>
<td>I</td>
<td>Solid tumours</td>
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</table>

### Planned studies

<table>
<thead>
<tr>
<th>Immuno-oncology Combination</th>
<th>Phase</th>
<th>Disease</th>
</tr>
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<tbody>
<tr>
<td>PD-L1 + tremelimumab</td>
<td>III</td>
<td>3L NSCLC</td>
</tr>
<tr>
<td>PD-L1 +/- tremelimumab</td>
<td>I/II/III</td>
<td>SCCHN</td>
</tr>
<tr>
<td>PD-L1 +/- tremelimumab</td>
<td>I/II</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>mOX40 + rituximab</td>
<td>I/II</td>
<td>Haematological</td>
</tr>
<tr>
<td>CD19 + PD-1</td>
<td>I/II</td>
<td>Haematological</td>
</tr>
<tr>
<td>PD-L1 + STAT3</td>
<td>I/II</td>
<td>Solid/haem tumours</td>
</tr>
<tr>
<td>PD-L1 + CXCR2i</td>
<td>I/II</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>PD-L1 + INCB024360 (IDO1)</td>
<td>I/II</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>PD-L1 + mogamulizumab (CCR4)</td>
<td>I/II</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>tremelimumab + mogamulizumab (CCR4)</td>
<td>I/II</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>PD-L1 + ADXS-HPV</td>
<td>I/II</td>
<td>HPV-cervical &amp; H&amp;N</td>
</tr>
<tr>
<td>mOX40 + tremelimumab</td>
<td>I/II</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>PD-L1 + ibrutinib (BTKi)</td>
<td>I/II</td>
<td>Haematological</td>
</tr>
<tr>
<td>PD-L1 + radiation</td>
<td>I</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>PD-L1 + tremelimumab + radiation</td>
<td>I</td>
<td>Solid tumours</td>
</tr>
<tr>
<td>PD-L1 + tremelimumab</td>
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Summary

Oncology poised to be transformational for the company

Broad pipeline addresses multiple mechanisms and allows for optimal combination therapies to improve patient benefit

Leadership in next-generation of science in oncology