FDA-AACR Public Workshop on

OPTIMIZING DOSAGES FOR ONCOLOGY DRUG PRODUCTS: QUANTITATIVE APPROACHES TO SELECT DOSAGES FOR CLINICAL TRIALS

February 15, 2024, 8 a.m. – 5 p.m.
February 16, 2024, 8 a.m. – 1 p.m.
Grand Hyatt Washington | Washington, D.C.
OPENING REMARKS
Marc Theoret, MD
U.S. Food and Drug Administration

WORKSHOP OVERVIEW
Stacy S. Shord, PharmD
U.S. Food and Drug Administration
FDA-AACR Public Workshop On
OPTIMIZING DOSAGES FOR ONCOLOGY DRUG PRODUCTS: QUANTITATIVE APPROACHES TO SELECT DOSAGES FOR CLINICAL TRIALS
February 15 and 16, 2024 | Washington, DC

Workshop Co-Chairs

Jiang Liu, PhD
FDA

Pat LoRusso, DO, PhD (hc), FAACR
Yale Cancer Center

Stacy Shord, PharmD
FDA

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#AACRSciencePolicy
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AstraZeneca

Bristol Myers Squibb

GSK

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Patricia M. LoRusso, DO, PhD (hc), FAACR
Yale Cancer Center

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Quantitative Approaches to Select Dosages for Clinical Trials

Dosage Optimization for Oncology

Products and Project Optimus

Stacy S. Shord, PharmD, BCOP, FCCP
Deputy Division Director
Division of Cancer Pharmacology II
Office of Clinical Pharmacology
OTS/CDER/FDA

February 15, 2024
Opinions presented are those of the speaker and should not be construed to represent FDA’s views or policies.
Project Optimus

**What?** Initiative to reform the dosage optimization and dose selection paradigm in oncology drug development

**Who?** A multidisciplinary team of medical oncologists, clinical pharmacologists, biostatisticians, toxicologists, and other scientists with expertise in key facets of dosage optimization

More Information: [Project Optimus](#)
Multidisciplinary Team

- **OCE**: Rick Pazdur, Marc Theoret
- **Leads**: Atik Rahman, Mirat Shah
- **RPM**: Pam Balcazar
- **Pharmacology/Toxicology**: Haleh Saber, Matthew Thompson
- **Clinical Pharmacology**: Brian Booth, Lanre Okusanya, Stacy Shord
- **Pharmacometrics**: Jiang Liu, Hao Zhu
- **OCP Policy**: Raj Madabushi
- **Clinical**: Brian Heiss, Jennifer Gao, Gwynn Ison, Elizabeth Duke, Shruti Gandhy, Cara Rabik, Pam Seam
- **Safety**: Abhi Nair
- **Biostatistics**: Joyce Cheng, Jonathon Vallejo, Gary Rosner
- **CBER**: Lianne Wu, Xiaofei Wang
- **Analysts**: Alex Akalu, Susan Jenney
Consequences of Not Optimizing Dosage

Before Approval

• Drug is poorly-tolerated at the approved recommended dosage
  – Patients may stop taking a potentially effective drug
  – Patients choose to try a different drug

• Drug does not make it to market or must be withdrawn from the market

• Takes long time to evaluate alternative dosages following approval
  – Patients may not want to participate in trial if commercially available
  – Disease area moves on to other treatments
Dosage Optimization PMRs

- 24 PMRs issued for 21 new drugs (15% of 138 total approved new drugs)
- Took a median of 6.5 years to fulfill or release
Project Optimus Supports Evaluating All Data to Inform Dosage Selection for Clinical Trials

• Consider all data: pharmacokinetic (PK), pharmacodynamic (PD), activity/efficacy, safety, and tolerability data at each step

• Evaluate safety information beyond DLTs, e.g., low-grade symptomatic toxicities, dosage modification frequencies, patient-generated data for treatment-related symptoms

• Identify a target dosage range early and then further evaluate several dosages (ideally in a randomized trial)

• Characterize dosage- and exposure-response relationships for efficacy and toxicity
Focusing on Dosage Before Approval

• Administering ‘optimized’ dosages in registration trial
  – Improves tolerability and adherence
  – Reduces dosage modifications (i.e., discontinuations)
  – Potentially increases likelihood of treatment response

• Earlier understanding of dose- and exposure-response relationships may allow for more rapid development of new therapies, such as
  – combination regimens, new dosing regimens & new formulations

• More efficient to evaluate multiple dosages early in development

• Challenging to conduct dosage optimization trials post-approval
Guidances and Tools
Tools Available to Support Dosage Selection and Optimization

- Guidance Documents
- MIDD & Formal Meetings
- Oncology Dosing Tool Kit

Formal Meetings
- Type A
- Type B
- Type B EOP
- Type C
- Type D

https://www.fda.gov/media/172311/download
• Dosages must have justification appropriate to development stage

• Evaluate all data to select and support dosages

• Randomized comparisons support identification of optimized dosage(s)

• Safety assessments should include low-grade symptomatic toxicities

• Important for all products, including those with anticipated rapid development timelines
Guidance Documents

1994

ICH Guideline

INTERNATIONAL CONFERENCE ON
HARMONISATION OF TECHNICAL
REQUIREMENTS FOR REGISTRATION OF
PHARMACEUTICALS FOR HUMAN USE
ICH Harmonised Tripartite
Guideline

Dose Response Information to
Support Drug Registration
ICH E4

Recommended for Adoption
at Step 4 of the ICH Process
on 10 March 1994
by the ICH Steering Committee

2003

Guidance for Industry
Exposure-Response Relationships — Study
Design, Data Analysis, and Regulatory
Applications

U.S. Department of Health and Human Services
Food and Drug Administration
Center for Drug Evaluation and Research (CDER)
Center for Biologics Evaluation and Research (CBER)
April 2003
CP

2022

Population Pharmacokinetics
Guidance for Industry

U.S. Department of Health and Human Services
Food and Drug Administration
Center for Drug Evaluation and Research (CDER)
Center for Biologics Evaluation and Research (CBER)
February 2022
Clinical Pharmacology
Optimized Dosages

Which dosages will be evaluated during dose escalation?

Which dosages will be chosen for further investigation?

What dosages will be selected for the registration trial(s)?

https://www.fda.gov/about-fda/oncology-center-excellence/oncology-dosing-tool-kit
Workshop
## Quantitative Approaches to Select Dosages for Clinical Trials

<table>
<thead>
<tr>
<th>Selecting Dosages for Dose-Escalation Portion of First-In-Human Trials</th>
<th>Selecting Dosages for Additional Exploration Based on Nonclinical and Early Clinical Data</th>
<th>Selecting Dosages for Registrational Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Selecting Dosages for Dose-Escalation Portion of First-In-Human Trials</td>
<td>• Evaluating and Modeling All Early Data to Select Recommended Phase II Dose</td>
<td>• Considering the Totality of Efficacy and Safety Data to Aide Registrational Trial Designs</td>
</tr>
<tr>
<td>• Alternative Designs for Dose-Finding Trials: Ending Reliance on Short-Term Safety</td>
<td>• Novel Trial Designs to Enhance Dose-Selection Decision Making</td>
<td>• Implementing Seamless and Adaptive Registrational</td>
</tr>
</tbody>
</table>
Acknowledgements

Jiang Liu
Atiqur Rahman
Mirat Shah
Marc Theoret
Project Optimus
SESSION 1: SELECTING DOSAGES FOR DOSE-ESCALATION PORTION OF FIRST-IN-HUMAN TRIALS
Dose Selection in Oncology First in Human Trials: Challenges and Opportunities

Hao Zhu, Ph.D., Mstat
Division Director
Division of Pharmacometrics,
FDA/CDER/OTS/OCP

FDA-AACR Public Workshop
(February 2024)
Dose Selection in Clinical Development

Preclinical Studies
FIH Study
(SAD and/or MAD)
Clinical Drug Development
Late Phase Approval
Life Cycle of a Drug
# FIH studies

<table>
<thead>
<tr>
<th>Subject</th>
<th>Non - Oncology</th>
<th>Oncology</th>
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</thead>
<tbody>
<tr>
<td>Subjects</td>
<td>Healthy volunteers</td>
<td>Cancer Patients</td>
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</table>

## Objectives

<table>
<thead>
<tr>
<th>Non - Oncology</th>
<th>Oncology</th>
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<tbody>
<tr>
<td>• Explore dose range based on short-term safety and tolerability findings.</td>
<td>• Explore dose range based on short-term safety and tolerability findings.</td>
</tr>
<tr>
<td></td>
<td>• Identify maximum tolerated dose (MTD)</td>
</tr>
<tr>
<td></td>
<td>• Proof of concept (POC)</td>
</tr>
<tr>
<td></td>
<td>• Identify potentially efficacious dose.</td>
</tr>
</tbody>
</table>

## Focus

<table>
<thead>
<tr>
<th>Non - Oncology</th>
<th>Oncology</th>
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<tbody>
<tr>
<td>Safety and tolerability</td>
<td>Safety and tolerability, efficacy-related findings</td>
</tr>
</tbody>
</table>

## Dose range

<table>
<thead>
<tr>
<th>Non - Oncology</th>
<th>Oncology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Explore a wide dose range that is reasonably safe and well-tolerated</td>
<td></td>
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</tbody>
</table>

## Dose titration

<table>
<thead>
<tr>
<th>Non - Oncology</th>
<th>Oncology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start low &amp; titrate gradually</td>
<td></td>
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</tbody>
</table>

[Image of a question mark]
Dose Selection in FIH Oncology Studies

**Expectation on safety**
Should not push the dose too high

**Expectation on efficacy**
Should not start the dose too low

- Limited treatment options
- Unmet medical needs
Oncology Dose Selection in the Past

Cytotoxic Chemotherapy

Toxicity
Efficacy

Dose/Drug Exposure vs Effect/Response

FIH Studies
Dose Escalation
Confirmation

Expansion / Efficacy Trials

Targeted Patients

MTD
Control

R

0 50 100 200 400 800

0 100

Patient with No DLT
Patient with DLT
Empirical Ways to Select Starting Dose

- FIH Trial
- NOAEL/NOEL
- STD10 (rodent)
- HNSTD (non-rodent)

*: ICH S9 Guidance
Novel Modalities in Oncology Development

- Target Therapy
- Monoclonal Antibodies
- Bispecific Antibodies
- Antibody Drug Conjugates
- RNA Compounds
- Novel Delivery System

Novel Mechanism of Actions

FIH Dose Selection
Changing Paradigm

Effect/Response vs. Dose/Drug Exposure

- Efficacy
- Toxicity

A) Low Ab conc.
B) Optimal Ab conc.
C) High Ab conc.
Model-Based Dose Selection

- Clinical Experience
- Preclinical Data
- Disease Progression
- Mechanism Of Action

Patients

Novel Modalities

Model as a holistic translational platform

FIH Dose
Good Start is Half the Job Done
SESSION 1A: UTILIZING NONCLINICAL DATA AND MODELING TO SUPPORT DOSAGE SELECTION FOR FIRST IN HUMAN TRIALS

MODERATOR
Hao Zhu, PhD
U.S. Food and Drug Administration

INTRODUCTORY SPEAKER
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U.S. Food and Drug Administration

Ralph Parchment, PhD
NCI Frederick National Laboratory

Manish Gupta, PhD
Genmab
Applications of model-based approaches to select the starting dosages for first in human trials

Alex Phipps, PhD
on behalf of the AZ Project Optimus Advisory group

Alex Phipps, Ph.D.,
Clinical Pharmacology and Pharmacometrics - Oncology,
AstraZeneca, Cambridge, U.K
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Join the conversation:
#AACRSciencePolicy
Disclosure Information

Alex Phipps has the following relevant financial relationships to disclose:
   Employee and Stockholder of: AstraZeneca

The views expressed in this presentation are my own and not necessarily reflective of AstraZeneca
Focus of the presentation

• Model-based approaches to impact starting doses in the following patient settings
  • Monotherapy
    • Case 1 – T-cell engager
    • Case 2 – Small molecule
  • a New Indication
    • Case 3 – Bispecific mAb
  • a New Drug Combination
    • Case 4 – Modelling of advanced cellular in vitro systems

Whilst working with the urgency that patients require
Defining the optimal clinical regimen (right starting dose, right dose, schedule and combination)

- Estimate of receptor occupancy
e.g., Most TKI’s Bispecific antibodies

- Target mediated drug disposition (TMDD)
e.g., rituximab, emactuzumab

- Biomarker response for combinations or tumour growth kinetics
Common approach

- Systems pharmacology models
e.g., bispecific mAb

- Direct translation of non-clinical efficacy models
e.g., osimertinib

Always balanced against safety
Model-based approaches to dose optimization in monotherapy: Balancing efficacy against safety

- Identify a safe starting dose with some predicted efficacy
- Identify an active dose range to explore
- Make an informed decision on dose to take forward to a Phase 3 trial
Case Study 1: Model based approach to optimizing Therapeutic Index – T-cell engagers

- Agonists of the immune system. *Blinatumomab approved in 2014*

- mAb binds T-cells and Tumour cells to form an active ‘trimer’ mimicking a synapse

- **Challenges to starting dose prediction**

- Nonclinical models can be limited by lack of antigen expression

- *In vitro* cytokine release experiments can overestimate clinical observation – resulting in starting doses up to 500x below activity

*Betts and van de Graaf 2020*
Selecting the starting dose for FIH – T-cell engagers

Betts et al. 2019 devised a QSP model which predicted the concentrations of the active ‘trimer’

This trimer may be more reflective of the ‘active’ moiety

The models also show that the trimer may saturate at higher concentrations. This may have implications on how high a dose is needed.
T-cell engagers – Trimer model-based starting dose approach for solid tumour

1. PK/PD modelling to predict in vitro trimer concentration-response relationship for cytotoxicity and cytokine release

2. PBPK and PK/PD modelling to predict trimer concentration in patients across a range of potential dose levels

3. Rational choice of appropriate PD biomarkers and upper limit of pharmacological activity level for starting dose selection

Ball, K et al mAbs 2023
T-cell engagers – Trimer model may enable cycle time reduction

Starting dose was 10x higher using Trimer than MABEL. - Well tolerated clinically

Model informed on single patient cohorts to predicted dose range (0.003 – 0.3 mg/kg)

Reduces number of patients exposed to ineffective doses

Informs on doses for expansion

By incorporating what is known about target expression can inform on doses across indications
Case Study 2: Using non-clinical and clinical data for starting dose and informing on dose expansion cohorts

Osimertinib

A brain penetrant mutant-selective EGFR inhibitor

Approved for the treatment of EGFR-mutant NSCLC harboring sensitizing EGFR mutations, including T790M

A ‘pre-Project Optimus’, Optimus like design
Translational modeling enabled a starting dose predicted to have efficacy

Preclinical studies shows 5 mg/kg is an efficacious dose in mouse

Translational PK-PD modeling to predict human efficacious exposure/dose

Starting dose of 20 mg is predicted to be efficacious; 80 mg for max efficacy.

BM = Brain metastases
Osimertinib dose selection followed an ‘Optimus like’ approach

AURA Phase I study - rolling six design

Dose escalation

Dose expansion at multiple dose levels in preselected efficacy population (T790M)

MTD not achieved

Treatment naïve cohort at 2 doses
## Considerations for selecting starting dose across new indications

<table>
<thead>
<tr>
<th>Considerations for starting dose</th>
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<tbody>
<tr>
<td><strong>Dose</strong></td>
</tr>
<tr>
<td>Established or expected in 1(^{st}) indication</td>
</tr>
<tr>
<td><strong>Mechanism of Action</strong></td>
</tr>
<tr>
<td>Supporting non-clinical E-R similarity. But be aware of modality specific concerns – e.g.,</td>
</tr>
<tr>
<td>. Bystander effect with ADC may vary</td>
</tr>
<tr>
<td>. Tumour penetration and blood flow per indication</td>
</tr>
<tr>
<td>. Receptor Expression (TMDD). Relevance to CAR-T?</td>
</tr>
<tr>
<td>. Immune cell expression across tumours – also, ‘cold’ and ‘hot’ tumours</td>
</tr>
<tr>
<td><strong>Clinical safety profile</strong></td>
</tr>
<tr>
<td>Is the AE profile to date likely to be tolerated in the second?</td>
</tr>
<tr>
<td><strong>DDI</strong></td>
</tr>
<tr>
<td>Understand differences in con-meds between indications</td>
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</table>

**Models may help with some of these questions**

**TMDD = target-mediated drug disposition**

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**FDA-AACR Public Workshop on**

**OPTIMIZING DOSAGES FOR ONCOLOGY DRUG PRODUCTS: QUANTITATIVE APPROACHES TO SELECT DOSAGES FOR CLINICAL TRIALS**
Case Study 3 - Rilvegostomig – PD1-TIGIT
Optimizing for two targets

• Bispecific antibody - binds two checkpoint targets
• PD-1 well established through Keytruda, Opdivo, etc
• Motivation was to build a model that would inform on
  • starting dose and optimal dose range
  • dose across indications and combinations

The anti-TIGIT component of rilvegostomig is derived from COM902 developed by Compugen Ltd

ACoP 2023 (Gong et al)
Rilvestromig – PD1-TIGIT Systems’ approach

Two TMDD modules - capture concentration and receptor binding dynamics

Couples PK and tumour dynamics - to simulate receptor occupancy (RO) across dose levels.

Compare RO for PD-1 and TIGIT drugs

Sensitivity analysis – effect of variable tumour uptake (PC) and concentration of receptor (CR)

RO = receptor occupancy
Rilvegostomig – Simulation results

Predicted RO for rilvegostomig and other PD-1 mAbs

The graph shows the predicted PD-1 and TIGIT %RO in TME across dose range. Compared to literature data for PD-1 drug.
Comparison to external meta-data informed on the dose range for expansion *a priori*

Starting dose of 70 mg (~ 70% RO of PD1 and TIGIT) informed by the model

Model was used to narrow the dose range for expansion (750 and 1500 mg)

Other PD-1 and TIGIT have same dose across indications - gives confidence to use the same dose to start indication expansions
Doses in combinations

To date we’ve considered first dose and dose optimization in monotherapy.

What about combinations?

I think the same approaches can apply.

An example - combining novel experimental systems with modeling to optimize combination doses \textit{a priori}

BENEFIT – Guides starting doses and can significantly reduce clinical combination dose ranging
Case Study 4

Modelling of advanced *in vitro* cellular systems is necessary for clinical translation

**Exposure-Response Relationships**

**Organoids**

**Systems Model**

*In vivo*

Quantitative systems models allow the prediction of time-series clinical responses with various what-if scenarios

**Patients**


draft diagrams and text...
Clinical Translation from MPS Data: Hematotoxicity

Human hematopoietic model

\[
\begin{align*}
\frac{dHSC}{dt} &= \phi_{HSC} = \lambda_{HSC} \cdot HSC \cdot (1 - f_1(HSC)) - (\sigma_{HSC} + \sigma_{mon}) \cdot HSC - \beta_{HSC} \cdot HSC \\
\frac{dMLP}{dt} &= \sigma_{SC} \cdot SC + \lambda_{SC} \cdot MLP \cdot (1 - f_2(CL)) + \beta_{SC} \cdot MLP - \sigma_{MLP} \cdot MLP - E_{reg} \cdot \lambda_{SC} \cdot MLP \\
\frac{dCMP}{dt} &= \sigma_{SC} \cdot SC + \lambda_{SC} \cdot CMP \cdot (1 - f_3(MEP)) + \beta_{CMP} \cdot CMP - \sigma_{CMP} \cdot CMP - E_{reg} \cdot \lambda_{SC} \cdot CMP \\
\frac{dCLP}{dt} &= \sigma_{SC} \cdot MLP + \lambda_{SC} \cdot CLP \cdot (1 - f_4(CL)) - \sigma_{CLP} \cdot CLP - E_{reg} \cdot \lambda_{SC} \cdot CLP \\
\frac{dMEP}{dt} &= \sigma_{SC} \cdot CMP + \lambda_{SC} \cdot MEP \cdot (1 - f_5(CL)) + \beta_{MEP} \cdot MEP - \sigma_{MEP} \cdot MEP - E_{reg} \cdot \lambda_{SC} \cdot MEP \\
\frac{dGMP}{dt} &= \sigma_{CMP} \cdot CMP + \lambda_{CMP} \cdot GMP \cdot (1 - f_6(CL)) - \sigma_{GMP} \cdot GMP - E_{reg} \cdot \lambda_{GMP} \cdot GMP \\
\frac{dMK}{dt} &= \sigma_{MEP} \cdot MEP + \lambda_{MEP} \cdot MK \cdot (1 - f_7(CL)) - \sigma_{MK} \cdot MK - E_{reg} \cdot \lambda_{MK} \cdot MK \\
\frac{dMKT}{dt} &= \sigma_{MK} \cdot MK - \tau_{MK} \cdot MKT \\
\frac{dCD71}{dt} &= \sigma_{GMP} \cdot CD71 + \lambda_{CD71} \cdot CD71 \cdot (1 - f_8(CL)) + \beta_{CD71} \cdot CD71 - \sigma_{CD71} \cdot CD71 - E_{reg} \cdot \lambda_{CD71} \cdot CD71 \\
\frac{dmyblast}{dt} &= \sigma_{GMP} \cdot myblast + \lambda_{myblast} \cdot myblast \cdot (1 - f_9(CL)) - \sigma_{myblast} \cdot myblast - E_{reg} \cdot \lambda_{myblast} \cdot myblast \\
\frac{dmyblastT}{dt} &= \sigma_{GMP} \cdot myblast + \lambda_{myblastT} \cdot myblastT - \sigma_{myblastT} \cdot myblastT \\
\frac{dmyblastT}{dt} &= \sigma_{GMP} \cdot myblast + \lambda_{myblastT} \cdot myblastT - \sigma_{myblastT} \cdot myblastT \\
\end{align*}
\]

Identification of Drug A + Drug B dosing schedule to avoid clinical hematotoxicity

Drug A: q3w dosing

Drug B: 7d off / 14d on

7d off / 7d on / 7d off

Stem cells (CD34+ CD38-)

Erythroid (CD13- CD36+ CD71+ CD235+)

Myeloid (CD13+ CD16+)

Megakaryocytes (CD13- CD41+)
The approach is currently being used to predict starting doses and schedule in combination. May result in significant reduction in clinical dose ranging.
Summary

Advantages of and considerations for model-based approaches

Advantages

- Can inform on rational dosing strategies
- Bolsters rationale for HA submissions
- Facilitates a more accurate starting dose
- Amenable to most modalities
- Aids decision making on when to expand cohorts/switch indication/combinations

Considerations

- Start early – data availability is key
- Discuss with teams the aims of and assumptions in the model
- Clear communication
- Capture learnings from one model to another – portfolio
- Keep models as simple as possible
Final thoughts

• Project Optimus has really helped focus the Industry on the question of the right dose/exposure, particularly in avoiding too high doses

• Model-based analysis, incorporating novel experimental systems, has the potential to decrease dose ranging across various indications and in combinations to speed drug development and reach patients sooner
Acknowledgements

Megan Gibbs
Kathryn Ball
Chang Gong
Song Ren
KyoungSoo Lim
Pavan Vajjah
Karthick Vishwanathan
Amal Ayyoub
Massimo Lei
Cesar Pichardo
Kevin Smart
Matt Hellmann
Susan Galbraith
Jayne Marshall
Deepa Subramaniam
Shaily Arora
Philip Overend
Owen Jones
Sonja Gill
Kainat Khan
Carmen Pin
Holly Kimko
Rhiannon David
Project Optimus Advisory Group - AZ
SESSION 1A: UTILIZING NONCLINICAL DATA AND MODELING TO SUPPORT DOSAGE SELECTION FOR FIRST IN HUMAN TRIALS

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