The human microbiome in precision medicine: (way) beyond fecal transplantation

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Overview

1. Translational microbiome research at Baylor College of Medicine

2. Translating the microbiome—application of appropriate models to examine causality

3. Translational Microbiome Research Vignettes
   - Maternal high fat diet induced social/synaptic defects in mice
   - Immunotherapy--microbiome and PD-1/PD-L1 blockade therapy

4. New enteroid co-culture system for the study of human/host-microbial relationships in vitro
Model for Translational Microbiome Research

- Compare microbiota in healthy and ill subjects
- ID organisms / genes associated with health / disease
- Develop applications / solutions

Community (100’s of strains, undefined composition)

Consortium (defined composition of more than one strain, which together perform a function of interest)

Single strain (one strain, pure isolate)

Bioactive (molecule produced by strain that mediates effect of host)

Diagnostic

Probiotic / antibiotic

Patient Stratification
- Clinical trials
- Personalized medicine
Mission: To understand how the microbiome impacts health and disease, to translate this understanding for better therapeutics and diagnostics, and to serve as a hub for these activities internationally.

Translational Microbiome Research at Baylor College of Medicine

- Microbiome Exploration (‘Omics’)
- Microbial Ecology Modeling and Dissection
- Center for Metagenomics And Microbiome Research
- Therapeutic development
- Education
- Translation
- Policy and outreach (Govt. and public)

Commercialization
Precision Medicine Paradigm

Keys to microbiome input on precision medicine

- How widely applicable is knowledge from one study or cohort
- How translatable is a pre-clinical model observation
Metagenomic/genomic applications in CMMR service center

Sample:
- Collection
- Storage
- Shipping
- Processing

Library preparation/QC
- Sequencing

Bioinformatics
- Community Structure
- Bacterial Pangenome
- Genome Assemblies
- Microbial RNASeq
- Viral Detection

OUTPUTS

Experimental Design

Microbial Biomass Enrichment

DNA / RNA Extraction and Purification

Sequencing Platform Flexibility
Advantages of metagenomics at scale

Studying multiple disease cohorts can facilitate dissection of disease mechanisms and lead to better diagnostics and therapeutics.

Universal factors:
- Common exposures
- Common genetic risks

*Universal target discovery

Unique regional factors:
- Diet
- Race/ethnicity
- Access to health care
- Environment

*Personalized target discovery

Uniformity in sample processing and data generation
Chronic Kidney Disease (Raj, George Washington University)
Biliary atresia (Shneider and Tessier, BCM)
Autism (Costa-Mattioli, BCM; Mazmanian, Cal Tech)
Malnutrition (Preidis, Texas Children’s)
Type 1 Diabetes (Krischer; Burkhardt, USF; Atkinson, UF)
Microbiome of death (Bucheli and Lynne, SHSU)
Microbial surveillance (Klotman (BCM-MC); Maresso (elementary school))
Synthetic probiotic organisms (Tabor, Rice)
Necrotizing enterocolitis (Burrin; Premkumar, BCM)
Fecal transplants (Graham, BCM; Wilkerson, MD Anderson; DuPont, UTHSC)
HIV (Vigil, UT; Bryson, UTH; San Juan, U. Norte; Klotman, BCM)
Colon cancer (El Serag, BCM; Daniel-MacDougall, MD Anderson)
Type 2 Diabetes (Fisher-Hoch, UTSPH)
COPD cancer (Liu, BCM)
Clostridium difficile (Graham and Koo, BCM; DuPont, UTSPH)
IBS/IBD (DuPont, BCM (adults); Versalovic, TCH/BCM (children); Dann UTMB; Round, Utah)
Immunotherapy (Wargo, MD Anderson)
Pancreatic cancer (Fisher, BCM)
Diet and cognition during development (Costa-Mattioli, BCM)
Anorexia and bulimia (Pinho, Albert Einstein Medicina Diagnóstica, Brazil)
Suicide and suicidal ideation (Madan, Methodist Research Institute; Salas, BCM)

Disease and Disease Model Projects
> 350 projects over the last 7 years, and growing…
Model systems for host-microbe interactions: Microbiome phenotyping pipeline

*In vitro* culturing in mini bioreactors

*In vivo* studies in *C. elegans*

Enteroids from various GI locations

*In vivo* studies in germ free mice

Robert Britton

Buck Samuel

Mary Estes

Alton Swennes
Microbial Reconstitution Reverses Maternal Diet-Induced Social and Synaptic Deficits in Offspring

Shelly A. Buffington,1,2 Gonzalo Viana Di Prisco,1,2 Thomas A. Auchtung,3,4 Nadim J. Ajami,3,4 Joseph F. Petrosino,3,4 and Mauro Costa-Mattioli1,2*

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2 Memory and Brain Research Center
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http://dx.doi.org/10.1016/j.cell.2016.06.001
Maternal high fat diet (MHFD) impairs sociability (and cognition and induces repetitive motion).
Maternal high fat diet (MHFD) impairs sociability (and cognition and induces repetitive motion)

A. Timeline:
- Begin maternal diet: 8wks
- Mating of dams: 3wks
- Subject offspring born: 3wks
- Wean offspring: 4wks
- Test offspring behavior

E. Testing:
- Habituation
- Sociability
- Social Novelty

B. Image of rodent

C. Interaction time (s):
- MRD: 200
- MHFD: 50

D. Contact duration (s):
- MRD: 1.5
- MHFD: 0.5

C. Graphs:
- Sociability: MRD (n=14) vs MHFD (n=14)
- Social Novelty: Interaction time (s) for Mouse 1, Mouse 2, Empty Mouse 1, Empty Mouse 2
The gut microbiome in mice born to MHFD dams is different from MRD-born mice

2016, Cell 165, 1762–1775
Cohousing MHFD offspring with MRD alleviates sociability defect in MHFD
Metagenomic sequencing reveals gut microbiome differences between MHFD and MRD born mice

<table>
<thead>
<tr>
<th>Species of interest</th>
<th>MRD Representation</th>
<th>MHFD Representation</th>
<th>Fold Change MRD/MHFD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactobacillus reuteri</td>
<td>7.49 ± 3.0</td>
<td>0.879 ± 0.21</td>
<td>9.24 ± 0.65</td>
</tr>
<tr>
<td>Parabacteroides distasonis</td>
<td>0.00709 ± 0.0055</td>
<td>0.00126 ± 0.0011</td>
<td>5.63 ± 1.17</td>
</tr>
<tr>
<td>Helicobacter hepaticus</td>
<td>7.35 ± 2.4</td>
<td>2.58 ± 1.3</td>
<td>2.84 ± 0.61</td>
</tr>
<tr>
<td>Bacteroides uniformis</td>
<td>5.49 ± 2.2</td>
<td>2.07 ± 0.78</td>
<td>2.65 ± 0.56</td>
</tr>
<tr>
<td>Olsenella unclassified</td>
<td>0.230 ± 0.064</td>
<td>0.121 ± 0.031</td>
<td>1.90 ± 0.38</td>
</tr>
<tr>
<td>Collinsella unclassified</td>
<td>0.0866 ± 0.031</td>
<td>0.0494 ± 0.016</td>
<td>1.75 ± 0.48</td>
</tr>
<tr>
<td>Blisobacterium pseudolongum</td>
<td>19.4 ± 3.3</td>
<td>11.3 ± 2.4</td>
<td>1.71 ± 0.27</td>
</tr>
<tr>
<td>Lactobacillus johnsonii</td>
<td>24.5 ± 6.2</td>
<td>17.1 ± 5.2</td>
<td>1.43 ± 0.40</td>
</tr>
</tbody>
</table>

Table 1. Species whose abundance is reduced in the gut microbiota of MHFD offspring.
Supplementation with live, not killed *L. reuteri* restores social behavior

A

B

C

Not shown: *L. johnsonii* does not restore social function
Oxytocin levels in the paraventricular nuclei (PVN) elevated in MRD and *L. reuteri*-treated MHFD offspring

Figure 4. Buffington et al.

Number of PVN is not increased in MRD vs. MHFD, however oxytocin production is increased, and is stimulated by *L. reuteri* treatment
Recent Immunotherapy Advances

Gut microbiome modulates response to anti-PD-1 immunotherapy in melanoma patients

V. Gopalarathnam,1,2,* C. N. Spencer,2,3 L. Neal,1 A. Reuben,1 M. C. Andrews,1 T. V. Karpinets,1 P. A. Prieto,1 D. Vicente,1 K. Hoffman,1 S. C. Wei,1 A. P. Cogbill,1,5 L. Zhao,3 C. W. Hodgson,3 D. S. Hutchinson,1 T. Manaz,2 M. Petruza de Macedo,2 T. Cotechin,1 T. Kumar,1 W. S. Chen,2 S. M. Roddy,2 R. Szczepanski,2 S. W. Moane,3 J. Galloway-Pena,1 H. Jiang,1 P. L. Chen,2 E. J. Shipp,2 K. Rervani,2 A. M. Aloni,12 R. F. Chemaly,1,3 S. Sheshyur,1 L. M. Vance,1 P. C. Ohrnyson,1 V. E. Jensen,1 A. G. Sweeney,1 F. McCaillter,3 E. Marcello Riquelme Sanchez,1 Y. Zhang,1 E. Le Chatelier,1,2 L. Zitvogel,1,8 N. Pons,1 J. L. Austin-Breneman,11 L. E. Haydon,1 E. M. Burton,1 J. M. Gardner,1 E. Sirmans,1 J. Hu,1 A. J. Lazar,1 T. Tsimpalakas,1 A. E. Hesel,1 H. F. Tsiolkas,1 E. I. Gillis,3 W. J. Hwu,2 S. P. Patel,3 R. E. Woodman,3 R. N. Amarilla,1,3 A. M. Davies,1 J. E. Gershonfeld,3 P. Hwu,3 J. E. Lee,1 J. Zhang,1 L. M. Cousins,1 Z. A. Cooper,1 P. A. Futreal,1 C. R. Daniel,1 N. J. Ailman,1 J. F. Petrossian,1 M. T. Teitell,1 P. Sharma,3,6 J. F. Allison,1 R. R. Jenq,3 J. A. Warago1,6,12

RESEARCH

CANCER IMMUNOTHERAPY

The commensal microbiome is associated with anti-PD-1 efficacy in metastatic melanoma patients

Yuwa Matsuo,7 Jessica Feuling,8 Rhyne Bao,4,10 Tara Chongswat,1 Yanxuan Zha,4 Maria-Luisa Alegre,4 Jason J. Luke,3 Thomas F. Gajewski1

Anti-PD-1–based immunotherapy has had a major impact on cancer treatment but has only benefited a subset of patients. Among the variables that could contribute to interpatient heterogeneity is differential composition of the patients’ microbiome, which has been shown to affect tumor immunity and immunotherapy efficacy in preclinical mouse models. We analyzed baseline stool samples from metastatic melanoma patients before immunotherapy treatment, through an integration of 365 choanal RNA gene sequencing, metagenomic shotgun sequencing, and quantitative polymerase chain reaction for selected bacteria. A significant association was observed between commensal microbial composition and clinical response. Bacterial species more abundant in responders included Bifidobacterium longum, Collinsella aerofaciens, and Enterococcus faecium. Reconstitution of germ-free mice with fecal material from responding patients could lead to improved tumor control, augmented T-cell responses, and greater efficacy of anti-PD-1 therapy. Our results suggest that the commensal microbiome may have a mechanistic impact on antitumor immunity in human cancer patients.

RESEARCH

CANCER IMMUNOTHERAPY

Gut microbiome influences efficacy of PD-1–based immunotherapy against epithelial tumors

Bertrand Ronty,1,3,5 Emmanuelle Le Chatelier,4 Lisa Derron,1,3,5 Conde C. M. Desai,5,14 Maryam Tjdani Abou,1,4,8 Romain Daillies,5,14 Aurélie Fischinger,1,5,6 Meriem Messaoudenc,1,6 Conrad Buerki,3,4,5,6 Maria P. Robert,1,6,8 Marie Fléthe,1,5,6 Caroline Flament,1,5,6 Véronne Péliré-Culame,1,5,6 Paul Opolon,1 Christophe Klein,1 Kristina Iribarren,1,3,4,5,12 Laura Mondragón,1,4,5,12 Nicolas Jhonneau,1,8,9,10 So Qin,1,12 Gladys Ferreres,3,5,6 Céline Clémence,1,4,5,6 Laura Massquilla,5,6 Jördel Remon Masoty,1,5,6 Charles Nait,1,5,6,12 Solenn Brosseau,1,5,6,12 Couruche Kaderbhal,1,5,6 Corentin Richard,1,5 Ilhia Rizzi,1,5 Florence Levene,4 Nathalie Galleron,1,5 René Quéguiner,1,5,6,12 Nicolaas Pons,5 Bernard Ryffel1,6 Véronique Minard-Collin,1,6 Patrick Gomim,1,6 Jean-Charles Soria,1,6,12 Eric Deutsch,1,6,8 Yohanes Lelio,5,6,12 François Girgensons,5,6,12 Gérard Zalcman,1,6 François Goldwasser,1,6,8,12 Bernard Escobed1,6,8,12,15 Matthew O. Hellmann,9,20 Alexander Eggensperger,1,6,12,15 Dédé Rouault,30 Laurence Allégro,1,5,12GUIDO KROESWY,9,30,31,32,36,14,12,14 LAURENCE ZITKOVEN1,3,6,12
Microbiome alone can improve an immunotherapy response

N = 54 responders, 35 non-responders

Science 05 Jan 2018: Vol. 359, Issue 6371, pp. 32-34
Enteroids: a unique opportunity for studying human disease at the epithelial level

Mechanistic investigations of host-microbe interactions in the gut are limited because of two principle challenges:

1. Epithelium is oxygen dependent while many gut bacteria are facultative or obligate anaerobes

2. Intestinal epithelium is in a state of chronic low-grade hypoxia which is exacerbated in inflammatory conditions such as Inflammatory Bowel Disease (IBD).

OXYGEN CONTROL IS IMPORTANT!

Physiologic and inflammatory hypoxia in the gut

Physiologic Epithelium = 2-5% $O_2$ in vivo
Hypoxic Epithelium = 1-3% $O_2$ in vivo
Monolayers in Standard Incubator = 7-18% $O_2$
Assembly and validation of a novel enteroid-anaerobe co-culture system

Anaerobic Chamber

Blood Gas

Anaerobic

Aerobic

O₂

O₂

O₂

Gas Permeable

Well Bottom

Tatiana Fofanova

Chris Stewart

Jenny Auchtung
**Experimental Design**

3D Jejunal enteroids, p10-p15

Seeded $5 \times 10^5$ / transwell

4 days differentiation

Fitted into Co-Culture System
1-2hr equilibration at 5% Oxygen
$O_2$ measurements

$O_2$ measurements
Contamination checks at 0h, 24hr
Transepithelial electronic resistance (TEER) at 0hr, 24hr
Endpoint: histology, RNA, survival
System recapitulates *in vivo* oxygen gradients

**Steep Oxygen Gradient:**
- delivery of 5.6% and 10.2% O₂ blood gas,
- basolateral side is oxygenated while the apical side is effectively anaerobic.

**Healthy phenotype:**
Enteroids monolayers imaged after 24hrs at 5% basolateral oxygen are polarized (Villin stain in brown), show an intact mucus layer (Alcian blue stain) and normal morphology.
Impact of physiological levels of oxygen

Hallmark of Physiological Hypoxia: HIF1α

- HIF1α expression
  - Increased barrier integrity
  - Activation of NFκB and other innate immunity genes
  - Increased mucin production

Physiologic Hypoxia = 2-5% O₂ in vivo
Inflammatory Hypoxia = 1-3% O₂ in vivo
Monolayers in Standard Incubator = 8.5-10% O₂
Trans-epithelial resistance increases in response to hypoxia, independent of patient line

Hallmark of Physiological Hypoxia: Increased Barrier Integrity

24 hr incubation in 5% basolateral oxygen

Reduced Oxygen Conditions (5% oxygen)
Increase Epithelial Barrier Integrity

All J-lines
Gene expression under physiologic hypoxia

Hallmark of Physiological Hypoxia: Antimicrobial Response and Barrier Integrity

- RT2 Profiler Arrays: Tight Junction Array, Anti-Microbial Response Array
- 168 genes (84 per pathway)
- 106 retained in analysis after QC

Up-regulated  Down-regulated
## Pathways differentially regulated during physiologic hypoxia

<table>
<thead>
<tr>
<th>GO-ID</th>
<th>Description</th>
<th>Corrected P-Value</th>
<th>Cluster Frequency</th>
<th>Genes</th>
</tr>
</thead>
<tbody>
<tr>
<td>45429</td>
<td>Positive regulation of nitric oxide biosynthesis process</td>
<td>1.9214 e-8</td>
<td>6/33 (18.1%)</td>
<td>HSP90AA1 IL1B AKT1 TICAM1 TLR4 TLR2</td>
</tr>
<tr>
<td>43123</td>
<td>Positive regulation of I-kB kinase/NF-kB cascade</td>
<td>5.1691 e-8</td>
<td>8/33 (24.2%)</td>
<td>VAPA IL1B TLR6 TICAM1 TLR4 RELA RHOA MYD88</td>
</tr>
<tr>
<td>2221</td>
<td>Pattern recognition receptor signaling pathway</td>
<td>6.9208 e-8</td>
<td>5/33 (15.1%)</td>
<td>TLR6, TICAM, TLR4, RELA, TLR2</td>
</tr>
<tr>
<td>326755</td>
<td>Regulation of IL-6 production</td>
<td>6.9208 e-8</td>
<td>6/33 (18.1%)</td>
<td>IL1B, TLR6, TICAM, TLR4, MYD88, TLR2</td>
</tr>
<tr>
<td>10647</td>
<td>Positive regulation of cell communication</td>
<td>1.1126 E-7</td>
<td>11/33 (33.3%)</td>
<td>VAPA IL1B RAC1 TLR6 TICAM1 HIF1A TLR4 RHOA RELA MYD88 TLR2</td>
</tr>
<tr>
<td>6954</td>
<td>Inflammatory Response</td>
<td>1.1785 e-7</td>
<td>10/33 (30.3%)</td>
<td>CXCL8 IL1B AKT1 CXCL1 RAC1 TLR6 TICAM1 HIF1A TLR4 RELA</td>
</tr>
<tr>
<td>7163</td>
<td>Establishment or maintenance of cell polarity</td>
<td>3.195 e-6</td>
<td>5/33 (15.1%)</td>
<td>CDC42 PRKCI PARD3 LLGL1 MARK2</td>
</tr>
</tbody>
</table>

Generated via Cytoscape using the BINGO Plugin

*(BINGO: a Cytoscape plugin to assess overrepresentation of gene ontology categories in biological networks)*
Summary: System Design and Induction of Hypoxia

- Precision control of delivered basolateral oxygen
- System reaches steady-state within 2 hours
- Operating range is approximately between 1-8% Oxygen
- Induction of physiological hypoxia in enteroid monolayers
  - HIF1a Expression
  - Barrier Integrity
  - Upregulation of tight junction and anti-microbial response genes
- Gene expression profile reflects activation of
  - NO biosynthesis pathway as a regulator of hypoxia
  - NFkB cascade
## Can we add bacteria to the mix

<table>
<thead>
<tr>
<th><strong>Bacteroides thetaiotaomicron</strong></th>
<th><strong>Blautia sp.</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Bacteroidetes</td>
<td>• Firmicutes</td>
</tr>
<tr>
<td>• Gram-negative nanoanaerobe</td>
<td>• Gram-positive obligate anaerobe</td>
</tr>
<tr>
<td>• Common, abundant commensal</td>
<td>• Common, abundant commensal</td>
</tr>
<tr>
<td>• Acetate production</td>
<td>• Lactate and acetate production</td>
</tr>
</tbody>
</table>

Clinical implications:
Bacteroides thetaiotaomicron: Associated with remission in UC/Crohns
Blautia sp.: Reduced incidence of GvH disease

Griffiths, S. FST Journal, 2015
Experimental Design

3D Jejunal enteroids, p10-p15

Seeded 5x10^5 / transwell

4 days Differentiation

Fitted into Co-Culture System
1-2hr equilibration at 5% Oxygen
+ 3x10^4 bacteria in 300uL for 24hr

CFU counts at 0h, 8hr, 24hr
TEER at 0hr, 8hr, 24hr
Endpoint: histology, RNA, survival
System supports enteroid-anaerobe co-culture for at least 24 hours: *B. theta*

16S FISH Stain

![Images of 5% Oxygen - 24hr, + B. theta, 8hr, + B. theta, 24hr](image)

![Graph showing B. thetaiotaomicron Growth](graph)

N=12/group
Unique hypoxia/tight-junction related gene expression changes with bacterial co-culture.
### Pathways differentially regulated during physiologic *B. theta* co-culture

<table>
<thead>
<tr>
<th>GO-ID</th>
<th>Description</th>
<th>Corrected P-Value</th>
<th>Cluster Frequency</th>
<th>Genes</th>
</tr>
</thead>
<tbody>
<tr>
<td>42127</td>
<td>Regulation of Cell Proliferation</td>
<td>9.79E-03</td>
<td>7/31 22.5%</td>
<td>MAP2K1 CXCL8 RIPK2 IL18 CXCL1 TLR4 RELA</td>
</tr>
<tr>
<td>6954</td>
<td>Inflammatory Response</td>
<td>3.26E-05</td>
<td>8/31 25.8%</td>
<td>CXCL8 RIPK2 CXCL1 F11R CXCL2 TLR4 RELA NFKB1</td>
</tr>
<tr>
<td>44419</td>
<td>Interspecies Interaction Between Organisms</td>
<td>7.92E-04</td>
<td>6/31 19.3%</td>
<td>IRF7 F11R CLDN1 TLR4 RELA TLR2</td>
</tr>
<tr>
<td>2758</td>
<td>Innate Immune Response Activating Signal Transduction</td>
<td>1.86E-05</td>
<td>4/31 12.9%</td>
<td>RIPK2 TLR4 RELA TLR2</td>
</tr>
<tr>
<td>1819</td>
<td>Positive Regulation of Cytokine Production</td>
<td>1.86E-05</td>
<td>6/31 19.3%</td>
<td>PYCARD RIPK2 CASP1 IL18 TLR4 TLR2</td>
</tr>
<tr>
<td>51092</td>
<td>Positive Regulation of NFkb TF Activity</td>
<td>1.86E-05</td>
<td>5/31 16.1%</td>
<td>PYCARD RIPK2 TLR4 RELA TLR2</td>
</tr>
<tr>
<td>2237</td>
<td>Response To Molecule of Bacterial Origin</td>
<td>2.41E-04</td>
<td>5/31 16.1%</td>
<td>RIPK2 CASP1 TLR4 RELA TLR2</td>
</tr>
</tbody>
</table>
Summary: Enteroid-anaerobe co-culture

- Short term co-culture of commensal nano-anaerobe (B. theta) and obligate anaerobe (Blautia sp.) with enteroid monolayers permits/enables survival of both
- Co-culture decreases TEER in a patient-line specific manner
- B. theta growth is independent of enteroid line
- Blautia sp. growth is potentially affected by enteroid line (DNS)
- Gene expression profile, in response to B. theta co-culture, similar between patient lines
  - Reduced expression of some genes previously up-regulated in physiological-hypoxia
  - Expression profile corroborates interaction with molecules of bacterial origin
- Gene expression profile, in response to Blautia co-culture, varies between lines
The enteroid system may be used to:

- reveal mechanism in host microbe associations
- predict translatability of animal model studies
- Characterize the individual responses to potential microbiome therapeutic and diagnostic candidates
- Amenable to: RNAsseq, metabolomics, proteomics, single-cell sequencing
Moving toward…

Treatment A
Treatment B
Treatment C

Human Genome Metabolome Proteome Microbiome Clinical Data

Environmental Data

Enteroid Monolayer

Undifferentiated
Differentiated

(+ immune cells!)

(+ 40x)

A B C
Acknowledgements

Petrosino Laboratory--CMMR
Current Members
Kristi Hoffman
Melissa Mezzari
Tao Lin
Chunxu Gao
Tulin Ayvaz
Lorenzo D’Amico
Tatiana Fofanova
Jonathan Gesell
Michael Holder
Matthew Ross
Daniel Smith
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Matthew Wong
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Harsha Doddapaneni
Yi Han

MD Anderson Cancer Center
Jen Wargo
Rob Jenq