Targeting acquired resistance in oncology with purpose-built drugs

August 2022
FORWARD-LOOKING STATEMENTS AND MARKET DATA

We caution you that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, research and development plans, the anticipated timing, costs, design and conduct of our ongoing and planned preclinical studies and planned clinical trials for our product candidates, the timing and likelihood of regulatory filings and approvals for our product candidates, our ability to commercialize our product candidates, if approved, the impact of the COVID-19 pandemic on our business, the pricing and reimbursement of our product candidates, if approved, the potential to develop future product candidates, the timing and likelihood of success, plans and objectives of management for future operations, and future results of anticipated product development efforts, are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplates,” “believes,” “estimates,” “predicts,” “potential” or “continue” or the negative of these terms or other similar expressions. The inclusion of forward-looking statements should not be regarded as a representation by us that any of our plans will be achieved. Actual results may differ from those set forth in this presentation due to the risks and uncertainties inherent in our business, including, without limitation: we are early in our development efforts, have not tested any of our product candidates in clinical trials and the approach we are taking to discover and develop drugs based on our SNAP platform is novel and unproven and it may never lead to product candidates that are successful in clinical development or approved products of commercial value; potential delays in the commencement, enrollment, and completion of preclinical studies and clinical trials; our dependence on third parties in connection with manufacturing, research and preclinical testing; unexpected adverse side effects or inadequate efficacy of our product candidates that may limit their development, regulatory approval, and/or commercialization; unfavorable results from preclinical studies; results from preclinical studies or early clinical trials not necessarily being predictive of future results; our ability to maintain undisrupted business operations due to the COVID-19 pandemic, including delaying or disrupting our preclinical studies, manufacturing, and supply chain; regulatory developments in the United States and foreign countries; our ability to obtain and maintain intellectual property protection for our product candidates and proprietary technologies; we may use our capital resources sooner than we expect; and other risks described in our prior filings with the Securities and Exchange Commission (SEC), including under the heading “Risk Factors” in our annual report on Form 10-K and any subsequent filings with the SEC. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us.
Why invest in Tyra?

What we do

Next gen product candidates
• Acquired drug resistance
• Improved tolerability

How we do it

SNÅP CHEMISTRY DESIGN

What we’re developing

FGFR3–selective inhibitor, FGFR2, achondroplasia and other FGFR-3 related skeletal dysplasias, FGFR4-related cancers, and RET

<table>
<thead>
<tr>
<th>NASDAQ: TYRA</th>
</tr>
</thead>
<tbody>
<tr>
<td>CASH:* $275.1M</td>
</tr>
<tr>
<td>Avg. Qtr. Burn: $15–20M</td>
</tr>
<tr>
<td>Common Stock O/S:* 41.9M</td>
</tr>
<tr>
<td>Fully diluted:* 53.6M</td>
</tr>
</tbody>
</table>

*June 30, 2022
We develop next gen products

Our accelerated approach to design

Our differentiated candidates
We need next gen drugs in targeted oncology

FGFR2 Example

**FGFR2 Example**

Goyal et. al., Cancer Discov. 2017 Mar;7(3):252-263
Sequence and structure inform what is driving resistance

FGFR2 Example

Gatekeeper mutation

<table>
<thead>
<tr>
<th></th>
<th>pemigatinib</th>
<th>infigratinib</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( -\text{O} -\text{Cl} )</td>
<td>( -\text{O} -\text{F} )</td>
</tr>
<tr>
<td></td>
<td>( -\text{Cl} )</td>
<td>( -\text{F} )</td>
</tr>
<tr>
<td>erdafitinib</td>
<td>( -\text{O} )</td>
<td>( -\text{O} )</td>
</tr>
<tr>
<td></td>
<td>( -\text{Cl} )</td>
<td>( -\text{O} )</td>
</tr>
<tr>
<td>TAS120</td>
<td>( -\text{O} )</td>
<td>( -\text{O} )</td>
</tr>
</tbody>
</table>
Structural insights provide a rational path to address recurrence

EGFR Example

<table>
<thead>
<tr>
<th>Gefitinib</th>
<th>Gefitinib occluded</th>
<th>Osimertinib</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wild type EGFR protein</td>
<td>PFS: 10.2 months</td>
<td>PFS: 18.9 months</td>
</tr>
</tbody>
</table>

Gatekeeper mutation
Precedent shows next gen drugs extend progression free survival

Durability (months)

EGFR (T790<sup>GK</sup>)

- gefitinib (67<sup>1</sup> | PFS)
  - 10.9
- osimertinib (77<sup>2</sup> | PFS)
  - 18.9

ROS1 (G2032<sup>SF</sup>)

- entrectinib (78<sup>2</sup> | DOR)
  - 12
- repotrectinib (91<sup>1</sup> | DOR)
  - 23.1

ALK (L1196<sup>GK</sup>)

- crizotinib (76<sup>1</sup> | PFS)
  - 10.4
- alectinib (83<sup>1</sup> | PFS)
  - 25.7

RET (V804<sup>GK</sup>)

- cabozantinib (28<sup>2</sup> | PFS)
  - 5.5
- selpercatinib (64<sup>2</sup> | DOR)
  - 17.5

FGFR3 (V555<sup>GK</sup>)

- erdafitinib (32<sup>1</sup> | DOR)
  - 5.4

FGFR2 (V565<sup>GK</sup>)

- pemigatinib (36<sup>2</sup> | DOR)
  - 9.1

Information provided in the table above is for illustrative purposes only and is not a head-to-head comparison. Differences exist between study or trial designs and subject characteristics, and caution should be exercised when comparing data across studies.
Alterations in the FGFR family represent a large opportunity

7% of all cancers have FGFR aberrations

- Head and Neck: 15% FGFR1 A
- Non-Small Cell Lung: 20% FGFR1 A, 4% FGFR2 M
- Intrahepatic cholangiocarcinoma: 10-20% FGFR2 F
- Gastric: 5-10% FGFR2 A, 4% FGFR2 M
- Urothelial: 10-60% FGFR3 M, 7% FGFR1 A, 6% FGFR3 F
- Small Cell Lung: 6% FGFR1 A
- Breast: 7-23% FGFR1/2 A
- Endometrial: 12% FGFR2 M
- Cervical: 5% FGFR3 M
- Rhabdomyosarcoma: 7-8% FGFR4 M

A: Amplifications
F: Fusions
M: Mutations
Alterations in the FGFR family represent a large opportunity

Approved pan-FGFRi: pemigatinib, infigratinib

Acquired Resistance
• Gatekeeper mutation
• Molecular brake

Head and Neck
15% FGFR1 A

Non-Small Cell Lung
20% FGFR1 A
4% FGFR2 M

Small Cell Lung
6% FGFR1 A

Breast
7-23% FGFR1/2 A

Endometrial
12% FGFR2 M

Cervical
5% FGFR3 M

Rhabdomyosarcoma
7-8% FGFR4 M

Gastric
5-10% FGFR2 A
4% FGFR2 M

Urothelial
10-60% FGFR3 M
7% FGFR1 A
6% FGFR3 F

Intrahepatic cholangiocarcinoma
10-20% FGFR2 F

Approved pan-FGFRi: pemigatinib, infigratinib

Acquired Resistance
• Gatekeeper mutation
• Molecular brake

A Amplifications
F Fusions
M Mutations
Alterations in the FGFR family represent a large opportunity

Approved pan-FGFRi: erdafitinib
Acquired Resistance: gatekeeper mutation

Isoform Selectivity / Tox
- FGFR1
- FGFR2
- FGFR3
- FGFR4

Intrahepatic cholangiocarcinoma
10-20% FGFR2 F

Gastric
5-10% FGFR2 A
4% FGFR2 M

Urothelial
10-60% FGFR3 M
7% FGFR1 A
6% FGFR3 F

Head and Neck
15% FGFR1 A

Non-Small Cell Lung
20% FGFR1 A
4% FGFR2 M

Small Cell Lung
6% FGFR1 A

Breast
7-23% FGFR1/2 A

Endometrial
12% FGFR2 M

Cervical
5% FGFR3 M

Rhabdomyosarcoma
7-8% FGFR4 M

A Amplifications
F Fusions
M Mutations
We’re building a pipeline of differentiated assets

<table>
<thead>
<tr>
<th>Program</th>
<th>Resistance alteration</th>
<th>US incidence</th>
<th>Anticipated Milestone</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGFR3: TYRA-300</td>
<td>V555\textsuperscript{GK}</td>
<td>28-33K</td>
<td>Commence patient dosing</td>
</tr>
<tr>
<td>FGFR2: TYRA-200</td>
<td>V565\textsuperscript{GK} N550\textsuperscript{MB}</td>
<td>3.5K</td>
<td>File IND 2H-2022</td>
</tr>
<tr>
<td>FGFR3 (ACH)</td>
<td>G380R\textsuperscript{1}</td>
<td>8-22K\textsuperscript{2}</td>
<td>Nominate lead candidate</td>
</tr>
<tr>
<td>FGFR4</td>
<td>V550\textsuperscript{GK} C552\textsuperscript{CYS}</td>
<td>2K</td>
<td>Nominate lead candidate</td>
</tr>
<tr>
<td>RET</td>
<td>V804\textsuperscript{GK} G810\textsuperscript{SF}</td>
<td>2-6K</td>
<td>Nominate lead candidate</td>
</tr>
</tbody>
</table>

\textsuperscript{1} Key activating mutation for ACH. \textsuperscript{2} Number represents US prevalence rather than incidence.
We develop next gen products

Our accelerated approach to design

Our differentiated candidates
Our unconventional approach accelerates discovery

Asynchronous design cycle takes several weeks

**DESIGN CYCLE**
- Chemistry design
- Crystallography
- Cell-based assays
- In vivo models

**CHEMISTRY DESIGN**
- Crystallography
- Cell-based assays
- In vivo models
We’ve optimized the drug design cycle in-house

- **CRYSTALLOGRAPHY**
  - New compound to structure in as little as 3 days

- **CELL-BASED ASSAYS**
  - New compound to cellular data in as little as 2 days

- **TUMOR MODELS**
  - New compound to TGI data in as little as 5 days
We develop next gen products

Our accelerated approach to design

Our differentiated assets
TYRA-300

FGFR3-selective inhibitor
Our lead candidate, TYRA-300, is an FGFR3-selective inhibitor
TYRA-300 retains potency in a FGFR3:TACC3-V555M cell line

<table>
<thead>
<tr>
<th>Inhibitor</th>
<th>RT112/84 IC\textsubscript{50} (nM)</th>
<th>Fold Difference from RT112/84 FGFR3:TACC3 in IC\textsubscript{50}</th>
</tr>
</thead>
<tbody>
<tr>
<td>erdafitinib</td>
<td>4.4</td>
<td>7.9</td>
</tr>
<tr>
<td>pemigatinib</td>
<td>5.3</td>
<td>14.5</td>
</tr>
<tr>
<td>futibatinib</td>
<td>11.0</td>
<td>19</td>
</tr>
<tr>
<td>infigratinib</td>
<td>14.5</td>
<td></td>
</tr>
<tr>
<td>TYRA-300</td>
<td>7.9</td>
<td></td>
</tr>
</tbody>
</table>
TYRA-300 is active \textit{in vivo} in bladder cancer models

**Bladder Cancer Xenograft $FGFR3:TACC3$**

- Vehicle
- TYRA-300 (12.5 mg/kg BID) 95% TGI

**Bladder Cancer Xenograft $FGFR3:TACC3-V555M$**

- Vehicle
- TYRA-300 (12.5 mg/kg BID) 77% TGI

- Vehicle
- erdafitinib (12.5 mg/kg BID) 73% TGI

- Vehicle
- erdafitinib (12.5 mg/kg BID) 12% TGI
We designed TYRA-300 to be FGFR3 selective

**FGFR isoform selectivity**

Subtle, but *distinct* differences at key interaction zones along the pocket walls

MOLECULAR MODEL

3.0Å cross-sections

CRystallography

FGFR3 specific inhibitor
TYRA-300 has shown selectivity for FGFR3 over other kinases

TYRA-300 was profiled in a scanMAX\textsuperscript{SM} (KINOMEscan) screen, IC\textsubscript{50} data generated by Reaction Biology Inc.

<table>
<thead>
<tr>
<th>Kinase</th>
<th>TYRA-300</th>
<th>FGFR3 selectivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGFR3</td>
<td>1.6</td>
<td>1.0x</td>
</tr>
<tr>
<td>FLT4</td>
<td>2.1</td>
<td>1.3x</td>
</tr>
<tr>
<td>FGFR2</td>
<td>6.5</td>
<td>4.0x</td>
</tr>
<tr>
<td>FGFR4</td>
<td>11.0</td>
<td>6.9x</td>
</tr>
<tr>
<td>JAK2</td>
<td>35.5</td>
<td>22x</td>
</tr>
<tr>
<td>LTK</td>
<td>65.1</td>
<td>41x</td>
</tr>
<tr>
<td>FGFR1</td>
<td>108</td>
<td>68x</td>
</tr>
<tr>
<td>FLT1</td>
<td>201</td>
<td>126x</td>
</tr>
<tr>
<td>JAK3</td>
<td>206</td>
<td>129x</td>
</tr>
</tbody>
</table>
TYRA-300 has shown selectivity for FGFR3 over other isoforms

TYRA-300 selectivity vs. approved or late-stage clinical compounds: Ba/F3 Cellular IC$_{50}$ (nM)

<table>
<thead>
<tr>
<th>FGFR</th>
<th>erdafitinib</th>
<th>futibatinib</th>
<th>pemigatinib</th>
<th>infigratinib</th>
<th>TYRA-300</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGFR1</td>
<td>5.5</td>
<td>3.9</td>
<td>12.3</td>
<td>15.3</td>
<td>113</td>
</tr>
<tr>
<td>FGFR2</td>
<td>1.8</td>
<td>1.0</td>
<td>4.3</td>
<td>5.8</td>
<td>34.9</td>
</tr>
<tr>
<td>FGFR3</td>
<td>1.3</td>
<td>0.8</td>
<td>5.2</td>
<td>6.9</td>
<td>1.8</td>
</tr>
<tr>
<td>FGFR4</td>
<td>17.7</td>
<td>6.1</td>
<td>142</td>
<td>459</td>
<td>98.4</td>
</tr>
</tbody>
</table>

Fold Selectivity for FGFR3

<table>
<thead>
<tr>
<th>FGFR</th>
<th>erdafitinib</th>
<th>futibatinib</th>
<th>pemigatinib</th>
<th>infigratinib</th>
<th>TYRA-300</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGFR1</td>
<td>4.2x</td>
<td>4.9x</td>
<td>2.4x</td>
<td>2.2x</td>
<td>63x</td>
</tr>
<tr>
<td>FGFR2</td>
<td>1.4x</td>
<td>1.3x</td>
<td>0.8x</td>
<td>0.8x</td>
<td>19x</td>
</tr>
<tr>
<td>FGFR4</td>
<td>14x</td>
<td>7.6x</td>
<td>27x</td>
<td>67x</td>
<td>55x</td>
</tr>
</tbody>
</table>

TYRA-300 shows significant isoform selectivity for FGFR3 over other FGFR isoforms.
TYRA-300 did not elevate phosphate relative to erdafitinib

Rat plasma phosphate at 24 hours after single dose

1. N=4 per group, pooled rat plasma; dotted line = pre-dose phosphate value of 3 dose groups
TYRA-300 is active \textit{in vivo} in bladder cancer models

Bladder Cancer Xenograft UM-UC-14 ($\text{FGFR3}^{S249C}$)

- Vehicle (30% HP-β-CD)
- TYRA-300 (9 mg/kg BID) – 90% TGI, 34% Regression
- Erdafitinib (12.5 mg/kg BID) – 91% TGI, 41% Regression
- TYRA-300 (18 mg/kg QD) – 96% TGI, 75% Regression
Our FGFR3 program addresses important, unmet needs

<table>
<thead>
<tr>
<th>FGFR Resistant&lt;sup&gt;1&lt;/sup&gt; includes V555&lt;sup&gt;GK&lt;/sup&gt;</th>
<th>1K</th>
<th>Locally advanced/metastatic urothelial cancer (UC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGFR Naïve&lt;sup&gt;1&lt;/sup&gt;</td>
<td>4K</td>
<td>Locally advanced/metastatic UC</td>
</tr>
<tr>
<td></td>
<td>5K</td>
<td>Tumor agnostic</td>
</tr>
<tr>
<td></td>
<td>5K</td>
<td>Localized MIBC</td>
</tr>
<tr>
<td></td>
<td>14-19K</td>
<td>Recurrent Non-MIBC</td>
</tr>
<tr>
<td><strong>Driver mutations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>S249C, R248C, Y373C, G370C, FGFR3-TACC3 fusion</td>
</tr>
</tbody>
</table>

1. Population sizes reflect US incidence estimates

**CDx**
- Liquid Biopsy
- NGS
- Fusion detection
Precedent paves a potential path for rapid development

Recently approved targeted oncology drugs

<table>
<thead>
<tr>
<th>Months to approval</th>
<th>N</th>
<th>Target</th>
<th>TYRA targets</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 20 40 60</td>
<td>187</td>
<td>RET</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>36</td>
<td>RET</td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>97</td>
<td>RET</td>
<td></td>
</tr>
<tr>
<td>87</td>
<td>107</td>
<td>FGFR</td>
<td></td>
</tr>
<tr>
<td>114</td>
<td>55</td>
<td>NTRK</td>
<td></td>
</tr>
<tr>
<td>127</td>
<td>43</td>
<td>PDGFRA</td>
<td></td>
</tr>
<tr>
<td>240</td>
<td>54</td>
<td>PDGFRA</td>
<td></td>
</tr>
<tr>
<td>257</td>
<td>58</td>
<td>MET</td>
<td></td>
</tr>
<tr>
<td>264</td>
<td>58</td>
<td>MET</td>
<td></td>
</tr>
<tr>
<td>290</td>
<td>60</td>
<td>MET</td>
<td></td>
</tr>
<tr>
<td>308</td>
<td>51</td>
<td>ROS1</td>
<td></td>
</tr>
<tr>
<td>312</td>
<td>51</td>
<td>ROS1</td>
<td></td>
</tr>
</tbody>
</table>

ENABLERS

Fast Track
Accelerate Approval
Breakthrough Therapy
Priority Review
Phase 1 design determines recommended phase 2 dose (RP2D)

Phase 1 Part A: What is the MTD?
Phase 1 Part B: What is the optimal dose?
TYRA-200

FGFR2 Inhibitor
Polyclonal acquired drug resistance occurs often in FGFR2

72% of patients showed clinical benefit

67% Had on-target resistance

MUTATION FREQUENCY

<table>
<thead>
<tr>
<th>Mutation Type</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular Brake</td>
<td>E566 17%</td>
</tr>
<tr>
<td></td>
<td>K642 4%</td>
</tr>
<tr>
<td>Gatekeeper</td>
<td>V565 57%</td>
</tr>
<tr>
<td>DFG Latch</td>
<td>L618 13%</td>
</tr>
<tr>
<td>A-loop Activator</td>
<td>K660 9%</td>
</tr>
<tr>
<td>αC Tether</td>
<td>M538 4%</td>
</tr>
<tr>
<td>P-Loop Cys</td>
<td>C492 4%</td>
</tr>
</tbody>
</table>

Data presented at EORTC (October 2020); N=46. Evaluable FGFR2-fusion Patients (ICC) on FGFR therapy w/ post-progression biopsy
TYRA-200 is potent across known FGFR2 resistance mutations


Enzymatic IC_{50} measurements generated at Reaction Biology Corp using Tyra enzymes. All experiments conducted under identical conditions, tested in duplicate on the same day.
Acquired resistance mutations alter FGFR2 protein structure

Molecular Brake and Gatekeeper mutation crystal structures reveal dynamic protein movement

Adaptable binding mode of TYRA-200
TYRA-200 maintains potency for mutations in cell-based assays

<table>
<thead>
<tr>
<th>FGFR2: Ba/F3 cell line</th>
<th>IC_{50} (nM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>wild type (WT)</td>
<td></td>
</tr>
<tr>
<td>pemigatinib</td>
<td>2.4</td>
</tr>
<tr>
<td>infigratinib</td>
<td>9.5</td>
</tr>
<tr>
<td>futibatinib</td>
<td>1.7</td>
</tr>
<tr>
<td>erdafitinib</td>
<td>7.5</td>
</tr>
<tr>
<td>TYRA-200</td>
<td>8.4</td>
</tr>
</tbody>
</table>

Fold Difference from WT FGFR2 for variants: Clinical fusion 1, Clinical fusion 2, N550K, V565F, V565I, K660E, K660N

All experiments conducted in identical conditions, tested same day, in duplicate.
TYRA-200 is active *in vivo* against gatekeeper mutation V565F.
TYRA-200 shows high selectivity for FGFR family, sparing R4

<table>
<thead>
<tr>
<th>IC$_{50}$ (nM)</th>
<th>TYRA-200</th>
<th>FGFR2 selectivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1</td>
<td>0.47</td>
<td>1.0x</td>
</tr>
<tr>
<td>1-5</td>
<td>0.66</td>
<td>1.4x</td>
</tr>
<tr>
<td>6-10</td>
<td>1.8</td>
<td>3.8x</td>
</tr>
<tr>
<td>11-50</td>
<td>30.5</td>
<td>65x</td>
</tr>
<tr>
<td></td>
<td>35.6</td>
<td>76x</td>
</tr>
</tbody>
</table>

TYRA-200 was profiled in a scanMAX$^\text{SM}$ (KINOMEscan) screen, IC$_{50}$ data generated by Reaction Biology Inc.
TYRA-200 maintains selectivity over R4 in cell-based assays

TYRA-200 selectivity vs. futibatinib: Ba/F3 Cellular IC$_{50}$ (nM)

<table>
<thead>
<tr>
<th></th>
<th>futibatinib</th>
<th>TYRA-200</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGFR1</td>
<td>2.7</td>
<td>17.2</td>
</tr>
<tr>
<td>FGFR2</td>
<td>1.7</td>
<td>8.4</td>
</tr>
<tr>
<td>FGFR3</td>
<td>0.5</td>
<td>1.2</td>
</tr>
<tr>
<td>FGFR4</td>
<td>9.9</td>
<td>151.6</td>
</tr>
</tbody>
</table>

Fold Selectivity for FGFR2

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>FGFR4</td>
<td>6x</td>
<td>18x</td>
</tr>
</tbody>
</table>

TYRA-200 shows isoform selectivity for FGFR2 over FGFR4
TYRA-200 is active \textit{in vivo} against molecular brake N550K.
Our FGFR2 program addresses important, unmet needs

<table>
<thead>
<tr>
<th>FGFR Resistant&lt;sup&gt;1&lt;/sup&gt;</th>
<th>FGFR Naïve&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Driver mutations</th>
</tr>
</thead>
<tbody>
<tr>
<td>includes V565&lt;sup&gt;gK&lt;/sup&gt; and N550&lt;sup&gt;MB&lt;/sup&gt;</td>
<td>0.6K</td>
<td>Fusion+, N550&lt;sup&gt;MB&lt;/sup&gt;, K650E, S252W, Y375C, C382R</td>
</tr>
<tr>
<td>FGFR Naïve&lt;sup&gt;1&lt;/sup&gt;</td>
<td>1.1K</td>
<td>1.1K</td>
</tr>
<tr>
<td></td>
<td>1.4K</td>
<td>ICC</td>
</tr>
<tr>
<td></td>
<td>1.0K</td>
<td>Uterine/Endometrial&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tumor Agnostic&lt;sup&gt;3&lt;/sup&gt; including Melanoma, NSCLC, CRC, Breast, Bladder, Ovarian, Gastro-esophageal</td>
</tr>
</tbody>
</table>

1. Population sizes reflect US incidence estimates
2. Includes newly diagnosed, metastatic, FGFR2+ cases (500 annually) in addition to 10-15% of Stage 2 and 3 patients who will recur with metastatic disease and/or peritoneal carcinomatosis within 3 years of treatment, representing up to an additional 2700 cases (approximately 900 annual cases of recurrent FGFR2+ endometrial cancer).
3. Estimates based on annual deaths to represent new and recurrent cases in each tumor indication listed and percentage of oncogenic/likely oncogenic FGFR2 mutations/fusions in Project Genie.
We’re building a pipeline of differentiated assets

<table>
<thead>
<tr>
<th>Program</th>
<th>Resistance alteration</th>
<th>US incidence</th>
<th>Anticipated Milestone</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGFR3: TYRA-300</td>
<td>V555&lt;sup&gt;GK&lt;/sup&gt;</td>
<td>28-33K</td>
<td>Commence patient dosing</td>
</tr>
<tr>
<td>FGFR2: TYRA-200</td>
<td>V565&lt;sup&gt;GK&lt;/sup&gt; N550&lt;sup&gt;MB&lt;/sup&gt;</td>
<td>3.5K</td>
<td>File IND 2H-2022</td>
</tr>
<tr>
<td>FGFR3 (ACH)</td>
<td>G380R&lt;sup&gt;1&lt;/sup&gt;</td>
<td>8-22K&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Nominate lead candidate</td>
</tr>
<tr>
<td>FGFR4</td>
<td>V550&lt;sup&gt;GK&lt;/sup&gt; C552&lt;sup&gt;CYS&lt;/sup&gt;</td>
<td>2K</td>
<td>Nominate lead candidate</td>
</tr>
<tr>
<td>RET</td>
<td>V804&lt;sup&gt;GK&lt;/sup&gt; G810&lt;sup&gt;SF&lt;/sup&gt;</td>
<td>2-6K</td>
<td>Nominate lead candidate</td>
</tr>
</tbody>
</table>

Program: FGFR3, FGFR2, FGFR4, RET  
Mutations: V555<sup>GK</sup>, V565<sup>GK</sup>, N550<sup>MB</sup>, G380R<sup>1</sup>, V550<sup>GK</sup>, C552<sup>CYS</sup>, V804<sup>GK</sup>, G810<sup>SF</sup>  
US incidence: 28-33K, 3.5K, 8-22K<sup>2</sup>, 2K, 2-6K  
Key activating mutation for ACH  
1. Number represents US prevalence rather than incidence

**Anticipated Milestone**
- Commence patient dosing
- File IND 2H-2022
- Nominate lead candidate
- Nominate lead candidate

**Discovery**
- FGFR3: TYRA-300
- FGFR2: TYRA-200
- FGFR3 (ACH)
- FGFR4
- RET

**IND-Enabling**
- FGFR3: TYRA-300
- FGFR2: TYRA-200
- FGFR3 (ACH)
- FGFR4
- RET

**Phase 1**
- FGFR3: TYRA-300
- FGFR2: TYRA-200
- FGFR3 (ACH)
- FGFR4
- RET

**Phase 2**
- FGFR3: TYRA-300
- FGFR2: TYRA-200
- FGFR3 (ACH)
- FGFR4
- RET

**Phase 3**
- FGFR3: TYRA-300
- FGFR2: TYRA-200
- FGFR3 (ACH)
- FGFR4
- RET

IND cleared

**SNAP**
CHEMISTRY  
DESIGN

ACH: Achondroplasia, GK: Gatekeeper, CYS: Cysteine Mutant, SF: Solvent Front, MB: Molecular Brake

1. Key activating mutation for ACH  
2. Number represents US prevalence rather than incidence

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Why invest in Tyra?

What we do
Next gen product candidates
• Acquired drug resistance
• Improved tolerability

How we do it
SNÅP CHEMISTRY DESIGN

What we’re developing
FGFR3-selective inhibitor, FGFR2, achondroplasia and other FGFR-3 related skeletal dysplasias, FGFR4-related cancers, and RET

<table>
<thead>
<tr>
<th>NASDAQ: TYRA</th>
</tr>
</thead>
<tbody>
<tr>
<td>CASH:* $275.1M</td>
</tr>
<tr>
<td>Avg. Qtr. Burn: $15–20M</td>
</tr>
<tr>
<td>Common Stock O/S:* 41.9M</td>
</tr>
<tr>
<td>Fully diluted:* 53.6M</td>
</tr>
</tbody>
</table>

*June 30, 2022