CM-352: a new, potent and safe molecule for the prevention and treatment of haemorrhage
Outline

• Institution: CIMA

• Project

• Partnering Opportunities
The Center for Applied Medical Research (CIMA) is a private non-profit biomedical research institution of the University of Navarra, based in Pamplona, Spain.

CIMA carries out high quality scientific work with a strong translational focus.
CIMA. De-risking Drug Discovery Process

• Target Rich
  - BioBank
  - Patient Data

• Translational Medicine
  Bidirectional data analysis to identify and/or prioritize clinically relevant molecular targets or pathways.

• Basic Science
  Advanced basic research to decipher MoA underlying clinical evidence.
  Implementation of in-vitro or/and in-vivo assays for unequivocal assessment: PoC

• Drug Discovery
  Proprietary tool(s), biologics or/and small molecules, for in-vivo PoC: efficacy & safety

CUN
Medical Center

CIMA
Translating Basic Science

Target ID and Validation
Basic Biological Research
- siRNA, MoA, KO mice, peptides, …
- Cellular & Animal Models

Drug Discovery

Gene Therapy
Molecular Therapeutics

Drug Discovery & Development
Clinical
FDA

Pharmaceutical Industry

Novel Antifibrinolytic Agents

XIV Encuentro de Cooperación FarmaIndustria
Madrid, November 2015
CIMA. De-risking Drug Discovery Process

• Expected Deliverables:
  i. Novel Target / MoA
  ii. In-vitro & In-vivo PoC with “drug-like” molecules or biologics: Efficacy & Safety → Advanced Lead(s)
  iii. Lead(s) with proprietary IP (Availability for further development)
  iv. “Know-how”
# Projects Overview

<table>
<thead>
<tr>
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**IP & “validated” targets**
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<td>C</td>
<td>Anti-neoplastic</td>
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<td>Assay established</td>
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<td>Immune regulation</td>
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<td>E</td>
<td>Huntington</td>
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<td>Assay to be defined</td>
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**IP & “validated” targets**
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Antihemorrhagic Agents: Current Standard of Care

**HAEMORRHAGE**
- Bleeding is a common complication in **surgical** (2.5 to 3.5 % of 100-120 million major surgeries every year in the 7MM) and **trauma patients** (50% deaths occurring within 24 h).

**STANDARD OF CARE**
- Lysine analogs (indirect inhibitors of fibrinolysis), such as **tranexamic acid** (TXA) reduces surgical bleeding and blood transfusion by about one third.
- **Aprotinin** (direct inhibitor of fibrinolysis) - withdrawn in 2008 due to cardiovascular side effects and increased mortality (BART study). *EMA recommendation (2012), suspension be lifted for a restricted range of indications*

**SHORTCOMINGS**
- Data suggest that TXA might be less effective than aprotinin in reducing blood loss.
- Allogenic transfusion risk is 23% increased in TXA when compared to Aprotinin.
- TXA side effects include seizures, renal impairment and thromboembolic complications

**UNMET NEED**
- Medical need for effective and safer agents to manage patients with major bleeding
- Intracranial hemorrhage (ICH); an important orphan indication
Antihemorrhagic Agents: Novel Approach

AIM

- Effective agents with impact on fibrinolytic function - *no involvement in hemostasis*
**Antihemorrhagic Agents: Novel Approach**

**AIM**
- Effective agents with impact on fibrinolytic function - *no involvement in hemostasis*

**APPROACH**
- Target identification from array analyses (Affymetrix®) – human cells: **MMP3 & MMP10**
  - MMP10 knock-out mice show the desired biological response
  - MMP10 *hMAb* shows the desired *in-vitro* functional response
  - Pharmacological tool compound identified (dual inhibitor: MMP10 & MMP3); PoC for *in-vivo* validation

- Knowledge based *de-novo* design, and synthesis
  - Workflow towards Lead ID
    - Synthesis
    - *In-vitro* binding assay
    - *In-vitro* functional assay; (Thromboelastometry) **decision point**
    - ADME profiling
    - Toxicology (Anatomopathological study @ 10x efficacious dose)
    - *In-vivo* efficacy model (tail bleeding)

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**cima**

Novel Antifibrinolytic Agents

XIV Encuentro de Cooperación FarmaIndustria
Madrid, November 2015
Hit to Lead: *In-vitro* binding assay

- 112 new proprietary compounds synthesized; all diversity points explored
- Biochemical assays vs MMP10 & MMP3
Lead ID: *In-vitro* functional assay

- 112 new proprietary compounds synthesized; all diversity points explored
- Biochemical assays vs MMP10 & MMP3
Optimized Lead: CM-352

- Multifactorial optimization process led to CM-352
- *In-Vitro* efficacy model: Thromboelastometry (*human whole blood*)

CM-352 is able to reduce by 50% lysis time at sub-nanomolar concentration.
<table>
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<tr>
<th><strong>Efficacy</strong></th>
<th>Binding affinities (MMP10 &amp; MMP3):</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Delay in Lysis Time (functional assay in human blood)</td>
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<tr>
<td></td>
<td>IC$_{50}$: 12nM &amp; 15nM</td>
</tr>
<tr>
<td></td>
<td>EC$_{50}$: 0.7nM</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>ADME</strong></th>
<th>P450s: 1A2, 2C19, 2C9, 2D6, 3A4 (&lt;50% @ 10µM)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>hPXR (EC$_{50}$ &gt; 100µM)</td>
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<tr>
<td></td>
<td>Plasma Protein Binding (% unbound)</td>
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<tr>
<td></td>
<td>Solubility (&gt;100 µg/mL)</td>
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<td></td>
<td>Caco-2 (Pe 10$^{-6}$ in cm/s) &amp; Efflux Ratio</td>
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<tr>
<td></td>
<td>Liver Microsomal Stability (t$_{1/2}$ estimation) <em>in minutes</em></td>
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<tr>
<td></td>
<td>S9 Stability (t$_{1/2}$ estimation) <em>in minutes</em></td>
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<tr>
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<td>&gt;145(H), &gt;145(M), &gt;145(R)</td>
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<tr>
<td></td>
<td>hERG binding (IC$_{50}$ &gt;100 µM)</td>
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<tr>
<td></td>
<td>Patch Clamp (IC$_{50}$ &gt;30 µM)</td>
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<tr>
<td></td>
<td>Mini Ames (<em>in 2 strains</em>)</td>
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<tr>
<td></td>
<td>THLE &amp; PBMC (LC$_{50}$ &gt; 100µM)</td>
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<tr>
<td></td>
<td>Anatomopathological analysis (@ 10 mg/kg)</td>
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<tr>
<td></td>
<td>Acute Toxicity</td>
</tr>
<tr>
<td></td>
<td>LD$_{50}$: 100 mg/Kg</td>
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<tr>
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<td>No alteration observed (<em>lung, brain, kidney &amp; liver</em>)</td>
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<tr>
<th><strong>Toxicity</strong></th>
<th>Pharmacokinetics (Vss, t$<em>{1/2}$ &amp; C$</em>{max}$) @ 1 mg/Kg <em>in mice</em> (i.v.)</th>
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<td>Brain tissue/Plasma Ratio @ T$_{max}$ from 1 mg/Kg <em>in mice</em> (i.v.)</td>
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<tr>
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<td>0.94 (L/Kg), 1.4 (h), 3.3 (µM)</td>
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<td>1.1 % (34 nM)</td>
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| **PK** | CM-352 |
**Lead Profiling (II)**

### MoA & off-target Selectivity

<table>
<thead>
<tr>
<th>Isoforms selectivity</th>
<th>Binding affinities vs 9 additional MMP isoforms (&gt;50% @ 10μM)</th>
<th>All isoforms (in fact, &gt;70%)</th>
</tr>
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### MoA & Hemost. & Fibrin.:

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<th>Binding affinities vs 89 add. targets bearing metal binding sites (≤ 50% @ 10μM)</th>
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<tr>
<td>Fibrinolysis</td>
<td>tPA, uPA, plasmin, PAI1, … @ 10μM</td>
<td>Inactive*</td>
</tr>
<tr>
<td>Primary Hemostasis</td>
<td>Platelet aggregation @ 10μM</td>
<td>Inactive*</td>
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<tr>
<td>Sec. Hemostasis</td>
<td>KLK-1, Factor X, … @ 10μM</td>
<td>Inactive*</td>
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<tr>
<td>Additional</td>
<td>Fibrinogen, TAFI, APC, … @ 10μM</td>
<td>Inactive*</td>
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### Off-Target

<p>| | |</p>
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<td>Binding affinities vs 89 add. targets bearing metal binding sites (≤ 50% @ 10μM)</td>
<td>✓</td>
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**Conclusion:** Effective antifibrinolytic agent; and, no impact on hemostasis

*No impact on hemostatic parameters; and, no coagulopathy induction*
Optimized Lead: CM-352

- **In-Vivo efficacy** model: Hyperfibrinolytic Tail Bleeding

\[ \text{CM-352 reduces bleeding time by } >89\% \text{ at } 10\mu g/kg \text{ in hyperfibrinolytic induced conditions} \]
Optimized Lead: CM-352

- \textit{In-Vivo} efficacy model: Hepatectomy Bleeding

Only CM-352 is effective at reducing blood loss in an aggressive bleeding model.
**Optimized Lead: CM-352**

- **In-Vivo efficacy model**: Intracranial hemorrhage model – an important unmet need

CM-352 is effective at reducing hematoma and lesion volume in intracranial hemorrhage.

- **Magnetic Resonance Imaging (MRI)**
  - Hematoma volume:
    - i. ~30% reduction in 3 hours
    - ii. ~45% reduction in 24 hours

- **Neurological recovery**
  - Bederson scale
    - Baseline
    - 24 h
    - day 14

**CM-352** is effective at reducing hematoma and lesion volume in intracranial hemorrhage.
Optimized Lead: CM-352

- **In-Vivo efficacy** model: **Antidote for Rivaroxaban** *(new generation of anticoagulants)*

---

**CM-352** is effective at reducing bleeding time after Rivaroxaban treatment

*Where DOAC means new generation of anticoagulants: Dabigatran and Rivaroxaban*
Small Molecules as novel antifibrinolytic agents

**AIM**
- Effective agents with impact on fibrinolytic function - no involvement in hemostasis

**APPROACH**

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  - In-vivo efficacy model (tail bleeding)

**ACHIEVEMENT**
- Proprietary chemical series, IP – patent applications filed: WO2014012964 & WO2015104343
- Preclinical Candidate, CM-352, showing optimal profiling:
  - Efficacy (4 in-vivo models), ADME, cardiovascular safety, Toxicity, PK, off-target selectivity, ….
• Critical point is currently **on-going, looking for investment** to move to:

  i.- IMPD-enabling studies *(based on EMA feedback)*

  ii.- Phase I
Outline

• Institution: CIMA

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• Partnering Opportunities
Value proposition

- Proprietary small molecules with in vivo proof of concept; e.g. optimized Lead Compound, CM-352:
  - ~4 times more efficacious at doses up to 30,000 times lower than TXA.
  - ~4 times more efficacious at doses up to 1,000 times lower than Aprotinin.
- Efficacious in aggressive bleeding model (hepatectomy). Aprotinin and TXA do not stop bleeding.
- Efficacious in ICH model (subarachnoid hemorrhage) → orphan indication (speeding up the process)
- Efficacious as antidote for new generation anticoagulant agent (Rivaroxaban; targeting FXa)
- Have no impact on hemostasis
- No thrombus formation
- Optimal ADME, off-target selectivity and PK profile; e.g. $t_{1/2}$ is 1.4 hours (optimal for acute treatment)
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Differentiation And Market

- Novel MoA and novel “Markush” formulas
- Potential to recover Aprotinin market niche ($600 Million) – major surgery
- Life plan also involves: first-aid/trauma & ICH (orphan indication to speed up the process)
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- Project at preclinical stage with follow-on products:
  - Target-based approach
  - Phenotypic-based discovery

- Time to IND estimated ~12 to 18 months
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  - 2 patents cover different novel “Markush” formulas (WO2014012964 and WO2015104343)
  - 1 patent claims known MMP inhibitors – acute treatment (WO2015107139)
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CM-352 first line of therapy to treat blood loss in major surgery, trauma and first-aid as well as ICH
• Partnering

Two scenarios are initially envisioned:

1. - Product license (IP)

2. - Stepwise research investment & first option (right of first refusal)
Acknowledgements

Jesús Hernández, PhD

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- J. Castillo, PhD

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