Advancing innovation towards breakthrough cancer therapies

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October 2, 2017
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INVESTMENT CASE
From cutting-edge science to breakthrough products

Today’s Science.
Tomorrow’s Cancer Treatments

**Proprietary Technologies**
with unique mechanisms of action in DNA-targeting with extended IP protection

**Translational Expertise**
to transform scientific opportunities into breakthrough treatments

**Oncology Experience**
& collaborations with 1st tier academic institutions

**Development**
of innovative agents in monotherapies as well as in combinations

Developing breakthrough DNA-targeting therapies for unmet cancer treatment needs
Growth value strategy

- **Build a diversified pipeline with first in class innovative programs**
  - 2 innovative programs, DBRi and Epigenetic, two of the most sought for classes
  - A unique chemistry platform generating new DNA-targeting compounds

- **Create multiple path to success through development in combo or monotherapies**

- **Leverage products value to PoC/ optimal inflexion points, compelling for pharma partners**

- **Capitalize on solid in/out licensing expertise to execute value creation transactions and enrich pipeline**

October 2017
DNA Break repair and epigenetics: 2 of the most sought-after MoAs

IO, DNA Break repair, Epigenetics & KI generate the highest deal values

Onxeo’s analysis based on Clarivate Cortellis (only licensing deals with publicly available financial information - 2014-2017):
https://www.cortellis.com/intelligence/
## Onxeo R&D Pipeline: Committed to advance leading-edge programs

<table>
<thead>
<tr>
<th>Program</th>
<th>INDICATION</th>
<th>PRECLINICAL</th>
<th>PHASE I</th>
<th>PHASE II</th>
<th>PHASE III</th>
<th>MILESTONES</th>
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<td>platON™ Proprietary platform of decoy oligonucleotides</td>
<td>GENERATION OF NEW DNA-TARGETING COMPOUNDS</td>
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<td>• Next compound Q1 18</td>
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<td>AsiDNA™ IV</td>
<td>Solid tumors</td>
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<td>• Phase I filing Q4 17&lt;br&gt;• Proof-of-Mechanism in man end 18</td>
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<td>AsiDNA™ + PARPi</td>
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<td>• Ready for Proof-of-Concept (PoC) in man end 18</td>
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<tr>
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<td>Solid tumors</td>
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<td>• PoC confirmed via IT¹ (DRIIM study)&lt;br&gt;• Ongoing for IV²</td>
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<tr>
<td>AsiDNA + belinostat /HDACi</td>
<td>Liquid &amp; solid tumors</td>
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<td>• Ready for PoC in man end 18</td>
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<td>Oral belinostat</td>
<td>Liquid &amp; solid tumors</td>
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<td>• Phase I filing Q4 17&lt;br&gt;• Proof-of-Mechanism in man end 18</td>
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<td>Beleodaq® + CHOP*</td>
<td>PTCL 1st line</td>
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<td>• Phase III required by the FDA from SPPI as MA holder in 2nd line</td>
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</table>

¹ IT : intratumoral – IV : intravenous  
² CHOP: Cyclophosphamide, Vincristine, Doxorubicine, Prednisone
platON™
Onxeo’s Chemistry Platform of Decoy Oligonucleotides

Leverage proprietary decoy oligonucleotides technology to generate new breakthrough compounds in oncology
platON™: Proprietary Chemistry Platform of Decoy Oligonucleotides

All three components of the platform can be acted upon to obtain new compounds with different properties and/or activities

A powerful and versatile platform to generate breakthrough compounds acting on intracellular DNA binding targets
platON™: expanding Onxeo’s ability to generate new candidates

- AsiDNA™, first drug candidate from platON™ to enter Phase 1 in Q1 2018
- Next compounds planned from Q1 2018

DNA binding proteins with clear cancer driver properties

- Translational capabilities
- Dedicated screening platform
- Vectorized oligonucleotides library

Best-in-class drug candidates

Combine deep biology insight with leading translational expertise and capabilities in oncology to deliver new, best-in-class drugs

October 2017
AsiDNA™
A first-in-class compound targeting tumor DNA break repair pathways

First compound issued from platON™ *decoy oligo-nucleotides platform* (acquired with DNA Therapeutics in 2016)

- Clinical proof-of-concept achieved (DRIIM phase 1 study - intratumoral)
- Preclinical proof-of-concept by systemic administration (IV) achieved
- Clinical translation ongoing (PK/PD relationship)
- Large potential in multiple indications, alone (stratification biomarkers under validation) & in combination (eg. radio/chemotherapy/ PARPi)

**NEXT MILESTONE**
AsiDNA™ IV
Phase 1 filing Q4 2017
Prevention of tumor DNA repair is a leading-edge field of research in oncology

- Many cancer treatments or genetic instabilities induce DNA damaging but tumor cells survive by repairing DNA damage leading to resistance.

- DNA break repair inhibitors treatments lead to cancer cell death alone (on genetic instable tumors) or in combination with DNA damaging agents.

- Potential new anti-cancer treatment paradigm: Nobel Prize 2015 on DNA repair mechanisms.

Last decade

- DNA damaging agents
- Genetic instabilities

Today

- DNA damaging agents
- Genetic instabilities
- DBR inhibition

DNA damage signaling
- ATM
- ATR
- CHK1
- CHK2
- PARP
- MRN
- RAD51
- DNA-PK

CANCER CELL

DNA damage

CELL DEATH

NO RESPONSE OR RESISTANCE

CELL DEATH

October 2017
AsiDNA™: first-in-class compound in DNA Break repair inhibition

32 bp DNA duplex with a 5´-Chol-TEG & a non-nucleotidic loop

Active 32 bp DNA duplex
- Binds and activates DNA-PK and PARP signaling enzymes
- Sequence not specific, chosen to be non-homologous
- Genomic DNA length optimize

Loop: Coupling Agent
Cholesterol: Vector to promote cellular uptake

Double-stranded 32 bp DNA is tethered with a loop to prevent disassociation\(^1\)
- Phosphorothioate substitutions at the 5’ and 3’ ends to prevent degradation\(^1\)
- Efficient nuclear uptake of the DNA is mediated via a covalently linked cholesterol molecule\(^2\)

Simple, elegant, unique and safe

AsiDNA™: a mechanism of action unlike any other DNA break repair inhibition through an agonist mechanism

Recruitment of DNA damage signaling and repair proteins to the sites of genomic damage is one of the early events in tumors DNA repair

AsiDNA™ is a double stranded DNA molecule (decoy oligo-nucleotide) that mimics double stranded DNA breaks to interfere with DNA repair, redirecting repair enzymes away from sites of tumor DNA damage\(^1,2,3\)

Tumoral Cells (deficient cell cycle control) keep dividing with damaged DNA leading to DNA fragmentation and loss and then mitotic death.

Healthy cells (proficient cell cycle control) stop dividing and reducing transcription, waiting for the disappearance of false signal to resume dividing

Healthy cells and tissues are insensitive to AsiDNA™

Healthy cells insensitivity
- AsiDNA™ triggers the activation of DNA PK and the inhibition of enzyme recruitment in normal cells as well in cancer cells, while not inducing the death of normal cells\(^1\)

Healthy tissues insensitivity
- AsiDNA™ shows no toxicity on healthy tissues even after irradiation\(^2\)

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AsiDNA™: compelling clinical outcomes of DRIIM Phase I study (Intratumoral administration + radiotherapy for metastatic melanoma)

- **Proof of concept established**
  - Overall response rate = 59%
  - Complete response = 30%
    (CR from low-dose radiotherapy alone less than 10%\(^2\))
  - Partial response = 29%
  - Durable response (up to 12 months follow up period)

- **Well tolerated compound**
  - Absence of immune response

- **PK/PD properties**
  - Tumor responses statistically correlated to plasmatic exposure of AsiDNA

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October 2017
AsiDNA™: efficacy on both BRCA mutated and non-mutated tumors in contrast to PARP inhibitors

Effect of AsiDNA and olaparib (PARPi) on BRCA -/- (mutated) Triple Negative Breast Cancer

In contrast to PARPi, AsiDNA displays strong efficacy in BRCA+/+ (non-mutated) tumors

Synergy on mutated BRCA, and no limitation to mutated tumors

AsiDNA™: therapeutic efficacy in **intravenous** murine model of Triple Negative Breast Cancer

- AsiDNA™ sequesters and sustainably hyperactivates the key DNA repair proteins DNA-PK thus preventing effective DNA repair in tumors

- Demonstrated therapeutic efficacy of AsiDNA™ alone administered intravenously

*as measured by gH2AX phosphorylation*
AsiDNA™: strong synergy in combination with PARP inhibitors

- Synergistic effect of AsiDNA™ combined with various PARP inhibitors
  - Increased unrepaired DNA break sites, DNA damages and cell lethality in different tumor cell lines
  - No lethality observed in healthy cells

- Cytotoxic synergistic effect obtained with talazoparib on two tumor cell lines (solid and liquid) without any cytotoxicity on healthy cells

AsiDNA™: strong synergy in combination with HDAC inhibitors

- Strong synergistic effect between AsiDNA™ and histone deacetylase inhibitors (HDACi)
  - The synergy remains over time after repeated administration: potential for new treatment schemes
  - Similar results obtained with HDACi such as vorinostat, entinostat, romidepsin

- Strong cytotoxic synergistic effect observed with belinostat on both solid and liquid tumor cell lines, without any cytotoxicity on healthy cells

% of cell survival

Source: preclinical results; please refer to the Company’s press release on September 28, 2017
AsiDNA™ development strategy

- **Confirm AsiDNA™ activity via systemic administration (IV)**
  - Biomarkers activity and stratification (micronuclei, gH2AX)
  - Ongoing in vivo preclinical in several tumors (combo with PARPi, HDACi...)

- **First-in human Phase I AsiDNA™ IV in advanced malignancies**
  - Confirm proof of mechanism in human, determine optimal clinical dose
  - Translational clinical outcomes: relationship between exposure, activity & safety

- **Clinical development program extension in solid/liquid tumors**
  - AsiDNA™ IV alone for any genetically unstable tumors
  - AsiDNA™ IV in combination with DNA damaging agents

- **Proprietary technology** (Method of Use) patent until 2024
- **Drug product and related compounds** protected until 2031

- **H1 2018**
- **CTA Filing Q4 2017**
- **End 2018/Early 2019**
AsiDNA™: our objectives

- **A new paradigm for cancer treatment**
  - Open a new avenue for cancer treatment with a breakthrough product

- **Strong and promising activity**
  - Capitalize on a set of preclinical results showing strong and promising activity, in monotherapy or in combination other anti-cancer therapies

- **Clear and efficient development plan**
  - Pursue a clear and efficient development plan with preliminary results in man expected from H2 2018

- **platON™**
  - Leverage our expertise to create new compounds with PlatON™ platform

A first-in-class drug candidate with significant competitive advantages and high value potential
belinostat (Beleodaq®)  
HDAC inhibitor

Acquired through the merger with Topotarget (2014)

Fast Track / Orphan Drug Designation (US/EU)

FDA conditional approval in 2nd line PTCL (2014) for Beleodaq® IV

Partnered to Spectrum (SPPI) for the US, & to Pint Pharma for South America

Ongoing development of an oral formulation
Ongoing exploration of combos in liquid & solid tumors

NEXT MILESTONE
Phase 1 (oral / mono) filing Q4 2017
Beleodaq® (belinostat IV) already approved and marketed in Peripheral T-cell lymphoma (PTCL)

**PTCL**
- A subtype of non-Hodgkin’s lymphoma (NHL) which affects T-cells with 17,000 to 27,000 incident cases\(^1\) in key pharmaceutical markets

**Regulatory status**
- Fast Track / Orphan Drug Designation (US & EU); FDA conditional approval in 2\(^{nd}\) line PTCL in 2014 following successful Phase II Belief Study
- Commercialized in the US by Spectrum Pharma (SPPI) since July 2014

**Clinical development**
- Successful Phase II Belief Study (n = 129)\(^2\)
  - Pivotal for FDA conditional approval
  - Favorable safety profile
- Successful Phase I study of Beleodaq® + CHOP (n=23) in 1\(^{st}\) line PTCL
- Phase III clinical study of Beleodaq® + CHOP in PTCL 1\(^{st}\) line under preparation

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\(^{1}\) International Peripheral T-Cell and Natural Killer/T-Cell Lymphoma Study, (J Clin Oncol 26 :4124-4130) and GLOBOCAN 2012, IARC data.

Beyond PTCL: combining belinostat with DNA Break Repair inhibitors

- **Rationale for combining HDACi + DBRi**
  - HDACi induce histone hyperacetylation and ROS* generation that cause structural alterations in chromatin which may expose portions of DNA that are normally protected by heterochromatin to DNA-damages

- **Rationale for belinostat**
  - Recognized efficacy and **superior safety profile** vs other products approved in R/R PTCL makes it more amenable to drug combination

Adapted from J.H. Lee et al, PNAS – vol 107, 33, 2010

*radical oxygen species*
Belinostat: strong synergy in combination with DNA break repair inhibitors (DBRi)

- Synergistic effect of belinostat combined with various DBRi:

  + LY2603618 CheK1 inhibitor

  + AsiDNA

  + AZD6738 ATR inhibitor

Increased cell lethality in different tumor cell lines (solid and liquid)
No lethality observed in healthy cells with belinostat

* Almost no detectable surviving tumor cells
Belinostat: strong increase of DNA breaks on tumor cells but not on healthy cells

\( \gamma H2AX \) quantification

October 2017
Oral belinostat formulation development

- New formulation technology (Amorphous spray dried dispersion)
  - Improved bioavailability by x4
  - Extension of indications (combos, maintenance) & patients’ access (outpatients)

Significant potential competitive advantages over IV formulations
belinostat / Beleodaq®: Build value potential beyond PTCL

- **Confirm belinostat activity via oral administration**
  - Biomarkers activity and stratification (micronuclei, gH2AX...)
  - Ongoing in vivo preclinical in combo with several DNA break repair inhibitors (including AsiDNA™)

- **First-in-human Phase I belinostat oral in advanced malignancies**
  - Confirm proof of mechanism in human, determine optimal clinical dose
  - Translational clinical outcomes: relationship between exposure, activity & safety

- **Clinical development program extension in advanced tumors**
  - belinostat oral in combination with other DNA break repair inhibitors (incl. belinostat oral + AsiDNA™)

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- **belinostat oral formulation extends IP protection until 2038** (vs. 2026/2027 for the IV formulation)

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- **October 2017**
- **H1 2018**
- **CTA Filing Q4 2017**
- **End 2018/Early 2019**
Oral belinostat: our objectives

- **Development plan**
  - Execute the successful ongoing development plan of a compound already approved in an orphan indication

- **Oral form and combination therapy**
  - The conjugation of an oral form with a combination protocol open a wide range of potential cancer indications

- **Patent protection**
  - An extended patent protection for a drug with high potential

Significant competitive advantages in this class of product with high value potential
Financial figures and next milestones
Diversified shareholder structure and financial profile

Shareholder structure (as of June 21, 2017)

- 50.1% Other free float
- 19.2% French institutions
- 17.5% International institutions
- 13.0% Financière de la Montagne
- 17.5% International institutions

Dual listing Euronext Paris & Nasdaq Copenhagen
Ticker: ONXEO

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<td>CASH RUNWAY</td>
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Q3 financial & business update on October 26, 2017
Multiple near to mid-term R&D value-creating milestones

**platON™**
*From early 2018 on*
New compounds and their potential applications

**Oral belinostat**
*H2 2017 to H1 2018*
Results of ongoing preclinical studies in other tumors/combinations
*End 2017*
Filing for a Phase I as monotherapy in all tumors types
*From H2 2018 on*
Intermediate findings from phase I

**AsiDNA™ (IV)**
*End 2017*
Filing for a Phase I as monotherapy in solid tumors
*Early 2018*
Results of ongoing in-vivo preclinical studies in other tumors/combinations
*From H2 2018*
Intermediate findings from phase I
*End 2018*
Proof-of-mechanism in man
Onxeo: advancing innovation towards breakthrough cancer therapies

- A clear value creating strategy by driving innovative programs to best inflexion points to generate deals

- R&D programs based on 2 of the most promising mechanisms of action, with multiple development paths and wide potential of applications

- A sound translational expertise to drive optimal compounds’ development

- A proven capacity to generate transactions and enrich the pipeline
Thank you for your attention

We welcome questions from the audience
Contacts
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Nicolas Fellmann – CFO
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Company Information
www.onxeo.com
A complementary and experienced management team...

Nicolas FELLMAN
CFO
Formerly Pfizer, Ernst & Young

Philippe MAITRE
EVP Onxeo Inc., CBDO
Leads the US subsidiary
Formerly Aventis, PPD, mAbRx

Judith GRECIET (Pharm.D)
CEO since 2011
Formerly Wyeth, Eisai
Eisai France President until 2011

Françoise BONO (PhD)
CSO
Sanofi, Evotec
Evotec’s EVP until 2016

Olivier DE BEAUMONT (MD)
CMO
Formerly Senior VP Stallergenes Greer, Quintiles, Aventis

Demonstrated track record for product development, M&A, partnering
...advised by an international board of industry experts

JOSEPH ZAKRZEWSKI  
Chairman of the Board

JUDITH GRECIET  
CEO

DANIELLE GUYOT-CAPARROS  
Independent director

CHRISTINE GARNIER  
Independent director

ELVIRA SANZ  
Independent director

JEAN-PIERRE BIZZARI  
Independent director

THOMAS HOFSTAETTER  
Independent director

JEAN-PIERRE KINET  
Independent director

NICOLAS TREBOUTA  
Director, representing Financière de la Montagne

October 2017
AsiDNA™ demonstrates strong synergy in combination with HDAC inhibitors

- **Solid tumor Cell line**
- **Liquid tumor Cell line**
- **Healthy Cell line**

- **AsiDNA**
- **Belinostat**
- **AsiDNA+Belinostat**

- **AsiDNA**
- **Vorinostat**
- **AsiDNA+Vorinostat**

- **AsiDNA**
- **Entinostat**
- **AsiDNA+Entinostat**

- **AsiDNA**
- **Romidepsin**
- **AsiDNA+Romidepsin**

* ns, * p < 0.05, ** p < 0.01, *** p < 0.001, **** p < 0.0001


Inhibition of DNA damage repair by artificial activation of PARP with siDNA. Croset A, Cordelières FP, Berthault N, Buhler C, Sun JS, Quanz M, Dutreix M. Nucleic Acids Res. 2013, 41:7344-55.


Chemosensitization of hepatocellular carcinoma by the novel DNA repair inhibitor DT01 in mouse and rabbit models. Herath N, et al., Submitted - European radiology (under review)


ASCO 2015 annual meeting. Abstract #143029.


Safe and Effective Treatment of Patients with Peripheral T-cell Lymphoma (PTCL) with the Novel HDAC Inhibitor, Belinostat, in Combination with CHOP: Results of the Bel-CHOP Phase 1 Trial. (Johnston, P., et al., ASH 2015).

A Phase II trial of Belinostat (PXD101) in patients with relapsed or refractory peripheral or cutaneous T-cell lymphoma. (Foss, F., et al., 2014, BJH)


Phase II activity of belinostat (PXD-101), carboplatin, and paclitaxel in women with previously treated ovarian cancer. (Dizon, D.S, et al., Int J Gynecol Cancer, 2012)

A Phase I/II Clinical Trial of Belinostat (PXD101) in Combination with Doxorubicin in Patients with Soft Tissue Sarcoma. (Vitfell-Rasmussen, J., et al., 2016, Sarcoma)

Paclitaxel/Carboplatin With or Without Belinostat as Empiric First-Line Treatment for Patients With Carcinoma of Unknown Primary Site: A Randomized, Phase 2 Trial. (Hainsworth, J.D., et al., 2015, Cancer, 121(10), 1654-61).


A randomized phase II trial of Doxorubicin-Transdrug™ (Livatag®) demonstrates significant overall survival increase in inoperable hepatocellular carcinoma (HCC) patients. Merle, P. et al. 2011 (In Book of abstracts)


Dose and infusion rates drive lung toxicity of Doxorubicin Transdrug (DT) in rats. Merle, P. et al. 2014 (In Book of abstracts)


Mechanistic study of the relative cytotoxicity of doxorubicin loaded nanoparticle formulation compared to free doxorubicin in hepatocellular carcinoma (HCC) cell lines. Trochon-Joseph, V et al. AACR 2016, Abstr nr 2143

A novel nanoparticle formulation of doxorubicin is clearly differentiated from free doxorubicin in overcoming resistance mechanism in chemo-resistant tumors. Trochon-Joseph, V et al. AACR 2017, Abstr nr 3076

Safety and efficacy results from the phase 3 relive study of doxorubicinloaded nanoparticles versus best standard of care in patients with advanced hepatocellular carcinoma after failure or intolerance to previous treatment including sorafenib. P. Merle et al, (O-020) (Summary of key findings on Onxeo’s website - [http://www.onxeo.com/pdf/170918_Onxeo_Support_Slides_Call_Relive.pdf](http://www.onxeo.com/pdf/170918_Onxeo_Support_Slides_Call_Relive.pdf) )