Press release
Cham, November 21, 2023

Biotech company Oncoteq further expands its pipeline, in-licensing a promising treatment for breast cancer

- **Oncoteq, a clinical stage biotech company specializing in innovative cancer treatments, in-licenses a novel compound with potential to shift the paradigm in breast cancer treatment**
- **The first-in-class compound TEQ103 (formerly SERA2), is a selective estrogen receptor activator, which specifically targets cancer cells expressing estrogen receptor alpha and in mouse models induces complete regression of tumors that are resistant to current treatments**
- **TEQ103 has the potential to deliver better efficacy and overall survival outcomes with fewer side effects as compared to current treatments**
- **Under the terms of the agreement, Oncoteq will be responsible for developing and commercializing TEQ103 globally**
- **This is in-licensing deal follows Oncoteq's strategy to build a development pipeline through deal-making and unlock the hidden value of assets through artificial intelligence.**

Oncoteq AG, a clinical stage biotech company specializing in innovative cancer treatments, expands its pipeline with the in-licensing of the small molecule TEQ103 (formerly SERA2) from US biotech incubator, Systems Oncology. The agreement represents Oncoteq's second in-licensing of a potential first-in-class or best-in-class cancer treatment following its transaction with Merck KGaA in 2022. The company will continue to seek opportunities with which to expand its growing oncology-focused pipeline.

TEQ103 is a first-in-class selective estrogen receptor activator (SERA) that potentially offers a paradigm-shifting treatment approach for patients with breast cancer. TEQ103 utilises a novel mechanism directed towards estrogen receptor alpha (ERα), a proven target for the treatment of patients with breast cancer. Through high-affinity binding to ERα, TEQ103 exerts lethal effects only in cells that have an activated stress response, so-called anticipatory unfolded protein response (aUPR), a feature absent in healthy cells. This pushes a normally protective cellular stress-response pathway into overdrive and rapidly and selectively kills ERα-expressing cancer cells. Current ERα-targeting ("endocrine") breast cancer treatments act to slow tumor growth by modulating or degrading ERα or by lowering estrogen levels and lack selectivity for cancer cells.

Breast cancer is among the most frequent of cancers and annually more than an estimated 350,000 patients lose their lives to the disease. Given that around 80% of all breast cancers express ERα, TEQ103 may represent a paradigm shift in treatment for a large group of breast cancer patients.

"*We are thrilled by the opportunity to bring forward a potentially highly effective treatment for breast cancer, a disease still characterized by significant unmet medical needs despite recent advances in treatment. We are excited to progress this molecule as fast as possible to clinical testing,*

knowing that it could be a potential game changer for breast cancer patients in great need", says Mads Dalsgaard, Chief Executive Officer of Oncoteq.

Spyro Mousses, Chief Executive Officer of Systems Oncology, comments: "*We are really impressed with the team at Oncoteq and their vision of how to take this molecule forward to a new groundbreaking breast cancer treatment. It is pleasure to hand over the future development to Oncoteq and have them build upon what we started, first and foremost to benefit patients*"

TEQ103 is currently in pre-clinical development and Oncoteq will complete the non-clinical data package before advancing the molecule into clinical development in 2025. Oncoteq will firstly prioritize development of TEQ103 as treatment of breast cancer and could later expand to other indications, as the compound has potential as a treatment for several other cancers. Currently, the global market for breast cancer treatments has a value of approximately USD 25-30 billion annually, thus TEQ103 has significant potential not only for patients but also commercially.

Through the deal, Oncoteq obtains a world-wide exclusive license to develop and commercialize TEQ103 in exchange for upfront payment, milestones and royalties.

For further information and interview with Chief Executive Officer Mads Dalsgaard, please contact:

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About Oncoteq

Established in 2022, Oncoteq is a clinical stage biotech company specializing in new innovative cancer treatments. Combining AI technology from sister company Innoplexus with Oncoteq's scientific and drug development expertise allows the company to identify and the unlock the value of compounds licensed from biopharma.

Oncoteq is founded and seeded by Cureteq AG and headquartered in Zug, Switzerland. Cureteq AG is an asset management and drug development company with focus on oncology, central nervous systems disorders, rare diseases and women's health.

For further information please visit Oncoteq website www.oncoteq.ch, Cureteq website www.cureteq.com. and our LinkedIn-page: https://www.linkedin.com/company/cureteq/

About TEQ103

TEQ103 (formerly SERA2) was the result of a collaborative program between Systems Oncology and University of Illinois, developing molecules to activate the anticipatory unfolded protein response. In general, UPR is the endoplasmic reticulum (EnR) stress sensor that ensures protein-folding quality control and maintains intracellular protein homeostasis, which are both necessary for cell viability.

Paul Hergenrother's Laboratory at the University of Illinois invented and optimized a series of small molecule selective estrogen receptor activator (SERA) compounds. David Shapiro's Laboratory at the

David Shapiro's Laboratory at the University of Illinois conducted the seminal research on this mechanism, showing that the anticipatory UPR is a normal physiologic stress reaction activated by pro-growth agents to promote cellular protein homeostasis. Cancer cells evolve the ability to drive elevation of the anticipatory unfolded protein response for survival and drug resistance. This puts cancer cells very close to the lethal threshold for UPR activation, making cancer cells selectively vulnerable to compounds that can overdrive this pathway. In UPR hyperactivation the cell enters a state of ATP-depleting overdrive resulting in cell swelling, sustained protein synthesis inhibition and rapid cell death. SERA compounds selectively drive ERα to hyperactivate phospholipase C gamma, leading to calcium efflux from the EnR into the cytosol, which consequently hyperactivates the UPR. In the context of cancer cells with anticipatory elevation in the UPR, the SERA class of compounds induce further UPR hyperactivation past the lethality threshold, driving rapid cancer cell death.

In collaboration with Systems Oncology, TEQ103 was developed and advanced as a clinical lead compound. Specifically, TEQ103-mediated ERα hyperactivation exerts irreversible lethal effects on ERα-expressing breast cancer cells. In animal models, TEQ103 is found to be both well-tolerated and highly active in ERα-expressing MCF-7 breast tumor models and induces durable responses. TEQ103 is also highly active in a breast cancer patient-derived xenograft model with ESR1 mutation and clinical resistance to selective estrogen receptor degraders and CDK4/6 inhibitors.

About breast cancer

The pathologic, diagnostic, prognostic, and therapeutic role of ERα in breast cancer is undisputed. However, despite recent advances in the treatment of patients with ERα-expressing advanced and/or metastatic breast cancer – adding up to roughly 80% of patients if the new category of "HER2-low" ERα-expressing disease is also considered – more effective and better tolerated treatment options are warranted, in particular as these patients benefit only modestly from available salvage chemotherapy regimens. In cases where ERα-expressing advanced and/or metastatic breast cancers no longer respond to available endocrine treatment options, there are unfortunately no effective treatments available for these patients.

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