



Amolyt Pharma Announces Research Agreement and Licensing Option with XOMA

Amolyt to advance pre-clinical development of anti-PTHrP monoclonal antibodies as potential treatments of primary hyperparathyroidism (PHPT) and humoral hypercalcemia of malignancy (HHM)

Amolyt has obtained the option to license one or more candidates from XOMA for further clinical development worldwide

PHPT and HHM are rare endocrine diseases with high unmet needs and these candidates from XOMA represent a natural expansion of Amolyt's current development pipeline

LYON, France, and Cambridge, MA, January 24, 2023 - Amolyt Pharma ("Amolyt," "Amolyt Pharma" or the "Company"), a global company specialized in developing innovative therapies for rare endocrine and related diseases, today announced that the Company has entered into a research agreement and licensing option with XOMA Corporation ("XOMA") for the pre-clinical evaluation of a select set of monoclonal antibodies that arose from XOMA's legacy discovery efforts as potential treatments for primary hyperparathyroidism ("PHPT") and humoral hypercalcemia of malignancy ("HHM"). Amolyt has the option to license one or more of these candidates from XOMA for further clinical development worldwide.

Both PHPT and HHM are rare endocrine diseases with high unmet needs and a common target, the parathyroid hormone 1 receptor ("PTH1R"), and are characterized by the hypersecretion of parathyroid hormone ("PTH") and parathyroid hormone-related peptide ("PTHrP"), respectively, resulting in continuous stimulation of the PTH1R, leading to bone loss, hypercalcemia and hypophosphatemia. The objective of the research agreement is to test the anti-PTH1R antibodies in relevant animal disease models with the aim of selecting a candidate that can successfully halt the over-stimulation of the PTH1R caused by excess PTH and PTHrP and eliminate the debilitating symptoms and serious complications associated with PHPT and HHM.

"We are continuing to build our portfolio for rare endocrine and related diseases while moving, in parallel, our lead program for hypoparathyroidism into a pivotal trial. This new program for primary hyperparathyroidism and humoral hypercalcemia of malignancy represents a natural expansion of our pipeline and leverages our clinical expertise in the field of calcium and bone metabolism and in the biology of parathyroid hormone and its receptor," stated Thierry Abribat, Ph.D., chief executive officer of Amolyt Pharma.

"We look forward to advancing this pre-clinical work and, if successful, entering into a license agreement with XOMA in order to bring the program to patients, to expand our development pipeline and to establish more opportunities for long-term value creation," Dr. Abribat concluded.

Brad Sitko, chief investment officer of XOMA, added, "Given Amolyt's established expertise in rare endocrine disorders, and specifically diseases stemming from dysregulation of PTH, we believe they are a great partner to advance the development of these promising antibodies. We



look forward to watching Amolyt's progress, as PHPT and HHM patients are underserved by current standards of care."

About Primary Hyperparathyroidism ("PHPT")

Primary hyperparathyroidism involves excessive PTH production due to enlargement of one or more of the parathyroid glands located at the front and base of the neck that leads to hypercalcemia. Excess serum calcium affects multiple body systems including the skeletal, gastrointestinal, renal (kidney), muscular, cardiovascular, and central nervous systems. Most commonly, primary hyperparathyroidism is seen in people over 60 years of age and is more common in women. Radiation to the head and neck increases risk as does the rare parathyroid carcinoma. In approximately 85% of cases, PHPT is caused by a single adenoma. In approximately 15% of cases, multiple glands are involved (either multiple adenomas or hyperplasia) and management of the disease is more challenging. Prevalence is estimated at ~233/100k women and 85/100k men.

About Humoral Hypercalcemia of Malignancy ("HHM")

Approximately 20% of all cancer patients develop hypercalcemia during their clinical course, and 80% of those cases are caused by excessive secretion of PTHrP from tumor cells (HHM). Squamous cell carcinomas of the head, neck, esophagus and lung, and cancers of the cervix, lung and colon are most commonly involved, in addition to renal cell, bladder, breast, endometrial, and ovarian cancers. It is associated with a wide spectrum of symptoms including nausea, vomiting, anorexia, abdominal pain, constipation, polyuria, hypotension, bone pain, bone loss fatigue and confusion. Renal failure or coma can occur; thus, this condition may be considered an oncologic emergency. Current standard of care is focused on bone anti-resorptive therapy, and intravenous hydration to enhance urinary calcium excretion. However there are many limitations to available therapy and unwanted effects include fever, bone pain, renal toxicity, hypocalcemia, and osteonecrosis of the maxilla and mandible. A better tolerated systemic therapy that directly targets the interaction between PTHrP and the PTH1R, rather than the downstream effect on bone resorption, has the potential to provide a safer, more effective alternative. Based on a prevalence study in the United States, HHM represented about 57,000 cases in 2013.

About Amolyt Pharma

Amolyt Pharma, a clinical stage biotechnology company, is building on its team's established expertise to deliver life-changing treatments to patients suffering from rare endocrine and related diseases. Its development portfolio includes eneboparatide (AZP-3601), a long-acting PTH1 receptor agonist as a potential treatment for hypoparathyroidism, and AZP-3813, a peptide growth hormone receptor antagonist for the potential treatment of acromegaly. Amolyt Pharma aims to further expand and develop its portfolio by leveraging its global network in the field of endocrinology and with support from a strong syndicate of international investors. To learn more, visit <https://amolytpharma.com/> or follow us on [Twitter](#) and [LinkedIn](#).

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