NEWS RELEASE

kymab

Kymab to Present Update on Lead Immunocytokine Program KY1043 at Two European Scientific Meetings in November 2019

- KY1043, a bifunctional, PD-L1-based, IL-2Rα (CD25)-directed immunocytokine, binds and blocks the activity of PD-L1 on PD-1 and preferentially stimulates T cells via preferential binding to the high affinity trimeric IL-2αβγ receptor (CD25/CD122/CD132)
- By engaging with both PD-L1 and IL-2αβγ receptor, KY1043 stimulates potent anti-tumor responses in a T cell tumor killing assay leading to enhanced tumor killing compared to a non-targeted immunocytokines
- *In vivo,* KY1043 produces rapid, dose-dependent tumor regression of established MC38 tumors
- Pharmacodynamic data indicates that KY1043 can increase CD8+ T cells in the tumor microenvironment with differential activity in the periphery that lead to the expansion of Treg cells
- Expanding data set continues to provide evidence for a dual mechanism-of-action of KY1043 *in vitro* and *in vivo*
- Development activities, including regulatory-enabling toxicological studies, are in progress

Cambridge, UK; November 14, 2019: Kymab, a clinical-stage biopharmaceutical company developing antibody-based therapeutics, will be delivering oral presentations detailing an expanding body of evidence for the activity of the company's novel immunocytokine KY1043 at both the Promise of Interleukin-2 Therapy Congress being held at the Centre international de Conférence Sorbonne Université in Paris, France November 13-16 and the 11th annual PEGS Europe Meeting at the Lisbon Center in Lisbon, Portugal November 18-22. These presentations follow the KY1043 poster presented at the Society of Immunotherapy for Cancer (SITC) 34th Annual Meeting held on November 8, in the United States.

KY1043 is a novel, fully-human, PD-L1-based, IL-2Rα (CD25)-directed immunocytokine that is designed to both inhibit the interaction between PD-L1 and the T cell checkpoint receptor PD-1 and stimulate T cells *via* interaction of a mutated form of IL-2 that preferentially activates antigen experienced T cells to promote the immune response against tumors.

Data presented will demonstrate that KY1043 retains the ability to effectively inhibit PD-1/PD-L1 binding and demonstrates preferential binding to the IL-2Rα (CD25)-containing high-affinity trimeric IL-2Rαβγ receptor present on antigen-experienced and activated T cells as well as on regulatory T cells (Treg). *In vitro*, KY1043 increases antigen-specific T cell-mediated killing of tumor cells. *In vivo*, KY1043 administration produces profound tumor growth suppression in an established MC38 tumor model, leading to the regression of tumors in a dose-dependent manner. Mice treated with KY1043 are resistant to subsequent tumor challenge indicating, the potential for long-term anti-tumor memory responses. Mechanism-of-action studies indicate that, while Treg cells are increased in the periphery as expected, in the tumor CD8 effector cells are increased without a concomitant increase in intratumoral Treg cells.

Meeting: Promise of Interleukin-2 Therapy Congress, Paris, France
Title: KY1043, a novel PD-L1 IL-2 immunocytokine directed towards IL-2Rα (CD25), delivers potent anti-tumour activity in vivo
Presenter: Tim Malcom
Date and Time: Friday 15th November, 4:30pm CET

Meeting: PEGS Europe Meeting, Lisbon, Portugal
Title: The Development of KY1043; a Highly-Differentiated PD-L1-Based IL-2Rα CD25-Biased Immunocytokine

Presenter: Matthew McCourt
Track: Tumour Microenvironment
Date and Time: Tuesday 19th November, 09:05 am GMT

###ENDS###

NOTES TO EDITORS

About KY1043

KY1043 is a novel immunocytokine based on a fully-human PD-L1 antibody discovered by Kymab. KY1043 a bi-functional PD-L1-based IL-2Rα (CD25)-directed immunocytokine and is designed to both inhibit the interaction between PD-L1 and the T cell checkpoint receptor PD-1 and stimulate T cells *via* interaction of a mutated form of IL-2 that preferentially activates antigen-experienced T cells to promote the immune response against tumors.

KY1043 has been investigated in highly illustrative *in vitro* and syngeneic models. *In vitro*, KY1043 increases antigen-specific T cell-mediated killing of tumor cells. *In vivo*, KY1043 administration produces profound effects in an established MC38 tumor model, leading to the regression of tumors in a dose-dependent manner.

For more information on Kymab please see http://www.kymab.com.

Forward-looking statements

This announcement includes forward-looking statements that involve risks, uncertainties and other factors, many of which are outside of our control, that could cause actual results to differ materially from the results discussed in the forward-looking statements. Forward-looking statements include statements concerning our plans, objectives, goals, future events, performance and/or other information that is not historical information. All such forward-looking statements are expressly qualified by these cautionary statements and any other cautionary statements which may accompany the forward-looking statements. We undertake no obligation to publicly update or revise forward-

looking statements to reflect subsequent events or circumstances after the date made, except as required by law.

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