X-Vax Technology, Inc.
601 Heritage Drive, Suite 138
Jupiter, FL 33458

NEWS RELEASE

FOR IMMEDIATE RELEASE

X-Vax Technology Raises $56 Million in Upsized Series A Financing to Advance Lead Herpes Vaccine Program

- *A new approach to beating herpes by inducing antibodies that mediate the killing of infected cells*
- *Participants include Johnson & Johnson Innovation – JJDC, Inc. (JJDC); Adjuvant Capital, an impact investment fund supported by the Bill & Melinda Gates Foundation as an anchor investor; Serum Institute of India; Alexandria Venture Investments; Founders Fund.*

JUPITER, FL, July 23, 2019. X-Vax Technology, Inc. (X-VAX ™), a biotechnology company developing vaccines based on a new approach that mediates the killing of infected cells, today announced that it has raised $56 million in an upsized Series A financing with participation from strategic and institutional investors, including Johnson & Johnson Innovation – JJDC, Inc. (JJDC); Adjuvant Capital, an impact investment fund supported by the Bill & Melinda Gates Foundation as an anchor investor; Serum Institute of India; Alexandria Venture Investments; and FF DSF VI, a scout investment vehicle out of Founders Fund. Proceeds from the financing will be used to advance X-VAX's lead program, a vaccine candidate against herpes, called ΔgD-2 (delta gD-2) for further development and production, including a Phase 1 clinical study.

"We are pleased to have the support of our new and existing investors as we continue to build our leadership position in the development of a herpes vaccine," said Ulf Wiinberg, President and Chief Executive Officer of X-VAX. "We are encouraged by the preclinical data for our new approach to beating herpes and creating a potentially world-changing vaccine."

"Herpes infections are a significant global health problem that affect all age groups from infants to the elderly. Infection is associated with a wide range of disease," said Betsy Herold, MD, co-Inventor and Professor of Pediatrics, Microbiology & Immunology at Albert Einstein College of Medicine in New York. "The ability of the virus to successfully escape clearance by the immune system and to establish a non-replicating state known as latency with periodic reactivation results in lifelong infection and ongoing risk of transmission."

"We believe that ΔgD-2 may be more promising than other previous vaccine candidates because it elicits a different type of immune response against HSV-1 and HSV-2 that is more effective in preclinical models at clearing virus and preventing the establishment of latency. In nonclinical models, immunization with ΔgD-2 elicits antibodies that facilitate the killing of infected cells, rapidly clearing the virus and thereby inducing sterilizing immunity," added William Jacobs, PhD, co-Inventor and Professor of Microbiology & Immunology at Albert Einstein College of Medicine.

Maxim Merchant Capital, a division of Maxim Group LLC, acted as sole placement agent for the financing.

About herpes, a global epidemic

There is no approved vaccine for herpes simplex. Herpes simplex virus is categorized into 2 types: herpes simplex virus type 1 (HSV-1) and herpes simplex virus type 2 (HSV-2). More than 3.7 billion people under the age of 50 around the world are infected with HSV-1, while over 400 million have HSV-2.[1, 2] Neonatal infection can be devastating, at 60% fatality without treatment.[3] Other complications include encephalitis or meningitis (inflammation of the brain or the tissue that covers the brain and spinal cord), and infectious blindness. HSV-2 is also known to contribute significantly to the spread of HIV.[4] Antiviral drug therapy shows only moderate efficacy and comes with significant side effects.[5] Attempts to develop an effective vaccine have repeatedly failed.

About X-Vax Technology, Inc.

We are a biotech company committed to developing vaccines against pathogens acquired by mucosal infection such as herpes. Our research leads us to believe that the new approach we are taking could succeed in defeating herpes. We have created a herpes vaccine candidate that we call ΔgD-2 (delta gD-2) because it is based on an HSV-2 virus genetically deleted for glycoprotein D (gD-2). With it, we have been able to prevent infections caused by herpes type 1 (HSV-1) and type 2 (HSV-2) in multiple preclinical models—with encouraging results. The vaccine induces Fc receptor activating antibodies that mediate antibody-dependent cell-mediated killing (ADCK) as the primary mechanism of protection. ADCK is induced to flag infected cells for destruction by natural immune cells. [6, 7]

Forward-looking statements

This news release contains express or implied forward-looking statements pursuant to U.S. Federal securities laws. For example, we are using forward-looking statements when we

discuss the proposed use of proceeds from the Series A financing, when we describe our belief that our new approach to beating herpes could succeed in potentially creating a world-changing vaccine, when we discuss the belief that our vaccine candidate is more promising than other previous vaccine candidates and when we state our belief that our new approach could succeed in defeating herpes. These forward-looking statements and their implications are based on the current expectations of the management of X-VAX only, and are subject to a number of factors and uncertainties that could cause actual results to differ materially from those described in the forward-looking statements. The following factors, among others, could cause actual results to differ materially from those described in the forward-looking statements: changes in technology and market requirements; we may encounter delays or obstacles in launching and/or successfully completing our clinical trials; our products may not be approved by regulatory agencies, our technology may not be validated as it progresses further and its methods may not be accepted by the scientific community; we may be unable to retain or attract key employees whose knowledge is essential to the development of our products; unforeseen scientific difficulties may develop with our process; our products may wind up being more expensive than we anticipate; results in the laboratory may not translate to equally good results in real clinical settings; results of preclinical studies may not correlate with the results of human clinical trials; our patents may not be sufficient; our products may harm recipients; changes in legislation may adversely impact us inability to timely develop and introduce new technologies, products and applications; loss of market share and pressure on pricing resulting from competition, which could cause the actual results or performance of X-VAX to differ materially from those contemplated in such forward-looking statements. Except as otherwise required by law, we undertake no obligation to publicly release any revisions to these forward-looking statements to reflect events or circumstances after the date hereof or to reflect the occurrence of unanticipated events.

Contact

Andreas Eggert

aeggert@x-vax.com

+1-561-517-XVAX (+1-561-517-9829)

x-vax.com

References

1. Looker, K.J., et al. Global and regional estimates of prevalent and incident herpes simplex virus type 1 infections in 2012. *PloS one* 10, e0140765 (2015).

2. Looker, K.J., et al. Global estimates of prevalent and incident herpes simplex virus type 2 infections in 2012. *PloS one* 10, e114989 (2015).
3. Looker, K.J., et al. First estimates of the global and regional incidence of neonatal herpes infection. *Lancet Glob Health* 5, e300-e309 (2017).
4. Looker, K.J., et al. Effect of HSV-2 infection on subsequent HIV acquisition: an updated systematic review and meta-analysis. *Lancet Infect Dis* 17, 1303-1316 (2017).
5. Sauerbrei, A. Optimal management of genital herpes: current perspectives. *Infect Drug Resist* 9, 129-141 (2016).
6. Petro C et al., Herpes simplex type 2 virus deleted in glycoprotein D protects against vaginal, skin and neural disease, *Elife*. 2015;4.
7. Petro C et al., HSV-2 ΔgD elicits FcγR-effector antibodies that protect against clinical isolates, *JCI Insight*. 2016;1(12).