DEPARTMENT OF HEALTH AND HUMAN SERVICES

Notification of Charter Renewal: National Preparedness and Response Science Board (Previously Known as the National Biodefense Science Board)

AGENCY: Office of the Secretary, Department of Health and Human Services.

ACTION: Notice.

SUMMARY: The Secretary of the Department of Health and Human Services has renewed the charter of the National Preparedness and Response Science Board (NPRSB), previously known as the National Biodefense Science Board, for an additional two-year period through July 3, 2016.

FOR FURTHER INFORMATION CONTACT: Please submit any inquiries to CAPT Charlotte Spires, DVM, MPH, DACVPM, Executive Director and Designated Federal Official, National Preparedness and Response Science Board, Office of the Assistant Secretary for Preparedness and Response, U.S. Department of Health and Human Services, Thomas P. O'Neill Federal Building, Room number 14F18, 200 C St. SW., Washington, DC 20024; Office: 202–260–0627, Email address: *charlotte.spires@hhs.gov.*

SUPPLEMENTARY INFORMATION: As stipulated by the Federal Advisory Committee Act (FACA), 5 U.S.C. App. 2 Section 9(c), the U.S. Department of Health and Human Services is hereby giving notice of the renewal of the NPRSB charter for an additional two-year period. The Board shall provide expert advice and guidance to the Secretary on scientific, technical, and other matters of special interest to the Department of Health and Human Services regarding current and future chemical, biological, nuclear, and radiological agents, whether naturally occurring, accidental, or deliberate. The Board may also provide advice and guidance to the Secretary on other matters related to public health emergency preparedness and response.

Dated: June 13, 2014.

Nicole Lurie,

Assistant Secretary for Preparedness and Response.

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

National Center for Advancing Translational Sciences (NCATS): Cooperative Research and Development Agreement (CRADA) and Licensing Opportunity for Small Molecule Inhibitors of the Human USP1/UAF1 Complex(1) for the Treatment of Cancer

SUMMARY: The National Center for Advancing Translational Sciences (NCATS) and its collaborator, the University of Delaware, are seeking Cooperative Research and Development Agreement (CRADA) partners to collaborate in the final stages of lead optimization, evaluation and preclinical development of a novel series of selective and potent small-molecule inhibitors of the human USP1/UAF1 complex(*1*) for the treatment of cancer. Interested potential CRADA partners will receive detailed information about the project after signing a confidential disclosure agreement (CDA) with NCATS and University of Delaware.

DATES: Interested candidate partners must submit a statement of interest and capability to the NCATS point of contact before July 24, 2014 for consideration. Guidelines for the preparation of a full CRADA proposal will be communicated shortly thereafter to all respondents with whom initial confidential discussions will have established sufficient mutual interest. CRADA applications submitted after the due date may be considered if a suitable CRADA collaborator has not been identified by NIH and its collaborator, the University of Delaware, among the initial pool of respondents. Licensing of background technology related to this CRADA opportunity is also available to potential collaborators.

ADDRESSES: Questions about licensing opportunities of related background technology should be addressed to Jenny Wong, M.S., Senior Licensing and Patenting Manager, Office of Technology Transfer, NIH, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852–3804, Telephone: (301) 435–4633; Email: *wongje@mail.nih.gov.* Respondents interested in licensing will be required to submit an ''Application for License to Public Health Service Inventions.'' An executed CDA will be required to receive copies of the patent applications.

FOR FURTHER INFORMATION CONTACT: Further details of this CRADA opportunity and statement of interest please contact Lili Portilla, M.P.A., Director of Strategic Alliances, National Center for Advancing Translational Sciences, NIH, 9800 Medical Center Drive, Room 311, Rockville, MD 20850; Telephone (301) 217–2589; Email: *Lilip@nih.gov* or Dr. Krishna Balakrishnan, Senior Technology Transfer Manager, NCATS, Telephone: (301) 217–2336; Email: *balakrik@mail.nih.gov.*

SUPPLEMENTARY INFORMATION: Ubiquitin-specific proteases (USPs) have in recent years emerged as a promising therapeutic target class in the ubiquitin-proteasome system (UPS). Velcade® (bortezomib), a small molecule proteasome inhibitor, has established the ubiquitin-proteasome system as a valid target for anticancer treatment. However, proteasome inhibitors in general suffer from a narrow therapeutic index and acquired resistance. A promising alternative to proteasome inhibition has been to target the enzymes upstream of proteasome-mediated protein degradation, i.e. the ubiquitin ligases and deubiquitinating enzymes (DUBs), to generate more specific, less toxic therapeutic agents.

The advantage of inhibiting DUB lies in the specificity of therapeutic intervention that can lead to better efficacy and reduced side effects. It has become clear that the DUB activities are indispensable for the normal cellular functions. Abnormal cellular expression of DUBs or the loss of function due to mutation in certain DUB genes have been linked to various human diseases(*2, 3*). Among the five DUB subfamilies, ubiquitin-specific protease (USP) is emerging as promising targets for pharmacological intervention because of their connection to many human diseases, including prostate, colon and breast cancer, pediatric acute lymphoblastic leukemia, and familial cylindromatosis(*2, 4*). From the past successes in targeting proteases with small molecule antagonists, it is expected that efforts of targeting human USPs will lead to potent and specific therapeutic agents.

The human ubiquitin-specific protease 1 (or USP1) occupies a special position because it has been implicated in DNA damage response in higher vertebrates and humans. Previous studies showed that disruption of USP1 in chicken DT40 cells resulted in increased sensitivity to DNA crosslinkers(*5*) and knockout of the murine USP1 gene in a mouse model resulted in hypersensitivity to mitomycin C(*6*). Previously we have demonstrated that inhibiting the cellular activity of human USP1 by