Request for Applications (RFA) on Novel Biomarkers for the Partnership for Accelerating Cancer Therapies

A. FNIH RFA NUMBER: 2018-PACT001	**B. DATE ISSUED:** December 15, 2018	
C. ISSUED BY:	**D. ADDRESS OFFERS TO:**	
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E. FOR INFORMATION REGARDING THIS SOLICITATION CONTACT:		
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IMPORTANT: **F.** To be considered for award, Offers must be received at the location specified in Block E.2. above by 11:59PM EST January 21, 2019. Offers must be clearly identified with the solicitation number provided in Block A above.		

The Foundation for the National Institutes of Health, a non-profit, 501(c) (3) charitable organization that supports the NIH in its mission to improve health by forming and facilitating public-private partnerships for biomedical research, is issuing a Request For Applications (RFA) for novel biomarkers to support the Partnership for Accelerating Cancer Therapies (PACT). PACT seeks to provide a systematic approach to immune and related oncology biomarker investigation in clinical trials by supporting development of standardized biomarkers and assays. PACT is executed by FNIH as a public-private partnership involving the National Cancer Institute (NCI), the U.S. Food and Drug Administration (FDA), multiple pharmaceutical companies, non-profits and patient advocates.

Purpose

This Request for Applications is associated with the Partnership for Accelerating Cancer Therapies (PACT), a project associated with the Beau Biden Cancer Moonshot ™ Initiative that is intended to accelerate cancer research. The overall goal for this Request for Applications (RFA) is to develop and validate novel biomarkers that accurately predict response to immuno-oncology interventions, including combination therapies, monotherapies, vaccines, and other novel immunotherapy treatments.

Background

Recent advances in cancer treatment have offered the prospect of greatly enhanced outcomes, prolonged survival, and cure for some patients. Much of the recent success has been driven by the development of

new immuno-oncology (IO) agents, leading to an explosion of translational research as well as investment in the field. To date, however, the improvements in outcomes and cure generated by the monotherapies of these agents are possible only for a minority of patients, and emerging data demonstrate that the greatest impact on cancer treatment will be achieved by combinations of multiple IO agents or of IO and non-IO agents. The successful pursuit of these combination therapies is complicated, however, by the sheer numbers of possible combinations, by high biologic complexity with the tumor and its host, and by the need for new translational biomarkers and assays to guide which patients should receive which combinations. These challenges are further compounded by the novelty and intensely competitive nature of the IO field, which has encouraged fragmented and at times duplicative research approaches.

To solve these challenges, a systematic cross-sector effort is required to identify and develop robust, standardized biomarkers and related clinical data that support the selection and testing of promising therapeutic combinations. To enable achievement of these goals, the National Institutes of Health (NIH) and multiple pharmaceutical companies have formed a 5-year, ~$220 million precompetitive public-private research collaboration called the Partnership for Accelerating Cancer Therapies (PACT).

PACT will facilitate robust, systematic, and uniformly conducted clinical testing of biomarkers that enable researchers and clinicians to better understand the mechanisms of response and resistance to treatment strategies. PACT will provide a systematic approach to immune and related oncology biomarker investigation in clinical trials by providing standardized and harmonized basic and exploratory biomarker assays, which can be utilized within the PACT programs and across the research community. These modules allow for (a) consistent generation of data, (b) access to uniform and harmonized assays to support data reproducibility, (c) comparability of data across trials, and (d) discovery/validation of new biomarkers for combination immunotherapies and related combinations.

This RFA has been released to drive the discovery and validation of new biomarkers in (d). To this point, PACT has selected three primary areas on which to focus this RFA:

1) Liquid Biopsy
2) Tumor Microenvironment
3) Microbiome

Biomarkers developed from this RFA funding in these key areas will be expected to take into account already ongoing biomarker assay development work that has been undertaken as part of the PACT/CIMAC-CIDC efforts, which have already been funded by the National Cancer Institute (NCI) and PACT in order to establish a network of Cancer Immune Monitoring and Analysis Centers (CIMACs) and a Cancer Immunologic Data Commons (CIDC) to provide consistent, standardized biomarker assays and data repository for NCI's extramural clinical trial networks. The NCI made awards to 4 academic research institutions: The University of Texas MD Anderson Cancer Center (PIs: Ignacio Wistuba, Chantale Bernatchez, Gheath Al-Atrash), Icahn School of Medicine at Mount Sinai (PI: Sacha Gnjatic), Stanford University (PIs: Holden Maecker, Sean Bendall), and Dana-Farber Cancer Institute (PIs: Catherine Wu and F. Stephen Hodi).

To this end, the PACT team is providing here the list of the initial assays that have been selected by the CIMACs for standardization, harmonization, and development (Figure 1). Those assays indicated as Tier 1 assays are those that will be prioritized for standardization and harmonization across centers and will be the priority focus for incorporation into trials. The data from these assays will also be the primary focus for initial incorporation into the CIDC. The Tier 2 assays will likely be run in individual CIMACs for the first 1-2 years of the project while the first round of standardization and harmonization are performed. Some of these assays, as well as the assays developed under this RFA, may be promoted to Tier 1 and expanded to all CIMAC laboratories for standardization, harmonization, and common use. The PACT team feels that this understanding is essential, so the applicant can understand the level of novelty necessary to be considered for this award.

Tier 1 = broadly recommended for most trials

Tier 2 = other assays, usage depends on trial

Currently being assessed for promotion to Tier 1

	Tier 1	Tier 2
Tissue Imaging	**Multiplex immunohistochemistry and immunofluorescence**	Conventional immunohistochemistry
		Multiplexed Ion-Beam Imaging (MIBI)

	Tier 1	Tier 2
Cell Profiling	**Mass Cytometry (CyTOF)**	High-dimensional flow cytometry
		ELISpot

	Tier 1	Tier 2
Sequencing	**RNA-Seq**	**TCR/BCR clonality**
	NanoString	Single-cell TCRseq
	Whole Exome Sequencing	HLA-Seq; Epitope prediction
		ISH DNA/RNA
		Neoantigen Prediction
		Cell-free DNA (circulating tumor DNA)
		Epigenomics (ATAC-Seq)
		HTG-EdgeSeq (gene expression)
		Microbiome (16S Deep Sequencing)
		Single-cell transcriptome

	Tier 1	Tier 2
Cytokines/Serum Analytes	**O-link serum cytokine analysis**	Luminex
		Seromics-ELISA/Grand serology
		MesoScale Discovery

Figure 1. Current CIMAC and PACT-selected Tier 1 and Tier 2 assays

The PACT team drafting this RFA has considered this ongoing work in selection of our topic areas of focus, which are further delineated below; therefore, the team would ask that applicants consider how their proposed assays could enhance this ongoing work or develop novel assays and biomarkers not already in the standardization and harmonization process.

Topic Area 1: Liquid Biopsy

Cancer as a genetic disease requires understanding of tumor genomics and epigenomics ((epi)genomics) to enable personalized medicine to become a reality. However, a significant barrier to understanding tumor (epi)genomics is the real-world limitation of tissue availability (including limitations on tissue quantity, quality, and tissue heterogeneity). Significant efforts towards the mitigation of this issue have been made with the advancement of ctDNA assessment, which is being evaluated in large prospective clinical trials and utilized in the clinic to select patients who are candidates for targeted therapies as well as to monitor response and resistance to such therapies. However, the implementation of "liquid biopsy" to define predictive biomarkers of response or resistance to immunotherapy and to monitor treatment response remains nascent (Gandara DR., 2018).

A holistic strategy centered on "liquid biopsy" that may give way to predictive signatures of IO response is the goal of this RFA, with an understanding that such signatures may be specific to a particular histology. The term "liquid biopsy" is used broadly in this RFA with the aim to use only blood sampling to capture tumor and tumor microenvironment (TME) characteristics associated with IO response or resistance.

The utilization of current or the development of novel technologies can enable identification of fundamental genomic characteristics from liquid biopsies that may be associated with response (e.g., high TMB, DDR pathway defects, *PBRM1* alterations, etc.,) or resistance (e.g., genomic alterations of IFN-g pathway genes, co-alteration of *KRAS/STK11* in NSCLC, defective core antigen processing and presenting machinery, etc.) to immunotherapy (Teo MY., 2018; Miao D., 2018; Gao J., 2016; Skoulidis F., 2018; Gettinger S., 2017). In addition, a blood-based assessment of tumor-derived exosomes (TEX) and non-tumor derived exosomes (non-TEX) to identify a molecular signature that can inform the immune status of the TME would be an ideal area of focus. TEX and non-TEX isolated from patients' plasma may be a relevant non-invasive biomarker that can inform disease, immune status, and potentially the presence of T-cell inflamed TME, and as such, may be used as a predictive biomarker of

immunotherapy response (Chen G., 2018). TEX identification and characterization of its molecular cargo is in its early stages and will require significant advancement and standardization of procedures used for TEX isolation from other plasma derived non-TEX isolates (Theodoraki MN., 2018). Characterization of the TEX and non-TEX molecular cargo (protein, DNA, RNA, miRNA) leading to generation of a signature predictive of IO therapy response or resistance would be the goal. Assessing TEX as a surrogate of inflammatory status of the TME, when coupled with ctDNA analysis, may significantly improve the ability to identify patients more likely to respond to IO (Cristescu R., 2018). Monitoring of TEX can also support a deeper understanding of PKPD relationship for non-IO and IO therapeutics in clinical development. As a result, TEX and non-TEX analysis is also a high priority biomarker target (Ludwig S., 2017; Marzia Del Re, 2018; Theodoraki MN., 2018; Theodoraki MN., 2018).

The scientific target for responses to this topic area would be to propose testing or assays that would help the IO field to understand tumor (epi)genomics, especially those that address identification of functional alterations from the "variants of unknown significance" in the core-DDR pathways and other cancer related genes, by sequencing circulating tumor cells (CTCs), ctDNA, and/or exosomes (DNA and RNA)and/or to understand the inflammatory status of the TME by characterization of TEX and non-TEX molecular cargo. To this end, the ideal response to this topic area would include plans for two primary assessments:

1) Generation of a ctDNA panel with IO focus, such as those referenced in paragraph three of this section, and
2) Development and standardization of protocols for TEX and Non-TEX analysis.

Topic Area 2: Tumor Microenvironment

The tumor microenvironment (TME) is a complex matrix of tumor, stroma and a diverse immune infiltrate. Many factors, both tumor-intrinsic and extrinsic, have been correlated with the patient response to cancer immunotherapy. Tumor-intrinsic determinants of response to immune checkpoint inhibition include the tumor mutational burden (TMB), neoantigen repertoire and tumor PD-L1 expression levels (Yarchoan et al., 2017; Hellman et al., 2018). Adaptive immune responses mediated by antigen-specific T cells are frequently directed against tumor neoantigens arising from cancer mutations (Tran et al., 2017; Danilova et al., 2018). Indeed, cancers that are deficient in DNA mismatch repair (MMR) are found to contain high mutational loads that are predictive of response to PD-1 blockade therapy (Le et al., 2015; Le et al., 2017; Overman et al., 2017); however, productive antitumor immunity is hampered by immune suppression in the TME and acquired resistance. Mechanisms of acquired resistance to adaptive immune responses and checkpoint blockade include TGF-β signaling/gene expression (Mariathasan et al., 2018), β-catenin (Jiang et al., 2018), HLA class I genotype (Chowell et al., 2017) and mutations in the genes encoding β2M, IFNGR1/2, JAK1/2 and STK11/LKB1 (Zaretsky et al., 2016; Sharma et al., 2017; Skoulidis et al., 2018).

Responses to checkpoint blockade have also been correlated with tumor-extrinsic factors such as the functional status and composition of the immune infiltrate including the degree of cytotoxic T cell (CTL) infiltration, the state of T cell dysfunction/anergy and the frequencies of regulatory T cells (Tregs), myeloid-derived suppressor cells (MDSCs) and tumor-associated macrophages (TAMs). A molecular profile of CD8+ T cell exhaustion was initially characterized using murine studies of chronic LCMV infection in which neutralization of the PD-1/PD-L1 axis was sufficient to restore proliferation, cytokine production and lytic function in exhausted LCMV-specific $CD8^+$ T cells (Barber et al., 2006; Wherry et al., 2007). The highest frequencies of tumor-reactive T cells in human non-small cell lung cancer (NSCLC) are found in the $CD8^+/PD\text{-}1^{high}$ tumor-infiltrating lymphocytes (TILs); however, the functional and transcriptomic profile of these cells only partially resembles that observed in chronic LCMV infection (Thommen et al., 2018).

To address the complexity and heterogeneity of the TME across cancer indications, systematic efforts have been undertaken to define the genomic and immunologic pathways that are operative in cancer and govern its interaction with innate and adaptive immunity (Thorsson et al., 2018; Hoadley et al., 2018; Campbell et al., 2018). Studies of T cell dysfunction and the development of TME transcriptomic profiles have been conducted to establish predictive biomarkers of response to immune checkpoint blockade (Jiang et al., 2018; Thommen and Schumacher, 2018; Thommen et al., 2018). Genomic, transcriptomic and proteomic signatures have the potential to be used as reliable immune checkpoint blockade biomarkers but require clinical trials with sufficient pretreatment samples to enable the validation of a multi-parameter biomarker. Additionally, data analytics and computational algorithms such as PRECOG (PREdiction of Clinical Outcomes from Genomic profiles), CIBERSORT (Cell type Identification By Estimating Relative Subsets Of known RNA Transcripts) and TIDE (Tumor Immune Dysfunction and Exclusion) warrant continued validation in patients treated with checkpoint modulation across a diversity of cancer indications. The examples of pathways and methodologies in this RFA are provided for context and should not be assumed as exclusive or preferential methodologies for the responses to this topic area. The development of novel and potentially transformative methods/endpoints are encouraged.

To further develop these prospective biomarkers, the goal of this topic area for proposals is to employ genomic (DNA), transcriptomic (RNA), and/or proteomic signatures of the TME to examine the prognostic relationship between response to cancer immunotherapy and the following:

1) The composition and functionality of the immune infiltrate.
    a. NK cell and $CD8^+$ TIL phenotype, metabolic disposition and functional status (e.g. proliferation, cytokine production, CTL activity and measures of stemness).
    b. Diversity and clonality of the TCR repertoire in the TIL fraction of primary human tumors.
    c. Relative frequencies and suppressive function of Tregs, MDSCs, and TAMs.
    d. Chemotactic networks (e.g. CXCR5/CXCL13 axis) and stromal extracellular matrix (e.g. TGF-β driven LOX gene expression) that govern the recruitment of innate and adaptive immune cells into the TME.

2) An assessment of the frequency and potency of tumor-intrinsic and acquired resistance mechanisms that limit the success of checkpoint blockade therapy.
    a. TMB, neoantigen repertoire, PD-L1 expression, APC processing/presentation machinery, and HLA class I genotype.
    b. Nucleic acid sensing pathway activation/regulation (e.g. RIG-I, MDA5, cGAS/STING) in tumor cells and innate immune infiltrates.
    c. Tumor-derived immunosuppressive soluble factors (e.g. TGF-β) that limit effector T cell function.
    d. Metabolic determinants of a productive vs. suppressive TME (e.g. hypoxia, pH, deprivation of nutrients (glucose and essential amino acids) and relative utilization of oxidative phosphorylation and glycolysis by tumor cells and immune cells.

Topic Area 3: Microbiome

In recent years, the microbiome has been emerging as a factor in the pathophysiology of multiple diseases, including metabolic syndromes, neurological diseases, autoimmune indications, and cancer. In addition to a role in the initiation of disease, data emerged a few years ago linking the gut microbiome with response to checkpoint inhibitors in animal models. Mice raised in different facilities with distinct gut microbiomes had differential responses to anti-PD-L1 therapy (Sivan, et al., 2015), while mice treated with broad spectrum antibiotics abrogated anti-CTLA-4 activity (Vetizou, et al., 2015). These findings have been extended into the clinic, with a number of studies demonstrating differences in the microbiome

of responders to PD-1 inhibition versus non-responders (Chaput, et al., 2017; Routy, et al., 2018). While no single species has emerged as a key contributor to any one disease or response to checkpoint inhibition, it has become clear that a lack of diversity or changes in the proportion of certain phyla are linked with lack of response to checkpoint inhibition.

One common criticism of studies undertaken thus far is that while they have generated valuable insights into the interplay between the microbiome in health and disease, few actionable hypotheses have emerged. The PACT project wishes to amend this by seeking proposals from investigators in the field of translational microbiome research for clinically applicable research projects aimed at identifying novel microbiome-related biomarkers associated with the immuno-oncology field. The microbial content of the colon is many fold greater than other parts of the GI tract, and the exposure of these microbes and their metabolic products to the immune system is greater in the colon, suggesting that this may be the anatomical region most worth exploring, not only for colorectal cancer but for other indications where checkpoint inhibitors have demonstrated activity. These biomarkers could fit into one of these areas:

1) The relationship between the diversity of the microbiome, or the presence or absence of specific genera or species, and outcome when patients are treated with immuno-oncology agents (including, but not limited to checkpoint inhibitors).
2) The relationship (if any) between the microbiome and the immune cell infiltration into the tumor microenvironment.
3) The relationship (if any) between the microbiome and the tumor genome.
4) Metabolites produced by specific genera that predict outcome and/or resistance to IO therapy.

Specific Research Objectives and Requirements

This RFA solicits applications for proposals to develop and analytically validate novel biomarkers that accurately predict response to immuno-oncology interventions, including combination therapies and monotherapies, with the goal of advancing cancer treatment and research. The overarching technical objective for responses to this RFA within the scientific topic areas above is to develop assays which satisfy the analytical validity definition as adopted by the Institute of Medicine. Any ctDNA assay proposed must accurately, reproducibly, and reliably detect and measure the variant(s) of interest, with statistical significance, (Merker JD., 2018). Standardization of the assay is critical, and the responses should outline the planned studies to be conducted to address analytical validity, such as evaluation of standardized reference materials with known characteristics along with cross-platform comparisons as recently outlined in a joint review by American Society of Clinical Oncology and College of American Pathologists (Merker JD., 2018). The ultimate eventual goal of the PACT effort is to assess the clinical validity and clinical utility of assays developed as part of this partnership. The proposed projects within the responses must be designed such that the assay developed could be incorporated into the clinical setting by the conclusion of the term of the award.

The conduct of clinical trials is outside of the scope of this current RFA, but applicants should take into consideration that the ultimate goal of the biomarkers developed will be clinical validation and eventually proof of clinical utility.

The topic areas of investigation targeted by this RFA are as follows:

1) For the topic area of Liquid Biopsy, a successful response to this area would include plans for at least one of these primary assessments with priority given to applications that address both:
    a. Generation of ctDNA panel with IO focus with priority given to those panels that address key areas of interest, including:

i. Assesses novel methods to define blood-based tumor mutational burden (bTMB) that are designed independent of existing NGS panels
ii. Inclusion of key genes within core-DDR pathways in addition to the genes currently available in standard gene panels that may be clinically actionable. Additionally, evidence to support that pathways/genes nominated for the panel inclusion are associated with a specific immune landscape would be ideal (Tissue based analysis of TME)
iii. Assesses functional impact of DDR alterations on surrogate genes (e.g., to observe microsatellite instability status) as a means to observe/confirm pathway defect
iv. Assesses functionality of the tumor APM and IFN-g pathway. Also includes genes that have demonstrated to potentially be predictors of response or resistance to IO.

b. Development and standardization of protocol for the characterization of the Tumor-derived exosome (TEX) and non-tumor derived exosome (non-TEX) molecular cargo (protein, DNA, RNA, miRNA) Ideally, association between the TEX and non-TEX immune status should be correlated with tissue-based analysis of the immune landscape.

2) For the topic of the Tumor Microenvironment, a successful response to this area would include assessments of at least one of the two overarching foci as well as at least one specific sub-topic:
   a. The composition and functionality of the immune infiltrate, with preferential focus on one of the following specified sub-topics:
      i. CD8+ TIL phenotype, metabolic disposition and functional status (e.g. proliferation, cytokine production, CTL activity and measures of stemness).
      ii. Diversity and clonality of the TCR repertoire in the TIL fraction of primary human tumors.
      iii. Relative frequencies and suppressive function of T-regs, MDSCs and TAMs.
      iv. Chemotactic networks (e.g. CXCR5/CXCL13 axis) and stromal extracellular matrix (e.g. TGF-β driven LOX gene expression) that govern the recruitment of innate and adaptive immune cells into the TME.

   b. Tumor-intrinsic and acquired resistance mechanisms that limit the success of checkpoint blockade therapy, with preferential focus on one of the following specified sub-topics:
      i. TMB, neoantigen repertoire, PD-L1 expression, APC processing/presentation machinery, and HLA class I genotype.
      ii. Nucleic acid sensing pathway activation/regulation (e.g. RIG-I, MDA5, cGAS/STING) in tumor cells and innate immune infiltrates.
      iii. Tumor-derived immunosuppressive soluble factors (e.g. TGF-β) that limit effector T cell function.
      iv. Metabolic determinants of a productive vs. suppressive TME (e.g. hypoxia, pH, deprivation of nutrients (glucose and essential amino acids) and relative utilization of oxidative phosphorylation and glycolysis by tumor cells and immune cells.

3) For the topic area of the Microbiome, a successful response to this area would include assessments of at least one of the following:
   a. The relationship between the diversity of the microbiome, or the presence or absence of specific genera or species, and outcome when patients are treated with immuno-oncology agents (including, but not limited to checkpoint inhibitors).
   b. The relationship (if any) between the microbiome and the immune cell infiltration into the tumor microenvironment.
   c. The relationship (if any) between the microbiome and the tumor genome.
   d. Metabolites produced by specific genera that predict outcome.

Award Information

I. Funds Available and Anticipated Number of Awards

The number of awards and the amount per award is contingent upon the submission of a sufficient number of meritorious applications and proper budget justification within the proposal.

II. Award Budget

Application budgets are limited to an amount up to $500,000 per year and need to reflect the actual needs of the development of the proposed biomarker/assay. Proper scientific and budget justification will need to be provided for evaluation. The committee reserves the right to award at a lower amount than requested.

III. Award Project Period

The scope of the proposed biomarker assay work should determine the award project period. The maximum project period is 2 years with the potential for a 1 year no-cost extension to complete the necessary analyses if the sample collection for the study lasts the duration of the original award.

Eligibility Information

Organizations eligible to apply are:

- Private or public sector
- US-based or international
- Able to comply with the necessary PACT IP, data sharing, and publication guidelines (Guidelines documents are available upon request).

Application and Submission Instructions

I. Submission Deliverables

Complete applications will include:

- Application write-up which should describe the information below, but more details can be provided in the application response template (Appendix 1):
  - biomarker assay(s) to be developed
  - rationale for why this biomarker would benefit the IO field, especially the advancement of patient selection for drug treatment
  - current state of the biomarker (i.e. – early stages, assay developed but need analytical validation, analytical validation done, but need clinical validation, etc.)
  - personnel that will conduct the work
- Detailed budget that delineates (An example budget table can be found in Appendix 1)
  - Personnel
  - Reagents and materials
  - Equipment
  - Sample acquisition (if necessary)
  - Other requirements for work proposed
- Detailed budget justification

- Proposed project timeline
- Biosketches from the Principal Investigators (Should not exceed 3 pages)

II. Data, Publications and Intellectual Property

All applicants will be expected to comply with the PACT Policies and Guidelines that have already been established for the partnership. These are available upon request and will be attached to any award agreements for those projects selected for funding.

III. Page Limit

Please keep your responses under 15 pages in length (single spaced, font 11 pt) not including biosketches. Further section length suggestions are provided in Appendix 1.

IV. Award Reporting

For those applications selected for award, the Principal Investigators on the award should expect to submit progress updates for the project every 6 months in a format that will be described in the award agreements.

V. Additional Information Required

Please provide any existing IP or patent information relevant to the assay that may affect its use in the partnership or the banking of any resulting data funded by this effort in a public controlled access database for use after initial publication of the findings. Further guidance available upon request.

VI. Submission Instructions

Send responses via e-mail to PACT@fnih.org with a copy to Dr. Stacey J. Adam, Director, Cancer Research Partnerships (sadam@fnih.org), and Jenny Peterson-Klaus, PACT Project Manager, Cancer Research Partnerships (jpeterson-klaus@fnih.org).

You may call 301-435-8364 with questions regarding the RFA or the submission process.

Key Dates

Application Due Date: January 21, 2019, 11:59 PM EST

Targeted Application Review Period: January 22, 2019 – February 22, 2019

Potential Oral Presentations from Finalists (*If Needed*): February 25-28, 2019
Applicants will be informed after initial review of proposals whether they will need to provide and oral presentation with the ability for Q&A to the PACT RFA Review Team

Targeted Award Announcement: March 2019*
Applicants will be notified by email of the outcome of the RFA.

**If no adequate submissions are received in this timeline, the FNIH reserves the right to extend the target deadline.*

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