Oportunidades en el Marco de financiación de EEUU: National Institutes of Health (NIH) y otras iniciativas internacionales.

XXV JORNADA DE PROYECTOS EUROPEOS DE LA UNIVERSIDAD DE MURCIA, 22 de Octubre de 2015

Dr. Ana Mª Blanco, Oficina Internacional IIS La Fe

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1.- <u>Financiación USA</u>: NIH (National Institutes of Health), CDRMP (U.S. Department of Defense), PCORI (Patient-Centered Outcomes Research Institute).

Financiación USA: National Institutes of Health (NIH).

Financiación USA: National Institutes of Health (NIH).

MAYOR ENTIDAD DE FINANCIACIÓN DE PROYECTOS CIENTÍFICO-TECNOLÓGICOS A NIVEL MUNDIAL.

Financiación USA: National Institutes of Health (NIH).

- NCI — National Cancer Institute
- NEI — National Eye Institute
- NHLBI — National Heart, Lung, and Blood Institute
- NIDDK — National Institute of Diabetes and Digestive and Kidney Diseases
- NHGRI — National Human Genome Research Institute
- NIAAA — National Institute on Alcohol Abuse and Alcoholism
- NIAID — National Institute of Allergy and Infectious Diseases
- NIA — National Institute on Aging
- NIAMS — National Institute of Arthritis and Musculoskeletal and Skin Diseases
- NIAMS — National Institute of Biomedical Imaging and Bioengineering
- NICHD — *Eunice Kennedy Shriver* National Institute of Child Health and Human Development
- NIDCD — National Institute on Deafness and Other Communication Disorders
- NLM — U.S. National Library of Medicine
- NIDCR — National Institute of Dental and Craniofacial Research
- NIDA — National Institute on Drug Abuse. *The Science of Drug Abuse & Addiction*
- NIGMS — National Institute of General Medical Sciences
- NIMH — National Institute of Mental Health
- NIMHD — National Institute on Minority Health and Health Disparities
- NINDS — National Institute of Neurological Disorders and Stroke
- NINR — National Institute of Nursing Research

Financiación USA: National Institutes of Health (NIH).

FINANCIACION TOTAL POR AÑOS

Fiscal Year	Application Type	NIH Institutes / Centers	Activity Code	Number of Applications Reviewed	Number of Applications Awarded	Success Rate[2]	Total Funding[3]
2010	FY Total	All Institutes	Total	45.983	9.455	20,6%	3.953.126.083 USD
2011	FY Total	All Institutes	Total	49.592	8.765	17,7%	3.751.173.768 USD
2012	FY Total	All Institutes	Total	51.313	9.032	17,6%	3.811.804.254 USD
2013	FY Total	All Institutes	Total	49.581	8.310	16,8%	3.513.047.712 USD
2014	FY Total	All Institutes	Total	51.073	9.241	18,1%	4.494.169,749 USD

FINANCIACION NON-US (FOREIGN) TOTAL POR AÑOS

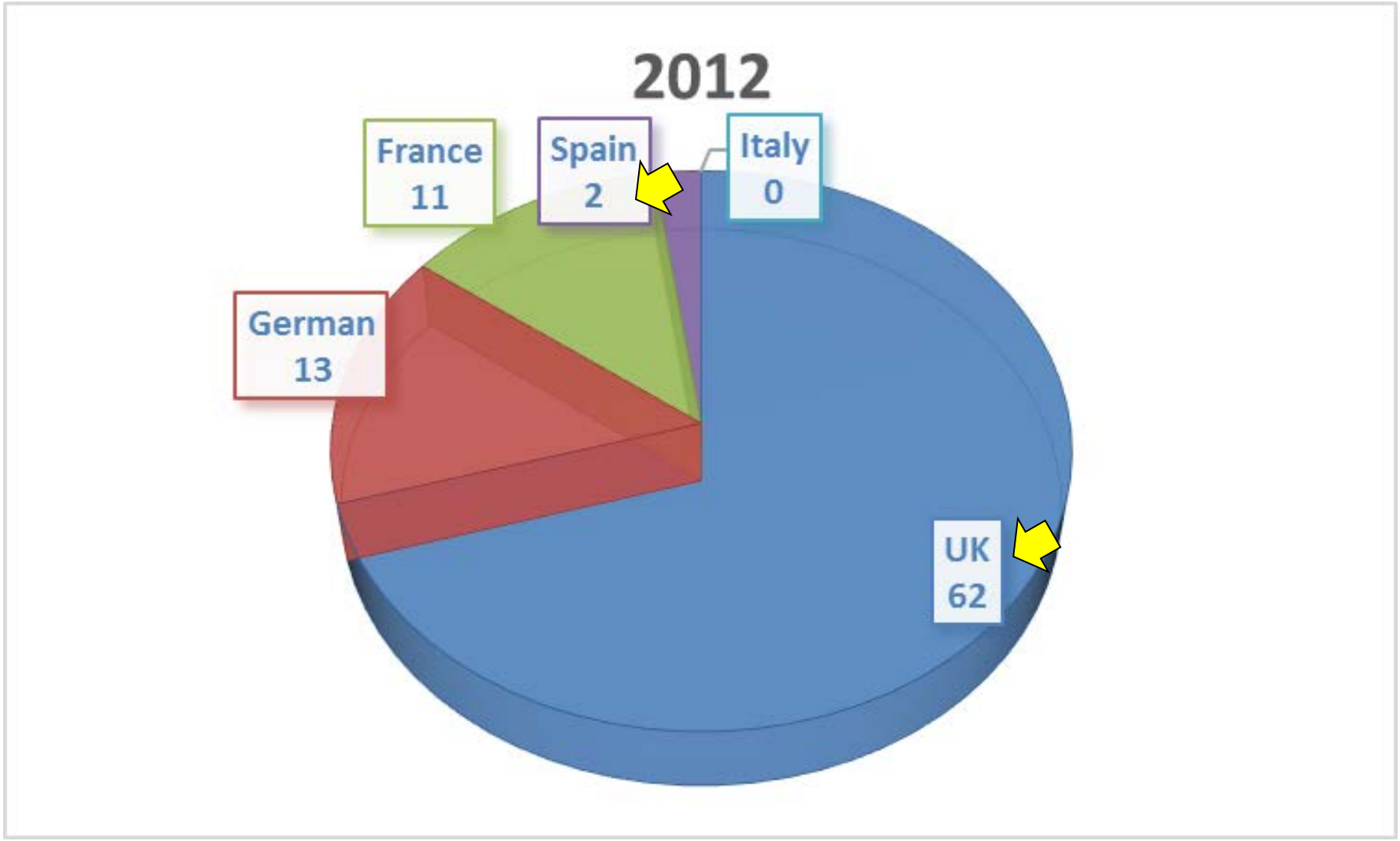
Fiscal Year	NIH Institutes / Centers	Activity Code	Number of Applications Awarded	Total Funding[3]
2012	All Institutes	Total	534	226.564.007 USD
2013	All Institutes	Total	480	232.230.209 USD
2014	All Institutes	Total	481	181.501.478 USD

Fiscal Year	NIH Institutes / Centers	Activity Code	Number of Applications Awarded	Total Funding[3]
Present 2015	All Institutes	Total	180	62.080.107 USD

ESPAÑA COMO SOLICITANTES??

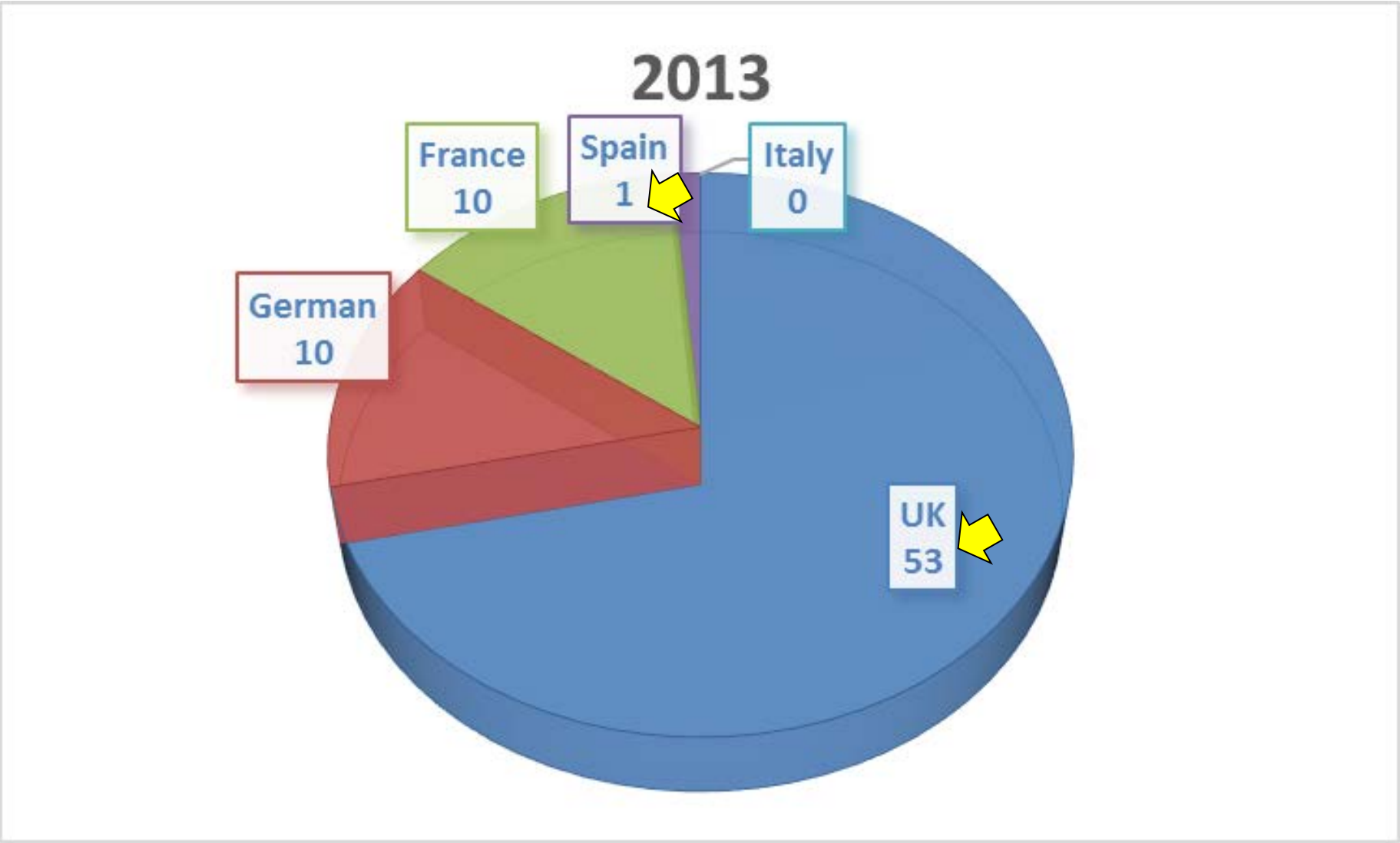
Fuente: Office of Research Information Systems (ORIS) / Office of Statistical Analysis and Reporting (OSAR) / www.report.nih.gov.

Financiación USA: National Institutes of Health (NIH).



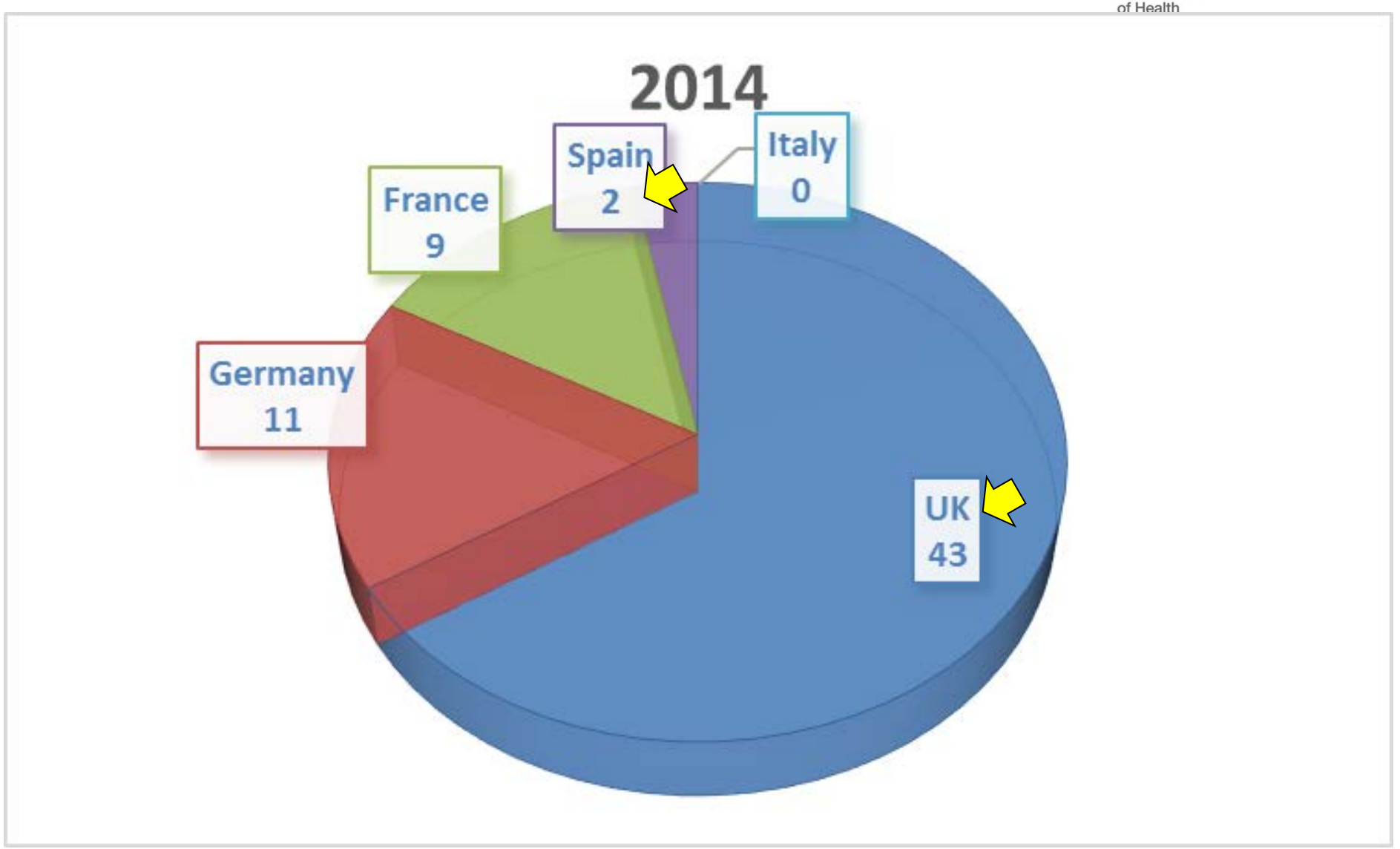
Fuente: Office of Research Information Systems (ORIS) / Office of Statistical Analysis and Reporting (OSAR) / www.report.nih.gov.

Financiación USA: National Institutes of Health (NIH).



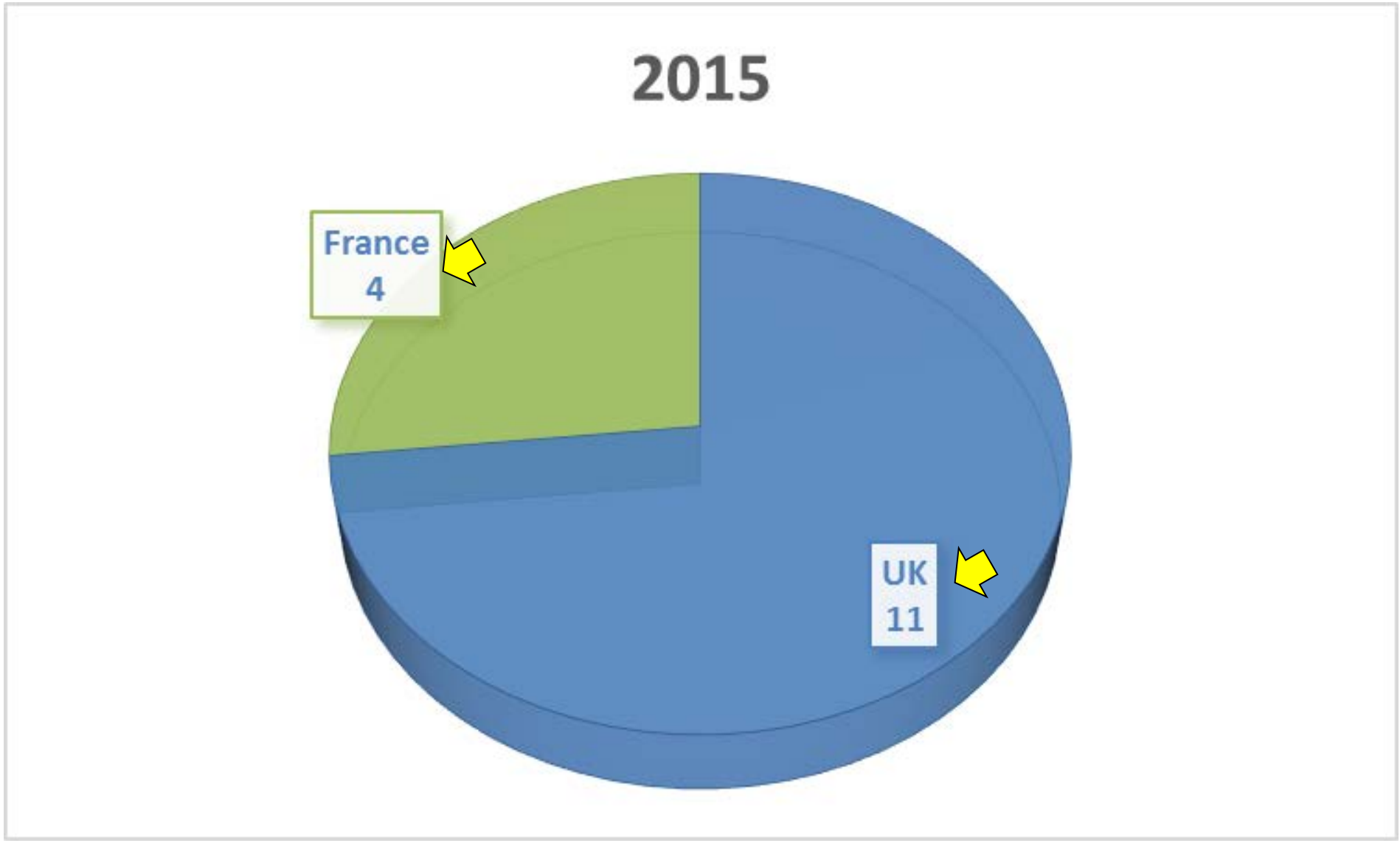
Fuente: Office of Research Information Systems (ORIS) / Office of Statistical Analysis and Reporting (OSAR) / www.report.nih.gov.

Financiación USA: National Institutes of Health (NIH).



Fuente: Office of Research Information Systems (ORIS) / Office of Statistical Analysis and Reporting (OSAR) / www.report.nih.gov.

Financiación USA: National Institutes of Health (NIH).



Fuente: Office of Research Information Systems (ORIS) / Office of Statistical Analysis and Reporting (OSAR) / www.report.nih.gov.

Financiación USA: National Institutes of Health (NIH).

Tipos de Grants:

RESEARCH GRANTS (R series)

CAREER DEVELOPMENT AWARDS (K series)

RESEARCH TRAINING & FELLOWSHIPS (T&F series)

PROGRAM PROJECT/CENTER GRANTS (P series)

NON U.S. ELEGIBILITY

R01
R03
R21

MODALIDADES DE FINANCIACIÓN

Financiación USA: NIH.

R01: NIH Research Project Grant Program (R01)

- Se utiliza para financiar un proyecto específico de investigación, es una **subvención**.
- **No hay límite** específico de presupuesto.
- Se requiere permiso si se va a solicitar $500K o más (costes directos) en un año.
- En general es para proyectos entre **3- 5 años**.
- Se requieren **resultados preliminares** contundentes.

R03: NIH Small Grant Program (R03)

- Subvención limitada a un **período corto de tiempo** para apoyar tipos de proyectos como:
estudios piloto o de factibilidad, ejecución de datos preliminares, análisis secundarios de datos existentes, proyectos de investigación cortos, desarrollo de una nueva tecnología, etc.
- Hay límite de presupuesto hasta $ 50.000 por año (**Máximo $ 100.000**).
- Máxima duración del proyecto son **2 años**.
- No es renovable.
- **No se requieren resultados preliminares**.

Financiación USA: NIH.

R21: NIH Exploratory/Developmental Research Grant Award (R21)

● La subvención R21 pretende fomentar **nuevos proyectos de investigación exploratorios y de desarrollo**.

- La viabilidad de una nueva área de investigación o de un nuevo sistema experimental que tiene el potencial de mejorar la salud.
- Uso único e innovador de una metodología existente para explorar un área científica nueva.

Estos estudios pueden implicar un riesgo considerable, pero puede dar lugar a un gran avance en un área en particular.

● Máxima duración del proyecto son **2 años**.

● El presupuesto total no puede exceder los **$275.000**. En un año el presupuesto máximo debe ser $ 200.000.

● **No se requieren resultados preliminares** porque en esta convocatoria se asume un **cierto riesgo**, pero se valora muy positivamente que se presenten estos resultados preliminares aunque no sean muy contundentes.

Instituto de Investigación Sanitaria La Fe

Financiación USA: NIH.

NIH National Institutes of Health

CONVOCATORIAS "PARENT CALLS"

R01: http://grants.nih.gov/grants/guide/pa-files/PA-13-302.html
R21: http://grants.nih.gov/grants/guide/pa-files/PA-13-303.html
R03: http://grants.nih.gov/grants/guide/pa-files/PA-13-304.html

Con 3 fechas límite/año

EXPIRAN EN EL AÑO 2016

R01: 5 febrero, 5 junio y 5 octubre.
R21 y R03: 16 febrero, 16 junio y 16 octubre.

Financiación USA: NIH.

Department of Health and Human Services

Part 1. Overview Information

Participating Organization(s)	National Institutes of Health (NIH)
Components of Participating Organizations	National Cancer Institute (NCI) National Eye Institute (NEI) National Heart, Lung, and Blood Institute (NHLBI) National Human Genome Research Institute (NHGRI) National Institute on Aging (NIA) National Institute on Alcohol Abuse and Alcoholism (NIAAA) National Institute of Allergy and Infectious Diseases (NIAID) National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) National Institute of Biomedical Imaging and Bioengineering (NIBIB) *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD) National Institute on Deafness and Other Communication Disorders (NIDCD) National Institute of Dental and Craniofacial Research (NIDCR) National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) National Institute on Drug Abuse (NIDA) National Institute of Environmental Health Sciences (NIEHS) National Institute of General Medical Sciences (NIGMS) National Institute of Mental Health (NIMH) National Institute on Minority Health and Health Disparities (NIMHD) National Institute of Neurological Disorders and Stroke (NINDS) National Institute of Nursing Research (NINR) National Library of Medicine (NLM) National Center for Complementary and Integrative Health (NCCIH formerly NCCAM) Division of Program Coordination, Planning and Strategic Initiatives, Office of Research Infrastructure Programs (ORIP)
Funding Opportunity Title	**Research Project Grant (Parent R01)**
Activity Code	R01 Research Project Grant
Announcement Type	Reissue of PA-11-260
Related Notices	• April 16, 2015 - Notice of Information: NIMH High-Priority Areas for Research on Women's Mental Health During Pregnancy and the Postpartum Period . See Notice NOT-MH-15-013. • September 25, 2014 - See Notice NOT-MH-14-033. Notice of Information on High-Priority Research Areas to Understand and Reduce Mental

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Financiación USA: NIH.

NIH National Institutes of Health

BÚSQUEDA DE CONVOCATORIAS ESPECÍFICAS NIH

POR PALABRAS CLAVE/DISCIPLINA/ENFERMEDAD

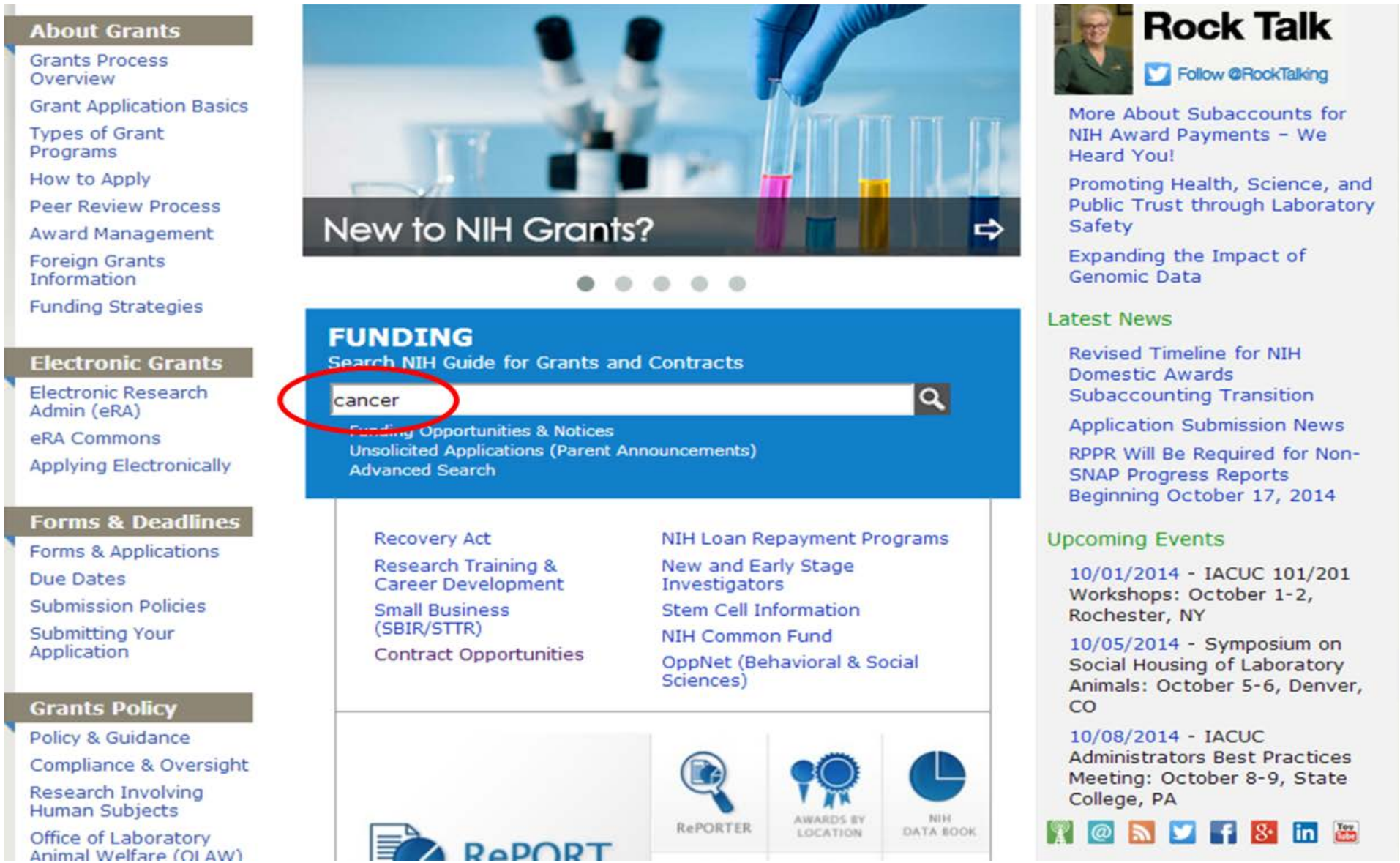
POR EJEMPLO

CANCER

Instituto de Investigación Sanitaria La Fe

http://grants.nih.gov/grants/oer.htm

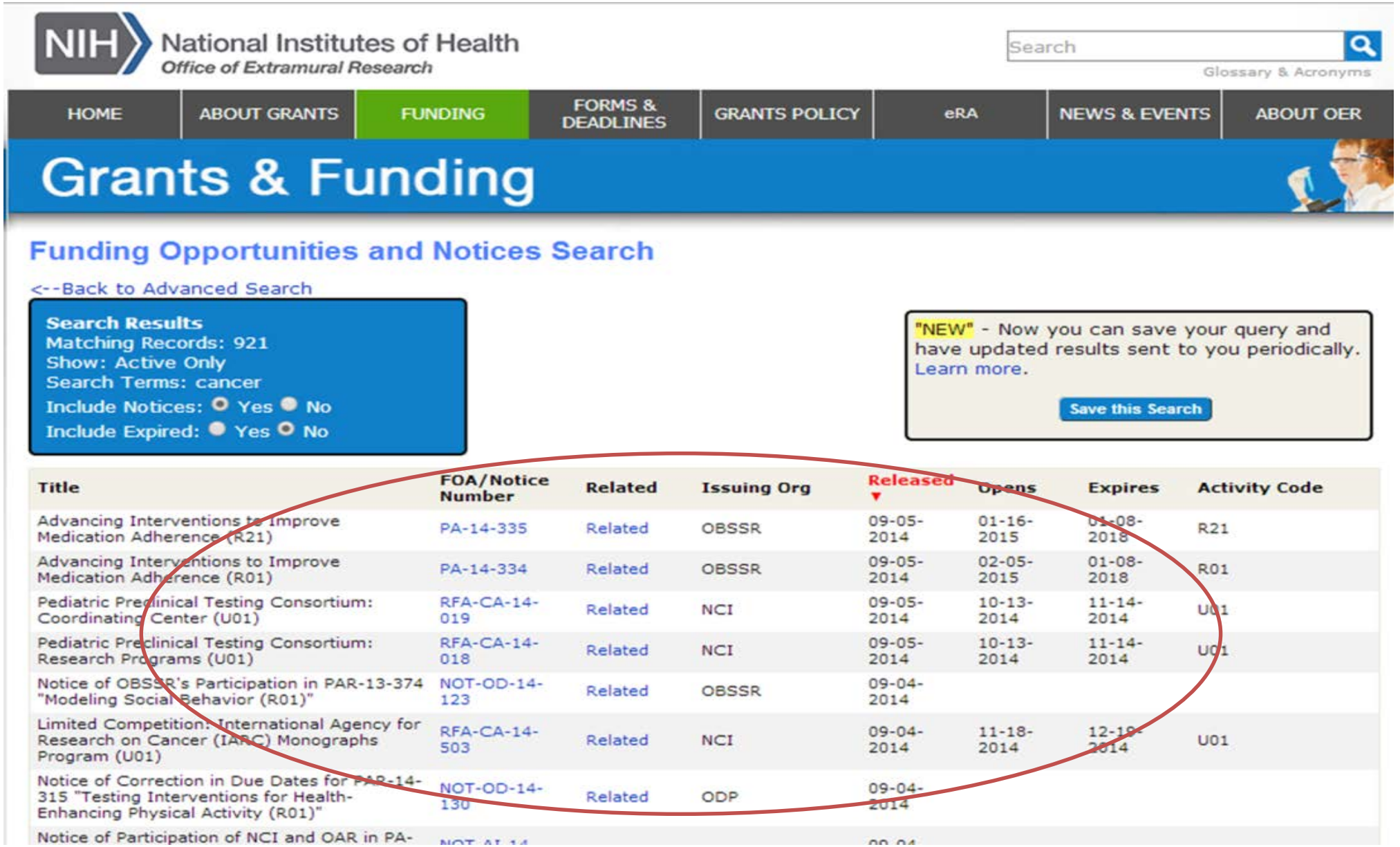
National Institutes of Health



Instituto de Investigación Sanitaria La Fe

http://grants.nih.gov/grants/oer.htm

National Institutes of Health



Title	FOA/Notice Number	Related	Issuing Org	Released	Opens	Expires	Activity Code
Advancing Interventions to Improve Medication Adherence (R21)	PA-14-335	Related	OBSSR	09-05-2014	01-16-2015	01-08-2018	R21
Advancing Interventions to Improve Medication Adherence (R01)	PA-14-334	Related	OBSSR	09-05-2014	02-05-2015	01-08-2018	R01
Pediatric Preclinical Testing Consortium: Coordinating Center (U01)	RFA-CA-14-019	Related	NCI	09-05-2014	10-13-2014	11-14-2014	U01
Pediatric Preclinical Testing Consortium: Research Programs (U01)	RFA-CA-14-018	Related	NCI	09-05-2014	10-13-2014	11-14-2014	U01
Notice of OBSSR's Participation in PAR-13-374 "Modeling Social Behavior (R01)"	NOT-OD-14-123	Related	OBSSR	09-04-2014			
Limited Competition: International Agency for Research on Cancer (IARC) Monographs Program (U01)	RFA-CA-14-503	Related	NCI	09-04-2014	11-18-2014	12-19-2014	U01
Notice of Correction in Due Dates for PAR-14-315 "Testing Interventions for Health-Enhancing Physical Activity (R01)"	NOT-OD-14-130	Related	ODP	09-04-2014			
Notice of Participation of NCI and OAR in PA-							

Financiación USA: NIH.

R01: **Early-life Factors and Cancer Development Later in Life**. PA-15-126.
http://grants.nih.gov/grants/guide/pa-files/PA-15-126.html

R21: **Early-life Factors and Cancer Development Later in Life**. PA-15-125.
http://grants.nih.gov/grants/guide/pa-files/PA-15-125.html

R03: **Early-life Factors and Cancer Development Later in Life**. PA-15-124.
http://grants.nih.gov/grants/guide/pa-files/PA-15-124.html

La convocatoria termina en el año 2018.

Tres fechas límite anuales:

R01: 5 febrero, 5 junio y 5 octubre
R21 y R03: 16 febrero, 16 junio y 16 octubre.

The purpose of this Funding Opportunity Announcement (FOA) is to stimulate **research focused on the role of early-life factors in cancer development later in life.** Given that current emerging evidence from limited research indicates a potentially important role for early-life events and exposures in cancer development, **it is necessary to better understand 1) the early-life (maternal-paternal, in utero, birth and infancy, puberty and adolescence, and teenage and young adult years) factors that are associated with later cancer development; 2) how early-life factors mediate biological processes relevant to carcinogenesis; and 3) whether predictive markers for cancer risk based on what happens biologically at early-life can be measured and developed for use in cancer prevention strategies.** Markers that predict malignancy or pre-malignant conditions would allow assessment of early-life exposures with relevant outcomes without having to wait 50 years for cancer development. Ultimately, a better mechanistic understanding of how early-life events and exposures contribute to the etiology of cancer later in life will allow for the **development of effective interventions during pregnancy or early life** that may have a profound impact on cancer prevention.

Department of Health and Human Services

Part 1. Overview Information

Participating Organization(s)	National Institutes of Health (NIH)
Components of Participating Organizations	National Cancer Institute (NCI) Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) National Institute of Environmental Health Sciences (NIEHS)
Funding Opportunity Title	Early-life Factors and Cancer Development Later in Life (R01)
Activity Code	R01 Research Project Grant
Announcement Type	New
Related Notices	None
Funding Opportunity Announcement (FOA) Number	PA-15-126
Companion Funding Opportunity	PA-15-125, R21 Exploratory/Developmental Grant PA-15-124, R03 Small Grant Program
Number of Applications	See Section III. 3. Additional Information on Eligibility.
Catalog of Federal Domestic Assistance (CFDA) Number(s)	93.393; 93.113; 93.865
Funding Opportunity Purpose	The purpose of this Funding Opportunity Announcement (FOA) is to stimulate research focused on the role of early-life factors in cancer development later in life. Given that current emerging evidence from limited research indicates a potentially important role for early-life events and exposures in cancer development, it is necessary to better understand 1) the early-life (maternal-paternal, in utero, birth and infancy, puberty and adolescence, and teenage and young adult years) factors that are associated with later cancer development; 2) how early-life factors mediate biological processes relevant to carcinogenesis; and 3) whether predictive markers for cancer risk based on what happens biologically at early-life can be measured and developed for use in cancer prevention strategies. Markers that predict malignancy or pre-malignant conditions would allow assessment of early-life exposures with relevant outcomes without having to wait 50 years for cancer development. Ultimately, a better mechanistic understanding of how early-life events and exposures contribute to the etiology of cancer later in life will allow for the development of effective interventions during pregnancy or early life that may have a profound impact on cancer prevention.

Part 2. Full Text of

Section I. Funding Opportunity

Purpose

The purpose of this Funding Opportunity Announceme
emerging evidence from limited research indicates a

1) the early-life (maternal-paternal, in utero, birth
development;

2) how early-life factors mediate biological proces

3) whether predictive markers for cancer risk bas
that predict malignancy or pre-malignant conditio
development.

Ultimately, a better mechanistic understanding of how
interventions during pregnancy or early life that may

Background

Most of the epidemiology research conducted to date
consequences for cancer development later in life.

- There is good evidence that prenatal exposure
vagina, and breast in the daughters of the wom
- Other evidence shows that early menarche has
period is critical in the etiology of this disease.
- Size at birth (i.e., birth weight and length) also
- In addition to early growth, having been breast
- Other in utero and early-life factors have been
have been associated with several adult malig
almost all invasive cervical cancer cases world
- A single acute exposure to radiation to those w
elevated risk of malignancies decades later.
- Animal studies have also provided support to li
epigenetic modifications and phenotypic changes in the off-spring that are potentially linked to cancer.

NIH Efforts

To explore the links between early life factors and cancer development and to explore ways to strategically support cancer research focused on early life factors and events a series

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Section II. Award Information
Section III. Eligibility Information
Section IV. Application and Submission Information
Section V. Application Review Information
Section VI. Award Administration Information
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Section VIII. Other Information

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NIH Efforts

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Section III. Eligibility Information

1. Eligible Applicants

Eligible Organizations

Higher Education Institutions

- Public/State Controlled Institutions of Higher Education
- Private Institutions of Higher Education

The following types of Higher Education Institutions are always encouraged to apply for NIH support as Public or Private Institutions of Higher Education:

- Hispanic-serving Institutions
- Historically Black Colleges and Universities (HBCUs)
- Tribally Controlled Colleges and Universities (TCCUs)
- Alaska Native and Native Hawaiian Serving Institutions
- Asian American Native American Pacific Islander Serving Institutions (AANAPISIs)

Foreign Institutions

Non-domestic (non-U.S.) Entities (Foreign Institutions) **are** eligible to apply.

Non-domestic (non-U.S.) components of U.S. Organizations **are** eligible to apply.

Foreign components, as defined in the *NIH Grants Policy Statement*, **are** allowed.

- Indian/Native American Tribal Governments (Federally Recognized)
- Indian/Native American Tribal Governments (Other than Federally Recognized)
- Eligible Agencies of the Federal Government
- U.S. Territory or Possession

Other

- Independent School Districts
- Public Housing Authorities/Indian Housing Authorities
- Native American Tribal Organizations (other than Federally recognized tribal governments)
- Faith-based or Community-based Organizations
- Regional Organizations
- Non-domestic (non-U.S.) Entities (Foreign Institutions)

Foreign Institutions

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Non-domestic (non-U.S.) components of U.S. Organizations are eligible to apply.
Foreign components, as defined in the *NIH Grants Policy Statement*, are allowed.

Required Registrations

Applicant Organizations

Instituto de Investigación Sanitaria La Fe

Financiación USA: NIH.

NIH National Institutes of Health

BÚSQUEDA DE CONVOCATORIAS ESPECÍFICAS NIH

POR PALABRAS CLAVE/DISCIPLINA/ENFERMEDAD

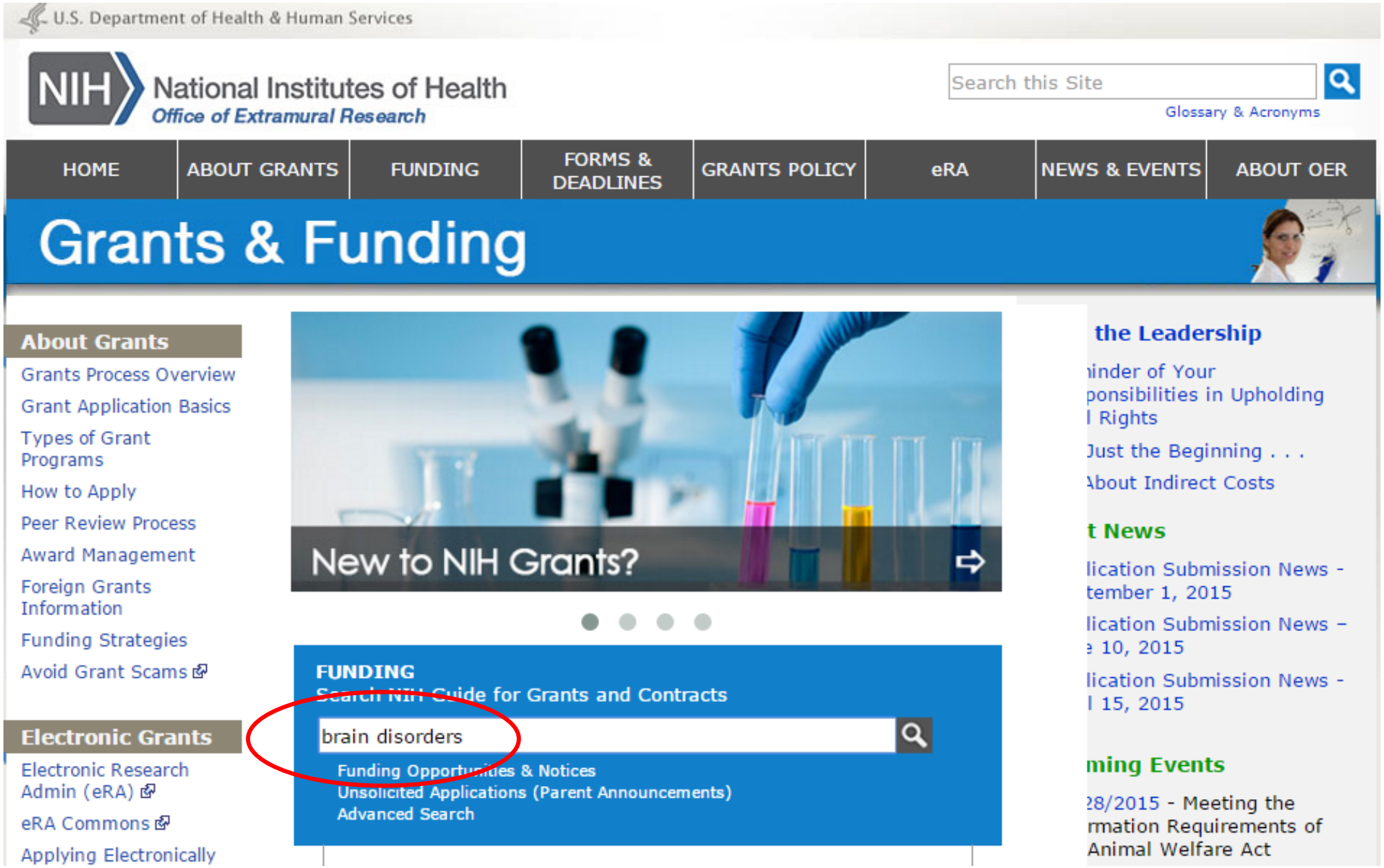
POR EJEMPLO

BRAIN DISORDERS

Instituto de Investigación Sanitaria La Fe

http://grants.nih.gov/grants/oer.htm

NIH National Institutes of Health



Financiación USA: NIH.

National Institutes of Health

http://grants.nih.gov/grants/oer.htm

MRI-Based Neuroimaging Datasets from Diverse Sources	010	[illegible]	[illegible]	2015			
Molecular and Cellular Substrates of Complex Brain Disorders (R01)	PAR-14-309	Related	NIMH	08-04-2014	09-05-2014	09-08-2017	R01
Molecular and Cellular Substrates of Complex Brain Disorders (R21)	PAR-14-310	Related	NIMH	08-04-2014	09-16-2014	09-08-2017	R21
Notice of Correction to PAR-14-153 "Temporal Dynamics of Neurophysiological Patterns as Potential Targets for Treating Cognitive Deficits in Brain Disorders (R01)"	NOT-MH-14-013	Related	NIMH	03-14-2014			
Temporal Dynamics of Neurophysiological Patterns as Potential Targets for Treating Cognitive Deficits in Brain Disorders (R01)	PAR-14-153	Related	NIMH	03-14-2014	05-05-2014	05-08-2017	R01
Temporal Dynamics of Neurophysiological Patterns as Potential Targets for Treating Cognitive Deficits in Brain Disorders (R21)	PAR-14-158	Related	NIMH	03-14-2014	05-16-2014	05-08-2017	R21
Notice of Informational Conference Calls for Prospective Applicants to NIH BRAIN Initiative Funding Opportunity Announcements	NOT-NS-14-005	Related	NINDS	01-06-2014			

Financiación USA: NIH.

R01: **Molecular and Cellular Substrates of Complex Brain Disorders**. PAR-14-309.
http://grants.nih.gov/grants/guide/pa-files/PAR-14-309.html

R21: **Molecular and Cellular Substrates of Complex Brain Disorders**. PAR-14-310.
http://grants.nih.gov/grants/guide/pa-files/PAR-14-310.html

This Funding Opportunity Announcement (FOA) encourages research grant applications directed toward the discovery of the **impact of alterations associated with complex brain disorders on the fundamental cellular and molecular substrates of neuronal function**.

La convocatoria termina en el año 2017.

Tres fechas límite anuales:

R01: 5 febrero, 5 junio y 5 octubre
R21: 16 febrero, 16 junio y 16 octubre.

Department of Health and Human Services
Part 1. Overview Information

Participating Organization(s)	National Institutes of Health (NIH)
Components of Participating Organizations	National Institute of Mental Health (NIMH)
Funding Opportunity Title	Molecular and Cellular Substrates of Complex Brain Disorders (R01)
Activity Code	R01 Research Project Grant
Announcement Type	Reissue of PAR-11-299
Related Notices	None
Funding Opportunity Announcement (FOA) Number	PAR-14-309
Companion Funding Opportunity	PAR-14-310, R21 Exploratory/Developmental Grant
Number of Applications	See Section III. 3. Additional Information on Eligibility.
Catalog of Federal Domestic Assistance (CFDA) Number(s)	93.242
Funding Opportunity Purpose	This Funding Opportunity Announcement (FOA) encourages research grant applications directed toward the discovery of the impact of alterations associated with complex brain disorders on the fundamental cellular and molecular substrates of neuronal function.

Key Dates

Posted Date	August 4, 2014
Open Date (Earliest Submission Date)	September 5, 2014
Letter of Intent Due Date(s)	Not Applicable
Application Due Date(s)	Standard dates apply, by 5:00 PM local time of applicant organization. All types of non-AIDS applications allowed for this funding opportunity announcement are due on these dates.

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Part 2. Full Text of

Section I. Funding Opp

Disorders of complex brain function such as schizoph
neuroscience. Impairments in complex brain function
developing effective treatments has been slow since
with the recent emergence of potential disease-assoc
assay technologies, neuroscientists are now poised t
perspective of hypothesis testing for disease relevanc
contribute to disorders of complex brain functions.

To facilitate progress towards understanding the biolo
processes act at the basic molecular, cellular, and cir
third messengers, neuromodulators, neurotrophins an
neuron-glia communication; oxidative, immunological
dynamics such as excitatory/inhibitory balance or pro

This funding opportunity encourages the submission
understanding the biological mechanisms behind puta
disorders. Applications submitted to this FOA should
mechanisms at the molecular, cellular and circuit leve
disorder). Rather, applicants are encouraged to addre

Examples of relevant research include, but are not lim

- Studies aimed at exploring the molecular, cellul
pathways associated with brain disorders.
- Investigations of epigenetic or environmental fa
- Exploratory studies including innovative in vitro
disorders and/or to identify novel potential treat
- Optimization and implementation of novel cell-b
associated alterations in cell processes.
- Studies to identify the functions of molecules li
neuroimmune/neuroinflammatory, environmenta
- Development/optimization of new biological too
to complex brain disorders.

Applications submitted to this FOA should propose w
the mission of NIMH (see: http://www.nimh.nih.gov/about/strategic-planning-reports/nimh-strategic-plan-2008.pdf). Projects with a primary focus on behavioral measures, pharmacology or drug discovery, modeling mental disorder symptoms, human subjects or clinical populations (except for generation of iPS cells) and the neural substrates of neurodegenerative disorders are not appropriate for support under this announcement. The NIMH has high interest in applications that incorporate the longitudinal trajectory of

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Financiación USA: NIH.

CARACTERÍSTICAS IMPORTANTES

- Registro USA: SAM, eRA y Grants.
-  Multiples IPs.
-  100% Subvención proyectos I+D
-  Posibilidad “Resubmission”
- NO Colaborador USA
-  Principios éticos
-  Único % de costes indirectos 8%
-  Special in USA

Financiación USA: NIH.

REGISTROS DE LA INSTITUCIÓN **(6 semanas antes del deadline)**

1) **Dun and Bradstreet Universal Numbering System (DUNS):** http://fedgov.dnb.com/webform

2) **System for Award Management (SAM):** https://www.sam.gov/portal/SAM/##11. Necesidad de renovación anual. España: necesaria obtención de NATO Commercial and Government Entity (NCAGE) https://eportal.nspa.nato.int/AC135Public/scage/CageList.aspx.

3) **eRA Commons:** https://**commons.era**.nih.gov/. Se registra la institución con un Signing Official (SO) y Accounts Administrator (AA). Todos los IPs e investigadores de la institución que vayan a participar en el proyecto deben registrarse.

4) **Grants.gov:** http://www.grants.gov/web/grants/applicants/organization-registration.html. Se designa un Authorized Organization Representative (AOR) para envoi de propuestas.

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TIMELINE

Mes 1-3

Recepción y remisión: Asignación de la propuesta a revisores específicos.

Mes 6-8

Proceso de revisión: Dos niveles de revisión, asignación de nota (10-90), Summary Statement (documento de críticas de revisores).

Mes 9-10

Proceso de concesión: Notificación del premio, Ajustes administrativos.

Mes 9-12

Proceso de resumisión: Contacto con NIH para condiciones de resumisión, Re-escritura del proyecto subsanando críticas de Revisores.

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DOCUMENTOS solicitud NIH:

- ❑ Research strategy (6 o 12 páginas)
- ❑ Abstract (30 líneas)
- ❑ Specific aims (1 página)
- ❑ Project Narrative (4 líneas)
- ❑ Bibliography
- ❑ Facilities
- ❑ Equipment
- ❑ Foreign justification
- ❑ Biographical sketches (5 páginas)
- ❑ Budget
- ❑ Cover letter
- ❑ Protection of human subjects
- ❑ Vertebrate animals
- ❑ Letters of support
- ❑ Resource sharing plan
- ❑ Leadership Plan*
- ❑ Consortium-Contractual Arrangements*

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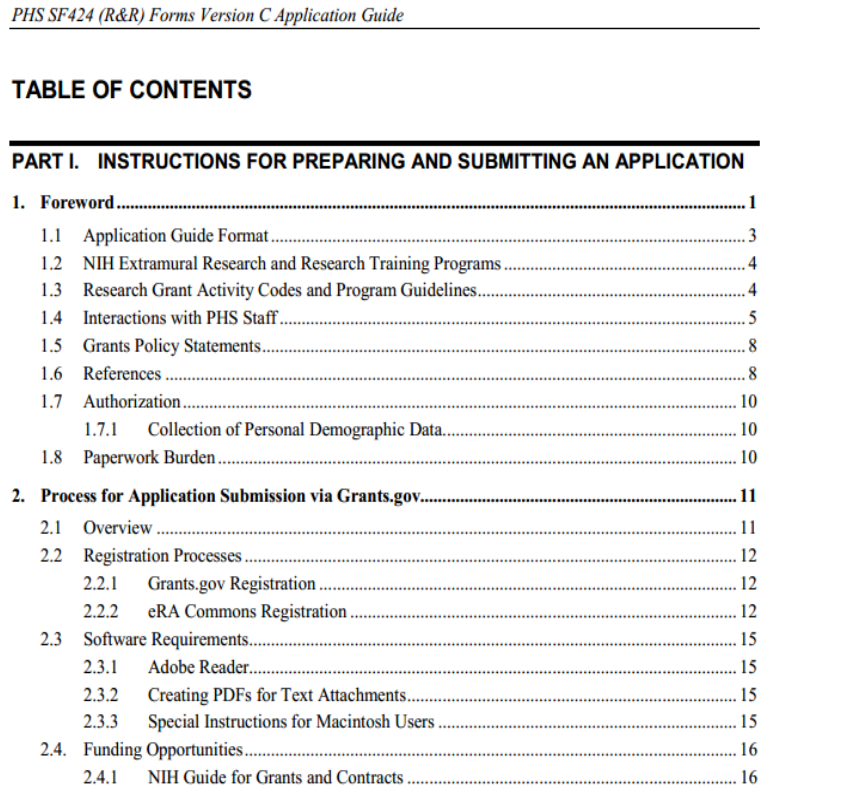
http://grants.nih.gov/grants/funding/424/index.htm#inst



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EJEMPLO RESEARCH STRATEGY R21 (6 pages)

RESEARCH STRATEGY

APPROACH

PRELIMINARY RESULTS

INNOVATION

RESEARCH STRATEGY

(a) SIGNIFICANCE The developing human immune system faces a balancing act that must be carefully timed. On the one hand, it must tolerate the presence of the surrounding mother and her non-inherited maternal alloantigens (NIMA) or otherwise risk the potential of engaging a fatal "graft vs. host" disease. On the other hand, novel antigens must be recognized as foreign when encountered after birth, triggering a vigorous adaptive immune response (*e.g.*, with cytolytic T cells and neutralizing antigens) against them. Otherwise, the newborn will be susceptible to diseases caused by multiple infectious agents.

In ongoing experiments, we have obtained preliminary data (see below) indicating that this switch from a fetal-type to an adult-type immune response is dependent upon the stage-specific appearance of distinct multilineage hematopoietic stem/progenitor cells (HSPC).[6] Thus, *in utero*, hematopoiesis in the first and second trimester is largely sustained by a fetal-type HSPC that gives rise to tolerogenic Tregs; later (and perhaps as early as the third trimester), an adult-type HSPC instead gives rise to immunoreactive T cells. The timing of this switch coincides with birth and normally allows the newborn to move from a stance of tolerance to one of active defence against all foreign antigens. In this manner, the "immune privileged" aspect of mammalian pregnancy is preserved while the ability of the newborn to fight infections is also permitted.

Nonetheless, infection remains a leading cause of death and morbidity in newborns. Not only are neonates susceptible to more severe forms of disease caused by human pathogens such as herpes simplex virus 1, respiratory syncytial virus, *Bordetella pertussis* and *Staphylococcus aureus*), they are also subject to serious infection by microbial entities that are commensal flora in adults. For example, even after implementation of intensive screening and prevention practices, the estimated rate of Group B Streptococcal sepsis in the first week of life is 0.34 per 1000 live births, resulting in 60-70 deaths per year.[7] In addition to the immediate impact of neonatal illness and death, the long-term disability resulting from these infections represents a profound public health burden.[8] Premature infants, in particular, are predisposed to more severe infections from all pathogens and can also succumb to fatal infection by microbes that infrequently cause severe disease in adults, such as *Staphylococcus epidermidis*.[9] Compared with adults and older children, newborns produce less, and generally less effective, antibody in response to most immunizations. They are also less able to generate T cells that mediate effective antimicrobial responses.[10-15] Together, these deficiencies render the neonate a vulnerable target for a host of invading pathogens.

If the switch to an "adult-type" immune system is incomplete or overly slow after birth, two other problems may also arise. First, the neonate may respond less well to immunizations provided during the first months of life, either generating low levels of an effective response or polarized features of a non-effective response.[10,11,16-24] Secondly, those neonates that are most likely to develop atopic disorders after birth are also those who are most likely to generate suboptimal (and/or strong Th2-type) response to vaccination.[21,25] Since fetal Tregs may suppress Th1-type (or other) immune responses to vaccines in a manner that is different than adult Tregs, we speculate that strong Th2 polarization of childhood responses to vaccines may in part be due to a higher than normal proportion of fetal Tregs at birth.

In the studies of this proposal, we hypothesize that the immune system "layering" that is necessary for effective *in utero* development and postnatal protection of the human fetus occurs at a dissimilar pace in different individuals, predisposing some at birth to less effective immune responses to childhood immunizations.

(b) INNOVATION Previous experiments have demonstrated that similar "layering" of the immune system can occur in avian and murine models.[1-3] In these species, however, the timing and/or anatomic constraints are entirely different. In particular, the murine immune system develops at a markedly different pace than does the human immune system, *e.g.*, with very few Tregs detectable until three days after birth[27] as compared to the late 1st trimester in the human. This study is innovative in two respects: this is the first time that human immune system layering has been studied *in utero* and at birth; in addition, we have identified and validated a set of genes that are uniquely expressed in fetal or adult T cells, allowing us to quantitatively and qualitatively study the kinetics of the two populations as a function of time. The proposed research has the potential to improve prevention (through improved vaccine strategies) and treatment of neonatal infection (by providing a better understanding of normal human fetal immune development), and should teach us how the developmental state of the fetus and newborn affects their ability to respond to pathogens or vaccines.

(c) RESEARCH PLAN We propose to test the hypothesis that the immune system of the human newborn is comprised of two distinct hematopoietic lineages, one derived from a multilineage HSPC that resides in the fetal liver and bone marrow, and another from an HSPC that begins to function later in pregnancy and that supplants the fetal lineage thereafter. The former lineage is endowed with tolerogenic T cells that allow the fetus to co-exist with the mother (including her NIMA and other foreign antigens circulating with her); the latter lineage is instead comprised of T cells that are more likely to develop effector functions against novel antigens. The basis for this hypothesis is found by precedent in avian and murine models, as well as by our own [illegible] largely unpublished Preliminary Data:

The human fetus can mount a vigorous immune response to exogenous antigens. Although the human fetal and neonatal adaptive immune systems are often described as "immature" (*i.e.*, dysfunctional or ineffective at mounting a response to antigenic challenge), there is substantial evidence that immune responses can develop at or before birth in species such as sheep and nonhuman primates (but, interestingly, not mice) (reviewed in refs.16 and 28). To determine whether human fetal T cells are responsive against alloantigens, fetal (~20 g.w.) lymphocytes from spleen or lymph node (LN) were labeled with carboxyfluorescein succinimidyl ester (CFSE) and co-cultured with irradiated antigen-presenting cells (APCs) that had been isolated from the peripheral blood of a single healthy adult donor. After five days substantive proliferative responses were observed for both CD4+ and CD8+ fetal T cells (Fig. 1). This finding raised the question: If fetal T cells respond so vigorously to alloantigens *in vitro*, do they not also respond to NIMA expressed by maternal cells that have moved into fetal LNs *in utero*?

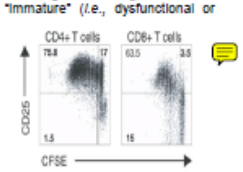

Figure 1. Representative plot of CD4+ and CD8+ total T cell proliferation after stimulation with allogeneic APCs from an unrelated donor for 5 days (3:1 ratio of fetal lymphocytes: allogeneic APCs)

The human fetal immune response to exogenous antigens can be actively suppressed by antigen-specific Tregs. We recently demonstrated that human fetal secondary lymphoid tissues contain significantly higher frequencies of $CD4^+CD25^{high}$ Tregs than those of adults.[4] Because Tregs have been shown to regulate maternal immunity to fetal alloantigens,[29] we reasoned that fetal Tregs might also play a role in suppressing fetal immune responses against invading maternal cells. To test this hypothesis, fetal LN cultures were either depleted or "mock-depleted" of Tregs before stimulation with self (autologous), maternal, or unrelated allogeneic APCs. Depletion of Tregs resulted in a highly significant increase in proliferation of $CD4^+$ and $CD8^+$ T cells responding to autologous or maternal APCs, but only a slight (yet statistically significant) increase in proliferation of those responding to unrelated APCs (Fig. 2). These data indicate that fetal T cells are not inherently deficient at responding to maternal alloantigens; rather, they are actively suppressed by fetal Tregs.

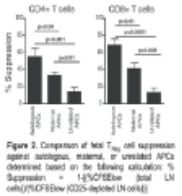

Figure 2. Comparison of fetal T_{reg} cell suppression against autologous, maternal, or unrelated APCs determined based on the following calculation: % Suppression = 1-[%CFSElow (total LN cells)/%CFSElow (CD25-depleted LN cells)]

The frequency of Tregs in peripheral lymphoid organs changes markedly during the course of gestation, falling from ~15–20% of total $CD4^+$ T cells at 12–20 g.w. to ~3–7% at birth.[30] To test the hypothesis that such a change in frequency reflects a greater propensity of fetal naïve $CD4^+$ and $CD8^+$ T cells to differentiate into Tregs in response to stimulation, fetal LN cells were depleted of $CD25^{high}$ Tregs and stimulated *in vitro*. After a five-day primary mixed lymphocyte reaction (MLR), a significant fraction of fetal, but not adult, $CD4^+$ and $CD8^+$ T cells had divided and upregulated FoxP3 expression to high levels (Fig. 3A, B).

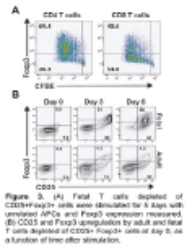

Figure 3. (A) Fetal T cells depleted of $CD25^{high}Foxp3^+$ cells were stimulated for 5 days with unrelated APCs and Foxp3 expression measured. (B) CD25 and Foxp3 upregulation by adult and fetal T cells depleted of $CD25^+$ $Foxp3^+$ cells at day 0, as a function of time after stimulation.

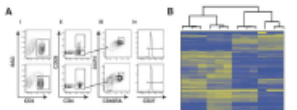

Figure 4. (A) Sort profiles for fetal (top) and adult (bottom) naïve $CD4^+$ T cells (panels i&iv) or $CD25^+$ Treg cells (panel ii). (B) Gene expression data showing the top 500 differentially expressed genes and clustering

Fetal Tregs are derived from a fetal-specific lineage of T cells. The above studies revealed profound differences in function between fetal and adult T cells that had otherwise indistinguishable phenotypes. To determine whether such differences are intrinsic to the T cell lineages found during these stages of ontogeny, $CD4^+CD25^{high}$ Tregs and $CD4^+CD45RA^+CCR7^+CD27^+$ naïve T cells from fetal and adult samples were sorted with a FACSDIVA (Fig. 4A). Microarray analysis (Fig. 4B) identified thousands of genes whose expression levels in adult and fetal $CD4^+$ naïve T cell populations differed significantly ($P<0.05$) and in a highly consistent manner between donors, including NOG, GZMA, and RGS1 were highly expressed (20-55 fold greater) by fetal cells whereas NAP1L2, NR3C2, and SYT4A were highly expressed by adult cells.

Fetal and adult HSPCs give rise to distinct populations of T cells. In avian and mouse models, there is strong evidence that fetal HSPCs give rise to unique subsets of lymphocytes that cannot be generated from adult HSPCs and that immune system "layering"[1] occurs during ontogeny.[2,3] To test whether a similar situation exists in humans, we performed a series of experiments in which fetal HSPCs from fetal liver and BM (18-22 g.w.) and adult BM samples were injected directly into the human Thy/Liv organ of the SCID-hu Thy/Liv mouse.[31] Following a 7-8 week maturation period, we were able to identify mature thymocyte populations derived from each HSPC population, based on the expression of a unique HLA type (typically HLA-A2 or A3) expressed by the donor (source of HSPCs) but not by the recipient thymic implant.[31,32] We isolated mature $CD3^+CD4^+CD8^-CD25^-$ thymocytes from thymic implants injected with fetal liver, fetal BM, or adult BM-derived HSPCs by FACS (FACS Aria), and performed microarray analysis on each population (Fig. 5A). We found that both HSPC populations from fetal liver and BM gave rise to identical populations of $CD4^+$ thymocytes on the basis of gene expression, with no differentially-expressed genes between them (Fig. 5B). By contrast, adult BM-derived HSPCs gave rise to $CD4^+$ thymocytes that showed substantial differences in gene expression patterns compared to each population of fetal HSPC-derived thymocytes (Fig. 5B: 1243 and 1162 differentially-expressed genes versus fetal liver and fetal BM, respectively). These data are consistent with the hypothesis that the developmental stage of HSPCs is in part responsible for the differences seen in peripheral T cell compartments in the fetus and adult, and that layering occurs during the ontogeny of the human immune system.

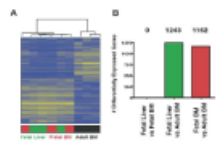

Figure 5. (A) Clustering of the top differentially expressed (DE) genes by CD4+ thymocytes from fetal liver HSPC (green), fetal BM HSPC (red), and adult BM HSPC (black). (B) Total number of DE genes (>2-fold difference expression difference and p<0.05) for each thymocyte population.

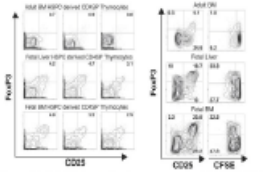

Figure 6. (A) CD25 and Foxp3 expression by $CD3^+CD4^+CD8^-$ thymocytes from adult BM, fetal liver, or fetal BM (3 SCID-hu thymic implants/group). (B) Expression of CD25 and Foxp3 (left) and proliferation (CFSE-dilution, right) following stimulation of $CD3^+CD4^+CD8^-$ thymocytes with allogeneic APCs for 7 days *in vitro*.

Fetal HSPC-derived T cells show an enhanced ability to generate Tregs during thymic maturation and upon exposure to foreign antigens *in vitro*. $CD4^+CD25^+Foxp3^+$ Tregs can be generated during thymic maturation or following activation of peripheral T cells. Some evidence indicates that Tregs may arise from a committed progenitor that is distinct from conventional T cell precursors.[33] We observed that fetal HSPC-derived thymocyte populations contained significantly greater frequencies of Tregs than those derived from adult HSPCs (Fig. 6A). In accordance with what we observed in peripheral fetal and adult T cell populations, we also noted that fetal HSPC-derived thymocytes were highly responsive to stimulation with allogeneic APCs and showed a propensity to differentiate into $FoxP3^+$ Tregs (Fig. 6B).

In sum, the above Preliminary Data indicate that the fetal

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Immune system is derived from a HSPC that gives rise to tolerogenic Tregs while the adult HSPC is more likely to give rise to immunoreactive T effector cells. At this point, we have very little information about the relative balance of these two compartments at birth. It is also not clear whether and to what extent variations in this balance may impact upon the response of the neonatal immune system to novel antigens, including those associated with routine vaccines or with environmental allergens. The experiments of this proposal are designed to explore these questions.

Specific Aim 1. To determine the normal range of fetal to adult T cells in the umbilical cord blood of neonates at birth

Hypothesis. Phys[illegible]ering of the human immune system during ontogeny leads to a normal range in the ratio of fetal to adult-type T cells at the time of birth, with some neonates born with a more tolerogenic immune system than others.

Rationale. As noted in the above Preliminary Data, the human fetal immune system is poised to generate a tolerogenic T[illegible] response upon stimulation, an attribute that is conferred by an HSPC that resides within the fetal liver and bone marrow. After birth, bone marrow-derived HSPC give rise instead to immunoreactive T cells with a reduced propensity to generate Tregs. Teleologically, such "layering" of the immune system would appear to be consistent with, and possibly necessary for, maintenance of the semi-allogeneic state of pregnancy and, reciprocally, for the generation of an active immune response against foreign (*e.g.*, infectious agents) after birth. Similar stage-specific waves of distinct hematopoietic progenitors have also been described in avian and murine models.[1-3] A key question that remains unanswered is the following: Is there inter-individual variation in the rate at which the fetal-type hematopoietic system is replaced by the adult-type system over time? In this Aim, we propose to determine whether and to what extent such variability may exist at the time of birth. Given known transcripts that uniquely identify tolerogenic fetal T cells (T_F) and immunoreactive adult T cells (T_A), the normal range of these two T cell subpopulations in the umbilical cord blood will be determined.

Experimental Approach. Co[illegible]sive phenotypic, transcriptional, and functional analyses will be carried out on umbilical cord blood (UCB) mononuclear cells from a total of 200 normal full-term deliveries. Over an 18-month time frame, 75 of these samples will be obtained on a recharge basis from the Human Cord Blood Bank of the UCSF Clinical and Translational Sciences Institute (see attached letter from Dr. William Balke), 75 will be obtained on a collaborative basis from Dr. Elizabeth Shpall of the University of Texas M.D. Anderson Cancer Center (see attached letter), and 50 will be obtained as part of a prospective study to be carried out with Dr. Shannon Thyne of the Child Health Center at SFGH (see attached letter). Initial studies will focus on naïve T cells obtained by a combination of ficoll hypaque gradient enrichment and FACS sorting; excess cells will be viably cryopreserved in liquid nitrogen for future experiments that may interrogate other subpopulations of cells. The following assays will be carried out:

1. Phenotypic analysis of T cell populations. The frequency of various T cell populations in the cord blood will be analyzed using standard markers of naïve $CD4^+$ T cells ($CD45RA^+CD27^+CCR7^+$), memory/effector T cells ($CD4^+CD45RO^+CD95^+HLA\text{-}DR^+$), and Tregs ($CD4^+CD25^{high}FoxP3^+CD127^{dim}$).[5] Absolute numbers will be quantified using TRUcount tubes (BD).
2. Transcriptional analysis of naïve T cell populations. Phenotypically-pure naïve $CD4^+$ T cells will be obtained by sort purification on a FACSAria (BD) and subjected to qRT-PCR assay to detect transcripts (transcript specific to cell-type Z denoted as $_TZ$) that are unique to fetal naïve T cells (*e.g.*, NOG, GZMA, and RGS1; $_TT_F$) or to adult naïve T cells (*e.g.*, NAP1L2, NR3C2, and SYT4A; $_TT_A$) as well as transcripts for house-keeping genes that are equivalently expressed in fetal and adult naïve T cells (*e.g.*, the β chain of the T cell receptor or HPRT; denoted by $_TX$). Each transcript will be quantitated in replicate and three standardized ratios of fetal/adult T cell transcripts (F/A-T) will be calculated based on the formula $(_TT_F/_TT_X)/(_TT_A/_TT_X) = (_TT_F/_TT_A)$. The three ratios will be F/A-T1 (NOG/NAP1L2), F/A-T2 (GZMA/NR3C2), and F/A-T3 (RGS1/SYT4A), and the mean of these ratios will be used to represent the fetal/adult T cell ratio (T_F/T_A).
3. Functional analysis of T cell populations. To test whether UCB T cells upregulate FoxP3 and adopt a Treg phenotype upon activation with alloantigens,[5] naïve $CD4^+$ T cells will be isolated by FACS and stimulated with allogeneic adult APCs plus or minus concurrent stimulation with cross-linking antibodies against CD3 and CD28. This type of stimulation reliably leads to Treg differentiation from naïve $CD4^+$ T_F (see Figure 3, Preliminary Data). Prior to stimulation, the cells will be labeled with CFSE for determination of proliferation. After six days of stimulation, the cells will be harvested and each T cell subpopulation will

be measured for proliferation (CFSE dilution) and for Treg differentiation (FoxP3 upregulation) by flow cytometry. Standard types of cytokine response (Th1, Th2, Th17, and Th22) will be measured by cytokine production after six days of differentiation *in vitro* by carrying out intracellular cytokine flow cytometry (CFC) for the following cytokines: IL-2, INF-γ, TNF-α, IL-4, IL-17, and IL-22. To test whether cord blood Tregs are better able to suppress Th1 vs. Th2 responses, $CD4^+$ naïve T cells will be sort-purified from a healthy adult (or cord blood) donor and cultured under Th1 or Th2 conditions in the presence of anti-CD3/anti-CD28. Th1 and Th2 polarization will be measured by cytokine production after six days of differentiation *in vitro*. These cells will subsequently be labeled with CFSE and cultured in the presence of different quantities of sort-purified $CD4^+CD25^+$ Treg from allogeneic adult blood or cord blood in the presence of anti-CD3/CD28. Suppression of Th1 or Th2 cells will be measured by inhibition of proliferation (by CFSE dilution) and suppression of cytokine secretion (by cytokine flow cytometry) after a six-day culture period.

Interpretation of Results. Th[illegible]ments described in this Aim extend observations that we have made in human fetal and adult samples [illegible] much larger number of samples of human cord blood. We anticipate that, at term, there will be a normative range of fetal- and adult-type Tregs and HSPCs in cord blood, representing variable kinetics by which layering of immune system ontogeny proceeds in different individuals. The relative frequency of fetal versus adult T cell-specific transcripts ($_TT_F/_TT_A$) will be taken as a measurement of the actual fetal/adult T cell ratio (T_F/T_A). Thus, we will make the assumption that $_TT_F/_TT_A = T_F/T_A$. In those cases in which the fetal/adult T cell ratio (T_F/T_A) is high, it is predictable that cord blood naïve T cells will be more likely to upregulate FoxP3 upon stimulation and that a predominant tolerogenic response to antigen will ensue.

Potential Problems and Alternative Approaches. Gi[illegible]isting data sets and the techniques that have already been established in the lab, the experiments of this Aim should be relatively straightforward. Though it is highly unlikely, it is possible that there will be no significant variability in the T_F/T_A ratio in full-term newborns. This would be an interesting finding, suggesting that the fetal-to-adult T cell transition occurs earlier during the third trimester of pregnancy and is complete at birth. Should this be the case, we will shift out attention to human premature infants and to nonhuman primates, each of which can be studied during the timeframe of the third trimester.

Specific Aim 2. To determine whether those full-term neonates with a high ratio of fetal/adult T cells are more likely to generate a Th2-polarized immune response to routine childhood immunizations.

Hypothesis. Infants with a high ratio of fetal/adult T cells will generate predominant Th2 responses to routine childhood vaccinations.

Rationale. In human neonates, T cell responses are often characterized by deficient Th1 responses,[16-19] a reduced capacity to induce T cell memory,[20] a high frequency of IgE and IgG4 production,[21] a skewed Th2 response,[22-24] and even the induction of hyporesponsiveness.[25] Such responsiveness to routine childhood vaccines has been found to vary within populations, possibly as a result of genetic and/or environmental factors[26,34,35] and is in part magnified by the formulation of vaccines with the Th2-polarizing adjuvant, alum.[36] In addition, those neonates that are most likely to develop atopic disorders after birth are also those who are most likely to generate suboptimal (and/or strong Th2-type) response to vaccination.[21,26,37] Since fetal Tregs may suppress Th1-type (or other) immune responses to vaccines in a manner that is different than adult Tregs, we speculate that strong Th2 polarization of childhood responses to vaccines may in part be due to a higher than normal proportion of fetal T cells at birth.

Recombinant Hepatitis B (HepB) vaccine is routinely given at birth in the United States and provides an ideal opportunity to investigate an *in vivo* response to antigenic stimulation in the newborn. The immune response to HepB vaccine is well studied and is characterized by a meager (10%) seroconversion rate with the first dose at birth.[38] Subsequent vaccine doses are then observed to yield a substantial antibody response.[39] The primary neonatal $CD4^+$ T cell response to HepB vaccine is characterized by both Th1 and Th2 cytokine production; interestingly, however, the HepB-specific memory $CD4^+$ recall response consists of robust Th2 cytokine production at one year of age.[40]

Experimental Approach. The experiments of this Aim will be carried out under the auspices of a study that has been approved by the UCSF Committee on Human Research protocol (H6325-26775. The effects of regulatory T cells on the development of the pediatric immune system; McCune PI). In a prospective study design, 50 pregnant women will be enrolled prior to delivery. Exclusion criteria will include previous/current

HepB infection (HepBsAg+), other immunomodulatory infections detected by prenatal screening (HepC, HIV), or plans for the use of cord blood for alternative purposes (*e.g.*, banking). With assistance from collaborators in the SFGH Child Health Center (see attached letter of collaboration from Dr. Shannon Thyne), cord blood will be obtained from these deliveries and each of the 50 infants will be followed with blood draws at 6 and 12 months. In all cases, infants will have received routine childhood immunizations, including those against HepB at birth, 1-2, and 6-12 months of age. UCB and infant peripheral blood will be processed by ficoll hypaque and concurrent purification of $CD4^+$ cells by negative selection (RosetteSep, StemCell). These cells will be tested for the following parameters over time:

1. The fraction of T_F vs. T_A in the umbilical cord blood. This will be carried out using the qRT-PCR-based approach described in Aim 1 to determine the ratio of fetal/adult T cells (T_F/T_A).
2. Analysis of the cellular immune response against HepB vaccine. Established flow cytometric assays will be used to assess the fraction of $CD4^+CD25^{high}FoxP3^+CD127^{dim}$ Tregs among $CD3^+CD4^+$ T cells at each time point. These fractions will be converted to absolute numbers using TruCount tube (BD) analysis of whole blood counts prior to $CD4^+$ cell isolation, as described.[41] In addition to quantitative phenotyping, the ability of circulating Tregs to suppress the proliferation of HepB-specific responder $CD4^+CD25^-$ T cells will be assessed using well-established methods.[5,41,42] UCB or peripheral mononuclear cells that have been depleted of $CD25^+$ cells (or mock depleted) will be stimulated with (1) polyclonal activators (cross-linking antibodies against CD3 and CD28), (2) soluble HepB antigen, and (3) peptides corresponding to HepB. Cytokine production in $CD4^+$ cells will be assessed by CFC, as described above in Aim 1, to determine whether antigen-specific stimulation yields a response that is predominantly Th1 or Th2 in type.
3. Levels of circulating immunoglobulin (Ig) isotypes generated against HepB vaccine. Established ELISAs will be used to quantitate circulating levels of vaccine-induced IgG1, IgG2, IgG3, IgG4, and IgE relative to total Ig.

Interpretation of Results. This study will relate the ratio of fetal to adult naïve T cells (T_F/T_A) to a number of immune parameters associated with routine childhood immunizations. In particular, it will be of interest to know whether a high T_F/T_A ratio is associated with a higher propensity towards a less effective "immature" response (*e.g.*, a Th2-predominant cytokine response to vaccine antigens and the predominance of less-mature, antigen-specific IgG1, IgG3, and IgE compared to IgG2). Infants will be studied both cross-sectionally as well as prospectively, and we anticipate that the T_F/T_A ratio and measures of immaturity (especially a bias towards a Th2 response) will decrease with age in tandem. The ability of Tregs to suppress antigen-specific responses against HepB will also be measured. Given the hypothesis that T_F in the newborn generate tolerance to antigens they encounter by becoming Tregs, more robust HepB-specific Treg suppression would be expected in those children with a higher T_F/T_A ratio.

Statistical Analysis. These data will be analyzed in consultation with biostatisticians in the Biostatistics Consultation Service, associated with the UCSF CTSI (see attached letter of support from Dr. Peter Bacchetti). The sample size that has been chosen will detect a 35% difference in cytokine production, with a study power of 0.8 and significance level of 0.05. The statistical approach will use an ANCOVA-type linear model approach for each outcome variable. Some data may be longitudinal as well as cross-sectional and we will include random effects to account for within-subject correlation. We will explore approaches that treat age as continuous, in particular, the Laird-Ware repeated measures model. If the measures exhibit strong skewness or outlying values, we will attempt to transform the outcome (*e.g.*, log-transformation) to mitigate these issues. If this is not adequate, we will compare the groups using non-parametric (*e.g.*, rank based) methods.

Potential Pitfalls and Alternative Approaches. The primary challenge in this study is that of recruiting and retaining 50 patients over an 18-month period. Given the experience of our collaborator, Dr. Shannon Thyne, we believe that this goal is attainable. The Birth Center at SFGH delivers 1250-1300 infants yearly, all of whom are under the care of the Division of Neonatology, in which Dr. Burt (an MD investigator in the PI's lab who will be working on this study) is an attending physician. He will therefore have the ability to recruit patients (under our CHR-approved protocol) as they are admitted to the hospital in labor. Furthermore, in previous studies carried out through the Birth Center, members of the McCune lab have successfully recruited similar numbers of patients in a period of approximately one year. Approximately 80% of infants born at SFGH receive their primary care in the Child Health Center at SFGH. If by three months of recruitment, we are not meeting expected goals, this study will be extended to the Birth Center at UCSF, which has a similar delivery rate and where we also have CHR approval to recruit.

Instituto de Investigación Sanitaria La Fe

Financiación USA: NIH.

National Institutes of Health

EJEMPLO BIOGRAPHICAL SKETCH (Max 5 pages)

OMB No. 0925-0001/0002 (Rev. 08/12 Approved Through 8/31/2015)

BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. DO NOT EXCEED FIVE PAGES.

NAME: Hunt, Morgan Casey

eRA COMMONS USER NAME (credential, e.g., agency login): huntmc

POSITION TITLE: Associate Professor of Psychology

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(If applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
University of California, Berkeley	B.S	05/1990	Psychology
University of Vermont	Ph.D.	05/1996	Experimental Psychology
University of California, Berkeley	Postdoctoral	08/1998	Public Health and Epidemiology

A. Personal Statement ← PERSONAL STATEMENT

I have the expertise, leadership, training, expertise and motivation necessary to successfully carry out the proposed research project. I have a broad background in psychology, with specific training and expertise in ethnographic and survey research and secondary data analysis on psychological aspects of drug addiction. My research includes neuropsychological changes associated with addiction. As PI or co-investigator on several university- and NIH-funded grants, I laid the groundwork for the proposed research by developing effective measures of disability, depression, and other psychosocial factors relevant to the aging substance abuser, and by establishing strong ties with community providers that will make it possible to recruit and track participants over time as documented in the following publications. In addition, I successfully administered the projects (e.g. staffing, research protections, budget), collaborated with other researchers, and produced several peer-reviewed publications from each project. As a result of these previous experiences, I am aware of the importance of frequent communication among project members and of constructing a realistic research plan, timeline, and budget. The current application builds logically on my prior work. During 2005-2006 my career was disrupted due to family obligations. However, upon returning to the field I immediately resumed my research projects and collaborations and successfully competed for NIH support.

1. Merrule, R.J. & Hunt, M.C. (2004). Independent living, physical disability and substance abuse among the elderly. Psychology and Aging, 23(4), 10-22.
2. Hunt, M.C., Jensen, J.L. & Crenshaw, W. (2007). Substance abuse and mental health among community-dwelling elderly. International Journal of Geriatric Psychiatry, 24(9), 1124-1135.
3. Hunt, M.C., Wiechelt, S.A. & Merrule, R. (2008). Predicting the substance-abuse treatment needs of an aging population. American Journal of Public Health, 45(2), 236-245. PMCID: PMC9162292 Hunt, M.C., Newlin, D.B. & Fishbein, D. (2009). Brain imaging in methamphetamine abusers across the life-span. Gerontology, 46(3), 122-145.

B. Positions and Honors ← POSITIONS & HONORS

Positions and Employment

1998-2000 Fellow, Division of Intramural Research, National Institute of Drug Abuse, Bethesda, MD
2000-2002 Lecturer, Department of Psychology, Middlebury College, Middlebury, VT
2001- Consultant, Coastal Psychological Services, San Francisco, CA
2002-2005 Assistant Professor, Department of Psychology, Washington University, St. Louis, MO
2007- Associate Professor, Department of Psychology, Washington University, St. Louis, MO

Other Experience and Professional Memberships

1995- Member, American Psychological Association
1998- Member, Gerontological Society of America
1998- Member, American Geriatrics Society
2000- Associate Editor, Psychology and Aging
2003- Board of Advisors, Senior Services of Eastern Missouri
2003-05 NIH Peer Review Committee: Psychobiology of Aging, ad hoc reviewer
2007-11 NIH Risk, Adult Addictions Study Section, members

Honors

2003 Outstanding Young Faculty Award, Washington University, St. Louis, MO
2004 Excellence in Teaching, Washington University, St. Louis, MO
2009 Award for Best in Interdisciplinary Ethnography, International Ethnographic Society

C. Contribution to Science ← CONTRIBUTION TO SCIENCE

1. My early publications directly addressed the fact that substance abuse is often overlooked in older adults. However, because many older adults were raised during an era of increased drug and alcohol use, there are reasons to believe that this will become an increasing issue as the population ages. These publications found that older adults appear in a variety of primary care settings or seek mental health providers to deal with emerging addiction problems. These publications document this emerging problem but guide primary care providers and geriatric mental health providers to recognize symptoms, assess the nature of the problem and apply the necessary interventions. By providing evidence and simple clinical approaches, this body of work has changed the standards of care for addicted older adults and will continue to provide assistance in relevant medical settings well into the future. I served as the primary investigator or co-investigator in all of these studies.
   a. Gruczynski, J., Shaft, B.M., Merrule, R., & Hunt, M.C. (2002). Community based participatory research with late-life addicts. American Journal of Alcohol and Drug Abuse, 15(3), 222-238.
   b. Shaft, B.M., Hunt, M.C., Merrule, R., & Venturi, R. (2003). Policy implications of genetic transmission of alcohol and drug abuse in female nonusers. International Journal of Drug Policy, 30(5), 46-58.
   c. Hunt, M.C., Marks, A.E., Shaft, B.M., Merrule, R., & Jensen, J.L. (2004). Early-life family and community characteristics and late-life substance abuse. Journal of Applied Gerontology, 28(2),26-37.
   d. Hunt, M.C., Marks, A.E., Venturi, R., Crenshaw, W. & Ratonian, A. (2007). Community-based intervention strategies for reducing alcohol and drug abuse in the elderly. Addiction, 104(9), 1436-1606. PMCID: PMC9000292

2. In addition to the contributions described above, with a team of collaborators, I directly documented the effectiveness of various intervention models for older substance abusers and demonstrated the importance of social support networks. These studies emphasized contextual factors in the etiology and maintenance of addictive disorders and the disruptive potential of networks in substance abuse treatment. This body of work also discusses the prevalence of alcohol, amphetamine, and opioid abuse in older adults and how networking approaches can be used to mitigate the effects of these disorders.
   a. Hunt, M.C., Merrule, R. & Jensen, J.L. (2005). The effect of social support networks on morbidity among elderly substance abusers. Journal of the American Geriatrics Society, 57(4), 15-23.
   b. Hunt, M.C., Pour, B., Marks, A.E., Merrule, R. & Jensen, J.L. (2005). Aging out of methadone treatment. American Journal of Alcohol and Drug Abuse, 15(6), 134-149.
   c. Merrule, R. & Hunt, M.C. (2007). Randomized clinical trial of cotinine in older nicotine addicts. Age and Ageing, 38(2), 9-23. PMCID: PMC9002364

3. Methadone maintenance has been used to treat narcotics addicts for many years but I led research that has shown that over the long-term, those in methadone treatment view themselves negatively and they gradually begin to view treatment as an intrusion into normal life. Elderly narcotics users were shown in carefully constructed ethnographic studies to be especially responsive to tailored social support networks that allow them to eventually reduce their maintenance doses and move into other forms of therapy. These studies also demonstrate the policy and commercial implications associated with these findings.

   a. Hunt, M.C. & Jensen, J.L. (2003). Morbidity among elderly substance abusers. Journal of the Geriatrics, 60(4), 45-61.
   b. Hunt, M.C. & Pour, B. (2004). Methadone treatment and personal assessment. Journal Drug Abuse, 45(5), 15-26.
   c. Merrule, R. & Hunt, M.C. (2005). The use of various nicotine delivery systems by older nicotine addicts. Journal of Ageing, 54(1), 24-41. PMCID: PMC9112304
   d. Hunt, M.C., Jensen, J.L. & Merrule, R. (2008). The aging addict: ethnographic profiles of the elderly drug user. NY, NY: W. W. Norton & Company.

Complete List of Published Work in MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbibliographies/public/1PpT7IEFIAJBtGMRDdWFmIWAO/?sort=date&direction=ascending

D. Research Support ← RESEARCH SUPPORT

Ongoing Research Support

R01 DA942367 Hunt (PI) 09/01/08-08/31/16
Health trajectories and behavioral interventions among older substance abusers
The goal of this study is to compare the effects of two substance abuse interventions on health outcomes in an urban population of older opiate addicts.
Role: PI

R01 MH922731 Merrule (PI) 12/15/07-11/30/15
Physical disability, depression and substance abuse in the elderly
The goal of this study is to identify disability and depression trajectories and demographic factors associated with substance abuse in an independently-living elderly population.
Role: Co-Investigator

Faculty Resources Grant, Washington University 08/15/09-08/14/15
Opiate Addiction Database
The goal of this project is to create an integrated database of demographic, social and biomedical information for homeless opiate abusers in two urban Missouri locations, using a number of state and local data sources.
Role: PI

Completed Research Support

R21 AA998075 Hunt (PI) 01/01/11-12/31/13
Community-based intervention for alcohol abuse
The goal of this project was to assess a community-based strategy for reducing alcohol abuse among older individuals.
Role: PI

Financiación USA: NIH.

EJEMPLO BUDGET

Check Form for Errors | Save

Next Period | RESEARCH & RELATED BUDGET - Budget Period 1 | Delete Period

OMB Number: 4040-0001
Expiration Date: 6/30/2016

ORGANIZATIONAL DUNS: 4771920870000 Enter name of Organization: RESEARCH FOUNDATION OF HOSPITAL LA FE

Budget Type: ☐ Project ☒ Subaward/Consortium Budget Period: 1 Start Date: 07/01/2016 End Date: 06/30/2017

A. Senior/Key Person — SENIOR/KEY PERSONNEL

	Prefix	First	Middle	Last	Suffix	Base Salary ($)	Months Cal.	Months Acad.	Months Sum.	Requested Salary ($)	Fringe Benefits ($)	Funds Requested ($)
x	Dr.				PhD		2.00			0.00	0.00	0.00
	Project Role: PD/PI											
x	Dr.				PhD		4.00			0.00	0.00	0.00
	Project Role: Postdoctoral researcher											

Add Additional Key Person

Additional Senior Key Persons: Add Attachment | Delete Attachment | View Attachment

Total Funds requested for all Senior Key Persons in the attached file:

Total Senior/Key Person: 0.00

B. Other Personnel

Number of Personnel	Project Role	Months Cal.	Months Acad.	Months Sum.	Requested Salary ($)	Fringe Benefits ($)	Funds Requested ($)
	Post Doctoral Associates						
	Graduate Students						
	Undergraduate Students						
	Secretarial/Clerical						
x							

Add Additional Other Personnel

Total Number Other Personnel | Total Other Personnel

Total Salary, Wages and Fringe Benefits (A+B): 0.00

C. Equipment Description — EQUIPMENT

List items and dollar amount for each item exceeding $5,000

	Equipment Item	Funds Requested ($)
x		

Add Additional Equipment

Additional Equipment: Add Attachment | Delete Attachment | View Attachment

Total funds requested for all equipment listed in the attached file

Total Equipment

D. Travel — TRAVEL

	Funds Requested ($)
1. Domestic Travel Costs (Incl. Canada, Mexico and U.S. Possessions)	
2. Foreign Travel Costs	2,500.00
Total Travel Cost	2,500.00

F. Other Direct Costs — OTHER DIRECT COSTS

	Funds Requested ($)
1. Materials and Supplies	2,500.00
2. Publication Costs	
3. Consultant Services	
4. ADP/Computer Services	
5. Subawards/Consortium/Contractual Costs	
6. Equipment or Facility Rental/User Fees	3,000.00
7. Alterations and Renovations	
8.	
9.	
10.	
Total Other Direct Costs	5,500.00

G. Direct Costs

	Funds Requested ($)
Total Direct Costs (A thru F)	8,000.00

H. Indirect Costs — INDIRECT COSTS

	Indirect Cost Type	Indirect Cost Rate (%)	Indirect Cost Base ($)	Funds Requested ($)
x	Non-will negotiate. Foreign institution	8.00	8,000.00	640.00

Add Additional Indirect Cost

Total Indirect Costs: 640.00

Cognizant Federal Agency (Agency Name, POC Name, and POC Phone Number): none

I. Total Direct and Indirect Costs

	Funds Requested ($)
Total Direct and Indirect Institutional Costs (G + H)	8,640.00

J. Fee

Funds Requested ($)

K. Budget Justification — BUDGET JUSTIFICATION

(Only attach one file.) Add Attachment | Delete Attachment | View Attachment

Financiación USA: NIH.

National Institutes of Health

SF424 (R & R)

PHS 398 Research Plan

PHS 398 Cover Page Supplement

Research and Related Senior/Key Person Profile (Expanded)

Research And Related Other Project Information

Project/Performance Site Location(s)

Optional

☒ Research & Related Budget

Instituto de Investigación Sanitaria La Fe

Financiación USA: NIH.

National Institutes of Health

CONSULTA PROYECTOS NIH CONCEDIDOS

http://report.nih.gov/nih_funding.aspx

Instituto de Investigación Sanitaria La Fe

Financiación USA: CDMRP.

Congressionally Directed Medical Research Programs
CDMRP
Department of Defense

Currently Funded Research Programs

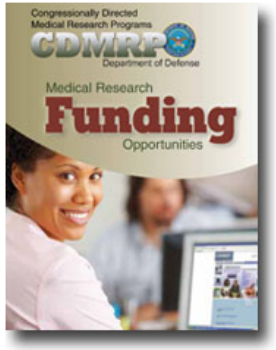
- Alcohol and Substance Abuse
- Amyotrophic Lateral Sclerosis
- Autism
- Bone Marrow Failure
- Breast Cancer
- Defense Medical Research and Development
- Duchenne Muscular Dystrophy
- Gulf War Illness
- Joint Warfighter Medical (Coming Soon)
- Lung Cancer
- Military Burn (Coming Soon)
- Multiple Sclerosis
- Neurofibromatosis
- Neurotoxin Exposure Treatment Parkinson's (Coming
- Orthotics and Prosthetics Outcomes
- Ovarian Cancer
- Peer Reviewed Alzheimer's
- Peer Reviewed Cancer
- Peer Reviewed Medical
- Peer Reviewed Orthopaedic
- Prostate Cancer
- Psychological Health/Traumatic Brain Injury
- Spinal Cord Injury
- Trauma Clinical Research Repository (Coming Soon)
- Tuberous Sclerosis Complex
- Vision

RESEARCH PROGRAMS | FUNDING OPPORTUNITIES | CONSUMER INVOLVEMENT | SEARCH AWARDS & OUTCOMES | MEDIA CENTER | ABOUT US

Home › Funding Opportunities

FUNDING OPPORTUNITIES

Start Pre-Application



» Click on Image to View Funding Opportunities Brochure

Funding Opportunities now available:

- Breast Cancer
- Defense Medical Research and Development Program
- Duchenne Muscular Dystrophy
- Epilepsy
- Gulf War Illness
- Military Burn
- Orthotics and Prosthetics Outcomes
- Peer Reviewed Alzheimer's
- Peer Reviewed Medical
- Peer Reviewed Orthopaedic
- Psychological Health/Traumatic Brain Injury
- Reconstructive Transplant Research
- Spinal Cord Injury
- Vision

BREAST CANCER

- Synopsis of Current Program Announcements

News

The Fiscal Year 2015 Appropriations Acts includes funding for programs managed by the DoD CDMRP

Financiación USA: CDMRP.

Congressionally Directed Medical Research Programs
CDMRP
Department of Defense

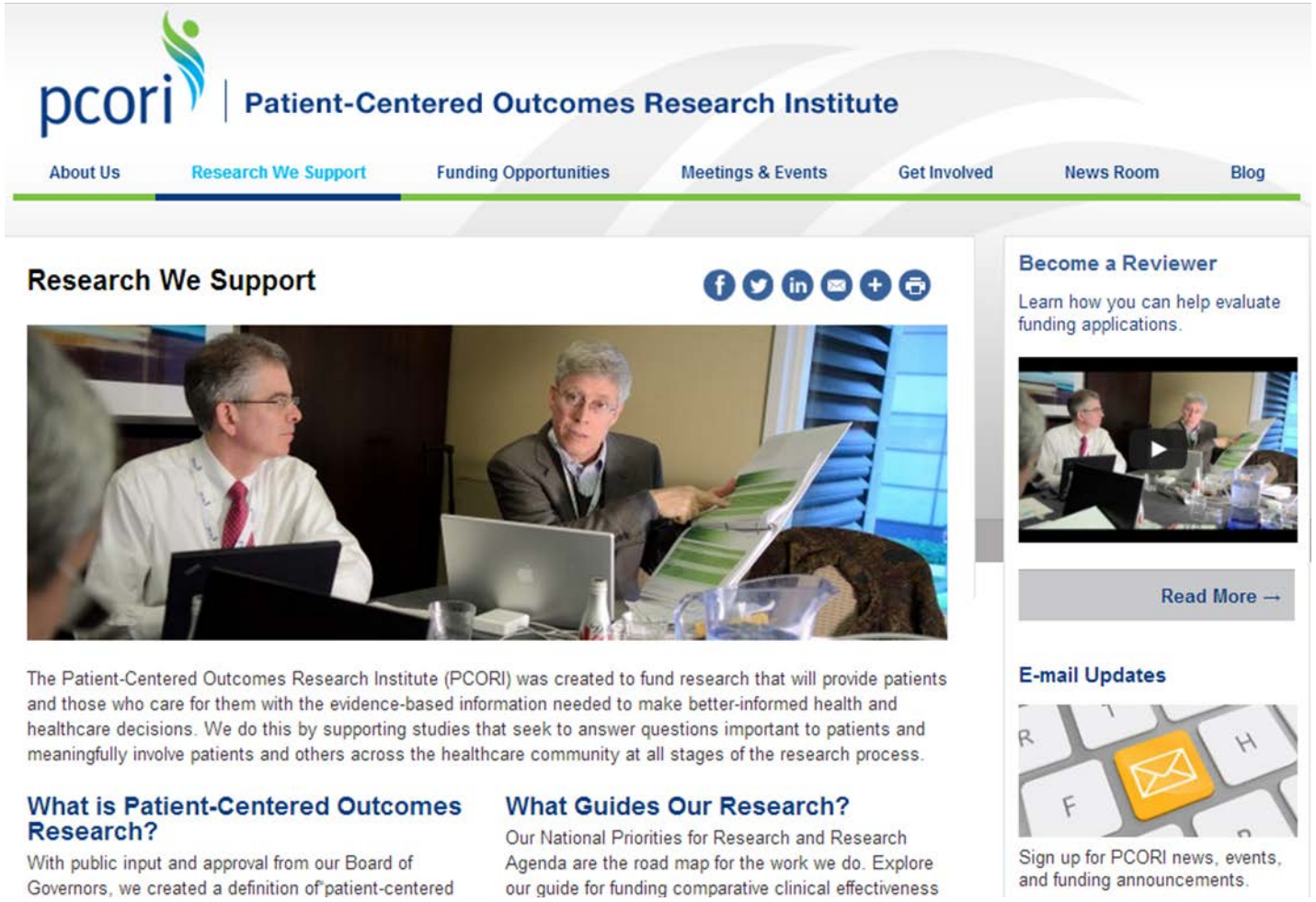
FY15 Breast Cancer Research Program (BCRP)

Synopsis of FY15 BCRP Award Mechanisms - (Adobe PDF) - provides a brief description and key elements of the award mechanism.

Mechanism	Release Date	Program Announcement/Instructions	Submission Deadline	
Breakthrough Award Level 1 and 2	August 25, 2015	Program Announcement Application Instructions (external link)	Pre-Application (Letter of Intent): November 18, 2015 Application: December 2, 2015	Submit Pre-Application
Breakthrough Award Level 3	July 23, 2015	Program Announcement Application Instructions (external link)	Pre-Application (Preproposal): September 18, 2015 Invited Application: December 21, 2015	Submit Pre-Application

Financiación USA: PCORI.

http://www.pcori.org/



Financiación USA: PCORI.

PCORI financia investigaciones encaminadas a que los **pacientes y sus cuidadores** obtengan la **información necesaria para tomar decisiones de salud**. En concreto, se financia **investigación comparativa de eficacia clínica**, así como el trabajo de apoyo que puede mejorar los métodos utilizados para llevar a cabo este tipo de estudios. Esta financiación se divide en cinco programas que reflejan las prioridades de investigación.

Evaluación de las Opciones de Prevención, Diagnóstico y Tratamiento.

Mejora de los Sistemas Sanitarios.

Comunicación y Difusión de la Investigación.

Abordar las disparidades.

Acelerar la investigación metodológica y productiva centrada en el paciente.

UPCOMING OPPORTUNITIES

Assessment of Prevention, Diagnosis, and Treatment Options - Cycle 1 2016

Key Deadlines	Type	Funds Available	Total Costs
LOI: March 2, 2016 **Application:** June 6, 2016 **Merit Review:** September 2016	Research Award		

Addressing Disparities - Cycle 1 2016

Key Deadlines	Type	Funds Available	Total Costs
LOI: March 2, 2016 **Application:** June 6, 2016 **Merit Review:** September 2016	Research Award		

Improving Healthcare Systems - Cycle 1 2016

Key Deadlines	Type	Funds Available	Total Costs
LOI: March 2, 2016 **Application:** June 6, 2016 **Merit Review:** September 2016	Research Award		

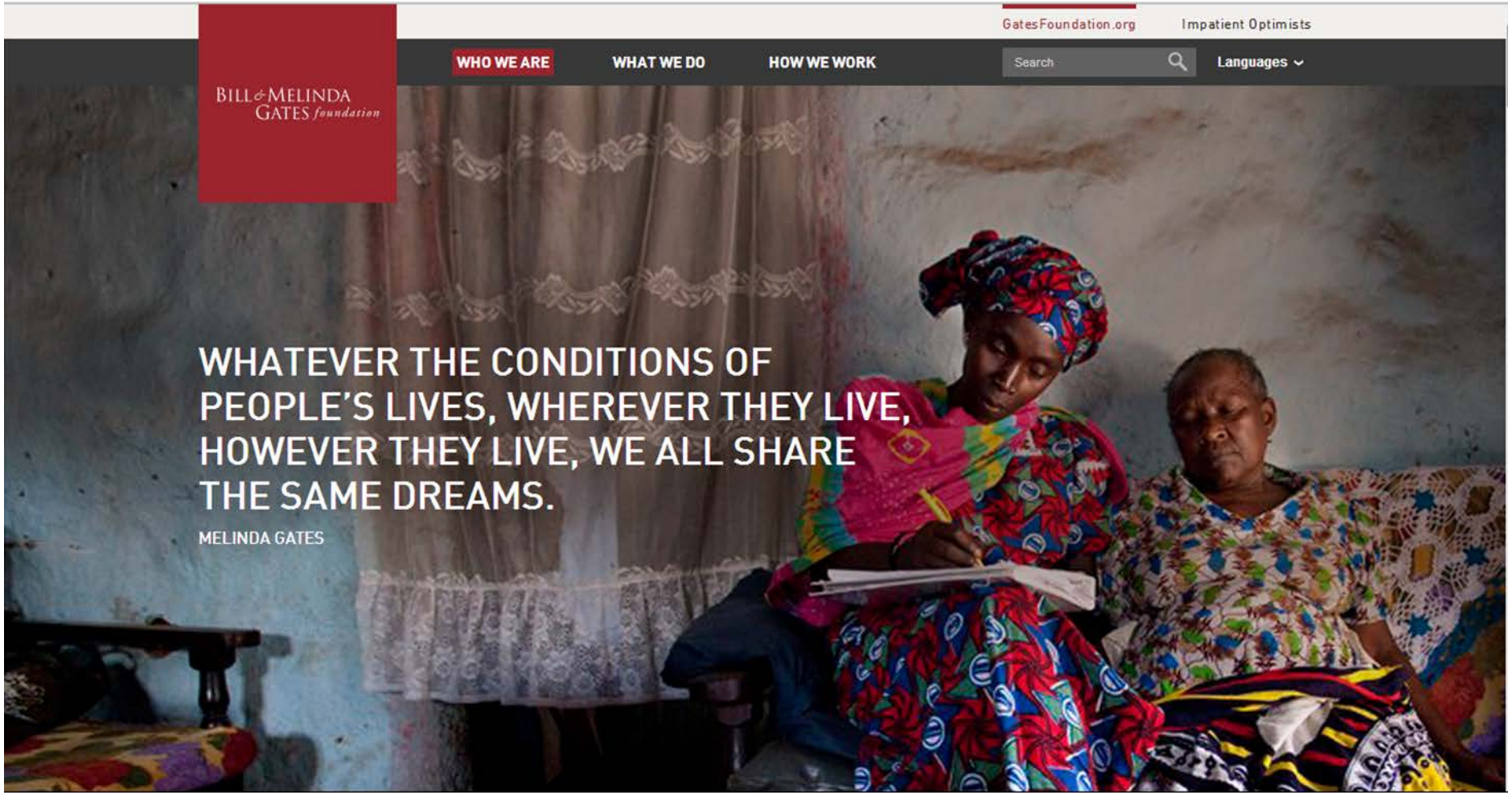
Foreign organizations and nondomestic components of organizations based in the United States may apply, **as long as there is demonstrable benefit to the US healthcare system**, and US efforts in the area of patient-centered research can be clearly shown.

Índice:

2.- Financiación internacional: Bill&Melinda Gates Foundation, Saving Lives At Birth, Grand Challenges Canada.

Financiación internacional: Bill&Melinda Gates Foundation.

http://www.gatesfoundation.org/



Financiación internacional: Bill&Melinda Gates Foundation.

Esta fundación subvenciona proyectos de I+D en beneficio del Tercer Mundo, sobretodo apoyando la lucha contra las enfermedades infecciosas, mejoras en la agricultura y Salud-Tecnología

GRANT OPPORTUNITIES

The foundation awards the majority of its grants to U.S. 501(c)(3) organizations and other tax-exempt organizations identified by our staff. (Tax status definitions) (Glossary of terms)

Request for Proposals (RFP)

- Grand Challenges Grant Opportunities
- Operations Research on Improving Paper-based Information Systems for Child Health
- School Networks for Evaluating and Improving the Efficacy of Digital Courseware
- Achieving Health Product Access through Market-Based Approaches
- Market Manager for HIV Prevention and HIV Treatment and Diagnostics
- Market Manager for Tuberculosis Treatment and Diagnostics
- Teacher Preparation Transformation Centers RFI
- Amplifying the Nutrition Impact of Agriculture in India RFP
- Data Innovation in US Education
- Accelerated Vaccine Introduction Rotavirus Vaccine Focus RFP
- Agriculture-Nutrition Impact Studies

Open Concept Memos

Qualified organizations may submit a Concept Memo for the funding area listed below:

- Global Health Grants

Global Health Grants

Financiación internacional: Bill&Melinda Gates Foundation.

How We Work

OPEN CONCEPT MEMO GLOBAL HEALTH GRANTS

← BACK Print

We accept concept memos for grants in our Global Health initiatives. These include:

- Enteric & Diarrheal Diseases
- Malaria
- Pneumonia

Getting Started

Please follow these instructions carefully when submitting a Concept Memo to the Global Health program. **Note:** Due to tax, legal, and reporting issues, we require that all Concept Memos be submitted in English.

Step 1: Download and Complete the Concept Memo

The Concept Memo includes questions regarding the scope, outcomes, and risks of your project as well as your organization's experience regarding the proposed work.

CONCEPT MEMO

• Concept Memo

Step 2: Submit your Concept Memo online

You must complete the online Concept Memo submission form and attach your completed Concept Memo in order for the foundation to process your request. Please do not mail a duplicate hard copy after submitting your Concept Memo online or send any additional attachments or information (videos, books, program materials, etc.)

Financiación internacional: Bill&Melinda Gates Foundation.

CONCEPT MEMO: MÁX. 4 PAGES

PROJECT SCOPE

INVESTMENT OUTCOMES

ORGANIZATIONAL FIT

RISKS/ CHALLENGES

Concept Details

1. Project Scope

Describe the concept and the overall approach you will take to achieve the intended results of this project. A detailed implementation plan is not needed at this stage.

2. Investment Outcomes

Explain what difference(s) this project will make by answering the following questions: What is the primary outcome(s) or result(s) this investment will achieve or significantly contribute to? How will you know when that result(s) has been achieved (how will the result be measured)? What are the critical outputs or intermediate outcomes that will lead to these results?

3. Organizational Fit

What experience does your organization have to implement the proposed work?

4. Risks/Challenges

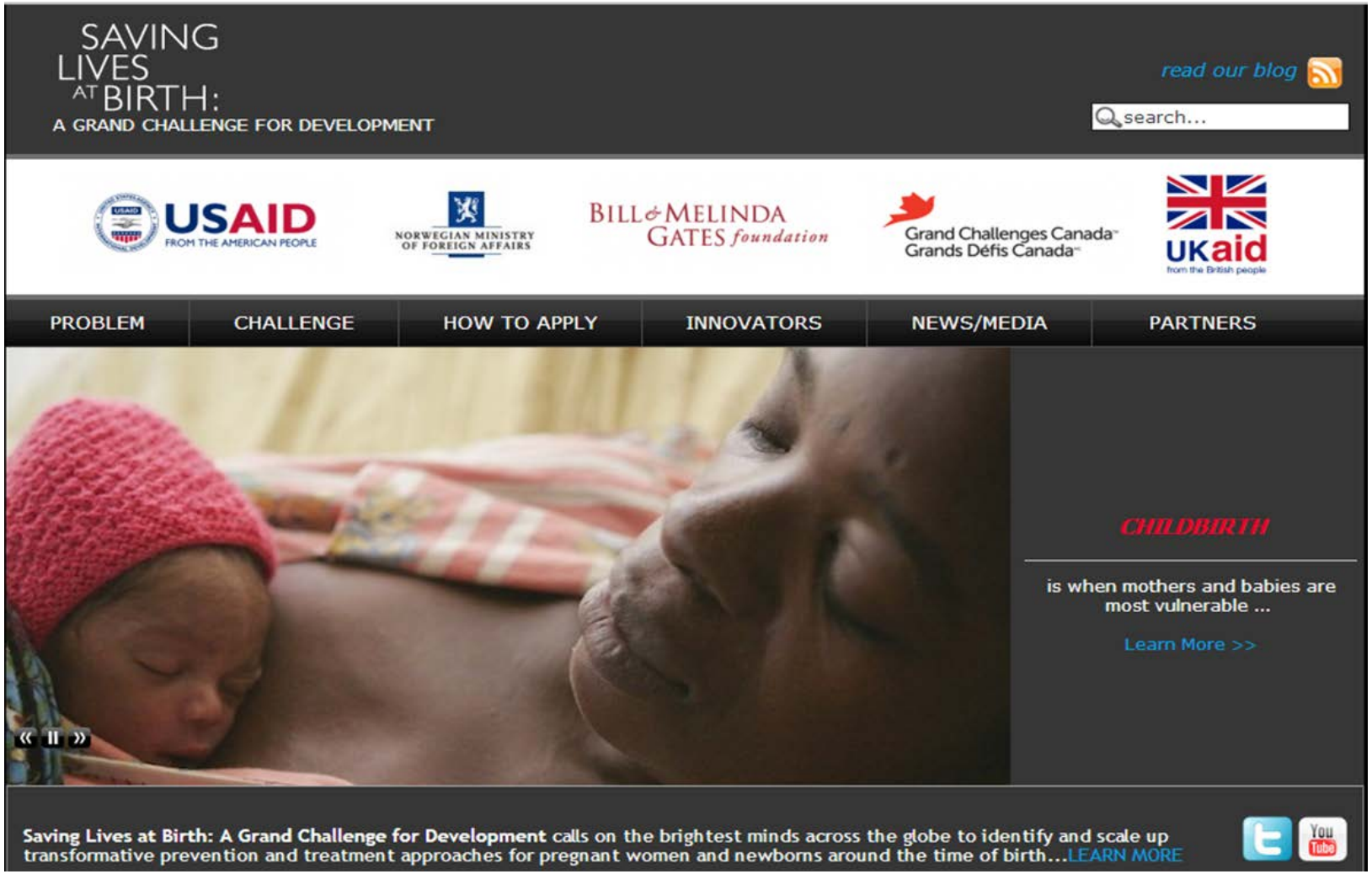
Describe your initial view of challenges to the success of this project and any early thoughts on mitigation.

Instituto de Investigación Sanitaria La Fe

Financiación internacional: Saving Lives at Birth.

SAVING LIVES AT BIRTH: A GRAND CHALLENGE FOR DEVELOPMENT

http://savinglivesatbirth.net/



Instituto de Investigación Sanitaria La Fe

Financiación internacional: Saving Lives at Birth.

SAVING LIVES AT BIRTH: A GRAND CHALLENGE FOR DEVELOPMENT

CIENCIA Y TECNOLOGÍAS

MEJORA DE CUIDADOS

MEJORA MÉTODOS COMPROMISO Y PARTICIPACIÓN POBLACIÓN

Estas convocatorias se abren para proyectos innovadores encaminados a la **prevención y el tratamiento de las mujeres embarazadas y los recién nacidos en los países del Tercer Mundo** o subdesarrollados alrededor del momento del parto.

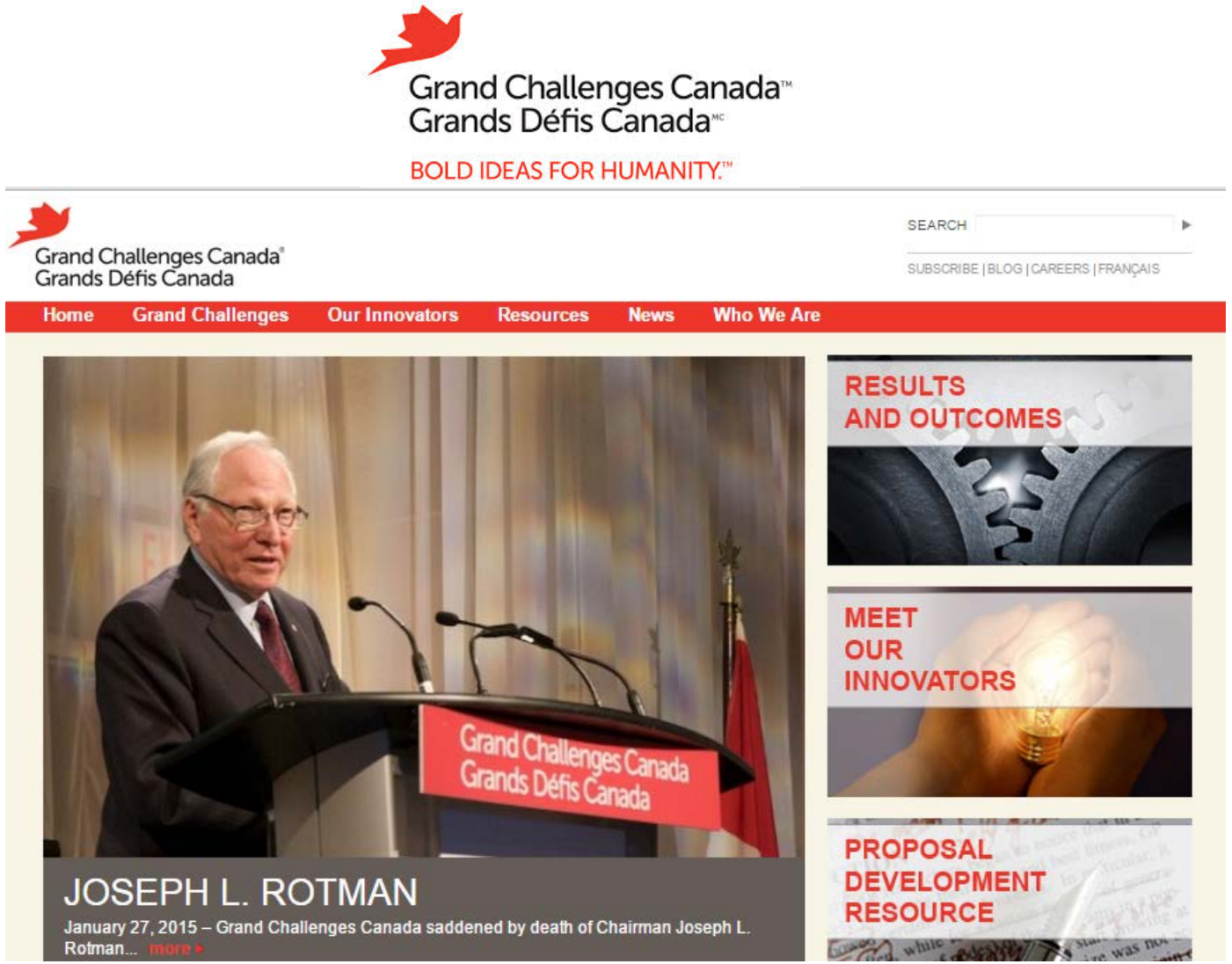
MODALIDADES DE FINANCIACIÓN:

Seed Funds/Validation Funds: $250,000 dólares/2 años, para apoyar el **desarrollo y la validación de ideas**.

Transition Funds: $2 millones dólares/4 años, para **desarrollar, refinar y testar rigurosamente el impacto** de soluciones integradas que evidencien una mejora en la salud muy significativa y una reducción de barreras.

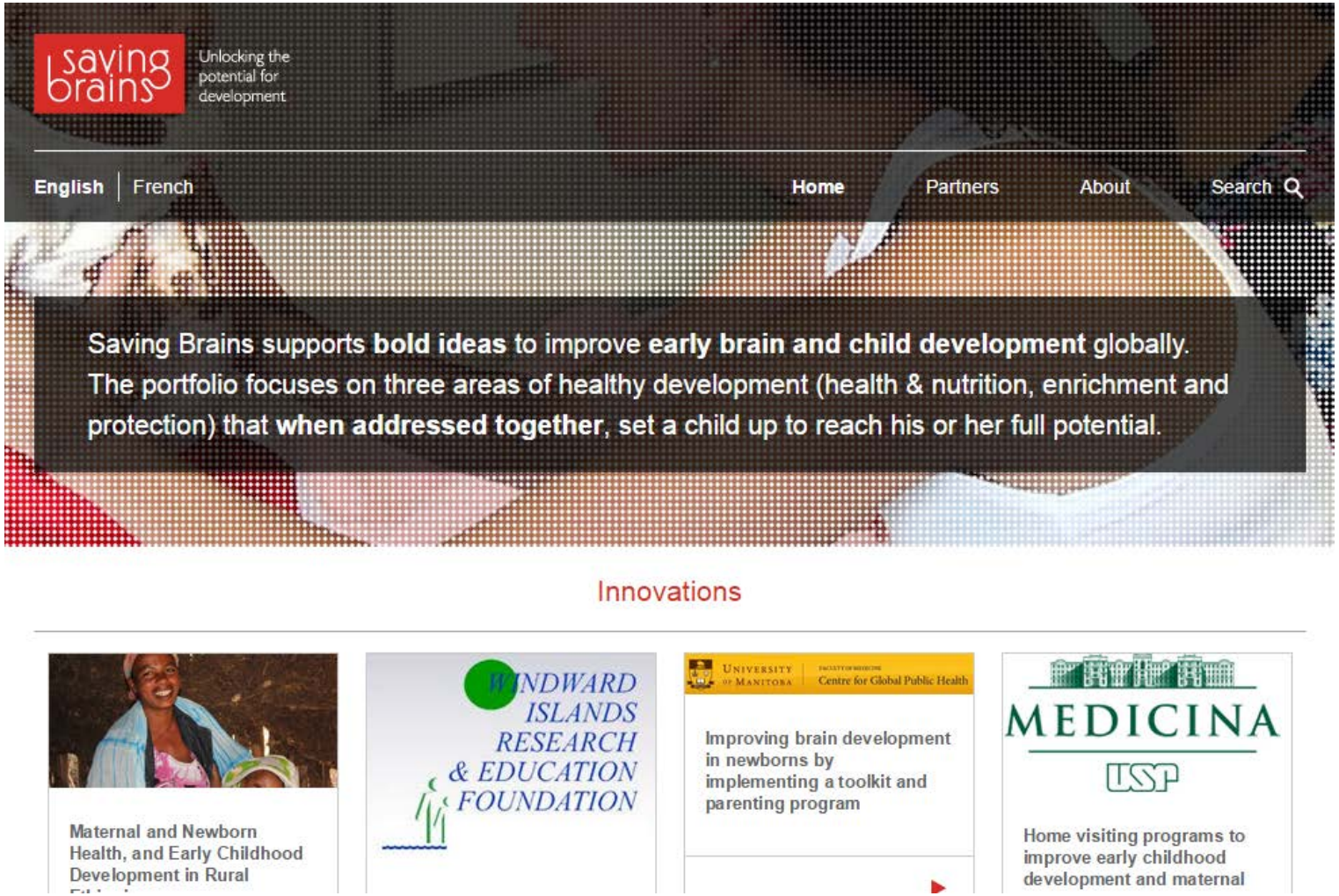
ROUND 6 : 2016 → **Expression of Interest (2-7 pages)**

Otras fuentes de financiación internacional: Canadá.



http://www.grandchallenges.ca/

Otras fuentes de financiación internacional: Canadá.



Instituto de Investigación Sanitaria La Fe

Alta competitividad a nivel europeo

3rd Health Programme

HORIZON 2020

AAL AMBIENT ASSISTED LIVING

eit European Institute of Innovation & Technology

erc

imi Innovative Medicines Initiative

E D C T P

MARIE CURIE ACTIONS

Unión Europea

Fondo Europeo de Desarrollo Regional "Una manera de hacer Europa"

Instituto de Investigación Sanitaria La Fe

Debemos intentarlo a nivel mundial

Instituto de Investigación Sanitaria La Fe

Tu hospital investiga para ti

Dra. Ana María Blanco Sánchez
Oficina Internacional IIS La Fe
e-mail: international@iislafe.es
+34 96 12 46617/+34 648 936 929