项目批准号	81571326
申请代码	H0920
归口管理部门	
依托单位代码	20003008C0839-1571

国家自然科学基金委员会

资助项目计划书

资助类别：面上项目

亚类说明：

附注说明：常规面上项目

项目名称：新生期应激损伤对炎症性肠病表观遗传易感性的潜在诱导及调控靶点

直接费用：57万元　　间接费用：11.4万元

项目资金：68.4万元　　执行年限：2016.01-2019.12

负 责 人：陈京红

通讯地址：上海市徐汇区宛平南路600号

邮政编码：200030　　电　　话：021-64387250

电子邮件：chenjh_008@hotmail.com

依托单位：上海市精神卫生中心

联 系 人：杨卫敏　　电　　话：021-34773239

填表日期：2015年09月07日

国家自然科学基金委员会制

Version：1.016.665

国家自然科学基金委员会资助项目计划书填报说明

一、项目负责人收到《关于国家自然科学基金资助项目批准及有关事项的通知》（以下简称《批准通知》）后，请认真阅读本填报说明，参照国家自然科学基金相关项目管理办法及《国家自然科学基金资助项目资金管理办法》（请查阅国家自然科学基金委员会官方网站首页“政策法规”-“管理办法”栏目），按《批准通知》的要求认真填写和提交《国家自然科学基金委员会资助项目计划书》（以下简称《计划书》）。

二、填写《计划书》时要求科学严谨、实事求是、表述清晰、准确。《计划书》经国家自然科学基金委员会相关项目管理部门审核批准后，将作为项目研究计划执行和检查、验收的依据。

三、《计划书》各部分填写要求如下：

（一）简表：由系统自动生成。

（二）摘要及关键词：各类获资助项目都必须填写中、英文摘要及关键词。

（三）项目组主要成员：计划书中列出姓名的项目组主要成员由系统自动生成，与申请书原成员保持一致，不可随意调整。如果批准通知中“项目评审意见及修改意见表”中“对研究方案的修改意见”栏目有调整项目组成员相关要求的，待项目开始执行后，按照项目成员变更程序另行办理。

（四）资金预算表：按批准资助的直接费用填报资金预算表和预算说明书，其中的劳务费、专家咨询费金额不应高于申请书中相应金额；间接费用及项目总经费由系统自动生成。国家重大科研仪器研制项目还应按照预算评审后批复的直接费用各科目金额填报资金预算表、预算说明书及相应的预算明细表。

（五）正文：

1. 面上项目、青年科学基金项目、地区科学基金项目：如果《批准通知》中没有修改要求的，只需选择“研究内容和研究目标按照申请书执行”即可；如果《批准通知》中“项目评审意见及修改意见表”中“对研究方案的修改意见”栏目明确要求调整研究期限和研究内容等的，须选择“根据研究方案修改意见更改”并填报相关修改内容。

2. 重点项目、重点国际（地区）合作研究项目、重大项目、国家重大科研仪器研制项目：须选择“根据研究方案修改意见更改”，根据《批准通知》的要求填写研究（研制）内容，不得自行降低、更改研究目标（或仪器研制的技术性能与主要技术指标以及验收技术指标）或缩减研究（研制）内容。此外，还要突出以下几点：

（1）研究的难点和在实施过程中可能遇到的问题（或仪器研制风险），拟采用的研究（研制）方案和技术路线；

（2）项目主要参与者分工，合作研究单位之间的关系与分工，重大项目还需说明课题之间的关联；

（3）详细的年度研究（研制）计划。

3. 国家杰出青年科学基金、优秀青年科学基金和海外及港澳学者合作研究基金项目：须选择“根据研究方案修改意见更改”，按下列提纲撰写：

（1）研究方向；

（2）结合国内外研究现状，说明研究工作的学术思想和科学意义（限两个页面）；

（3）研究内容、研究方案及预期目标（限两个页面）；

（4）年度研究计划；

（5）研究队伍的组成情况。

4. 对于其他类型项目，参照面上项目的方式进行选择和填写。

简表

申请者信息	姓　　名	陈京红	性 别	女	出生年月	1965年08月	民 族	汉族
	学　　位	博士			职称	副研究员		
	电　　话	021-64387250		电子邮件		chenjh_008@hotmail.com		
	传　　真			个人网页				
	工 作 单 位	上海市精神卫生中心						
	所 在 院 系 所							
依托单位信息	名　　称	上海市精神卫生中心					代码	20003008C0839
	联 系 人	杨卫敏		电子邮件		yang08631@163.com		
	电　　话	021-34773239		网站地址		www.smhc.org.cn		
合作单位信息	单　位　名　称							代　码
项目基本信息	项 目 名 称	新生期应激损伤对炎症性肠病表观遗传易感性的潜在诱导及调控靶点						
	资 助 类 别	面上项目			亚 类 说 明			
	附 注 说 明	常规面上项目						
	申 请 代 码	H0920:神经症和应激相关障碍			H0308:消化系统内分泌及神经体液调节异常			
	基 地 类 别							
	执 行 年 限	2016.01-2019.12						
	直 接 费 用	57万元			间 接 费 用	11.4万元		
	项 目 资 金	68.4万元						

项目摘要

中文摘要(500字以内)：

炎症性肠病IBD是复杂的胃肠道疾病，常与早期创伤和精神症状相关联。生命早期感染可导致免疫及神经内分泌长期改变，增加疾病易感性。我们前期研究证实慢性应激增加肠道敏感性和IBD的焦虑抑郁并发症，加之患者肠粘膜中DNA甲基化片段的发现，我们推测新生期炎性或母婴分离环境因素会损伤内皮屏障，炎性环境多重刺激，激活神经免疫系统，当成年环境再次改变时，表观遗传易感性改变，增加引发IBD形成风险。但尚无多重应激交互作用诱导遗传易感性导致IBD的具体研究。我们尝试用阶段刺激免疫强化的方式，探讨新生炎性应激后，NF-κB信号转导异常、交感神经系统致敏、内皮通透性增加，导致成年期表观遗传易感性IBD的发展。细胞因子基因编码的表观遗传调控和内皮连接蛋白对炎性环境再应激的反应可介导转录异常，其中组蛋白乙酰化酶抑制可作为IBD的潜在治疗靶标。早期应激的神经免疫调控可阐释诱导IBD发生的分子机制，为其防治提供科学依据。

关键词：新生期应激；炎症性肠病；表观遗传易感性；免疫激活；HDAC抑制剂

Abstract(limited to 4000 words)：

Inflammatory bowel disease (IBD) is a complex and common gastrointestinal disease often associated with early trauma and mental illness. Early-life infection may contribute to long lasting changes in the function of immune and neuroendocrine system,and increase adult disease susceptibility. Recent experiments in animals we also found that chronic stress can increase the visceral hypersensitivity and ulcerative colitis; and IBD also comorbidity with anxiety and depression. We hypothesized that the environmental changes of the neonatal inflammation and maternal deprivation that can damage the endothelial barrier, activates the immune system, as the environment challenges in adult, it can easily lead to the development of IBD. In accordance with the finding of methylated DNA segments in mucosal sample of IBD patients, we hypothesize the multiple stimulations and inflammatory environment interactions may lead to the epigenetic susceptibility which is the potential cause of IBD. However, there are no any experimental studies about the inflammatory environment interaction inducing epigenetic susceptibility about IBD risk so far. We suggested that the neonate-environment interaction or stress target multiple systems/organs, such as the SAM-axis, innate immunity and epithelial barrier, whose dysfunction together aggravates the immune response to adult-environment interaction. Dysfunction of the NF-κB signaling and sensitization of the SAM-axis are critical to aggravated immune response and increase of epithelial permeability. It will aggravate enteric immune, and lead to the development of epigenetic susceptibility. Epigenetic modulations of genes encoding proinflammatory cytokines and epithelial junction proteins in response to environmental challenges mediate aberrant transcription, and HDAC inhibitors show potential as therapeutic agents to IBD. Our proposal is significant in identifying epigenetic susceptibility to aggravated immune response. Epigenetic modulations for early-life stress may interpret the molecular mechanism which inducing the development of IBD, and provide scientific basis for the prevention and treatment of IBD.

Keywords: neonatal stress; inflammatory bowel disease, IBD; Epigenetic susceptibility; aggravated immune response; HDAC inhibitor

项目组主要成员

编号	姓名	出生年月	性别	职称	学位	单位名称	电话	证件号码	项目分工	每年工作时间（月）
1	陈京红	1965.08	女	副研究员	博士	上海市精神卫生中心	021-64387250		项目负责人	8
2	傅迎美	1978.12	女	助理研究员	博士	上海市精神卫生中心	13564112063		分子生化实验及细胞生物学实验	5
3	鞠培俊	1981.10	女	研究实习员	博士	上海市精神卫生中心	13585502759		实验设计、多重应激模型建立	5
4	王建玉	1974.08	女	教员	硕士	上海市精神卫生中心	18602187489		建立多重应激模型	8
5	胡昊	1986.10	男	博士生	硕士	上海市精神卫生中心	18516182631		分子生化实验及细胞实验	8
6	张灏	1989.10	女	硕士生	学士	上海市精神卫生中心	17717312279		分子生化实验及细胞实验	8

总人数	高级	中级	初级	博士后	博士生	硕士生
6	1	1	2		1	1

国家自然科学基金项目资金预算表（定额补助）

项目名称：　新生期应激损伤对炎症性肠病表观遗传易感性的潜在诱导及调控靶点

项目负责人：陈京红　　金额单位：万元

序号	科目名称	金额	备注
	(1)	(2)	(3)
1	**一、 项目资金支出**	68.4000	/
2	（一）直接费用	57.0000	
3	1、 设备费	0.0000	无
4	(1)设备购置费	0.0000	
5	(2)设备试制费	0.0000	
6	(3)设备改造与租赁费	0.0000	
7	2、 材料费	27.7190	包括原材料、试剂、耗材、药品、实验动物等
8	3、 测试化验加工费	2.0250	用于动物行为学及流式细胞测试
9	4、 燃料动力费	0.0000	无
10	5、 差旅费	4.3200	参加神经科学、消化道方面的国内学术会议
11	6、 会议费	0.7800	课题启动会、中期会议、专家咨询会及总结会
12	7、 国际合作与交流费	8.5000	参加神经科学、消化道方面的国际学术会议
13	8、 出版/文献/信息传播/知识产权事务费	3.2160	论文版面费、查新费用、图书购买费、打印
14	9、 劳务费	7.5600	用于直接参加项目研究的研究生的劳务费用
15	10、 专家咨询费	2.8800	用于神经科学、消化道专家咨询费用
16	11、 其他支出	0.0000	无
17	（二）间接费用	11.4000	
18	其中：绩效支出	2.8500	
19	**二、 自筹资金**	0.0000	

预算说明书

（请对各项支出的主要用途和测算理由及合作研究外拨资金等内容进行详细说明，可根据需要另加附页。）

（一）直接费用：57.0000

1、设备费　无

（1）购置设备费 无

（2）试制设备费 无

（3）设备改造与租赁费　无

2、材料费：27.7190

（1)试剂/药品：22.0190

=①试剂盒（RNA 抽提 500 元+反转录试剂盒 5860 元+SYBR Green Master Mix 试剂盒 3666 元+ECL 荧光试剂盒 800 元+总蛋白提取 450 元+蛋白定量盒 2000 元+WB 试剂 2000 元）×4 盒+cocktail 蛋白酶抑制剂 5000 元/支×1 支+PVDF5000/卷=7.1104 万元

②抗体：{RNA pol II 多抗+CREB-binding protein 多抗+acetyl-Histone H3 (Lys18)抗体+acetyl-Histone H4 (Lys 8) 抗体+NF-κB p65 多抗+ICAM-1 单抗+histone H4 多抗+histone H4K8Ac 抗体+histone H4K12Ac 抗体+histone H4K16Ac 抗体 +HDAC3 多抗+α-tubulin 抗体+histone H1 抗体+E-cadherin 抗体+β-actin 单抗}×3500 元/支×15 支+辣根标记二抗 500 元/支×6 支=5.55 万元。

③药物：共计 3.5384 万元。TNBS 5000 元/100ml×3 份＝1.75 万元，sodium butyrate1200 元/份×4 份＝0.48 万元,麻醉剂戊巴比妥钠:8.50 元/克×200 克=0.17 万元,Isoflurane 340 元/瓶 x20 瓶=0.68 万元，DSS 葡聚糖硫酸钠盐 MP 3543/100G×2 份=0.7086 万元。

④引物：(RelA 24 元+occludin 24 元＋IL-1β 24 元＋NF-κB 24 元+E-cadherin24 元)×10 对＝0.12 万

⑤siRNA、RNAi 干扰、过表达细胞株构建，用于在体转染监测细胞通透性改变 5000 元/批×3 批+构建肠腔粘膜通透蛋白 RNAi 干扰 1.23 万元+构建炎症细胞因子过表达细胞株 2.07=4.80 万元

⑥实验耗材：无 RNA 酶 EP 管 500 元×8 盒＋普通 EP 管 300 元×4 盒＋注射包 76 元/套×50 套＝0.90 万元

(2)动物饲养与购买：共计 5.70 万元

①实验动物饲养费：SPF 级，IVC 系统饲养每笼动物每天 6 元，每天平均饲养的动物为 10 只即 5 笼，3 年合计：6 元/笼×5 笼×300 天/年×4 年=3.60 万元。② Sprague Dawley（SD）实验大鼠及孕鼠购买：大鼠 50 元/只×100 只+孕鼠和窝鼠 400 元×40 只=2.10 万元。

3、测试化验加工费：2.025 ①动物行为学实验：25 元/只/次×50 只×9 次=1.125 万元。

②流式细胞测试：30 元/样本/次×50 样本×6 次=0.90 万元

4、燃料动力费：无

5、国内差旅费：共计 4.32 万元。①组员参加神经科学、消化道相关会议学术交流，共 2 次，每次 3 人。

（注册费 800 元+往返交通费 1500 元+住宿费 300 元×5 天＋伙食补贴 100 元×5 天＋市内交通费 80 元×5 天）×3 人×2 次=2.82 万元。②实验所需交通费 5000 元/年×3 年＝1.5 万元

6、会议费：0.78 课题启动及四次中期会议，每次 6 人共 5 次。伙食费 130 元×人数 6 人×天数 2 天×会议次数 5 次＝0.78 万元。

7、国际合作与交流费：8.50　美国神经科学及消化道疾病会议，做学术交流，每年 2 次，每次 2 人。（会议注册费 3000 元＋往返机票 15600 元＋住宿费 350 元×5 天＋伙食补贴 100 元×5 天＋市内交通费 80 元×5 天）×2 人次×3 年=8.50 万元。

8、出版物/文献/信息传播费：共计3.216 万元。论文版面费：中文版面费3000元×2 篇=0.60 万元。SCI论文6000元/篇×3 篇=1.80 万元。查新费用： 0.416 万元。图书购买费：用于购买神经科学、脑科学图书，大约200元/本×10本=0.20 万元。打印复印成果及参会海报：1元×1000张＋100元×10张＝0.2 万元。

9、劳务费：共计 7.56 万元。用于直接参加项目研究的研究生及技术人员劳务费用，研究生 1200 元/月×8 月×3 年×2 人次=5.76 万元，临时雇佣一名技术人员 1500 元/月×6 月×2 年×1 人次=1.8 万元。

10、专家咨询费：用于支付正高职称，神经科学、消化道专家咨询费，共计 6 人次，2 天，800 元/人/2 天×6 人 X3 次=2.88 万元。

11. 其他支出：无

（二）间接费用：11.4000

其中：绩效支出 2.85

二、自筹资金：无

项目负责人签字：　　科研部门公章：　　财务部门公章：

报告正文

研究内容和研究目标按照申请书执行。

国家自然科学基金资助项目签批审核表

<table>
<tr>
<td colspan="2">我接受国家自然科学基金的资助，将按照申请书、项目批准意见和计划书负责实施本项目（批准号：81571326），严格遵守国家自然科学基金委员会关于资助项目管理、财务等各项规定，切实保证研究工作时间，认真开展研究工作，按时报送有关材料，及时报告重大情况变动，对资助项目发表的论著和取得的研究成果按规定进行标注。

项目负责人（签章）：
年　　月　　日</td>
<td>我单位同意承担上述国家自然科学基金项目，将保证项目负责人及其研究队伍的稳定和研究项目实施所需的条件，严格遵守国家自然科学基金委员会有关资助项目管理、财务等各项规定，并督促实施。

依托单位（公章）
年　　月　　日</td>
</tr>
<tr>
<td rowspan="3">本栏目由基金委填写</td>
<td colspan="2">科学处审查意见：

建议年度拨款计划（本栏目为自动生成，单位：万元）：
<table>
<tr><td>年度</td><td>总额</td><td>第一年</td><td>第二年</td><td>第三年</td><td>第四年</td><td>第五年</td></tr>
<tr><td>金额</td><td></td><td></td><td></td><td></td><td></td><td></td></tr>
</table>
负责人（签章）：
年　　月　　日</td>
</tr>
<tr>
<td colspan="2">科学部审查意见：

负责人（签章）：
年　　月　　日</td>
</tr>
<tr>
<td>本栏目主要用于重大项目等</td>
<td colspan="2">相关局室审核意见：

负责人（签章）：
年　　月　　日</td>
</tr>
<tr>
<td></td>
<td colspan="2">委领导审批意见：

委领导（签章）：
年　　月　　日</td>
</tr>
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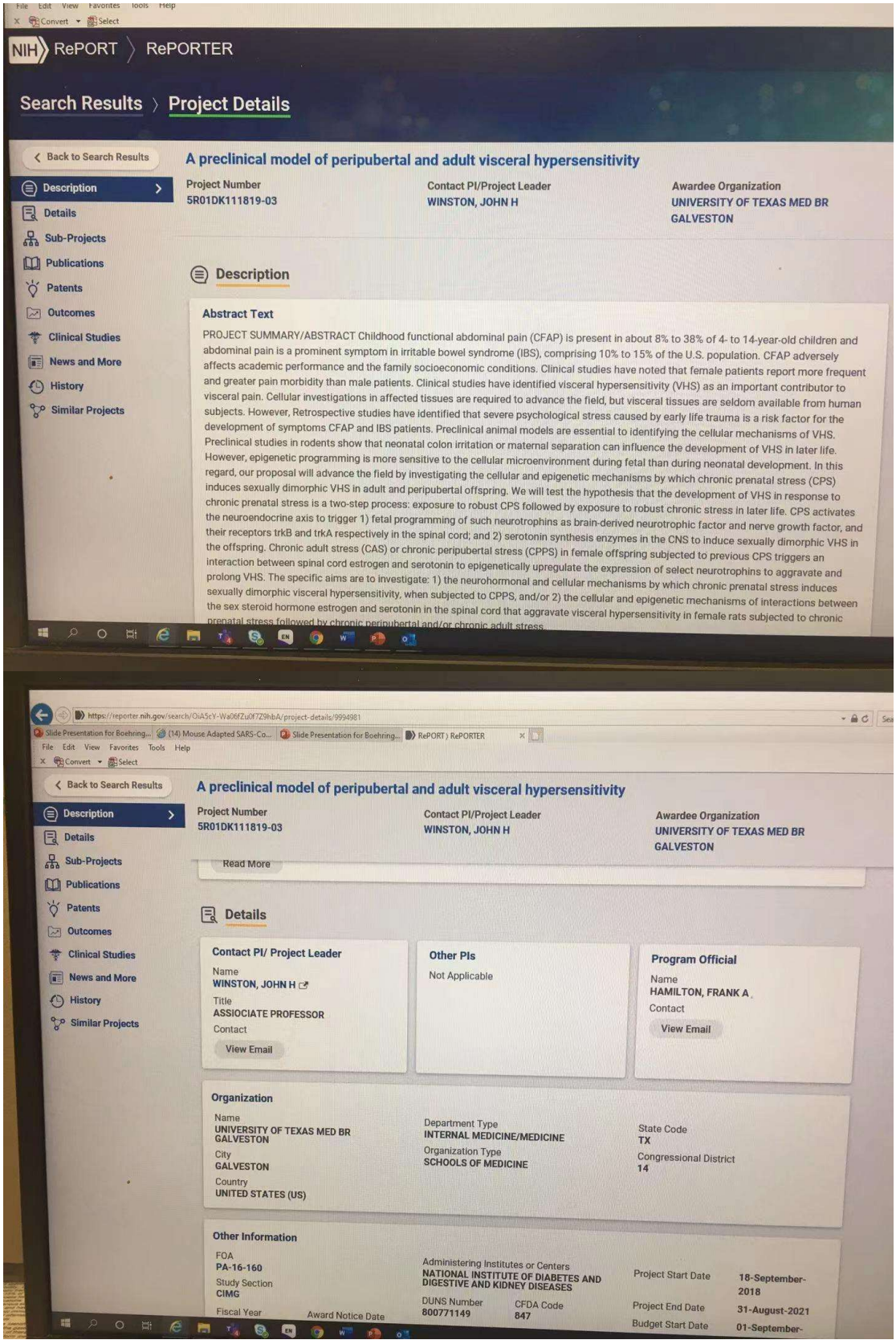
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A preclinical model of peripubertal and adult visceral hypersensitivity
Project Number
5R01DK111819-03
Contact PI/Project Leader
WINSTON, JOHN H
Awardee Organization
UNIVERSITY OF TEXAS MED BR GALVESTON
Description
Abstract Text
PROJECT SUMMARY/ABSTRACT Childhood functional abdominal pain (CFAP) is present in about 8% to 38% of 4- to 14-year-old children and abdominal pain is a prominent symptom in irritable bowel syndrome (IBS), comprising 10% to 15% of the U.S. population. CFAP adversely affects academic performance and the family socioeconomic conditions. Clinical studies have noted that female patients report more frequent and greater pain morbidity than male patients. Clinical studies have identified visceral hypersensitivity (VHS) as an important contributor to visceral pain. Cellular investigations in affected tissues are required to advance the field, but visceral tissues are seldom available from human subjects. However, Retrospective studies have identified that severe psychological stress caused by early life trauma is a risk factor for the development of symptoms CFAP and IBS patients. Preclinical animal models are essential to identifying the cellular mechanisms of VHS. Preclinical studies in rodents show that neonatal colon irritation or maternal separation can influence the development of VHS in later life. However, epigenetic programming is more sensitive to the cellular microenvironment during fetal than during neonatal development. In this regard, our proposal will advance the field by investigating the cellular and epigenetic mechanisms by which chronic prenatal stress (CPS) induces sexually dimorphic VHS in adult and peripubertal offspring. We will test the hypothesis that the development of VHS in response to chronic prenatal stress is a two-step process: exposure to robust CPS followed by exposure to robust chronic stress in later life. CPS activates the neuroendocrine axis to trigger 1) fetal programming of such neurotrophins as brain-derived neurotrophic factor and nerve growth factor, and their receptors trkB and trkA respectively in the spinal cord; and 2) serotonin synthesis enzymes in the CNS to induce sexually dimorphic VHS in the offspring. Chronic adult stress (CAS) or chronic peripubertal stress (CPPS) in female offspring subjected to previous CPS triggers an interaction between spinal cord estrogen and serotonin to epigenetically upregulate the expression of select neurotrophins to aggravate and prolong VHS. The specific aims are to investigate: 1) the neurohormonal and cellular mechanisms by which chronic prenatal stress induces sexually dimorphic visceral hypersensitivity, when subjected to CPPS, and/or 2) the cellular and epigenetic mechanisms of interactions between the sex steroid hormone estrogen and serotonin in the spinal cord that aggravate visceral hypersensitivity in female rats subjected to chronic prenatal stress followed by chronic peripubertal and/or chronic adult stress.
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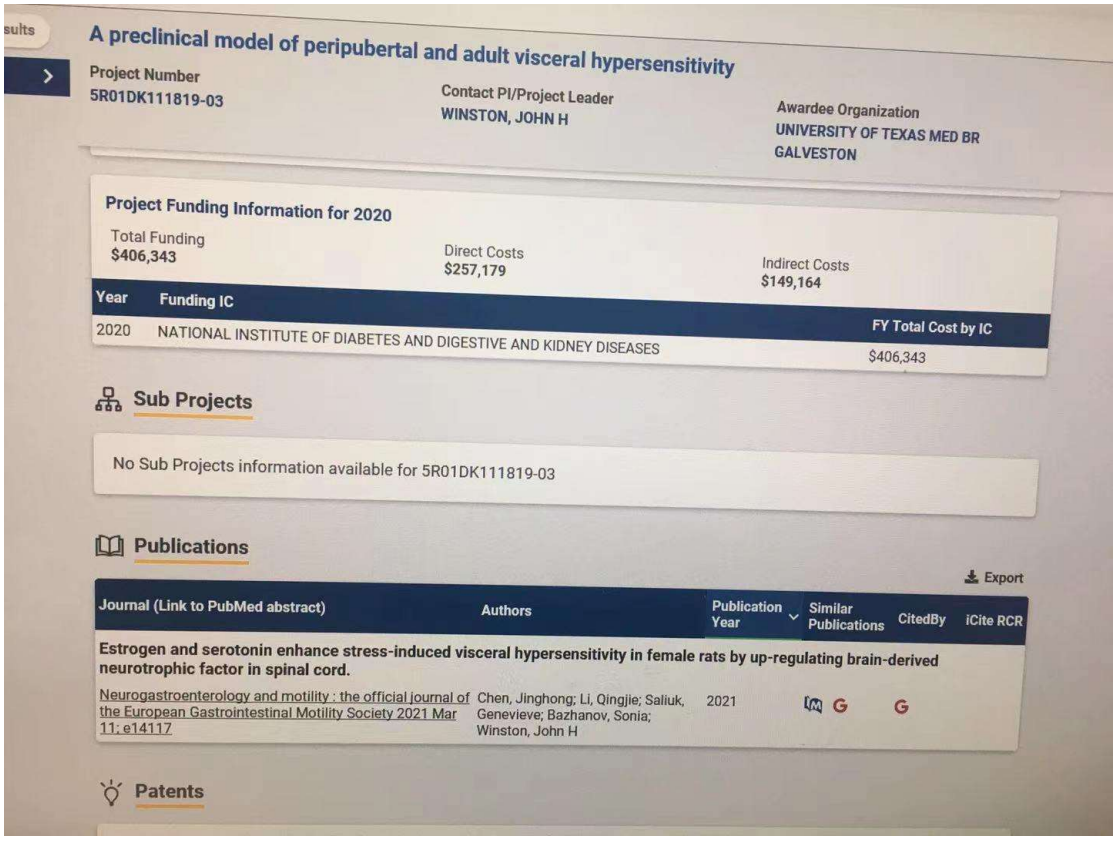
Read More
Details
Contact PI/ Project Leader
Name
WINSTON, JOHN H
Title
ASSIOCIATE PROFESSOR
Contact
View Email
Other PIs
Not Applicable
Program Official
Name
HAMILTON, FRANK A
Contact
View Email
Organization
Name
UNIVERSITY OF TEXAS MED BR GALVESTON
City
GALVESTON
Country
UNITED STATES (US)
Department Type
INTERNAL MEDICINE/MEDICINE
Organization Type
SCHOOLS OF MEDICINE
State Code
TX
Congressional District
14
Other Information
FOA
PA-16-160
Study Section
CIMG
Fiscal Year
Award Notice Date
Administering Institutes or Centers
NATIONAL INSTITUTE OF DIABETES AND DIGESTIVE AND KIDNEY DISEASES
DUNS Number
800771149
CFDA Code
847
Project Start Date
18-September-2018
Project End Date
31-August-2021
Budget Start Date
01-September-



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A preclinical model of peripubertal and adult visceral hypersensitivity

Project Number
5R01DK111819-03

Contact PI/Project Leader
WINSTON, JOHN H

Awardee Organization
UNIVERSITY OF TEXAS MED BR GALVESTON

Project Funding Information for 2020

Total Funding
$406,343

Direct Costs
$257,179

Indirect Costs
$149,164

Year	Funding IC	FY Total Cost by IC
2020	NATIONAL INSTITUTE OF DIABETES AND DIGESTIVE AND KIDNEY DISEASES	$406,343

Sub Projects

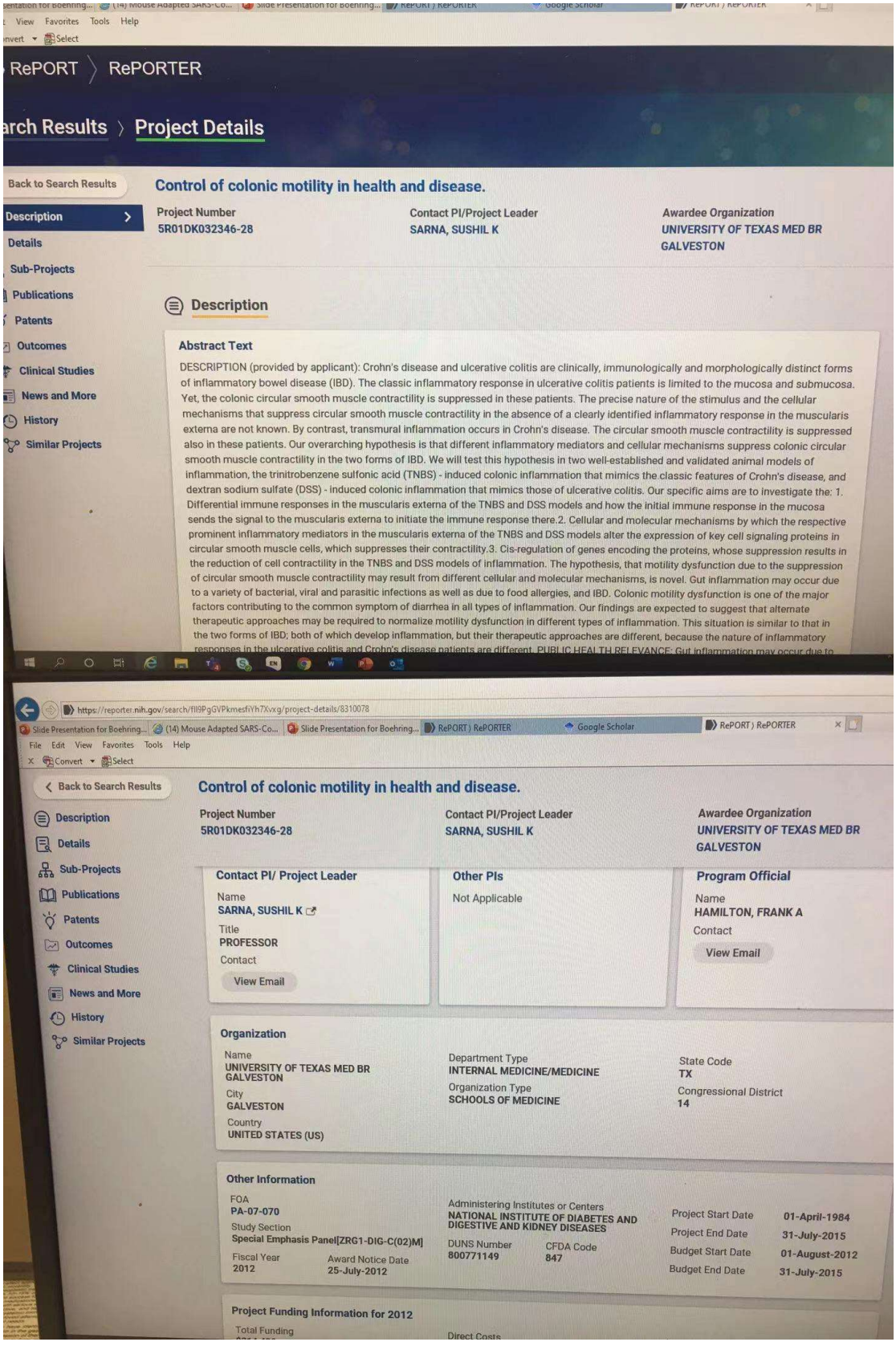
No Sub Projects information available for 5R01DK111819-03

Publications

Export

Journal (Link to PubMed abstract)	Authors	Publication Year	Similar Publications	CitedBy	iCite RCR
Estrogen and serotonin enhance stress-induced visceral hypersensitivity in female rats by up-regulating brain-derived neurotrophic factor in spinal cord.					
Neurogastroenterology and motility : the official journal of the European Gastrointestinal Motility Society 2021 Mar 11: e14117	Chen, Jinghong; Li, Qingjie; Saliuk, Genevieve; Bazhanov, Sonia; Winston, John H	2021			

Patents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Similar Projects
Control of colonic motility in health and disease.
Project Number
5R01DK032346-28
Contact PI/Project Leader
SARNA, SUSHIL K
Awardee Organization
UNIVERSITY OF TEXAS MED BR GALVESTON
Description
Abstract Text
DESCRIPTION (provided by applicant): Crohn's disease and ulcerative colitis are clinically, immunologically and morphologically distinct forms of inflammatory bowel disease (IBD). The classic inflammatory response in ulcerative colitis patients is limited to the mucosa and submucosa. Yet, the colonic circular smooth muscle contractility is suppressed in these patients. The precise nature of the stimulus and the cellular mechanisms that suppress circular smooth muscle contractility in the absence of a clearly identified inflammatory response in the muscularis externa are not known. By contrast, transmural inflammation occurs in Crohn's disease. The circular smooth muscle contractility is suppressed also in these patients. Our overarching hypothesis is that different inflammatory mediators and cellular mechanisms suppress colonic circular smooth muscle contractility in the two forms of IBD. We will test this hypothesis in two well-established and validated animal models of inflammation, the trinitrobenzene sulfonic acid (TNBS) - induced colonic inflammation that mimics the classic features of Crohn's disease, and dextran sodium sulfate (DSS) - induced colonic inflammation that mimics those of ulcerative colitis. Our specific aims are to investigate the: 1. Differential immune responses in the muscularis externa of the TNBS and DSS models and how the initial immune response in the mucosa sends the signal to the muscularis externa to initiate the immune response there.2. Cellular and molecular mechanisms by which the respective prominent inflammatory mediators in the muscularis externa of the TNBS and DSS models alter the expression of key cell signaling proteins in circular smooth muscle cells, which suppresses their contractility.3. Cis-regulation of genes encoding the proteins, whose suppression results in the reduction of cell contractility in the TNBS and DSS models of inflammation. The hypothesis, that motility dysfunction due to the suppression of circular smooth muscle contractility may result from different cellular and molecular mechanisms, is novel. Gut inflammation may occur due to a variety of bacterial, viral and parasitic infections as well as due to food allergies, and IBD. Colonic motility dysfunction is one of the major factors contributing to the common symptom of diarrhea in all types of inflammation. Our findings are expected to suggest that alternate therapeutic approaches may be required to normalize motility dysfunction in different types of inflammation. This situation is similar to that in the two forms of IBD; both of which develop inflammation, but their therapeutic approaches are different, because the nature of inflammatory responses in the ulcerative colitis and Crohn's disease patients are different. PUBLIC HEALTH RELEVANCE: Gut inflammation may occur due to
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Similar Projects
Control of colonic motility in health and disease.
Project Number
5R01DK032346-28
Contact PI/Project Leader
SARNA, SUSHIL K
Awardee Organization
UNIVERSITY OF TEXAS MED BR GALVESTON
Contact PI/ Project Leader
Name
SARNA, SUSHIL K
Title
PROFESSOR
Contact
View Email
Other PIs
Not Applicable
Program Official
Name
HAMILTON, FRANK A
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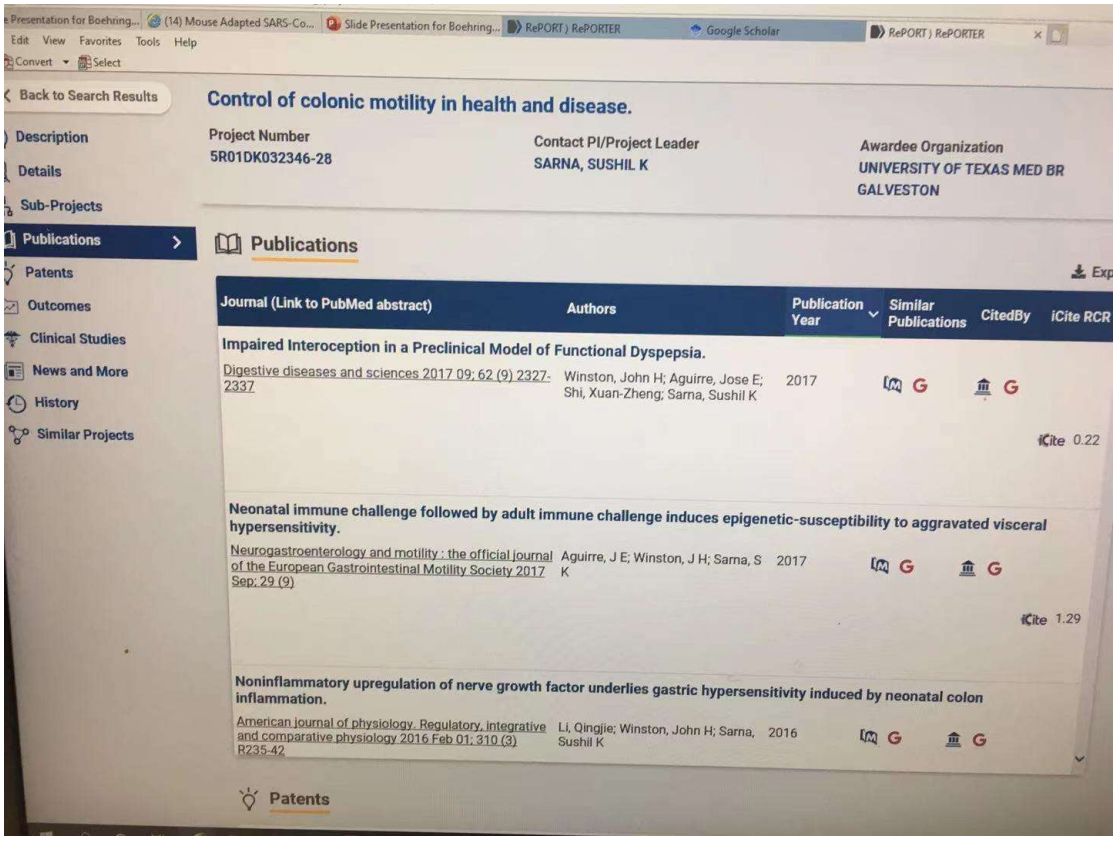
View Email
Organization
Name
UNIVERSITY OF TEXAS MED BR GALVESTON
City
GALVESTON
Country
UNITED STATES (US)
Department Type
INTERNAL MEDICINE/MEDICINE
Organization Type
SCHOOLS OF MEDICINE
State Code
TX
Congressional District
14
Other Information
FOA
PA-07-070
Study Section
Special Emphasis Panel[ZRG1-DIG-C(02)M]
Fiscal Year
2012
Award Notice Date
25-July-2012
Administering Institutes or Centers
NATIONAL INSTITUTE OF DIABETES AND DIGESTIVE AND KIDNEY DISEASES
DUNS Number
800771149
CFDA Code
847
Project Start Date
01-April-1984
Project End Date
31-July-2015
Budget Start Date
01-August-2012
Budget End Date
31-July-2015
Project Funding Information for 2012
Total Funding
Direct Costs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SARNA, SUSHIL K
Awardee Organization
UNIVERSITY OF TEXAS MED BR GALVESTON
Publications
Journal (Link to PubMed abstract)
Authors
Publication Year
Similar Publications
CitedBy
iCite RCR
Impaired Interoception in a Preclinical Model of Functional Dyspepsia.
Digestive diseases and sciences 2017 09; 62 (9) 2327-2337
Winston, John H; Aguirre, Jose E; Shi, Xuan-Zheng; Sarna, Sushil K
2017
iCite 0.22
Neonatal immune challenge followed by adult immune challenge induces epigenetic-susceptibility to aggravated visceral hypersensitivity.
Neurogastroenterology and motility : the official journal of the European Gastrointestinal Motility Society 2017 Sep; 29 (9)
Aguirre, J E; Winston, J H; Sarna, S K
2017
iCite 1.29
Noninflammatory upregulation of nerve growth factor underlies gastric hypersensitivity induced by neonatal colon inflammation.
American journal of physiology. Regulatory, integrative and comparative physiology 2016 Feb 01; 310 (3) R235-42
Li, Qingjie; Winston, John H; Sarna, Sushil K
2016
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