Director's Report

to the

NATIONAL ADVISORY COUNCIL ON DRUG ABUSE

May 2012

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Director
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** These sections contain select information. More comprehensive information will be posted in the May 2012 Staff Report to the Director.*

RESEARCH HIGHLIGHTS

An Oxycodone Conjugate Vaccine Elicits Drug-Specific Antibodies that Reduce Oxycodone Distribution to Brain and Hot-Plate Analgesia. Pravetoni M, Le Naour M, Harmon TM, Tucker AM, Portoghese PS, Pentel PR. J Pharmacol Exp Ther. 2012 Apr; 341(1):225-32.

Abstract: Opioid conjugate vaccines have shown promise in attenuating the behavioral effects of heroin or morphine in animals. The goal of this study was to extend this approach to oxycodone (OXY), a commonly abused prescription opioid. Haptens were generated by adding tetraglycine $(Gly)_4$ or hemisuccinate (HS) linkers at the 6-position of OXY. Immunization of rats with $OXY(Gly)_4$ conjugated to the carrier proteins bovine serum albumin (BSA) or keyhole limpet hemocyanin (KLH) produced high-titer antibodies to OXY and its metabolite oxymorphone with substantially lower affinities for other structurally related opioid agonists and antagonists. There was no measurable binding of antibody by the $(Gly)_4$ linker alone or off-target opioids methadone and buprenorphine. OXY(HS) conjugates were less immunogenic despite achieving protein haptenation ratios comparable to $OXY(Gly)_4$ BSA. In rats given a single intravenous dose of OXY, immunization with $OXY(Gly)_4$ KLH increased OXY protein binding and retention in serum while decreasing its unbound (free) concentration in plasma and distribution to brain. Vaccine efficacy correlated with serum antibody titers, and it was greatest in rats given the lowest OXY dose (0.05 mg/kg) but was significant even after a larger OXY dose (0.5 mg/kg), equivalent to the high end of the therapeutic range in humans. These effects of $OXY(Gly)_4$ -KLH on drug disposition were comparable to those of nicotine or cocaine vaccines that are in clinical trials as addiction treatments. Immunization with $OXY(Gly)_4$ -KLH also reduced OXY analgesia in a thermal nociception test. These data support further study of vaccination with the $OXY(Gly)_4$ -KLH immunogen as a potential treatment option for OXY abuse or addiction.

Generation Of A Synthetic Memory Trace. Garner AR, Rowland DC, Hwang SY, Baumgaertel K, Roth BL, Kentros C, Mayford M. Science. 2012 Mar 23; 335(6075):1513-6.

Abstract: We investigated the effect of activating a competing, artificially generated, neural representation on encoding of contextual fear memory in mice. We used a c-fos-based transgenic approach to introduce the hM_3D_q DREADD receptor (designer receptor exclusively activated by designer drug) into neurons naturally activated by sensory experience. Neural activity could then be specifically and inducibly increased in the hM_3D_q -expressing neurons by an exogenous ligand. When an ensemble of neurons for one context (ctxA) was artificially activated during conditioning in a distinct second context (ctxB), mice formed a hybrid memory representation. Reactivation of the artificially stimulated network within the conditioning context was required for retrieval of the memory, and the memory was specific for the spatial pattern of neurons artificially activated during learning. Similar stimulation impaired recall when not part of the initial conditioning.

Cannabinoid Receptor 2-Mediated Attenuation of CXCR4-Tropic HIV Infection in Primary CD4+ T Cells. Costantino CM, Gupta A, Yewdall AW, Dale BM, Devi LA, Chen BK. PLoS One. 2012;7(3):e33961. Epub 2012 Mar 20.

Abstract: Agents that activate cannabinoid receptor pathways have been tested as treatments for cachexia, nausea or neuropathic pain in HIV-1/AIDS patients. The cannabinoid receptors (CB_1R and CB_2R) and the HIV-1 co-receptors, CCR5 and CXCR4, all signal via Gαi-coupled pathways. We hypothesized that drugs targeting cannabinoid receptors modulate chemokine co-receptor function and regulate HIV-1 infectivity. We found that agonism of CB_2R, but not CB_1R, reduced infection in primary CD4+ T cells following cell-free and cell-to-cell transmission of CXCR4-tropic virus. As this change in viral permissiveness was most pronounced in unstimulated T cells, we investigated the effect of CB_2R agonism on to CXCR4-induced signaling following binding of chemokine or virus to the co-receptor. We found that CB_2R agonism decreased CXCR4-activation mediated G-protein activity and MAPK phosphorylation. Furthermore, CB_2R agonism altered the cytoskeletal architecture of resting CD4+ T cells by decreasing F-actin levels. Our findings suggest that CB_2R activation in CD4+ T cells can inhibit actin reorganization and impair productive infection following cell-free or cell-associated viral acquisition of CXCR4-tropic HIV-1 in resting cells. Therefore, the clinical use of CB_2R agonists in the treatment of AIDS symptoms may also exert beneficial adjunctive antiviral effects against CXCR4-tropic viruses in late stages of HIV-1 infection.

Cocaine Abstinence Alters Nucleus Accumbens Firing Dynamics During Goal-Directed Behaviors For Cocaine And Sucrose. Cameron CM, Carelli RM. Eur J Neurosci. 2012 Mar; 35(6):940-51.

Abstract: Distinct subsets of nucleus accumbens (NAc) neurons differentially encode goal-directed behaviors for natural vs. drug rewards, and the encoding of cocaine-seeking is altered following cocaine abstinence. Here, electrophysiological recording procedures were used to determine if the selective encoding of natural vs. cocaine reward by NAc neurons is: (i) maintained when the natural reinforcer is a highly palatable sweet tastant and (ii) altered by cocaine abstinence. Rats (n = 14) were trained on a multiple schedule of sucrose reinforcement and cocaine self-administration (2-3 weeks) and NAc activity was recorded during the task before and after 30 days of cocaine abstinence. Of 130 cells recorded before abstinence, 82 (63%) displayed patterned discharges (increases or decreases in firing rate, termed phasic activity) relative to operant responding for sucrose or cocaine. As in previous

reports, the majority of those cells displayed nonoverlapping patterns of activity during responding for sucrose vs. cocaine. Specifically, only 17 (21%) showed similar patterns of activity (i.e. overlapping activity) across the two reinforcer conditions. After abstinence, this pattern was largely maintained, 23 of 70 phasic cells (33%) were overlapping. However, cocaine abstinence altered the overall percentage of selectively active neurons across reinforcer conditions. Specifically, significantly more neurons became selectively activated during cocaine-directed behaviors than during sucrose-directed behaviors. The results indicate that, although the selective encoding of cocaine and natural rewards is maintained even with a highly palatable substance, 30 days of cocaine abstinence dynamically alters the overall population encoding of natural and drug rewards by NAc neurons.

Childhood Maltreatment is Associated with Reduced Volume in the Hippocampal Subfields CA3, Dentate Gyrus, and Subiculum. Teicher MH, Anderson CM, Polcaria A. PNAS. February 28, 2012; 109(9): E563-E572.
Abstract: Childhood maltreatment or abuse is a major risk factor for mood, anxiety, substance abuse, psychotic, and personality disorders, and it is associated with reduced adult hippocampal volume, particularly on the left side. Translational studies show that the key consequences of stress exposure on the hippocampus are suppression of neurogenesis in the dentate gyrus (DG) and dendritic remodeling in the cornu ammonis (CA), particularly the CA3 subfield. The hypothesis that maltreatment is associated with volume reductions in 3-T MRI subfields containing the DG and CA3 was assessed and made practical by newly released automatic segmentation routines for FreeSurfer. The sample consisted of 193 unmedicated right-handed subjects (38% male, 21.9 ± 2.1 y of age) selected from the community. Maltreatment was quantified using the Adverse Childhood Experience study and Childhood Trauma Questionnaire scores. The strongest associations between maltreatment and volume were observed in the left CA2-CA3 and CA4-DG subfields, and were not mediated by histories of major depression or posttraumatic stress disorder. Comparing subjects with high vs. low scores on the Childhood Trauma Questionnaire and Adverse Childhood Experience study showed an average volume reduction of 6.3% and 6.1% in the left CA2-CA3 and CA4-DG, respectively. Volume reductions in the CA1 and fimbria were 44% and 60% smaller than in the CA2-CA3. Interestingly, maltreatment was associated with 4.2% and 4.3% reductions in the left presubiculum and subiculum, respectively. These findings support the hypothesis that exposure to early stress in humans, as in other animals, affects hippocampal subfield development.

Provider And Clinic-Level Correlates Of Deferring Antiretroviral Therapy For People Who Inject Drugs: A Survey Of North American HIV Providers. Westergaard RP, Ambrose BK, Mehta SH, Kirk GD. J Int AIDS Soc. 2012 Feb 23; 15:10.
Abstract: Injection drug users (IDUs) face numerous obstacles to receiving optimal HIV care, and have been shown to underutilize antiretroviral therapy (ART). We sought to estimate the degree to which providers of HIV care defer initiation of ART because of injection drug use and to identify clinic and provider-level factors associated with resistance to prescribing ART to IDUs. We administered an Internet-based survey to 662 regular prescribers of ART in the United States and Canada. Questionnaire items assessed characteristics of providers' personal demographics and training, site of clinical practice and attitudes about drug use. Respondents then rated whether they would likely prescribe or defer ART for hypothetical patients in a series of scenarios involving varying levels of drug use and HIV disease stage. Survey responses were received from 43% of providers invited by email and direct mail, and 8.5% of providers invited by direct mail only. Overall, 24.2% of providers reported that they would defer ART for an HIV-infected patient with a CD4+ cell count of 200 cells/mm3 if the patient actively injected drugs, and 52.4% would defer ART if the patient injected daily. Physicians were more likely than non-physician providers to defer ART if a patient injected drugs (adjusted odds ratio 2.6, 95% CI 1.4-4.9). Other predictors of deferring ART for active IDUs were having fewer years of experience in HIV care, regularly caring for fewer than 20 HIV-infected patients, and working at a clinic serving a population with low prevalence of injection drug use. Likelihood of deferring ART was directly proportional to both CD4+ cell count and increased frequency of injecting. Many providers of HIV care defer initiation of antiretroviral therapy for patients who inject drugs, even in the setting of advanced immunologic suppression. Providers with more experience of treating HIV, those in high injection drug use prevalence areas and non-physician providers may be more willing to prescribe ART despite on-going injection drug use. Because of limitations, including low response rate and use of a convenience sample, these findings may not be generalizable to all HIV care providers in North America.

Attention-Deficit/Hyperactivity Disorder (ADHD) Symptoms, Craving To Smoke, And Tobacco Withdrawal Symptoms In Adult Smokers With ADHD. Berlin I, Hu MC, Covey LS, Winhusen T. Drug Alcohol Depend. Epub 2012 Feb 22.
Abstract: Tobacco withdrawal symptoms may be confounded with attention-deficit/hyperactivity disorder (ADHD) symptoms among smokers with ADHD. The objectives of this study were (1) To assess overlap between ADHD symptoms and tobacco/nicotine withdrawal symptoms and craving; (2) to assess the relationship between craving or withdrawal symptoms and the effect of osmotic-release oral system methylphenidate (OROS-MPH) on ADHD symptoms; (3) to assess the association of ADHD symptoms, craving, and withdrawal symptoms with abstinence. This was a secondary analysis of a randomized, placebo controlled smoking cessation trial assessing the efficacy of

OROS-MPH taken in addition to nicotine patch among individuals with ADHD. ADHD symptoms, withdrawal symptoms, and craving were assessed at baseline and 2, 4 and 6 weeks after a target quit day. Withdrawal symptoms and craving showed limited and modest overlap with ADHD symptoms prior to abstinence but more extensive and stronger correlation after quit day. Compared to placebo, OROS-MPH reduced ADHD symptoms; this effect was attenuated by controlling for withdrawal symptoms, but not by craving. Craving, but not ADHD symptoms and withdrawal symptoms, was associated with abstinence during the trial. When treating smokers with ADHD (1) craving, rather than tobacco withdrawal symptoms or ADHD symptoms may be the more effective therapeutic smoking cessation targets; (2) careful distinction of craving, withdrawal symptoms, and ADHD symptoms when assessing withdrawal phenomena is needed.

Higher Binding of the Dopamine D3 Receptor-Preferring Ligand [11C]-(+)-Propyl-Hexahydro-Naphtho-Oxazin in Methamphetamine Polydrug Users: A Positron Emission Tomography Study. Boileau I, Payer D, Houle S, Behzadi A, Rusjan PM, Tong J, Wilkins D, Selby P, George TP, Zack M, Furukawa Y, McCluskey T, Wilson AA, Kish SJ. J. Neurosci. 2012 Jan; 32(4):1353–1359.

Abstract: Positron emission tomography (PET) findings suggesting lower D2-type dopamine receptors and dopamine concentration in brains of stimulant users have prompted speculation that increasing dopamine signaling might help in drug treatment. However, this strategy needs to consider the possibility, based on animal and postmortem human data, that dopaminergic activity at the related D3 receptor might, in contrast, be elevated and thereby contribute to drug-taking behavior. We tested the hypothesis that D3 receptor binding is above normal in methamphetamine (MA) polydrug users, using PET and the D3-preferring ligand [^{11}C]-(+)-propyl-hexahydro-naphtho-oxazin ([^{11}C]-(+)-PHNO). Sixteen control subjects and 16 polydrug users reporting MA as their primary drug of abuse underwent PET scanning after [^{11}C]-(+)-PHNO. Compared with control subjects, drug users had higher [^{11}C]-(+)-PHNO binding in the D3-rich midbrain substantia nigra (SN;+46%; P<0.02) and in the globus pallidus (+9%; P=0.06) and ventral pallidum (+11%; P=0.1), whereas binding was slightly lower in the D2-rich dorsal striatum (approximately -4%, NS;-12% in heavy users, p=0.01) and related to drug-use severity. The [^{11}C]-(+)-PHNO binding ratio in D3-rich SN versus D2-rich dorsal striatum was 55% higher in MA users (p=0.004), with heavy but not moderate users having ratios significantly different from controls. [^{11}C]-(+)-PHNO binding in SN was related to self-reported “drug wanting.” We conclude that the dopamine D3 receptor, unlike the D2 receptor, might be upregulated in brains of MA polydrug users, although lower dopamine levels in MA users could have contributed to the finding. Pharmacological studies are needed to establish whether normalization of D3 receptor function could reduce vulnerability to relapse in stimulant abuse.

Association between Marijuana Exposure and Pulmonary Function Over 20 Years. Pletcherm M, Vittinghoffm E, Kalhanm R, Richmanm J, Saffordm M, Sidney S., Lin F., Kertesz S. JAMA. 2012; 307 (2): 173-181.

Abstract: Marijuana smoke contains many of the same constituents as tobacco smoke, but whether it has similar adverse effects on pulmonary function is unclear. The objective of this study was to analyze associations between marijuana (both current and lifetime exposure) and pulmonary function. Data are from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a longitudinal study collecting repeated measurements of pulmonary function and smoking over 20 years (March 26, 1985-August 19, 2006) in a cohort of 5115 men and women in 4 US cities. Mixed linear modeling was used to account for individual age-based trajectories of pulmonary function and other covariates including tobacco use, which was analyzed in parallel as a positive control. Lifetime exposure to marijuana joints was expressed in joint-years, with 1 joint-year of exposure equivalent to smoking 365 joints or filled pipe bowls. The primary outcome measure was forced expiratory volume in the first second of expiration (FEV(1)) and forced vital capacity (FVC). Marijuana exposure was nearly as common as tobacco exposure but was mostly light (median, 2-3 episodes per month). Tobacco exposure, both current and lifetime, was linearly associated with lower FEV(1) and FVC. In contrast, the association between marijuana exposure and pulmonary function was nonlinear (P < .001): at low levels of exposure, FEV(1) increased by 13 mL/joint-year (95% CI, 6.4 to 20; P < .001) and FVC by 20 mL/joint-year (95% CI, 12 to 27; P < .001), but at higher levels of exposure, these associations leveled or even reversed. The slope for FEV(1) was -2.2 mL/joint-year (95% CI, -4.6 to 0.3; P = .08) at more than 10 joint-years and -3.2 mL per marijuana smoking episode/mo (95% CI, -5.8 to -0.6; P = .02) at more than 20 episodes/mo. With very heavy marijuana use, the net association with FEV(1) was not significantly different from baseline, and the net association with FVC remained significantly greater than baseline (e.g., at 20 joint-years, 76 mL [95% CI, 34 to 117]; P < .001). Occasional and low cumulative marijuana use was not associated with adverse effects on pulmonary function.

Role for mTOR Signaling And Neuronal Activity In Morphine-Induced Adaptations In Ventral Tegmental Area Dopamine Neurons. Mazei-Robison MS, Koo JW, Friedman AK, Lansink CS, Robison AJ, Vinish M, Krishnan V, Kim S, Siuta MA, Galli A, Niswender KD, Appasani R, Horvath MC, Neve RL, Worley PF, Snyder SH, Hurd YL, Cheer JF, Han MH, Russo SJ, Nestler EJ. Neuron. 2011 Dec 22; 72(6):977-90.

Abstract: While the abuse of opiate drugs continues to rise, the neuroadaptations that occur with long-term drug exposure remain poorly understood. We describe here a series of chronic morphine-induced adaptations in ventral

tegmental area (VTA) dopamine neurons, which are mediated via down-regulation of AKT-mTORC2 (mammalian target of rapamycin complex-2). Chronic opiates decrease the size of VTA dopamine neurons in rodents, an effect seen in humans as well, and concomitantly increase the excitability of the cells but decrease dopamine output to target regions. Chronic morphine decreases mTORC2 activity, and overexpression of Rictor, a component of mTORC2, prevents morphine-induced changes in cell morphology and activity. Further, local knockout of Rictor in VTA decreases DA soma size and reduces rewarding responses to morphine, consistent with the hypothesis that these adaptations represent a mechanism of reward tolerance. Together, these findings demonstrate a novel role for AKT-mTORC2 signaling in mediating neuroadaptations to opiate drugs of abuse.

Satiating Effects Of Cocaine Are Controlled By Dopamine Actions In The Nucleus Accumbens Core. Suto N, Wise RA. Journal of Neuroscience. 2011 Dec 7; 31(49):17917-22.
Abstract: Intravenous cocaine intake in laboratory animals is characterized by periods of apparent drug satiety between regularly spaced earned injections. The reinforcing properties of cocaine are linked primarily to dopaminergic neurotransmission in the shell and not the core of nucleus accumbens. To determine whether the satiating effects of cocaine are similarly mediated, we perfused dopamine receptor agonists into the core or the shell during intravenous cocaine self-administrations by rats. Neither D1-type (SKF38393) nor D2-type (quinpirole) agonist was effective when given alone. However, a combination of the two agonists perfused into the core but not the shell significantly increased the time between cocaine self-injections, decreasing the amount of earned intake. Together with previous findings, the current data suggest that the satiating and reinforcing effects of cocaine are mediated by different ventral striatal output neurons.

Truncated G Protein-Coupled Mu Opioid Receptor MOR-1 Splice Variants Are Targets For Highly Potent Opioid Analgesics Lacking Side Effects. Majumdar S, Grinnell S, Le Rouzic V, Burgman M, Polikar L, Ansonoff M, Pintar J, Pan YX, Pasternak GW. Proc Natl Acad Sci U S A. 2011 Dec 6; 108(49):19778-83.
Abstract: Pain remains a pervasive problem throughout medicine, transcending all specialty boundaries. Despite the extraordinary insights into pain and its mechanisms over the past few decades, few advances have been made with analgesics. Most pain remains treated by opiates, which have significant side effects that limit their utility. We now describe a potent opiate analgesic lacking the traditional side effects associated with classical opiates, including respiratory depression, significant constipation, physical dependence, and, perhaps most important, reinforcing behavior, demonstrating that it is possible to dissociate side effects from analgesia. Evidence indicates that this agent acts through a truncated, six-transmembrane variant of the G protein-coupled mu opioid receptor MOR-1. Although truncated splice variants have been reported for a number of G protein-coupled receptors, their functional relevance has been unclear. Our evidence now suggests that truncated variants can be physiologically important through heterodimerization, even when inactive alone, and can comprise new therapeutic targets, as illustrated by our unique opioid analgesics with a vastly improved pharmacological profile.

Cocaine Hydrolase Encoded in Viral Vector Blocks the Reinstatement of Cocaine Seeking in Rats for 6 Months. Anker JJ, Brimijoin S, Gao Y, Geng L, Zlebnik NE, Parks RJ, Carroll ME. Biol Psychiatry. Epub 2011 Dec.
Abstract: Cocaine dependence is a pervasive disorder with high rates of relapse. In a previous study, direct administration of a quadruple mutant albumin-fused butyrylcholinesterase that efficiently catalyzes hydrolysis of cocaine to benzoic acid and ecgonine methyl ester acutely blocked cocaine seeking in an animal model of relapse. In the present experiments, these results were extended to achieve a long-duration blockade of cocaine seeking with a gene transfer paradigm using a related butyrylcholinesterase-based cocaine hydrolase (CocH). Male and female rats were allowed to self-administer cocaine under a fixed-ratio 1 schedule of reinforcement for approximately 14 days. Following the final self-administration session, rats were injected with CocH vector or a control injection (empty vector or saline), and their cocaine solutions were replaced with saline for 14 days to allow for extinction of lever pressing. Subsequently, they were tested for drug-primed reinstatement by administering intraperitoneal injections of saline (S), cocaine (C) (5, 10, and 15 mg/kg), and d-amphetamine according to the following sequence: S, C, S, C, S, C, S, d-amphetamine. Rats then received cocaine-priming injections once weekly for 4 weeks and, subsequently, once monthly for up to 6 months. Administration of CocH vector produced substantial and sustained CocH activity in plasma that corresponded with diminished cocaine-induced (but not amphetamine-induced) reinstatement responding for up to 6 months following treatment (compared with high-responding control animals). These results demonstrate that viral transfer of CocH may be useful in promoting long-term resistance to relapse to cocaine addiction.

Endocannabinoid Hydrolysis Generates Brain Prostaglandins That Promote Neuroinflammation. Nomura DK, Morrison BE, Blankman JL, Long JZ, Kinsey SG, Marcondes MC, Ward AM, Hahn YK, Lichtman AH, Conti B, Cravatt BF. Science. 2011 Oct 24.
Abstract: Endocannabinoid Hydrolysis Generates Brain Prostaglandins That Promote NaPhospholipase A2(PLA2) enzymes are considered the primary source of arachidonic acid for cyclooxygenase (COX)–mediated biosynthesis of prostaglandins. Here, we show that a distinct pathway exists in brain, where monoacylglycerol lipase (MAGL)

hydrolyzes the endocannabinoid 2-arachidonoylglycerol to generate a major arachidonate precursor pool for neuroinflammatory prostaglandins. MAGL-disrupted animals show neuroprotection in a parkinsonian mouse model. These animals are spared the hemorrhaging caused by COX inhibitors in the gut, where prostaglandins are instead regulated by cytosolic PLA2. These findings identify MAGL as a distinct metabolic node that couples endocannabinoid to prostaglandin signaling networks in the nervous system and suggest that inhibition of this enzyme may be a new and potentially safer way to suppress the proinflammatory cascades that underlie neurodegenerative disorders.

Cost-Benefit Analysis of Communities That Care Outcomes at Eighth Grade. Kuklinski, M., Briney, J., Hawkins, J., Catalano, R. Prev Sci. 2012 Apr;13(2):150-61.Epub 2011.
Abstract: This paper presents a cost-benefit analysis of the Communities That Care (CTC) prevention system, a public health approach to reducing risk, enhancing protection, and reducing the prevalence of adolescent health and behavior problems community wide. The analysis is based on outcomes from a panel of students followed from Grade 5 through Grade 8 in a randomized controlled trial involving 24 communities in 7 states. Previous analyses have shown that CTC prevented the initiation of cigarette smoking, alcohol use, and delinquency by the end of 8th grade in CTC communities compared to controls. This paper estimates long-term monetary benefits associated with significant intervention effects on cigarette smoking and delinquency as compared to the cost of conducting the intervention. Under conservative cost assumptions, the net present benefit is $5,250 per youth, including $812 from the prevention of cigarette smoking and $4,438 from the prevention of delinquency. The benefit-cost ratio indicates a return of $5.30 per $1.00 invested. Under less conservative but still viable cost assumptions, the benefit-cost ratio due to prevention of cigarette smoking and delinquency increases to $10.23 per $1.00 invested. Benefits from CTC's reduction in alcohol initiation as well as broader inclusion of quality-of-life gains would further increase CTC 's benefit-cost ratio. Results provide evidence that CTC is a cost-beneficial preventive intervention and a good investment of public dollars, even under very conservative cost and benefit assumptions.

Association of Childhood Adversities and Early-Onset Mental Disorders with Adult-Onset Chronic Physical Conditions. Scott K, Von Korff M, Angermeyer M, Benjet C, Bruffaerts R, de Girolamo G, Haro J, Lépine J, Ormel J, Posada-Villa J, Tachimori H, Kessler R. Arch Gen Psychiatry. 2011; 68 (8): 838-844.
Abstract: The physical health consequences of childhood psychosocial adversities may be as substantial as the mental health consequences, but whether this is the case remains unclear because much prior research has involved unrepresentative samples and a selective focus on particular adversities or physical outcomes. The association between early-onset mental disorders and subsequent poor physical health in adulthood has not been investigated. To investigate whether childhood adversities and early-onset mental disorders are independently associated with increased risk of a range of adult-onset chronic physical conditions in culturally diverse samples spanning the full adult age range. The design was cross-sectional community surveys of adults in 10 countries. Participants were adults (i.e., aged 18 years; N = 18 303), with diagnostic assessment and determination of age at onset of DSM-IV mental disorders, assessment of childhood familial adversities, and age of diagnosis or onset of chronic physical conditions. Main outcome measures included risk (i.e., hazard ratios) of adult-onset (i.e., at age >20 years) heart disease, asthma, diabetes mellitus, arthritis, chronic spinal pain, and chronic headache as a function of specific childhood adversities and early-onset (i.e., at age <21 years) DSM-IV depressive and anxiety disorders, with mutual adjustment. Results showed that a history of 3 or more childhood adversities was independently associated with onset of all 6 physical conditions (hazard ratios, 1.44 to 2.19). Controlling for current mental disorder made little difference to these associations. Early-onset mental disorders were independently associated with onset of 5 physical conditions (hazard ratios, 1.43 to 1.66). These results are consistent with the hypothesis that childhood adversities and early-onset mental disorders have independent, broad-spectrum effects that increase the risk of diverse chronic physical conditions in later life. They require confirmation in a prospectively designed study. The long course of these associations has theoretical and research implications.

Involvement Of Dopamine Receptors In Binge Methamphetamine-Induced Activation Of Endoplasmic Reticulum And Mitochondrial Stress Pathways. Beauvais G, Atwell K, Jayanthi S, Ladenheim B, Cadet JL. PLoS One. 2011; 6(12):e28946. Epub 2011 Dec 13.
Abstract: Single large doses of methamphetamine (METH) cause endoplasmic reticulum (ER) stress and mitochondrial dysfunctions in rodent striata. The dopamine D(1) receptor appears to be involved in these METH-mediated stresses. The purpose of this study was to investigate if dopamine D(1) and D(2) receptors are involved in ER and mitochondrial stresses caused by single-day METH binges in the rat striatum. Male Sprague-Dawley rats received 4 injections of 10 mg/kg of METH alone or in combination with a putative D(1) or D(2) receptor antagonist, SCH23390 or raclopride, respectively, given 30 min prior to each METH injection. Rats were euthanized at various timepoints afterwards. Striatal tissues were used in quantitative RT-PCR and western blot analyses. We found that binge METH injections caused increased expression of the pro-survival genes, BiP/GRP-78 and P58(IPK), in a SCH23390-sensitive manner. METH also caused up-regulation of ER-stress genes, Atf2, Atf3, Atf4, CHOP/Gadd153 and Gadd34. The expression of heat shock proteins (HSPs) was increased after METH injections. SCH23390

completely blocked induction in all analyzed ER stress-related proteins that included ATF3, ATF4, CHOP/Gadd153, HSPs and caspase-12. The dopamine D(2)-like antagonist, raclopride, exerted small to moderate inhibitory influence on some METH-induced changes in ER stress proteins. Importantly, METH caused decreases in the mitochondrial anti-apoptotic protein, Bcl-2, but increases in the pro-apoptotic proteins, Bax, Bad and cytochrome c, in a SCH23390-sensitive fashion. In contrast, raclopride provided only small inhibition of METH-induced changes in mitochondrial proteins. These findings indicate that METH-induced activation of striatal ER and mitochondrial stress pathways might be more related to activation of SCH23390-sensitive receptors.

CONGRESSIONAL AFFAIRS SECTION
(Prepared April 26, 2012)

Appropriations/Budget

In the President's Fiscal Year 2013 budget, the request for NIH is $30.62 billion, identical to the enacted level in FY 2012 of 30.62 billion. For NIDA, the Fiscal Year 2013 request is $1.054 billion, compared to an enacted level in FY 2012 of $1.052 billion.

Congressional Briefings/Meetings of Interest

Friends of NIDA Coalition Hosts Congressional Briefing on Developing Medications to Treat Addiction. On March 1, the FoN organized a briefing on the Hill focused on the development of new medications to treat addictions and the challenges in getting such medications FDA-approved and marketed (see Developing Medications to Treat Addiction: Challenges for Science, Policy, and Practice). Although the drug abuse fields has made great strides in advancing the science needed to develop medications, and there are promising new approaches already in clinical trials (e.g., vaccines and an implantable form of buprenorphine), the past few years have seen some of the largest pharmaceutical companies reduce or completely eliminate their research and development in this area. After discussing the current and future directions of the science, there was a round-table discussion on the reasons for the present state of affairs in the pharmaceutical industry and what the future may hold. With Dr. Volkow at this briefing were Dr. Phil Skolnick, Director of NIDA's Division of Pharmacotherapies and Medical Consequences of Drug Abuse; Dr. David Gastfriend of Alkermes, Inc., the maker of Vivitrol (the depot injection of naltrexone); and Shaun Thaxter of Reckitt Benckiser Pharmaceuticals, Inc., maker of Suboxone. Past collaboration between NIDA and drug companies were instrumental in making drugs like Vivitrol and Suboxone a reality. Although the market for addiction medications presents unique challenges to drug companies, NIDA remains committed to creating new opportunities to engage the pharmaceutical industry in addiction treatment research.

Member Meetings

Dr. Volkow had a few meetings recently with members of Congress:

- 3/7/12 with John Sullivan (R-OK). Mr. Sullivan was interested in discussing research from NIDA's treatment and medications portfolios.
- 3/8/12 with John Larson (D-CT). Mr. Larson was interested in discussing evidence based prevention approaches focused on youth.
- 4/11/12 with Harold Rogers (R-KY). As part of the Prescription Drug Abuse Summit, Mr. Rogers wanted to thank Dr. Volkow for her participation, and to discuss prescription drug abuse issues. He also addressed the pending reorganization of alcohol and drug abuse and addiction research at the NIH.
- 4/12/12 with Mary Bono Mack (R-CA) (at the Prescription Drug Summit, as part of a larger discussion) to discuss prescription drug abuse issues.

Federal Regulations/Investigations/Reports Requested by Congress

Drug Prevention and Treatment Programs – On December 12, 2011, GAO notified the Secretary of HHS that they are initiating a review of drug prevention and treatment programs within HHS. The review is in response to a request by the Senate Caucus on International Narcotics Control. Following are the key questions/objectives of the review:

(1) To what extent do federal agencies fund drug prevention and treatment programs? (2) To what extent do federal agencies evaluate the drug prevention and treatment programs they fund? HHS agencies involved in the review include the Centers for Disease Control and Prevention (CDC), the Health Resources and Services Administration (HRSA), the Indian Health Service (IHS), the National Institutes of Health (NIH), and the Substance Abuse and Mental Health Services Administration (SAMHSA). Work began on this review in January 2012.

National Drug Control Strategy – On February 9, 2012, GAO notified the Secretary of HHS that they were initiating a review of the National Drug Control Strategy. The review is in response to a request by the Senate Caucus on International Narcotics Control. Following are the key questions/objectives of the review: (1) To what extent has the 2010 National Drug Control Strategy been implemented, and how does the Office on National Drug Control Prevention (ONDCP) assess the effectiveness of the Strategy in preventing and reducing drug use? (2) To what extent does ONDCP have metrics in place to assess drug treatment and prevention programs? (3) What does the available research suggest about the effect of societal factors, such as state laws allowing the use of marijuana for medical purposes, the drug legalization movement, and pro-drug media, on youth drug use? HHS agencies involved in the review include SAMHSA, NIH, and CDC. In addition, GAO plans to contact several federal agencies that engage in drug control activities in order to obtain information and perspectives on the status and implementation of the national drug control strategy and performance metrics to assess drug prevention and treatment programs. Work began on this review in February 2012.

Inmate Reentry Programs - On March 5, 2012 GAO notified the Secretary of HHS that in response to a Congressional mandate, they were initiating a review of Inmate Reentry Programs. This will focus on: 1) What federal programs exist to support former inmates' reentry into society; 2) To what extent is there duplication of efforts; 3) what efforts the Federal Interagency Reentry Council and key agencies have taken to reduce duplication and provide effective coordination; and 4) what do federal agencies know about the effectiveness of programs to help former inmates re-enter society. Within HHS, participating agencies include ACF, SAMHSA, IHS and NIH. The DOJ will also be consulted. NIDA, NIMH, and NIAAA are participating from NIH. Work began on this review in April 2012.

NIH/HHS POLICY UPDATES

For a complete list see http://grants.nih.gov/grants/policy/policy.htm

March 30

Notices relating to the 2011 revised regulation on Promoting Objectivity in Research

April 18

March 21

PROGRAM ACTIVITIES/FOAS

New NIDA RFAs

On April 24, 2012, NIDA issued an RFA entitled **FY13 NIDA Avant-Garde Award Program for HIV/AIDS Research (DP1) RFA-DA-13-002**.

New NIDA Program Announcements

On March 21, 2012, NIDA issued a PAS entitled **Strategic Alliances for Medications Development to Treat Substance Use Disorders (R01) PAS-12-122.**

On March 23, 2012, NIDA issued a PA entitled **Technology-Based Interventions to Promote Engagement in Care and Treatment Adherence for Substance Abusing Populations with HIV (R01) PA-12-117; (R34) PA-12-118.** The purpose of this FOA

Pilot and Feasibility Studies in Preparation for Drug and Alcohol Abuse Prevention Trials (R34) PA-12-171.

Pre-Application for the **FY13 NIDA Avant-Garde Award Program for HIV/AIDS Research (X02) PAR-12-164.**

New FOAs Issued by the NIH Roadmap

On February 16, 2012, the NIH Common Fund issued a Roadmap PA entitled **Assays for High Throughput Screening (HTS) to Discover Chemical Probes in the Molecular Libraries Probe Production Centers Network (MLPCN) (X01) PAR-12-108.** The purpose of this FOA is to encourage the investigators to form collaborations with the Molecular Libraries Probe Production Centers Network (MLPCN) to implement HTS-ready assays for the discovery and development of small molecule chemical probes. Open date: March 16, 2012. Application due dates: April 16, 2012; August 15, 2012; by 5:00 PM local time of applicant organization. AIDS application due date: Not Applicable.

On March 19, 2012, the NIH Common Fund issued a Roadmap PA entitled **Use-Oriented Basic Research: Change Mechanisms of Behavioral Social Interventions (Admin Supp) PA-12-119.** The purpose of this FOA is to solicit administrative supplement applications to study possible mechanisms of action of behavioral or social interventions, with the ultimate aim of informing the simplification or other modifications of behavioral or social interventions to improve use in target settings and by target interventionists. Open date: April 15, 2012. Application due date: May 15, 2012 by 5:00 PM local time of applicant organization. AIDS application due date: Not Applicable.

New Administrative Supplement Program Announcements Issued by NIH

On February 13, 2012, NIDA, in collaboration with numerous other NIH components, issued an administrative supplement entitled **Administrative Supplements to Existing NIH Grants and Cooperative Agreements (Parent Admin Supp) PA-12-100**. The Office of Research on Women's Health (ORWH), participating Institutes and Centers (ICs) of the National Institutes of Health (NIH), and the Office of Dietary Supplements (ODS) announce the continuation of the program for administrative supplements to research grants to support individuals with high potential to re-enter an active research career after an interruption for family responsibilities or other qualifying circumstances. The purpose of these supplements is to encourage such individuals to re-enter research careers within the missions of all the program areas of NIH. Open date: Due dates may vary by awarding IC. Application due date(s): Due dates may vary by awarding IC. AIDS application date: Not applicable.

On April 6, 2012, NIDA, in collaboration with numerous other NIH components, issued an administrative supplement entitled **Research Supplements to Promote Diversity in Health-Related Research (Admin Supp) PA-12-149.** The National Institutes of Health (NIH) and the Centers for Disease Control and Prevention (CDC) hereby notify Program Director(s)/Principal Investigator(s) (PD(s)/PI(s)) holding specific types of NIH research grants, listed in the full Funding Opportunity Announcement (FOA) that funds are available for administrative supplements to improve the diversity of the research workforce by supporting and recruiting students, postdoctorates, and eligible investigators from groups that have been shown to be underrepresented in health-related research. Open date: Due dates may vary by awarding IC. Application due date(s): Due dates may vary by awarding IC. AIDS application date: Not applicable.

On April 6, 2012, NIDA, in collaboration with numerous other NIH components, issued an administrative supplement entitled **Research Supplements to Promote Re-Entry into Biomedical and Behavioral Research Careers (Admin Supp) PA-12-150.** The Office of Research on Women's Health (ORWH), participating Institutes and Centers (ICs) of the National Institutes of Health (NIH), and the Office of Dietary Supplements (ODS) announce the continuation of the program for administrative supplements to research grants to support individuals with high potential to re-enter an active research career after an interruption for family responsibilities or other qualifying circumstances. The purpose of these supplements is to encourage such individuals to re-enter research careers within the missions of all the program areas of NIH. Open date: Due dates may vary by awarding IC. Application due date(s): Due dates may vary by awarding IC. AIDS application date: Not applicable.

New RFAs Issued by Other NIH/HHS Components in which NIDA is a participant

On February 21, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **NIH Blueprint for Neuroscience Research Grand Challenge: Discovering Novel Drugs for Disorders of the Nervous System (U01) RFA-NS-13-003.** The National Institutes of Health (NIH) announces a unique opportunity for investigators working with small molecule compounds to gain access to a robust 'virtual pharma' network to discover neurotherapeutic drugs. Successful applicants to this FOA will become collaborative participants in this network, receiving both funding and no-cost access to contracted drug discovery services that are not typically available to the academic research community. Open Date: September 8, 2012. Application due date: October 8, 2012, by 5:00 PM local time of applicant organization. AIDS application date: Not Applicable.

On February 29, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Small Business Alzheimer's Disease Research (SBBR [R34/R44]) RFA-OD-12-003; (STTR [R41/R42]) RFA-OD-12-004**. The purpose of this funding opportunity announcement is to solicit Small Business Innovation Research (SBIR) and Small Business Technology Transfer Research (STTR) applications from eligible small business concerns in the area of Alzheimer's disease. Open Date: March 30, 2012. Application due date: April 30, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On April 20, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Clinical Sequencing Exploratory Research (U01) RFA-HG-12-009.** Applications submitted in response to this FOA will address critical questions about the application of genomic sequencing to clinical care of individual patients, from generation of genomic sequence data, to interpretation and translation of the data for the physician, to communication to the patient, including an examination of the ethical, legal and psychosocial implications of bringing broad genomic data into the clinic. Open date: June 26, 2012. Application due date: July 26, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

New PAs Issued with Other NIH/HHS Components in which NIDA is a participant

On February 23, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Research on the Health of LGBTI Populations (R01) PA-12-111; (R03) PA-12-112; (R21) PA-12-113.** The National Institutes of Health (NIH) is committed to supporting research that will increase scientific understanding of the health status of various population groups and improve the effectiveness of health interventions and services for individuals within those groups. This Funding Opportunity Announcement (FOA) highlights a particular community: lesbian, gay, bisexual, transgender, intersex, and related populations (designated here as LGBTI populations).Open Date: May 5, 2012. Application due dates: Standard dates apply. AIDS application due date: Standard dates apply.

On March 19, 2012, NIDA issued a PA entitled **Pilot Health Services and Economic Research on the Treatment of Drug, Alcohol, and Tobacco Abuse (R34) PA-12-130.** This Funding Opportunity Announcement (FOA) issued by the National Institute on Drug Abuse (NIDA) and the National Institute on Alcohol Abuse and Alcoholism (NIAAA) encourages pilot and preliminary research on (1) organizational and/or systems-level interventions that may optimize access, utilization, delivery, quality, and/or cost of treatment services for drug, tobacco, or alcohol abuse or dependence through the use of evidence-based practices; (2) organizational and/or systems-specific adaptations to existing evidence-based practices necessary to facilitate their implementation in these new contexts; and (3) novel service delivery models to be pilot tested in preparation for larger-scale effectiveness trials. Open date: May 16, 2012. Application due date: standard dates apply. AIDS application due date: standard dates apply.

On March 19, 2012, NIDA issued a PA entitled **Health Services and Economic Research on the Prevention and Treatment of Drug, Alcohol, and Tobacco Abuse (R01) PA-12-127; (R21) PA-12-128; (R03) PA-12-129 .** This Funding Opportunity Announcement (FOA) issued by the National Institute on Drug Abuse (NIDA) and the National Institute on Alcohol Abuse and Alcoholism (NIAAA) encourages Research Project Grant (R01), Exploratory/Developmental (R21), and Small Grant (R03) applications on health services and economic research to improve the quality of prevention, treatment, and recovery support services for drug, alcohol and tobacco abuse. Such research projects might emphasize any of the following subjects: (1) clinical quality improvement; (2) organization and delivery of services; (3) implementation research; (4) economic and cost studies; or (5) development or improvement of research methodology, analytic approaches, and measurement instrumentation used in the study of drug, alcohol, and tobacco prevention, treatment, and recovery services. Open date: May 16, 2012. Application due date: standard dates apply. AIDS application due date: standard dates apply.

On March 30, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Senior Scientist Research Award (K05) PA-12-148.** The purpose of the Senior Scientist Research (K05) is intended to provide protected time for outstanding senior scientists who have demonstrated a sustained high level of productivity conducting biomedical research relevant to the scientific mission of the appropriate institute to focus on their research and to provide mentoring of new investigators. Open Date: May 12, 2012. Application due dates: Standard dates apply. AIDS application due date: Standard dates apply.

On April 24, 2012, NIDA, in collaboration with numerous other NIH components, issued a PAR entitled **Multidisciplinary Studies of HIV/AIDS and Aging (R21) PAR-12-174; (R01) PAR-12-175; (R03) PAR-12-176.** This FOA invites applications proposing to study HIV infection, HIV-associated conditions, HIV treatment, and/or biobehavioral or social factors associated with HIV/AIDS in the context of aging and/or in older adults. Research approaches of interest include clinical translational, observational, and intervention studies in domestic and international settings. Open date: July 7, 2012. Application due dates: August 7, 2012; December 7, 2012; April 9, 2013; August 7, 2013; December 6, 2013; April 9, 2014; August 7, 2014; December 9, 2014; April 7, 2015, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

New NIH FOAs Issued in Collaboration with the FDA Center for Tobacco Products

On January 20, 2012, NIDA, in collaboration with numerous other NIH components and with the FDA Center for Tobacco Products, issued a Notice of Intent entitled **Administrative Supplements to NIH-funded Program Projects/Center Grants (with FDA) (P01, P50, and P60) NOT-CA-12-007.** The administrative supplements program and other FDA-NIH initiatives (for details, see http://cancercontrol.cancer.gov/nih-fda) are intended to provide a rapid mechanism for the FDA to promote research and generate findings needed to inform the development of regulations pertaining to the manufacture, distribution, and marketing of tobacco products. Consistent with the FDA CTP mission, this Notice seeks administrative supplements that expand, enhance, or facilitate research relevant to these issues. Receipt date: April 6, 2012. Earliest anticipated start date: September 2012.

On February 13, 2012, NIDA, in collaboration with numerous other NIH components and with the FDA Center for Tobacco Products, issued a Notice of Intent entitled **Notice of Intent to Publish a Request for Applications for Centers of Excellence for Research Relevant to the Family Smoking Prevention and Tobacco Control Act (P50) NOT-DA-12-007**. This Notice is provided to allow prospective applicants sufficient time to develop essential collaborations and plans prior to submitting an application in response to the published FOA. Projects resulting from this FOA are expected to serve the FDA by generating relevant findings and data needed to inform the regulation of the manufacture, distribution, and marketing of tobacco products to protect public health. Consistent with the FDA CTP mission, this FOA seeks research centers that address such topics as: the diversity of tobacco products, reducing addiction, reducing toxicity and carcinogenicity, adverse health consequences, communications, marketing of tobacco products, and economics and policies. Estimated publication date of announcement: Spring 2012. First estimated application due date: Fall 2012. Earliest estimated start date: July 2013.

NIDA PUBLICATIONS

NIDA Notes
The final print issue of *NIDA Notes* was mailed in early April. The first all-Web *NIDA Notes* articles have been posted, and new articles are slated to appear biweekly.

Addiction Science & Clinical Practice
Addiction Science & Clinical Practice, the journal founded by NIDA to promote research-practice dialogue, posted its first articles under its new owner, *Biomed Central*, and new Editors, Richard Saitz, MD, MPH and Jeffrey Samet, MD, MA, MPH. NIDA continues to support the journal during this start-up period.

PRESS RELEASES

February 21, 2012 – NIDA creates easy-to-read website on drug abuse

March 12, 2012 – National Inhalant Prevention Coalition to highlight dangers of helium

March 21, 2012 – Study provides clues for designing new anti-addiction medications

MEETINGS/CONFERENCES

Select Meetings and Conferences in which NIDA played a significant role

On April 12, 2012, NIDA, FDA, and CDC held a meeting titled Role of Naloxone in Opioid Overdose Fatality Prevention to discuss whether naloxone should be made more widely available to trained individuals who are not part of the healthcare system to reduce opioid overdose fatalities. Participants included academic, government and industry experts and patient advocates. Among the topics discussed were: populations at risk; successful public health interventions for preventing and treating opioid overdose; issues related to product development; and the social and legal aspects of wider naloxone availability.

Dr. Nora Volkow gave a keynote address titled "It's *Not* What the Doctor Ordered" at the inaugural National Prescription Drug Abuse Summit in Orlando, FL. This summit, held April 10-12, 2012, brought together state and national leaders, including the Surgeon General, law enforcement officials, medical professionals, community advocates, treatment experts, educators and others to share strategies to combat this devastating problem. NIDA also showcased resources for healthcare professionals and teens; including NIDA's PEERx, an online peer-to-peer initiative that educates teens and helps them spread the word about the dangers of prescription drug abuse.

On April 19-22, 2012, NIDA sponsored a Blending Conference on SBIRT at the ASAM Annual Meeting held in Atlanta, GA. The Blending meeting provided a forum to discuss emerging research findings and their implications in clinical practice. Workshops were offered on a variety of topics including: Motivational Interviewing in primary care; New pharmacotherapies for SUD; Screening, Brief Intervention, and Referral to Treatment (SBIRT); and HIV testing and intervention in integrated treatment settings.

NIDA held a research track at the American Psychiatric Association Annual Meeting in Philadelphia, PA, May 5-9, 2012. NIDA convened a number of sessions on topics unique to addiction science. A special performance of NIDA's *Addiction Performance Project*, with a dramatic reading by Dianne Wiest and other professional actors, and chaired by NIDA Director, Dr. Nora Volkow, was also featured at this year's meeting.

Upcoming Conferences/Exhibits

NIDA will participate the 74th annual meeting of the College on Problems of Drug Dependence (CPDD) - June 9-14, 2012 in Palm Springs, CA.

NIDA will participate in the American Psychological Association annual convention - August 2-5, 2012, in Orlando, FL.

COMMUNITY AND PRESS EVENTS

USA Science & Engineering Festival
April 28-29, 2012 in Washington, D.C. Designed to foster an enthusiasm for science among our Nation's youth.

Addiction Performance Project (APP)
April 16, 2012 in Chicago, IL, and May 8-9, 2012 in Philadelphia, PA. APP is a CME & CE program to help break down the stigma associated with addiction and promote a healthy dialogue that fosters compassion, cooperation, and understanding for patients living with this disease. This project is part of NIDA's outreach to practicing health professionals and those in training.

Screening of the motion picture documentary "Addiction, Inc."
June 1, 2012 in the Lipsett Auditorium on the NIH campus in Bethesda, MD, from 2-5 pm. Dr. Nora Volkow will host a free screening of the motion picture documentary "Addiction, Inc." The 100 minute film will start at 2 pm and the film's producer (Charles Evans) will be in the auditorium to answer questions. The film chronicles the experience of research scientist Victor DeNoble, who became a whistleblower for tobacco company activities in the 1980s. Dr. DeNoble will be available for questions via videocast from his home in California.

STAFF HONORS AND AWARDS

Dr. Thomas Brady, DESPR, was awarded the 2012 Joint Meeting on Adolescent Treatment Effectiveness (JMATE) Government Facilitation of Evidence-Based Practice Award posthumously on April 11, 2012 for his work as a government official dedicated to moving the field towards evidence-based practice related to adolescent substance abuse treatment. Key review criteria include evidence of: (1) facilitating the development, adoption, and/or use of evidence-based practices in community-based settings; (2) a sustained commitment to work with community-based treatment providers to help them actually use the knowledge/skills/materials/technologies; and (3) a specific focus on adolescent treatment.

Dr. Harold Perl, DESPR, was awarded a 2011 Meritorious Research Service Commendation by the American Psychological Association's Board of Scientific Affairs at the December 2011 APA Board of Directors meeting in Washington, DC. The award recognizes individuals who have made outstanding contributions to psychological science through their service as employees of the federal government or other organizations.

STAFF CHANGES

New Employees

Helio Chaves joined NIDA as the Deputy Executive Officer in March 2012. Helio is a seasoned professional who possesses more than 15 years of diverse experience in both private and government sectors. Helio's immediate past assignment was with the PSC where he served as the Director of the Division of (Grants) Payment Management. Other prior roles include Director of the Division of User Fees at the FDA, Budget Officer at the PSC, Accounting Operations Supervisor at the Holocaust Memorial Museum and Staff Accountant at the CPA firm Watkins, Meegan, Drury & Co. Helio has a Bachelor of Science degree in accounting from the University of Massachusetts at Dartmouth.

Retirements and Departures

Thomas Hilton, Ph.D. joined NIDA's Services Research Branch as a Program Official in 1999. His grant portfolio at NIDA originally emphasized organizational effectiveness in health service delivery and he recently started a portfolio focusing on addiction recovery and services reengineering. Tom also had a portfolio of grants advancing research methodology and psychological measurement. His role as PROMIS Science Officer also kept expanding as he convinced the Military and VA to adopt PROMIS. This is a position for which he is doubly well-suited because he is a retired Navy Medical Service Corps Captain. His military service began in 1968 when he joined the Navy Reserves. On April 30, 2012, Tom retired after 44 years of government service.

New Roles within NIDA

Dr. Harold Perl joined DESPR as Acting Branch Chief for the Prevention Research Branch, effective March 19, 2012. Dr. Perl moved to the Prevention Branch in DESPR from NIDA's CCTN where he had been Senior Lead for Behavioral Research, Dissemination and Training since 2005. Prior to joining NIDA, he served in various

programmatic and management capacities at the National Institute on Alcohol Abuse and Alcoholism (NIAAA) between 1989 and 2005, culminating as Chief of the Health Services Research Branch, with responsibility for developing and overseeing programs that focused on alcohol health services research and the dissemination and implementation of science-based substance abuse treatment practices.

Jennifer Bidle was selected as the as the new Chief of the Management Analysis Branch (MAB), Office of Management, NIDA in April 2012. Jennifer began her government career at the NIH in the Office of Management Assessment 10 years ago and has worked for NIDA in the Management Analysis Branch since August 2008. She earned an undergraduate degree in Health Administration and Policy from the University of Maryland Baltimore County and a Master of Business Administration from Mount St. Mary's University. During her time at NIDA, Jennifer has accomplished several tasks including developing standard Office of Management communication methods, conducting workload analyses and risk management reviews, and participating in strategic planning efforts.