Director's Report

to the

NATIONAL ADVISORY COUNCIL ON DRUG ABUSE

February 2012

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

RESEARCH HIGHLIGHTS

Dopamine D4 receptor, but not the ADHD-associated D4.7 variant, forms functional heteromers with the dopamine D2S receptor in the brain. González S, Rangel-Barajas C, Peper M, Lorenzo R, Moreno E, Ciruela F, Borycz J, Ortiz J, Lluís C, Franco R, McCormick PJ, Volkow ND, Rubinstein M, Floran B, Ferré S. Mol Psychiatry. 2011 Aug 16.

Abstract: Polymorphic variants of the dopamine D4 receptor have been consistently associated with attention-deficit hyperactivity disorder (ADHD). However the functional significance of the risk polymorphism (variable number of tandem repeats in exon 3) is still unclear. Here we show that whereas the most frequent 4-repeat (D4.4) and the 2-repeat (D4.2) variants form functional heteromers with the short isoform of the dopamine D2 receptor (D2S), the 7-repeat risk allele (D4.7) does not. D2 receptor activation in the D2S-D4 receptor heteromer potentiates D4 receptor-mediated MAPK signaling in transfected cells and in the striatum, which did not occur in cells expressing D4.7 or in the striatum of knock-in mutant mice carrying the 7 repeats of the human D4.7 in the third intracellular loop of the D4 receptor. In the striatum D4 receptors are localized in cortico-striatal glutamatergic terminals, where they selectively modulate glutamatergic neurotransmission by interacting with D2S receptors. This interaction shows the same qualitative characteristics than the D2S-D4 receptor heteromer-mediated MAPK signaling and D2S receptor activation potentiates D4 receptor-mediated inhibition of striatal glutamate release. It is therefore postulated that dysfunctional D2S-D4.7 heteromers may impair presynaptic dopaminergic control of corticostriatal glutamatergic neurotransmission and explain functional deficits associated with ADHD. Press Release: NIH study in mice shows a potential new target for the treatment of ADHD

Linking context with reward: a functional circuit from hippocampal CA3 to ventral tegmental area. Luo AH, Tahsili-Fahadan P, Wise RA, Lupica CR, Aston-Jones G. Science. 2011 Jul 15;333(6040):353-7.

Abstract: Reward-motivated behavior is strongly influenced by the learned significance of contextual stimuli in the environment. However, the neural pathways that mediate context-reward relations are not well understood. We have identified a circuit from area CA3 of dorsal hippocampus to ventral tegmental area (VTA) that uses lateral septum (LS) as a relay. Theta frequency stimulation of CA3 excited VTA dopamine (DA) neurons and inhibited non-DA neurons. DA neuron excitation was likely mediated by disinhibition because local antagonism of γ-aminobutyric acid receptors blocked responses to CA3 stimulation. Inactivating components of the CA3-LS-VTA pathway blocked evoked responses in VTA and also reinstatement of cocaine-seeking by contextual stimuli. This transsynaptic link between hippocampus and VTA appears to be an important substrate by which environmental context regulates goal-directed behavior.

Mirtazapine to Reduce Methamphetamine Use: a Randomized Controlled Trial Colfax GN, Santos GM, Das M, Santos DM, Matheson T, Gasper J, Shoptaw S, Vittinghoff E. Arch Gen Psychiatry. 2011 Nov;68(11):1168-75.

Abstract: No approved pharmacologic treatments for methamphetamine dependence exist. Methamphetamine use is associated with high morbidity and is a major cofactor in the human immunodeficiency virus epidemic among men who have sex with men (MSM). The objective of the study is to determine whether mirtazapine would reduce methamphetamine use among MSM who are actively using methamphetamine. A Double-blind, randomized, controlled, 12-week trial of mirtazapine vs placebo conducted from September 5, 2007, to March 4, 2010, San Francisco Department of Public Health. Participants were actively using, methamphetamine-dependent, sexually active MSM seen weekly for urine sample collection and substance use counseling and were randomly assigned to daily oral mirtazapine (30 mg) or placebo; both arms included 30-minute weekly substance use counseling. The primary study outcome was reduction in methamphetamine-positive urine test results. Secondary outcomes were study medication adherence (by self-report and medication event monitoring systems) and sexual risk behavior. Sixty MSM were randomized, 85% of follow-up visits were completed, and 56 participants (93%) completed the final visit. In the primary intent-to-treat analysis, participants assigned to the mirtazapine group had fewer methamphetamine-positive urine test results compared with participants assigned to the placebo group (relative risk, 0.57; 95% CI, 0.35-0.93, $P = .02$). Urine positivity decreased from 67% (20 of 30 participants) to 63% (17 of 27) in the placebo arm and from 73% (22 of 30) to 44% (12 of 27) in the mirtazapine arm. The number needed to treat to achieve a negative weekly urine test result was 3.1. Adherence was 48.5% by medication event monitoring systems and 74.7% by self-report; adherence measures were not significantly different between arms (medication event monitoring systems, $P = .82$; self-report, $P = .92$). Most sexual risk behaviors decreased significantly more among participants taking mirtazapine compared with those taking placebo (number of male partners with whom methamphetamine was used, $P = .009$; number of male partners, $P = .04$; episodes of anal sex with serodiscordant partners, $P = .003$; episodes of unprotected anal sex with serodiscordant partners, $P = .003$; episodes of insertive anal sex with serodiscordant

partners, $P = .001$). There were no serious adverse events related to study drug or significant differences in adverse events by arm ($P \geq .99$). The addition of mirtazapine to substance use counseling decreased methamphetamine use among active users and was associated with decreases in sexual risk despite low to moderate medication adherence. Trial Registration clinicalTrials.gov Identifier NCT00497081.

Novel Cocaine Vaccine Linked to a Disrupted Adenovirus Gene Transfer Vector Blocks Cocaine Psychostimulant and Reinforcing Effects. Wee S, Hicks MJ, De BP, Rosenberg JB, Moreno AY, Kaminsky SM, Janda KD, Crystal RG, Koob GF. Neuropsychopharmacology. 14 September 2011.
Abstract: Immunotherapy is a promising treatment for drug addiction. However, insufficient immune responses to vaccines in most subjects pose a challenge. In this study, the authors tested the efficacy of a new cocaine vaccine (dAd5GNE) in antagonizing cocaine addiction-related behaviors in rats. This vaccine used a disrupted serotype 5 adenovirus (Ad) gene transfer vector coupled to a third-generation cocaine hapten, termed GNE (6-(2R,3S)-3-(benzoyloxy)-8-methyl-8-azabicyclo [3.2.1] octane-2-carboxamido-hexanoic acid). Three groups of rats were immunized with dAd5GNE. One group was injected with (3)H-cocaine, and radioactivity in the blood and brain was determined. A second group was tested for cocaine-induced locomotor sensitization. A third group was examined for cocaine self-administration, extinction, and reinstatement of responding for cocaine. Antibody titers were determined at various time-points. In each experiment, a control group was added that was immunized with dAd5 without a hapten. The vaccination with dAd5GNE produced long-lasting high titers (>10(5)) of anti-cocaine antibodies in all of the rats. The vaccination inhibited cocaine-induced hyperlocomotor activity and sensitization. Vaccinated rats acquired cocaine self-administration, but showed less motivation to self-administer cocaine under a progressive-ratio schedule than control rats. When cocaine was not available in a session, control rats exhibited 'extinction burst' responding, whereas vaccinated rats did not. Moreover, when primed with cocaine, vaccinated rats did not reinstate responding, suggesting a blockade of cocaine-seeking behavior. These data strongly suggest that dAd5GNE vector-based vaccine may be effective in treating cocaine abuse and addiction.

Molecular mechanism for a gateway drug: epigenetic changes initiated by nicotine prime gene expression by cocaine. Levine A, Huang Y, Drisaldi B, Griffin EA Jr, Pollak DD, Xu S, Yin D, Schaffran C, Kandel DB, Kandel ER. Sci Transl Med. 2011 Nov 2;3(107):107ra109.
Abstract: In human populations, cigarettes and alcohol generally serve as gateway drugs, which people use first before progressing to marijuana, cocaine, or other illicit substances. To understand the biological basis of the gateway sequence of drug use, we developed an animal model in mice and used it to study the effects of nicotine on subsequent responses to cocaine. We found that pretreatment of mice with nicotine increased the response to cocaine, as assessed by addiction-related behaviors and synaptic plasticity in the striatum, a brain region critical for addiction-related reward. Locomotor sensitization was increased by 98%, conditioned place preference was increased by 78%, and cocaine-induced reduction in long-term potentiation (LTP) was enhanced by 24%. The responses to cocaine were altered only when nicotine was administered first, and nicotine and cocaine were then administered concurrently. Reversing the order of drug administration was ineffective; cocaine had no effect on nicotine-induced behaviors and synaptic plasticity. Nicotine primed the response to cocaine by enhancing its ability to induce transcriptional activation of the FosB gene through inhibition of histone deacetylase, which caused global histone acetylation in the striatum. We tested this conclusion further and found that a histone deacetylase inhibitor simulated the actions of nicotine by priming the response to cocaine and enhancing FosB gene expression and LTP depression in the nucleus accumbens. Conversely, in a genetic mouse model characterized by reduced histone acetylation, the effects of cocaine on LTP were diminished. We achieved a similar effect by infusing a low dose of theophylline, an activator of histone deacetylase, into the nucleus accumbens. These results from mice prompted an analysis of epidemiological data, which indicated that most cocaine users initiate cocaine use after the onset of smoking and while actively still smoking, and that initiating cocaine use after smoking increases the risk of becoming dependent on cocaine, consistent with our data from mice. If our findings in mice apply to humans, a decrease in smoking rates in young people would be expected to lead to a decrease in cocaine addiction. Press Release: NIH study examines nicotine as a gateway drug

Cocaine-induced cortical microischemia in the rodent brain: clinical implications. Ren H, Du C, Yuan Z, Park K, Volkow ND, Pan Y. Mol Psychiatry. 2011 Nov 29.

Abstract: Cocaine-induced stroke is among the most serious medical complications associated with its abuse. However, the extent to which acute cocaine may induce silent microischemia predisposing the cerebral tissue to neurotoxicity has not been investigated; in part, because of limitations of current neuroimaging tools, that is, lack of high spatiotemporal resolution and sensitivity to simultaneously measure cerebral blood flow (CBF) in vessels of different calibers (including capillaries) quantitatively and over a large field of view. Here we combine ultrahigh-resolution optical coherence tomography to enable tracker-free three-dimensional (3D) microvascular angiography and a new phase-intensity-mapping algorithm to enhance the sensitivity of 3D optical Doppler tomography for simultaneous capillary CBF quantization. We apply the technique to study the responses of cerebral microvascular networks to single and repeated cocaine administration in the mouse somatosensory cortex. We show that within 2-3 min after cocaine administration CBF markedly decreased (for example, ~70%), but the magnitude and recovery differed for the various types of vessels; arterioles had the fastest recovery (~5 min), capillaries varied drastically (from 4-20 min) and venules showed relatively slower recovery (~12 min). More importantly, we showed that cocaine interrupted CBF in some arteriolar branches for over 45 min and this effect was exacerbated with repeated cocaine administration. These results provide evidence that cocaine doses within the range administered by drug abusers induces cerebral microischemia and that these effects are exacerbated with repeated use. Thus, cocaine-induced microischemia is likely to be a contributor to its neurotoxic effects.

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 Nov 11;334(6057):809-13. Epub 2011 Oct 20.

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.

Early-Life Experience Decreases Drug-Induced Reinstatement of Morphine CPP in Adulthood via Microglial-Specific Epigenetic Programming of Anti-Inflammatory IL-10 Expression. Schwarz JM, Hutchinson MR, Bilbo SD. J Neurosci. 2011 Dec 7;31(49):17835-47.

Abstract: A critical component of drug addiction research involves identifying novel biological mechanisms and environmental predictors of risk or resilience to drug addiction and associated relapse. Increasing evidence suggests microglia and astrocytes can profoundly affect the physiological and addictive properties of drugs of abuse, including morphine. We report that glia within the rat nucleus accumbens (NAcc) respond to morphine with an increase in cytokine/chemokine expression, which predicts future reinstatement of morphine conditioned place preference (CPP) following a priming dose of morphine. This glial response to morphine is influenced by early-life experience. A neonatal handling paradigm that increases the quantity and quality of maternal care significantly increases baseline expression of the anti-inflammatory cytokine IL-10 within the NAcc, attenuates morphine-induced glial activation, and prevents the subsequent reinstatement of morphine CPP in adulthood. IL-10 expression within the NAcc and reinstatement of CPP are negatively correlated, suggesting a protective role for this specific cytokine against morphine-induced glial reactivity and drug-induced reinstatement of morphine CPP. Neonatal handling programs the expression of IL-10 within the NAcc early in development, and this is maintained into adulthood via decreased methylation of the IL-10 gene specifically within microglia. The effect of neonatal handling is mimicked by pharmacological modulation of glia in adulthood with ibudilast, which increases IL-10 expression, inhibits morphine-induced glial activation within the NAcc, and prevents reinstatement of morphine CPP. Taken together, we have identified a novel gene × early-life environment interaction on morphine-induced glial activation and a specific role for glial activation in drug-induced reinstatement of drug-seeking behavior.

Effect of secondhand smoke on occupancy of nicotinic acetylcholine receptors in brain. Brody AL, Mandelkern MA, London ED, Khan A, Kozman D, Costello MR, Vellios EE, Archie MM, Bascom R, Mukhin AG. Arch Gen Psychiatry. 2011 Sep;68(9):953-60. Epub 2011 May 2.

Abstract: Context: Despite progress in tobacco control, secondhand smoke (SHS) exposure remains prevalent worldwide and is implicated in the initiation and maintenance of cigarette smoking. OBJECTIVE: To determine whether moderate SHS exposure results in brain α(4)β(2)* nicotinic acetylcholine receptor (nAChR) occupancy. Design, Setting, and Participants: Positron emission tomography scanning and the radiotracer 2-[18F]fluoro-3-(2(S)azetidinylmethoxy) pyridine (also known as 2-[(18)F]fluoro-A-85380, or 2-FA) were used to determine α(4)β(2)* nAChR occupancy from SHS exposure in 24 young adult participants (11 moderately dependent cigarette smokers and 13 nonsmokers). Participants underwent two bolus-plus-continuous-infusion 2-FA positron emission tomography scanning sessions during which they sat in the passenger's seat of a car for 1 hour and either were exposed to moderate SHS or had no SHS exposure. The study took place at an academic positron emission tomography center. Main Outcome Measure Changes induced by SHS in 2-FA specific binding volume of distribution as a measure of α(4)β(2)* nAChR occupancy. Results: An overall multivariate analysis of variance using specific binding volume of distribution values revealed a significant main effect of condition (SHS vs control) ($F(1,22) = 42.5$, $P < .001$) but no between-group (smoker vs nonsmoker) effect. Exposure to SHS led to a mean 19% occupancy of brain α(4)β(2)* nAChRs (1-sample t test, 2-tailed, $P < .001$). Smokers had both a mean 23% increase in craving with SHS exposure and a correlation between thalamic α(4)β(2)* nAChR occupancy and craving alleviation with subsequent cigarette smoking (Spearman $\rho = -0.74$, $P = .01$). Conclusions: Nicotine from SHS exposure results in substantial brain α(4)β(2)* nAChR occupancy in smokers and nonsmokers. Study findings suggest that such exposure delivers a priming dose of nicotine to the brain that contributes to continued cigarette use in smokers. This study has implications for both biological research into the link between SHS exposure and cigarette use and public policy regarding the need to limit SHS exposure in cars and other enclosed spaces.

Short-term meditation induces white matter changes in the anterior cingulate. Tang YY, Lu Q, Geng X, Stein EA, Yang Y, Posner MI. Proc Natl Acad Sci U S A. 2010 Aug 31;107(35):15649-52. Epub 2010 Aug 16.

Abstract: The anterior cingulate cortex (ACC) is part of a network implicated in the development of self-regulation and whose connectivity changes dramatically in development. In previous studies we showed that 3 h of mental training, based on traditional Chinese medicine (integrative body-mind training, IBMT), increases ACC activity and improves self-regulation. However, it is not known whether changes in white matter connectivity can result from small amounts of mental training. We here report that 11 h of IBMT increases fractional anisotropy (FA), an index indicating the integrity and efficiency of white matter in the corona radiata, an important white-matter tract connecting the ACC to other structures. Thus IBMT could provide a means for improving self-regulation and perhaps reducing or preventing various mental disorders.

Adjunctive Counseling during Brief and Extended Buprenorphine-Naloxone Treatment for Prescription Opioid Dependence: A 2-phase Randomized Controlled Trial. Weiss RD, Potter JS, Fiellin DA, Byrne M, Connery HS, Dickinson W, Gardin J, Griffin ML, Gourevitch MN, Haller DL, Hasson AL, Huang Z, Jacobs P, Kosinski AS, Lindblad R, McCance-Katz EF, Provost SE, Selzer J, Somoza EC, Sonne SC, Ling W. Arch Gen Psychiatry. 2011 Dec;68(12):1238-46. Epub 2011 Nov 7.

Abstract: Context: No randomized trials have examined treatments for prescription opioid dependence, despite its increasing prevalence. Objective: To evaluate the efficacy of brief and extended buprenorphine hydrochloride-naloxone hydrochloride treatment, with different counseling intensities, for patients dependent on prescription opioids. Design: Multisite, randomized clinical trial using a 2-phase adaptive treatment research design. Brief treatment (phase 1) included 2-week buprenorphine-naloxone stabilization, 2-week taper, and 8-week postmedication follow-up. Patients with successful opioid use outcomes exited the study; unsuccessful patients entered phase 2: extended (12-week) buprenorphine-naloxone treatment, 4-week taper, and 8-week postmedication follow-up. Setting: Ten US sites. Patients: A total of 653 treatment-seeking outpatients dependent on prescription opioids. Interventions: In both phases, patients were randomized to standard medical management (SMM) or SMM plus opioid dependence counseling; all received buprenorphine-naloxone. Main Outcome Measures: Predefined "successful outcome" in each phase: composite measures indicating minimal or no opioid use based on urine test-confirmed self-reports. Results: During phase 1, only 6.6% (43 of 653) of patients had successful outcomes, with no difference between SMM and SMM plus opioid dependence counseling. In contrast, 49.2% (177 of 360) attained successful outcomes in phase 2 during extended buprenorphine-naloxone treatment (week 12), with no difference between counseling conditions. Success rates 8 weeks after completing the buprenorphine-naloxone taper (phase 2, week 24) dropped to 8.6% (31 of 360), again with no counseling difference. In secondary analyses, successful phase 2 outcomes were more

common while taking buprenorphine-naloxone than 8 weeks after taper (49.2% [177 of 360] vs 8.6% [31 of 360], P < .001). Chronic pain did not affect opioid use outcomes; a history of ever using heroin was associated with lower phase 2 success rates while taking buprenorphine-naloxone. Conclusions: Prescription opioid-dependent patients are most likely to reduce opioid use during buprenorphine-naloxone treatment; if tapered off buprenorphine-naloxone, even after 12 weeks of treatment, the likelihood of an unsuccessful outcome is high, even in patients receiving counseling in addition to SMM. Trial Registration clinicaltrials.gov Identifier: NCT00316277. Press Release: Painkiller Abuse Treated by Sustained Buprenorphine/Naloxone

Repressive LTR nucleosome positioning by the BAF complex is required for HIV latency. Rafati H, Parra M, Hakre S, Moshkin Y, Verdin E, Mahmoudi T. PLoS Biol. 2011 Nov;9(11):e1001206. Epub 2011 Nov 29.
Abstract: Persistence of a reservoir of latently infected memory T cells provides a barrier to HIV eradication in treated patients. Several reports have implicated the involvement of SWI/SNF chromatin remodeling complexes in restricting early steps in HIV infection, in coupling the processes of integration and remodeling, and in promoter/LTR transcription activation and repression. However, the mechanism behind the seemingly contradictory involvement of SWI/SNF in the HIV life cycle remains unclear. Here we addressed the role of SWI/SNF in regulation of the latent HIV LTR before and after transcriptional activation. We determined the predicted nucleosome affinity of the LTR sequence and found a striking reverse correlation when compared to the strictly positioned in vivo LTR nucleosomal structure; sequences encompassing the DNase hypersensitive regions displayed the highest nucleosome affinity, while the strictly positioned nucleosomes displayed lower affinity for nucleosome formation. To examine the mechanism behind this reverse correlation, we used a combinatorial approach to determine DNA accessibility, histone occupancy, and the unique recruitment and requirement of BAF and PBAF, two functionally distinct subclasses of SWI/SNF at the LTR of HIV-infected cells before and after activation. We find that establishment and maintenance of HIV latency requires BAF, which removes a preferred nucleosome from DHS1 to position the repressive nucleosome-1 over energetically sub-optimal sequences. Depletion of BAF resulted in de-repression of HIV latency concomitant with a dramatic alteration in the LTR nucleosome profile as determined by high resolution MNase nucleosomal mapping. Upon activation, BAF was lost from the HIV promoter, while PBAF was selectively recruited by acetylated Tat to facilitate LTR transcription. Thus BAF and PBAF, recruited during different stages of the HIV life cycle, display opposing function on the HIV promoter. Our data point to the ATP-dependent BRG1 component of BAF as a putative therapeutic target to deplete the latent reservoir in patients.

CONGRESSIONAL AFFAIRS
Prepared February 3, 2011

Appropriations/Budget

On December 23, 2011, the President signed into law H.R. 2055, the Consolidated Appropriations Act, 2012 (P.L. 112-74). This bill, a 9-bill Omnibus which includes funding for the Departments of Labor, HHS, and Education, provides funding for NIH in the amount of $30.689 billion, which is $299 million above last year's level and $758 million below the President's request. For NIDA, the enacted amount is $1.053 billion, which is $3.4 million above last year's level and $27 million below the President's request. (NOTE: The President released his FY 2013 budget too late for inclusion in this report.)

Also note: P.L. 112-74 officially created NIH's newest Center, the National Center for Advancing Translational Sciences (NCATS). See **http://ncats.nih.gov** for more details.

Legislation of Interest

SBIR/STTR - On December 31st, the President signed into law H.R. 1540, the National Defense Authorization Act for FY 2012. This bill included provisions to reauthorize the SBIR and STTR programs for six years. Setaside levels increase – the SBIR setaside increases to 3.2% from 2.5%, and the STTR setaside goes to .45% from .3%. The increases occur over time, reaching these levels in FY 2017. Further, the law allows 1) NIH, the Department of Energy, and the National Science Foundation (NSF) to award up to 25% of the SBIR setaside to venture capital

companies, hedge funds, or private equity firms; 2) agencies to apply for waivers to exceed the hard cap on awards (Hard Cap for Phase I-$225,000 and Phase II $1,500,000); and 3) NIH and NSF to make final decisions on proposals not later than 1 year after the solicitation closes.

Congressional Briefings of Interest

Marijuana -- On October 20, 2011, NIDA staff participated in a briefing focused on marijuana abuse and addiction, with a focus on issues around "medical marijuana." The briefing was requested by staff of the U.S. Senate Caucus on International Narcotics Control. Dr. Susan Weiss led the NIDA team, and focused on NIDA (and NIH) research on the adverse health effects of marijuana use and the variety of cannabinoid-focused research efforts underway. Other HHS participants in the briefing were the FDA, SAMHSA, and the Office of the Assistant Secretary for Health.

Methamphetamine -- At the request of Representative Denny Rehberg (R-MT), Chair, House Appropriations Subcommittee on Labor, HHS, and Education, NIDA Director Dr. Nora Volkow briefed House staffers on NIDA's research on methamphetamine addiction and treatment (November 16, 2011). Representatives from the Meth Project, a national methamphetamine prevention effort that started in Montana, also participated in the briefing. Dr. Volkow also briefed Senate staffers at a second, similar briefing at the request of Senator Michael Bennett (D-CO).

NIH/HHS POLICY UPDATES

For a comprehensive list of policy notices, see **http://grants.nih.gov/grants/policy/policy.htm**

2012

February 8	Request for Information (RFI): Input into the Scientific Strategic Plan for the proposed National Institute of Substance Use and Addiction Disorders
January 27	New NIH Policy on Efficient Spending Related to Grants Supporting Conferences and Meetings
January 20	Ruth L. Kirschstein National Research Service Award (NRSA) Stipends, Tuition/Fees and Other Budgetary Levels Effective for Fiscal Year 2012
January 20	Notice of Legislative Mandates in Effect for FY2012

The *Consolidated Appropriations Act, 2012* (Public Law 112-74) signed into law on December 23, 2011 provides funding to NIH for the fiscal year ending September 30, 2012. The intent of this Notice is to provide information on the following statutory provisions that limit the use of funds on NIH grant, cooperative agreement, and contract awards for FY2012. The Notice of Legislative Mandates for FY2011 was published on May 4, 2011, NIH Guide Notice NOT-OD-11-072.

Among the amended legislative mandates is the restriction on Distribution of Sterile Needles (Section 523)

Removed references to the purposes of needle distribution and to public health and law enforcement determinations. Now prohibits all programs to distribute sterile needles or syringes for the hypodermic injection of any illegal drug.

Among the additional mandates in effect is the limitation on Use of Funds for Promotion of Legalization of Controlled Substances (Section 509)

"(a) None of the funds made available in this Act may be used for any activity that promotes the legalization of any drug or other substance included in schedule I of the schedules of controlled substances established under section 202 of the Controlled Substances Act except for normal and recognized executive-congressional communications. (b)The limitation in subsection (a) shall not apply when there is significant

medical evidence of a therapeutic advantage to the use of such drug or other substance or that federally sponsored clinical trials are being conducted to determine therapeutic advantage."

January 20 NIH Fiscal Policy for Grant Awards – FY 2012

2011

December 21 Notice of Two Pilot Processes for Submitting Administrative Supplement Requests Electronically to NIH

December 21 NIH Research Involving Chimpanzees

December 14 Ruth L. Kirschstein National Research Service Awards (NRSA) and Other Fellowship Applications: New Policy on Post-Submission Information on Sponsor's Research Funding

December 13 Recovery Act: Notice of Revised Term of Award for All Recovery Act Awards to Ensure Project Completion by September 30, 2013

December 2 Adoption and Implementation of the Guide for the Care and Use of Laboratory Animals: Eighth Edition

November 10 Notice of Expanded Transparency Act Subaward and Executive Compensation Reporting Requirements for FY2012 and Beyond

October 20 Publication of the Revised NIH Grants Policy Statement (Rev. 10/1/2011): Policy Changes, Clarifications and Document Enhancements

October 5 Update of NIH Late Application Policy to Reflect Change in Due Dates for New Investigator R01 Resubmission Applications

September 28 Revised Multiple Program Director(s)/Principal Investigator(s) Policy to Allow Change with Prior Approval

Requests for Information

January 13 Request for Information (RFI): Input into the Deliberations of the Advisory Committee to the NIH Director Working Group on Diversity in the Biomedical Research Workforce

January 10 Request for Information (RFI): Input into the Deliberations of the Advisory Committee to the NIH Director Working Group on Data and Informatics

PROGRAM ACTIVITIES/FOAS

New NIDA RFAs

On February 6, 2012, NIDA issued an RFA entitled **Phased Services Research Studies of Drug Use Prevention, Addiction Treatment, and HIV in an Era of Health Care Reform (R21/R33) RFA-DA-13-001**. This funding opportunity announcement (FOA) solicits applications for Phased Innovation (R21/R33) research projects to conduct rigorous, objective services research to monitor and examine changes in drug use prevention, addiction treatment, and associated HIV and viral hepatitis services that may occur as a result of healthcare reform. Open date: July 22, 2012. Application due date: August 22, 2012, by 5:00 PM local time of applicant organization.

New NIDA Program Announcements

On August 16, 2011, NIDA issued a PA entitled **Drug Abuse Prevention Intervention Research PA-11-311 (R01); PA-11-312 (R21); PA-11-313 (R03).** The purpose of this FOA is to encourage applications from institutions/organizations that propose to advance the science of drug abuse and drug-related HIV prevention through 1) the development of novel prevention approaches, 2) the testing of novel and adapted prevention intervention approaches 3) the elucidation of processes associated with the selection, adoption, adaptation, implementation, sustainability, and financing of empirically validated interventions, and 4) the development of new methodologies suitable for the design and analysis of prevention research studies.

November 29, 2011, NIDA issued a PA entitled **International Research Collaboration on Drug Abuse and Addiction Research PA-12-040 (R01); PA-12-041 (R21); PA-12-042 (R03).** This Funding Opportunity Announcement (PA) encourages collaborative research applications on drug abuse and addiction that take advantage of special opportunities that exist outside the United States. Special opportunities include access to unusual talent, resources, populations, or environmental conditions in other countries that will speed scientific discovery. Projects should have relevance to the mission of NIDA and where feasible should address NIDAs scientific priority areas.

On December 21, 2011, NIDA issued a Program Announcement (PA) entitled **Imaging - Science Track Award for Research Transition (I/START) [R03] PAR-12-066.** This funding opportunity announcement (FOA) encourages Small Research Grant (R03) applications to facilitate the entry of investigators to the area of neuroimaging, including both new investigators and established investigators seeking to adopt neuroimaging methodologies in their research programs. Open Date: January 16, 2012.

On January 27, 2012, NIDA issued a Program Announcement (PA) entitled **Cutting-Edge Basic Research Awards (CEBRA) (R21) PAR-12-086.** The Cutting-Edge Basic Research Award (CEBRA) is designed to foster highly innovative or conceptually creative research related to drug abuse and addiction and how to prevent and treat them. It supports research that is high-risk and potentially high-impact that is underrepresented or not included in NIDA's current portfolio. Open Date: July 20, 2012. Application due dates: August 20, 2012; December 20, 2012; August 20, 2013, December 20, 2013, August 20, 2014, and December 19, 2014 , by 5:00 PM local time of applicant organization.

New FOAs Issued by the NIH Common Fund

On October 7, 2011, the NIH Common Fund issued an RFA entitled **Economic Studies Ancillary to Completed or Ongoing Health Care Delivery and Financing Pilots, Demonstrations, and Other Experiments (R01) RFA-RM-11-023.** This funding opportunity announcement (FOA) solicits applications for Research Project (R01) grant awards to support health economics research ancillary to completed or ongoing large-scale health care delivery and financing pilots, demonstrations, and other experiments (PDEs) that are intended to reduce health care costs or cost growth while maintaining or improving patient outcomes. Open date: January 8, 2012. Application due date: February 8, 2012, by 5:00 PM local time of applicant organization. EXPIRED: February 9, 2012.

On October 7, 2011, the NIH Common Fund issued an RFA entitled **Phased Economic Studies Ancillary to Planned Health Care Delivery and Financing Pilots, Demonstrations, and Other Experiments (R21/R33) RFA-RM-11-024.** This funding opportunity announcement (FOA) solicits applications for Phased Innovation (R21/R33) grant awards to support health economics research conducted alongside planned large-scale health care delivery and financing pilots, demonstrations, and other experiments (PDEs) that are intended to reduce health care costs or cost growth while maintaining or improving patient outcomes. Open date: January 8, 2012. Application due date: February 8, 2012, by 5:00 PM local time of applicant organization. EXPIRED: February 9, 2012.

On December 2, 2011, the NIH Common Fund issued an RFA entitled **Regional Comprehensive Metabolomics Resource Cores (RCMRC) (U24) RFA-RM-11-016.** This FOA invites cooperative agreement applications to establish Regional Comprehensive Metabolomics Resource Cores (RCMRC) to increase the national capacity for utilizing metabolomics in biomedical research. To facilitate the biological and clinical application of metabolomics, technology service cores, pilot/feasibility projects, training activities, and outreach activities will be supported. Application due dates: February 15, 2012; February 15, 2013.

On December 2, 2011, the NIH Common Fund issued an RFA entitled **Metabolomics Data Repository and Coordinating Center (DRCC) (U01) RFA-RM-11-020.** The purpose of this FOA is to provide a coordinating center and data repository for the accompanying metabolomics FOAs and metabolomics research community. One goal of the Data Repository Coordinating Center (DRCC) is to enhance metabolomics research by broadly disseminating data to researchers across the metabolomics community. Therefore the repository will store published primary data for examination and analysis with high performance computation methods in a cloud computing environment. It will help develop the necessary tools to allow access to data and available analytical tools. Open date: January 15, 2012. Application due date: February 15, 2012, by 5:00 PM local time of applicant organization.

On December 21, 2011, the NIH Common Fund issued an RFA entitled **Human Heredity and Health in Africa (H3Africa): H3Africa Biorepository Grants (UH2/UH3) RFA-RM-12-003.** The purpose of this FOA is to call for applications for UH2/UH3 cooperative agreements that will provide funding to develop plans, building upon existing infrastructure, for initial two-year feasibility studies (Phase I) and then for an additional five years of support for full-scale H3Africa Biorepositories (Phase II), beginning in 2014. Open date: December 21, 2012. Application due date: February 21, 2012, by 5:00 PM local time of applicant organization.

On December 21, 2011, the NIH Common Fund issued an RFA entitled **Human Health and Heredity in Africa (H3Africa): Research Grants (U01) RFA-RM-12-004**. The purpose of this FOA is to call for applications for research projects. Awards will focus on supporting research on the genetic/environmental contributors to health and disease in Africa that fall within the mission of the NIH, which is to seek fundamental knowledge about the nature and behavior of living systems and to apply that knowledge to enhance health, lengthen life, and reduce the burdens of illness and disability. Open Date: December 21, 2012. Application due date: January 6, 2012, by 5:00 PM local time of applicant organization.

On January 24, 2012, the NIH Common Fund issued an RFA entitled **NIH Health Care Systems Research Collaboratory - Coordinating Center (U54) RFA-RM-11-021**. The purpose of this FOA is to solicit applications for a Coordinating Center to provide national leadership for the NIH Health Care Systems (HCS) Research Collaboratory program. The Coordinating Center will 1) develop, adapt, and adopt technical and policy guidelines and best practices for the effective conduct of research studies in partnership with health care systems; 2) work collaboratively with each Demonstration Project team, including their partnering health care systems, to develop and test an implementation plan for the proposed Demonstration Projects while providing technical, design, and coordination support; and 3) disseminate widely Collaboratory endorsed policies and practices and lessons learned in the Demonstration Projects to inform best practices for broad participation of health care systems and their patients,

practitioners, and staff in research studies to improve health and care delivery. Open date: March 27, 2012. Application due date: April 27, 2012.

On January 24, 2012, the NIH Common Fund issued an RFA entitled **NIH Health Care Systems Research Collaboratory - Pragmatic Clinical Trials Demonstration Projects (UH2/UH3) RFA-RM-12-002**. The purpose of this FOA is to solicit applications for UH2/UH3 cooperative agreements for Demonstration Projects for efficient, large-scale pragmatic clinical trials to be conducted within the NIH Health Care Systems (HCS) Research Collaboratory supported through the NIH Common Fund. (See http://commonfund.nih.gov/hcscollaboratory/). Open date: April 2, 2012. Application due date: May 2, 2012 by 5:00pm local time of the applicant organization.

On February 1, 2012, the NIH Common Fund issued a PAR entitled **Resource Access for the Bridging Interventional Development Gaps Program (X01) PAR-12-092.** The purpose of this Funding Opportunity Announcement (FOA) is to invite investigators to apply for access to government-funded contract resources needed for the preclinical development of therapeutic agents. Open date: March 3, 2012. Application due date: April 3, 2012, by 5:00 PM local time of applicant organization.

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

On November 29, 2011, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Limited Competition - Women's Interagency HIV Study (WIHS-V) (U01) RFA-AI-12-002**. The purpose of the Women's Interagency HIV Study V (WIHS-V) is to characterize the long-term, natural and treated history of HIV infection in the current cohort of women, and recruit and retain new women into the cohort to provide insight into the changing demographics of the HIV epidemic among women in the United States (U.S.). Open Date: December 22, 2011. Application due date: February 22, 2012, by 5:00 PM local time of applicant organization.

On December 12, 2011, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Limited Competition: The Medical Education Partnership Initiative Linked Awards (MEPI) (R25) RFA-TW-11-004**. This NIH Funding Opportunity Announcement (FOA), supported by funds from the participating NIH Institutes and Centers (ICs), invites applications from foreign Institutions in Sub-Saharan African countries who are a part of the Medical Education Partnership Initiative Network of institutions (http://www.fic.nih.gov/Grants/Search/Pages/Awards-Program-MEPI.aspx) to develop research capacity and research education opportunities in priority health areas related to and/or beyond HIV/AIDS. Open Date: January 10, 2012. Application due date: February 10, 2012, by 5:00 PM local time of applicant organization. EXPIRED: February 11, 2012.

On December 21, 2011, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Tools to Enhance Studies of Glial Cell Development, Aging, Disease and Repair (R21) RFA-HD-12-211**. This Funding Opportunity Announcement (FOA) is issued as an initiative of the NIH Blueprint for Neuroscience Research. The Neuroscience Blueprint is a collaborative framework through which 16 NIH Institutes, Centers and Offices jointly support neuroscience-related research, with the aim of accelerating discoveries and reducing the burden of nervous system disorders (for further information, see http://neuroscienceblueprint.nih.gov/). The goal of this FOA is to encourage research grant applications that propose to develop or substantially modify existing cutting edge technologies that will advance glial cell research, discovery-based research on glial cell diversity, development and/or function in the central (CNS) and peripheral (PNS) nervous systems. Open Date: February 29, 2012. Application due date: March 29, 2012, by 5:00 PM local time of applicant organization.

On January 27, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Leadership Group for a Clinical Research Network on Therapeutics for HIV/AIDS and HIV-associated Infections in Adults (UM1) RFA-AI-12-004**. The purpose of this FOA is to encourage applications for the Leadership Group for a Clinical Research Network on Therapeutics for HIV/AIDS and HIV-associated Infections in Adults. The Leadership Group (LG) will have overall responsibility for developing, implementing and adapting the network's clinical research agenda to address NIAID's HIV/AIDS scientific priorities. Application due date: September 28, 2012.

On January 27, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Leadership Group for a Clinical Research Network on Integrated Strategies to Prevent HIV Infection (UM1) RFA-AI-12-011**. The purpose of this FOA is to solicit applications for the Leadership Group for a Clinical Research Network on Integrated Strategies to Prevent HIV Infection. The Leadership Group (LG) will have overall responsibility

for developing, implementing and adapting the network's clinical research agenda to address NIAID's HIV/AIDS scientific priorities. Application due date: September 28, 2012.

On January 27, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Development of Tools to Study the Synaptome (R21) RFA-MH-12-140**. This Funding Opportunity Announcement (FOA) issued by the National Institutes of Health, encourages applications that will develop novel technologies and/or tools to facilitate the study of genes and proteins at the synapse on a large scale. Open date: March 16, 2012. Application due date: April 16, 2012, by 5:00 PM local time of applicant organization.

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

On September 1, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Clinical Research Education and Career Development (CRECD) in Minority Institutions (R25) PAR-11-325.** The purpose of the Clinical Research Education and Career Development (CRECD) Program is to expand the national capability to improve diversity for research in the health sciences by developing the research workforce in clinical and translational sciences though providing grant support to minority institutions that offer doctorate degrees in the health professions or in a health-related science. Open Date: October 2, 2011. Application due dates: November 2, 2011, November 2, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: Not Applicable.

On September 22, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Interventions for Health Promotion and Disease Prevention in Native American Populations (R01) PAR-11-346**. The purpose of this funding opportunity announcement (FOA) is to develop, adapt, and test the effectiveness of health promotion and disease prevention interventions in Native American (NA) populations. NA populations are exposed to considerable risk factors that significantly increase their likelihood of chronic disease, substance abuse, mental illness, and HIV-infection. Open Date: April 15, 2012. Application due dates: May 15, 2012; May 15, 2013; May 15, 2014. AIDS application due date: Not Applicable.

On November 2, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Academic Research Enhancement Award (Parent R15) PA-12-006.** The purpose of the Academic Research Enhancement Award (AREA) program is to stimulate research in educational institutions that provide baccalaureate or advanced degrees for a significant number of the Nation's research scientists, but that have not been major recipients of NIH support. Open Date: January 25, 2012.

On November 9, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **NIH Competitive Revision Applications for Research Relevant to the Family Smoking Prevention and Tobacco Control Act (U01) PAR-12-011.** This Funding Opportunity Announcement (FOA) invites cooperative agreement research (U01) Revision applications from investigators with active U01 research project awards issued by one of the participating Institute/Centers (ICs) listed in this FOA to support an expansion of the scope of approved and funded U01 projects involving smoking and tobacco-related products and/or their constituents. Open Date: January 17, 2011. Application due date: February 17, 2012, by 5:00 PM local time of applicant organization.

On November 9, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **NIH Competitive Revision Applications for Research Relevant to the Family Smoking Prevention and Tobacco Control Act (R01) PAR-12-010.** This Funding Opportunity Announcement (FOA) invites Revision research project grant (R01) applications from investigators with active R01 research project awards issued by one of the participating Institute/Centers (ICs) listed in this FOA to support an expansion of the scope of approved and funded R01 projects involving smoking and tobacco-related products and/or their constituents. Open Date: January 17, 2011. Application due date: February 17, 2012, by 5:00 PM local time of applicant organization.

On November 18, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Behavioral Interventions to Address Multiple Chronic Health Conditions in Primary Care (R01) PA-12-024**. This funding opportunity announcement (FOA) seeks Research Project Grant (R01) applications that propose to use a common conceptual model to develop behavioral interventions to modify health behaviors and improve health outcomes in patients with comorbid chronic diseases and health conditions. Specifically, this FOA will support research in primary care that uses a multi-disease care management approach to behavioral interventions

with high potential impact to improve patient-level health outcomes for individuals with three or more chronic health conditions. Open Date: January 5, 2011.

On December 13, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Solicitation of Validated Hits for the Discovery of in vivo Chemical Probes (R01) PAR-12-060**. This Funding Opportunity Announcement (FOA) intends to support investigators who have interest and capability to join efforts for the discovery of in vivo chemical probes. It is expected that applicants will have in hand the starting compounds ("validated hits") for chemical optimization and bioassays for testing new analog compounds. Open Date: January 5, 2011.

On December 13, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Solicitation of Assays for High Throughput Screening (HTS) to Discover Chemical Probes (R21) PAR-12-059 (R01) PAR-12-058.** This Funding Opportunity Announcement (FOA) encourages investigators to form collaborations with an established academic, nonprofit, or commercial high throughput screening (HTS) facility that has the requisite expertise and experience to implement HTS-ready assays for the discovery and development of small molecule chemical probes. Open Date: January 5, 2011.

On December 22, 2011, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Fogarty HIV Research Training Program for Low- and Middle-Income Country Institutions (D43) PAR-12-068.** The purpose of this FOA is to encourage applications for research training programs to strengthen the HIV research capacity at low- and middle-income country (LMIC) institutions. Each application should propose a training program that will build or strengthen research capacity in an applicant-defined HIV-related scientific topic at an identified LMIC institution. Open Date: June 24, 2012. Application due dates: July 24, 2012, July 24, 2013, July 24, 2014, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On January 24, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Jointly Sponsored Ruth L. Kirschstein National Research Service Award Institutional Predoctoral Training Program in the Neurosciences (T32) PAR-12-084**. The Jointly Sponsored NIH Predoctoral Training Program in the Neurosciences supports broad and fundamental research training in the neurosciences via institutional NRSA research training grants (T32) at domestic institutions of higher education. Trainees appointed to this training grant are financially supported for either one or two years, during the first 2 years of their graduate research training. The primary objective is to prepare individuals for careers in neuroscience that have a significant impact on the health-related research needs of the Nation. Open Date: April 25, 2012. Application due dates: May 25, 2012, May 25, 2013, and May 25, 2014, by 5:00 PM local time of applicant organization. AIDS application due date: Not Applicable.

On January 31, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **PHS 2012-02 Omnibus Solicitation of the NIH, CDC, FDA and ACF for Small Business Innovation Research Grant Applications (Parent SBIR [R43/R44]) PA-12-088**. This Funding Opportunity Announcement (FOA) issued by the National Institutes of Health (NIH), the Centers for Disease Control and Prevention (CDC), the Food and Drug Administration (FDA) and the Administration for Children and Families (ACF) invites eligible United States small business concerns (SBCs) to submit Small Business Innovation Research (SBIR) grant applications. United States SBCs that have the research capabilities and technological expertise to contribute to the R&D mission(s) of the NIH, CDC, FDA or ACF awarding components identified in this FOA are encouraged to submit SBIR grant applications in response to identified topics (see PHS 2012-2 SBIR/STTR Program Descriptions and Research Topics for NIH, CDC, FDA and ACF). Open Date: March 5, 2012.

On January 31, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **PHS 2012-02 Omnibus Solicitation of the NIH for Small Business Technology Transfer Grant Applications (Parent STTR [R41/R42]) PA-12-089**. This Funding Opportunity Announcement (FOA) issued by the National Institutes of Health (NIH) invites eligible United States small business concerns (SBCs) to submit Small Business Technology Transfer (STTR) grant applications. United States SBCs that have the research capabilities and technological expertise to contribute to the R&D mission(s) of the NIH awarding components identified in this FOA are encouraged to submit STTR grant applications in response to identified topics (see PHS 2012-2 SBIR/STTR Program Descriptions and Research Topics for NIH). Open Date: March 5, 2012.

NIDA PUBLICATIONS

CTN Bulletin Board
Eight editions of the CTN Bulletin Board were distributed. The Bulletin Board is an electronic report on the progress of the protocols, committees, and node activity in the CTN.

CTN Data Sharing Web Site
Data from 24 CTN studies are now available on the CTN Data Sharing Web Site. 1,200 data sets have been downloaded by researchers from 19 countries

Principles of Drug Abuse Treatment for Criminal Justice Populations - A Research-Based Guide
NIH Pub Number: 06-5316
Published Sep 2006. Revised Jan 2012.
Presents research-based principles of addiction treatment that can inform drug treatment programs and services in the criminal justice setting.

Spice (InfoFacts)
Published Jan 2011. Revised Dec 2011.
Provides information about spice, a family of herbal mixtures that produce effects similar to that of marijuana, including how it is used, potential health effects, and public health concerns. En Español

Marijuana (Topics in Brief)
Revised Dec 2011.
Provides a brief update on the research on marijuana use, including its health effects, the potential for addiction, treatment, and marijuana in medicine.

Prescription Drug Abuse (Topics in Brief)
Revised Dec 2011.
Provides a brief overview of the non-medical use of prescription drugs, examining prevalence, health risks associated with use, treatment options, and research underway on the topic.

Tobacco Addiction (Topics in Brief)
Revised Dec 2011.
Examines the scope of tobacco use in the U.S., what makes it addictive, and potential treatment options for tobacco addiction.

Media Guide
Published Dec 2009. Revised Dec 2011.
Provides journalists with the latest findings on the science of drug abuse and addiction and commonly abused drugs, and lists resources for more information.

Seeking Drug Abuse Treatment: Know What To Ask
NIH Pub Number: 12-7764
Published Dec 2011.
Offers guidance in seeking drug abuse treatment and lists five questions to ask when searching for a treatment program.

Inhalants (InfoFacts)
Revised Dec 2011.
Provides an overview of inhalants, such as types of products commonly inhaled, how they affect the brain, other adverse effects on health, and the scope of use of inhalants in the U.S. En Español

Methamphetamine Addiction: Progress, but Need to Remain Vigilant (Topics in Brief)
Published Mar 2007. Revised Nov 2011.
Declines in Methamphetamine Abuse by Youth

Volume 24, Number 1 (NIDA Notes)
NIH Pub Number: 12-7831
Published Nov 2011.
Reports on innovative research, including active brain circuits that shed light on addiction; a new therapeutic skin patch; the disruption of neuron growth on cocaine relapse; genetic changes and vulnerability to relapse; regulators for addiction genes, and more.

Commonly Abused Prescription Drugs Chart
Published Sep 2002. Revised Oct 2011.
Offers a list prescription drugs commonly abused, including depressants, opioids and morphine derivatives, and stimulants, and provides their common and street names, how they are generally administered, and their potential health effects.

Prescription Drugs: Abuse and Addiction (Research Reports)
NIH Pub Number: 11-4881
Published Jul 2001. Revised Oct 2011.
Examines the non-medical use of prescription drugs-opioids, central nervous system depressants, and stimulants-describing adverse health effects of their use and the prevention and treatment of addiction. En Español

Marijuana: Download the Facts Poster
Published Sep 2011.
Emphasizes some of the harmful effects of marijuana use and encourages youth to get the facts at NIDA's web site for teens, teens.drugabuse.gov.

Lessons from Prevention Research (InfoFacts)
Published Feb 2004. Revised Aug 2011.
Describes principles important to consider when developing drug abuse prevention programs and discusses issues relevant for family, school, and community settings. En Español

Heads Up: Real News About Drugs and Your Body
Published Aug 2003. Revised Aug 2011.
An educational collaboration between the National Institute on Drug Abuse (NIDA) and Scholastic Inc., this series provides science-based information about the effects of drugs and drug abuse on the brain and body through a variety of print and Web formats, including teen articles, classroom lessons, student worksheets, and posters. All of these materials are free.

National Drug IQ Challenge
Revised Aug 2011.
Encourages people to test their knowledge about drug abuse and addiction by asking them to take a 15-question quiz. Accompanying answers shed light on the science of drug abuse. En Español

Screening for Drug Use in General Medical Settings
NIH Pub. No.: 11-7384
This is an at-a-glance booklet based on the NIDA-Modified Alcohol, Smoking, and Substance Involvement Screening Test.

NIDA International Program E-News
October 2011, December 2011

PRESS RELEASES

September 20, 2011 - NIDA Avant-Garde-Medications Development Award winners announced

September 27, 2011 - NIH to fund development of K-12 neuroscience education programs

October 6, 2011 - FDA and NIH announce joint study on tobacco use and risk perceptions

November 2, 2011 - NIH study examines nicotine as a gateway drug

November 7, 2011 - Teen musicians in drug treatment win 2012 GRAMMY® experience

November 8, 2011 - Painkiller Abuse Treated by Sustained Buprenorphine/Naloxone

December 9, 2011 - National Institute on Drug Abuse to Announce Results of 2011 Monitoring the Future Survey

December 14, 2011 - Cigarette and alcohol use at historic low among teens

January 17, 2012 - New NIDA resource helps families navigate addiction treatment options

MEETINGS/CONFERENCES

Select Meetings and Conferences in which NIDA played a significant role

From August 30 through November 8, 2011, Dr. Jonathan D. Pollock organized and chaired a weekly webinar series on molecular neuroanatomy with leaders in the field. These webinars were set up to facilitate discussion at the November 11-12, 2011 meeting "Molecular Neuroanatomy: the Next Decade of Progress" held at the Embassy Suites Hotel, Washington, DC.

September 21-23, 2011, the 8th Annual International Network on Brief Interventions for Alcohol Problems (INEBRIA) Conference was held in Boston, MA. NIDA CTN members and CCTN staff chaired and participated in several workshops and symposiums.

October 17 -18, 2011 in Toronto, Canada: Canada – Finland – USA Joint Workshop on "The Early Origins of Addiction" co-chaired by Dr. Cheryl Anne Boyce, DCNBR

October 19, 2011 at the 58th Annual Meeting American Academy of Child and Adolescent Psychiatry in Toronto, Canada, Drs. Cheryl Anne Boyce and Dr. Kevin Conway, along with other NIH staff, chaired sessions and workshops.

November 3-5, 2011, the 35th Annual National Conference of the Association for Medical Education and Research in Substance Abuse (AMERSA) was held in Washington, DC

November 7-8, 2011, the Special Populations Office (SPO) convened the "Diversity Alumni Meeting" in Bethesda, Maryland. Coordinated by Flair Lindsey, Program Analyst, SPO.

November 11, 2011, NIDA's Neuroscience Consortium organized the 10th annual Frontiers in Addiction Research Mini-convention at the Society for Neuroscience Meeting.

November 11-14, 2011, NIDA was sponsor and co-sponsor for lectures, training programs, symposia, and workshops organized by staff at the annual Society for Neuroscience meeting.

Nov. 16, 2011, Dr. Peter Hartsock, DESPR, co-chaired, with NIDA grantee Dr. Sheryl McCurdy (University of Texas School of Public Health), the inaugural meeting on health held by the African Studies Association (ASA), Washington, DC

December 4-8, 2011, NIDA staff chaired and co-chaired several events at the 50th Annual Meeting of the American College of Neuropsychopharmacology.

December 9-10, 2011, NIDA's African American Researchers and Scholars Workgroup sponsored a two-day "Grant Writing Booster Session" at Johns Hopkins University in Baltimore, Maryland.

February 6-9, 2012, Community Anti-Drug Coalitions of American National Leadership Forum XXII was held in National Harbor, MD.

Upcoming Conferences/Exhibits

"NIH Pain Research: Optimizing Funding through Grant Writing" and "Acupuncture for Chronic Low Back Pain: Clinical Evidence, the Science, and the Challenge" workshops at the 28th annual conference of the American Academy of Pain Medicine conference -- Palm Springs, California -- February 23-26, 2012.

2012 National Science Teachers Association National Conference on Science Education
Indianapolis, IN -- March 29-April 1, 2012

National CTN Steering Committee Meetings, in conjunction with the ASAM Pre-conference session (mini-blending sessions). -- Atlanta, Georgia -- April 17-18, 2012

Blending Conference on SBIRT at the ASAM Annual Meeting -- Atlanta, GA -- April 19-22, 2012

American Psychiatric Association Annual Meeting -- Philadelphia, PA -- May 5-9, 2012

COMMUNITY AND PRESS EVENTS

Addiction Performance Project (APP)
November 6th and 7th. Performances were held in Denver, CO for the APP.

National Drug Facts Week (NDFW)
October 31st through November 6th. New 2011 approaches included targeted outreach through Facebook, partnerships with *Radio One* network and *AOL,* an event on the *Today Show* plaza, and the creation of a Spanish version of the IQ Challenge. Former American Idol judge Kara DioGuardi announced the winner of the MusiCares® and GRAMMY Foundation® Teen Substance Abuse Awareness through Music contest on *Fox and Friends*.

Monitoring the Future (MTF) Press Conference
On December 14, 2011NIDA hosted its annual MTF survey results press conference at the National Press Club. NIDA Director Dr. Nora Volkow presented the results of the survey, and top tier columnists and reporters, trade press and bloggers were contacted to encourage press coverage. ONDCP Director Gil Kerlikowske, Assistant Secretary for Health at HHS Dr. Howard Koh, and Principal Investigator Dr. Lloyd Johnston also spoke.

STAFF HONORS AND AWARDS

Dr. Jag Khalsa, DPMCDA, received the Lifetime Achievement Award for his 46+ years of contributions to the field of drug research from the Board of Regents of Marathwada Institute of Technology in India (December 14, 2011).

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

Dr. Nadine Rogers, OEA, received a Meritorious Honor Award on December 12, 2011 from the U.S. Department of the State, Embassy—Phnom Penh

STAFF CHANGES

New Employees

Glenda Conroy joined NIDA in January to serve as our Executive Officer. Ms. Conroy comes to us from the HHS Program Support Center, where she served as Deputy Chief Financial Officer. Prior to that, she served as Director of Financial Enterprise Solutions and Deputy Director of Financial Management at the Food and Drug Administration. She has a wealth of experience with a background in executive leadership, technology, finance, organizational development, and team building. Glenda has an MBA from St. Francis University and several other academic certifications in project management, contracting, and accounting. She also served in the U.S. military as a Capitan in the Air Force Reserves.

Dr. Michelle Rankin joined the Office of Science Policy and Communications (OSPC) in December. Michele received her Ph.D. from Louisiana State University, where she focused on characterizing the signal transduction pathways of olfactory receptor neurons. She performed her post-doctoral research in the intramural program at NINDS in the Molecular Neuropharmacology Section, focusing on the molecular mechanisms that govern dopamine D_1 receptor signaling. In 2010 she joined the NINDS Extramural Research Program as a Health Program Specialist in the Neurodegeneration Cluster. She is also an Adjunct Associate Professor for UMUC and has taught and guest lectured for the NIH graduate school offered through FAES.

Dr. Comfort Boateng joined the Medicinal Chemistry Section in November as an IRTA fellow and recipient of a NIDA Scientific Director Diversity in Research Fellowship.

Retirements and Departures

Mary Affeldt retired on December 31, 2011. Mary had done an outstanding job as the NIDA Associate Director for Management (Executive Officer) since September 30, 2007. In the process she had transformed NIDA's business operations through innovations in communication, increased efficiencies, coalition, team building and her relentless determination to deliver effective solutions. She had also played a central role in many trans-NIH activities, initiatives, and workgroups that have helped NIH as a corporation carry its mission. Mary's career stretches 35 years and prior to her role as the NIDA Associate Director for Management, she had been the Executive Officer for NHGRI and the Chief of Administrative Management in the NIDA Intramural Program.

Dr. Laurence Stanford has retired from NIDA as the Deputy Director of the Division of Clinical Neuroscience and Behavioral Research on December 31, 2011. He had a long and distinguished career of Federal service working at the NSF and the NIH. Dr. Stanford was a tenured faculty at the University of Wisconsin for over 15 years, where his lab conducted a Federally-funded research program investigating the structure and function of the visual system. He did a sabbatical at the NSF to run the basic developmental neurobiology program within their Division of Integrative Biology and Neuroscience. He then joined the CSR at the NIH, where he was instrumental in the creation of the new neuroscience study sections, becoming the first Chief of the Integrative and Functional Neuroscience IRG. He then headed the Neuroscience and Behavioral Science Review Branch at the NIMH and was the Director of the Division of Scientific Review at the NICHD before coming to NIDA, where he directed the developmental neurobiology program for the division, and then served as the Deputy Director of DCNBR since 2004. He has worked tirelessly on many workgroups and committees, both across NIDA and the NIH, and has been extremely involved in research training, particularly with underrepresented groups and through the NIH Neuroscience Blueprint. He has overseen NIDA's very successful I/START Program, which has helped jump-start the careers of many junior investigators. His accomplishments are many, and he will be missed at NIDA.

Dr. Nicolette Borek, Division of Clinical Neuroscience and Behavioral Research, has accepted a position to the Office of Science in the Center for Tobacco Products at the Food and Drug Administration (FDA).

Janelle Barth will be a Supervisory Program Analyst for the Center for Tobacco Prevention and help to build their new organization. She had been at NIH for 10 years, almost half of which was spent with NIDA. During that time, she helped to establish the Management Analysis Branch and supported many new initiatives for our Institute. She was also an active leader and participant on many NIH committees and workgroups.

Patricia Anderson, after 32 years of federal service, retired at the end of 2011. Pat began her federal career at the IRS in their Field Operations Division. She moved to the NIDA Intramural Research Program's Clinical Pharmacology Branch in 1986 where she served as secretary to Branch Chief, Jack Henningfield. In 1994 she joined the Office of Science Policy and Communications (OSPC). Since 1996, Pat has served as publications assistant to Joan Nolan, NIDA's longtime publications and exhibits manager. Pat has always been committed to NIDA and its mission, motivated to reach our audiences, and willing to help people in any way she can. Through her hard work and dedication, she has engendered respect and admiration from her peers for her professionalism.

New Roles within NIDA

In June 2011, Dr. Antonello Bonci established the **Office of Education and Career Development (OECD)** within the Office of the Scientific Director (OSD). **Dr. Stephen Heishman** is Director, OECD, **Dr. Mary Pfeiffer** is Assistant Director, and **Ms. Stacey Saunders** is Administrative Assistant. The OECD serves as a visible and central office to provide the optimal training experience for our postdoctoral fellows, graduate students, post-baccalaureate students, and summer interns. Activities of the OECD include:

- Establishing training and career development programs for NIDA trainees
- Coordinating training activities with NIH Office of Intramural Training and Education
- Implementing a Mentoring Plan for postdoctoral fellows
- Implementing a Graduate Student Incentives Program
- Coordinating the NIH Summer Internship Program
- Coordinating the IRP's involvement in Towson University's Baltimore Excellence in STEM Teaching Project

Geoffrey Schoenbaum, M.D., Ph.D. has been named as Chief, Cellular Neurobiology Research Branch, IRP. Dr. Schoenbaum is renowned as a world authority in the field of cognitive neuroscience.

Markus Heilig, M.D., Ph.D. has been appointed as the Clinical Director for the joint NIDA-NIAAA clinical program. Dr. Heilig is a world leader in the fields of stress and translational neuroscience.

Dr. Brandon Harvey has been appointed as Director of the Optogenetic and Transgenic Technology Center at NIDA. This center is a unique resource available to all NIH intramural programs and will help develop projects related to the use of optogenetics and transgenic rats.

Details

Dr. Cece Spitznas, DCNBR, is on a 6-month detail to the Office of National Drug Control Policy (ONDCP), Executive Office of the President. Dr. Spitznas will provide support and assistance to the ONDCP in the development and implementation of substance abuse policies and programs to place greater emphasis on recovery, treatment and prevention.

Dr. Lula Beatty, Director, Special Populations Office, is on a 12-month detail to the American Psychological Association, Public Interest Directorate where she is serving as a Senior Advisor on health disparities and will work with the APA to establish a substance abuse focus and develop its health disparities strategy to include plans for research, meetings, publications, training and professional development.