

# Director's Report

to the

## NATIONAL ADVISORY COUNCIL ON DRUG ABUSE

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*\* These sections contain select information. More comprehensive information will be posted in the [September 2012 Staff Report to the Director](#).*



## RESEARCH HIGHLIGHTS

### [Retention on Buprenorphine Is Associated with High Levels of Maximal Viral Suppression among HIV-Infected Opioid Dependent Released Prisoners.](#)

Springer SA, Qiu J, Saber-Tehrani SA, Altice FL. PloS ONE. E-pub May 2012; 7(5): e38335

HIV-infected prisoners lose viral suppression within the 12 weeks after release to the community. This prospective study evaluates the use of buprenorphine/naloxone (BPN/NLX) as a method to reduce relapse to opioid use and sustain viral suppression among released HIV-infected prisoners meeting criteria for opioid dependence (OD). From 2005–2010, 94 subjects meeting DSM-IV criteria for OD were recruited from a 24-week prospective trial of directly administered antiretroviral therapy (DAART) for released HIV-infected prisoners; 50 (53%) selected BPN/NLX and were eligible to receive it for 6 months; the remaining 44 (47%) selected no BPN/NLX therapy. Maximum viral suppression (MVS), defined as HIV-1 RNA < 50 copies/mL, was compared for the BPN/NLX and non-BPN/NLX (N = 44) groups. The two groups were similar, except the BPN/NLX group was significantly more likely to be Hispanic (56.0% v 20.4%), from Hartford (74.4% v 47.7%) and have higher mean global health quality of life indicator scores (54.18 v 51.40). MVS after 24 weeks of being released was statistically correlated with 24-week retention on BPN/NLX [AOR = 5.37 (1.15, 25.1)], having MVS at the time of prison-release [AOR = 10.5 (3.21, 34.1)] and negatively with being Black [AOR = 0.13 (0.03, 0.68)]. Receiving DAART or methadone did not correlate with MVS. In recognition that OD is a chronic relapsing disease, strategies that initiate and retain HIV-infected prisoners with OD on BPN/NLX is an important strategy for improving HIV treatment outcomes as a community transition strategy.

### [Early Identification of HIV: Empirical Support for Jail-Based Screening.](#)

De Voux A, Spaulding AC, Beckwith C, Avery A, Williams C, Messina LC, Ball S, Altice FL. PloS ONE. E-pub May 2012; 7(5): e37603

Although routine HIV testing is recommended for jails, little empirical data exist describing newly diagnosed individuals in this setting. Client-level data (CLD) are available on a subset of individuals served in EnhanceLink, for the nine of the 10 sites who enrolled newly diagnosed persons in the client level evaluation. In addition to information about time of diagnosis, the authors analyzed data on initial CD4 count, use of antiretroviral therapy (ART), and linkage to care post discharge. Baseline data from newly diagnosed persons were compared to data from persons whose diagnoses predated jail admission. CLD were available for 58 newly diagnosed and 708 previously diagnosed individuals enrolled between 9/08 and 3/11. Those newly diagnosed had a significantly younger median age (34 years) when compared to those previously diagnosed (41 years). In the 30 days prior to incarceration, 11% of those newly diagnosed reported injection drug use and 29% reported unprotected anal intercourse. Median CD4 count at diagnosis was 432 cells/mL (range: 22–1,453 cells/mL). A minority (21%, N = 12) of new diagnoses started antiretroviral treatment (ART) before release; 74% have evidence of linkage to community services. Preliminary results from a cross-sectional analysis of this cohort suggest testing in jails finds individuals early on in disease progression. Most HIV<sup>+</sup> detainees did not start ART in jail; therefore screening may not increase pharmacy costs for jails. Detainees newly diagnosed with HIV in jails can be effectively linked to community resources. Jail-based HIV testing should be a cornerstone of “test and treat” strategies.

### [Astrocyte Glypicans 4 And 6 Promote Formation Of Excitatory Synapses Via GluA1 AMPA Receptors.](#)

Allen NJ, Bennett ML, Foo LC, Wang GX, Chakraborty C, Smith SJ, Barres BA. Nature. 2012 May 27; 486(7403): 410-414.

In the developing central nervous system (CNS), the control of synapse number and function is critical to the formation of neural circuits. The authors previously demonstrated that astrocyte-secreted factors powerfully induce the formation of functional excitatory synapses between CNS neurons. Astrocyte-secreted thrombospondins induce the formation of structural synapses, but these synapses are postsynaptically silent. Here they use biochemical fractionation of astrocyte-conditioned medium to identify glypican 4 (Gpc4) and glypican 6 (Gpc6) as astrocyte-secreted signals sufficient to induce functional synapses between purified retinal ganglion cell neurons, and show that depletion of these molecules from astrocyte-conditioned medium significantly reduces its ability to induce postsynaptic activity. Application of Gpc4 to purified neurons is sufficient to increase the frequency and amplitude of glutamatergic synaptic events. This is achieved by increasing the surface level and clustering, but not overall cellular protein level, of the GluA1 subunit of the AMPA ( $\alpha$ -amino-3-hydroxy-5-methyl-4-isoxazole propionic acid) glutamate receptor (AMPA). Gpc4 and Gpc6 are expressed by astrocytes in vivo in the developing CNS, with Gpc4 expression enriched in the hippocampus and Gpc6 enriched in the cerebellum. Finally, the authors demonstrate that Gpc4-deficient mice have defective synapse formation, with decreased amplitude of excitatory synaptic currents in the

developing hippocampus and reduced recruitment of AMPARs to synapses. These data identify glypicans as a family of novel astrocyte-derived molecules that are necessary and sufficient to promote glutamate receptor clustering and receptivity and to induce the formation of postsynaptically functioning CNS synapses.

**[Rac1 Is Essential In Cocaine-Induced Structural Plasticity Of Nucleus Accumbens Neurons.](#)** Dietz DM, Sun H, Lobo MK, Cahill ME, Chadwick B, Gao V, Koo JW, Mazei-Robison MS, Dias C, Maze I, Damez-Werno D, Dietz KC, Scobie KN, Ferguson D, Christoffel D, Ohnishi Y, Hodes GE, Zheng Y, Neve RL, Hahn KM, Russo SJ, Nestler EJ. *Nat Neurosci.* E-pub 2012 Apr 22.

Repeated cocaine administration increases the dendritic arborization of nucleus accumbens neurons, but the underlying signaling events remain unknown. Here the authors show that repeated exposure to cocaine negatively regulates the active form of Rac1, a small GTPase that controls actin remodeling in other systems. Further, they show, using viral-mediated gene transfer, that overexpression of a dominant negative mutant of Rac1 or local knockout of Rac1 is sufficient to increase the density of immature dendritic spines on nucleus accumbens neurons, whereas overexpression of a constitutively active Rac1 or light activation of a photoactivatable form of Rac1 blocks the ability of repeated cocaine exposure to produce this effect. Downregulation of Rac1 activity likewise promotes behavioral responses to cocaine exposure, with activation of Rac1 producing the opposite effect. These findings establish that Rac1 signaling mediates structural and behavioral plasticity in response to cocaine exposure.

**[Effects of Perinatal Exposure To Palatable Diets On Body Weight and Sensitivity To Drugs Of Abuse In Rats.](#)**

Bocarsly ME, Barson JR, Hauca JM, Hoebel BG, Leibowitz SF, Avena NM. *Physiol Behav.* E-pub 2012 May 4. The aim of the present study was to determine the effects of fat- and sugar-rich diets *in utero* and during the pre-weaning period on bodyweight and responses to drugs of abuse. In Exp. 1, dams were fed a balanced control diet or high-fat diet (HFD), and female offspring were cross-fostered to dams consuming the balanced diet. The HFD-exposed offspring, compared to controls, were heavier in body weight, had increased circulating triglyceride levels, and consumed more alcohol and HFD in adulthood. In Exp. 2, dams were fed standard chow alone or standard chow plus a 16% high-fructose corn syrup (HFCS) or 10% sucrose solution. Sets of offspring from each group were cross-fostered to dams in the other groups, allowing for the effects of HFCS or sucrose exposure during the gestational period or pre-weaning period to be determined. The offspring (both female and male) exposed to HFCS or sucrose *in utero* had higher body weights in adulthood and exhibited increased alcohol intake as shown in female offspring and increased amphetamine-induced locomotor activity as shown in males. Exposure to HFCS or sucrose only during the pre-weaning period had a similar effect of increasing amphetamine-induced locomotor activity in males, but produced no change in circulating triglycerides or alcohol intake. Collectively, these data suggest that prenatal as well as pre-weaning exposure to fat- and sugar-rich diets, in addition to increasing body weight, can affect responses to drugs of abuse.

**[Function of Human  \$\alpha 3\beta 4\alpha 5\$  Nicotinic Acetylcholine Receptors is Reduced by the  \$\alpha 5\$ \(Asp398Asn\) Variant.](#)**

George AA, Lucero LM, Damaj MI, Lukas RJ, Chen X, Whiteaker P. *J. Biological Chemistry.* E-pub 2012 Jun 4. Genome-wide studies have strongly associated a non-synonymous polymorphism (rs16969968), which changes the 398th amino acid in the nAChR  $\alpha 5$  subunit from aspartic acid to asparagine (Asp398Asn), with greater risk for increased nicotine consumption. The authors have used a pentameric concatamer approach to express defined and consistent populations of  $\alpha 3\beta 4\alpha 5$  nAChR in *Xenopus* oocytes.  $\alpha 5$ (Asn398; risk) variant incorporation reduces ACh-evoked function compared to inclusion of the common  $\alpha 5$ (Asp398) variant, without altering agonist or antagonist potencies. Unlinked  $\alpha 3$ ,  $\beta 4$  and  $\alpha 5$  subunits assemble to form a uniform nAChR population with pharmacological properties matching those of concatameric  $\alpha 3\beta 4^*$  nAChRs.  $\alpha 5$  subunit incorporation reduces  $\alpha 3\beta 4^*$  nAChR function following coinjection with unlinked  $\alpha 3$  and  $\beta 4$  subunits, but increases that of  $\alpha 3\beta 4\alpha 5$  vs.  $\alpha 3\beta 4$ -only concatamers.  $\alpha 5$  subunit incorporation into  $\alpha 3\beta 4^*$  nAChR also alters the relative efficacies of competitive agonists and changes the potency of the non-competitive antagonist mecamylamine. Additional observations indicated that, in the absence of  $\alpha 5$  subunits, free  $\alpha 3$  and  $\beta 4$  subunits form at least two further subtypes. The pharmacological profiles of these free subunit  $\alpha 3\beta 4$ -only subtypes are dissimilar, both to each other, and to those of  $\alpha 3\beta 4\alpha 5$  nAChR. The  $\alpha 5$  variant-induced change in  $\alpha 3\beta 4\alpha 5$  nAChR function may underlie some of the phenotypic changes associated with this polymorphism.

[\*\*Dopaminergic Mechanisms of Individual Differences in Human Effort-Based Decision-Making.\*\*](#) Treadway MT, Buckholtz JW, Cowan RL, Woodward ND, Li R, Ansari MS, Baldwin RM, Schwartzman AN, Kessler RM, Zald DH. *J. Neurosci.* 2012 May; 32(18): 6170–6176.

Preferences for different combinations of costs and benefits are a key source of variability in economic decision-making. However, the neurochemical basis of individual differences in these preferences is poorly understood. Studies in both animals and humans have demonstrated that direct manipulation of the neurotransmitter dopamine (DA) significantly impacts cost/benefit decision-making, but less is known about how naturally occurring variation in DA systems may relate to individual differences in economic behavior. In the present study, 25 healthy volunteers completed a dual-scan PET imaging protocol with [(18)F]fallypride and d-amphetamine to measure DA responsivity and separately completed the effort expenditure for rewards task, a behavioral measure of cost/benefit decision-making in humans. The authors found that individual differences in DA function in the left striatum and ventromedial prefrontal cortex were correlated with a willingness to expend greater effort for larger rewards, particularly when probability of reward receipt was low. Additionally, variability in DA responses in the bilateral insula was negatively correlated with willingness to expend effort for rewards, consistent with evidence implicating this region in the processing of response costs. These findings highlight the role of DA signaling in striatal, prefrontal, and insular regions as key neurochemical mechanisms underlying individual differences in cost/benefit decision-making.

[\*\*Tobacco Smoking Produces Greater Striatal Dopamine Release in G-allele Carriers with Mu Opioid Receptor A118G Polymorphism.\*\*](#) Domino EF, Evans CL, Ni L, Guthrie SK, Koeppe RA, Zubieta J-K. *Prog Neuro-Psychopharm Biol Psychiat.* E-pub 2012 April 10.

The purpose of this study was to determine if carriers of the allelic expression of the G variant of the human mu opioid receptor (OPRM1) A118G polymorphism have greater increases in striatal dopamine (DA) release after tobacco smoking. Nineteen of 20 genotyped male tobacco smokers, after overnight abstinence, smoked denicotinized (denic) and average nicotine (nic) containing tobacco cigarettes in a PET brain imaging study using [<sup>11</sup>C]raclopride. The right striatum had more free D<sub>2</sub> receptors than the left striatum pre- and post-tobacco smoking. After smoking the nic cigarettes, mean decreased DA binding was observed in the left dorsal caudate (– 14.6 ± 1.1; *t* = 3.77), left and right ventral putamen (– 26.3 ± 0.8; *t* = 4.27; 28.2 ± 1.1; *t* = 4.25, respectively), and right caudate (17.18 ± 1.1; *t* = 3.92). The effects of A118G genotype on the binding potentials for these four regions were then analyzed. Carriers of the G allele had larger magnitudes of DA release in response to nic smoking than those homozygous for the more prevalent AA allele in the right caudate and right ventral pallidum (*t* = 3.03; *p* = 0.008 and *t* = 3.91; *p* = 0.001). A voxel by voxel whole brain SPM analysis using an independent samples *t* test did not reveal any other differences between genotype groups. In addition, the venous plasma cortisol levels of the volunteers from 8:30 am to 12:00 noon were lower in the AG/GG allele carriers. Nic smoking increased plasma cortisol in both groups, but they were higher in the AA group. This preliminary study indicates a difference in both brain striatal DA release and plasma cortisol in A118G polymorphic male tobacco smokers.

[\*\*Family-Centered Program Deters Substance Use, Conduct Problems, and Depressive Symptoms In Black Adolescents.\*\*](#) Brody G, Chen Y, Kogan S, Yu T, Molgaard V, DiClemente R, Wingood G. *Pediatrics.* 2012; 129 (1): 108-115.

The present research addressed the following important question in pediatric medicine: Can participation in a new family-centered preventive intervention, the Strong African American Families-Teen (SAAF-T) program, deter conduct problems, substance use, substance use problems, and depressive symptoms among rural black adolescents across 22 months? Data were collected from 502 black families in rural Georgia, assigned randomly to SAAF-T or an attention control condition. The prevention condition consisted of 5 consecutive meetings at community facilities with separate, concurrent sessions for caregivers and adolescents followed by a caregiver-adolescent session in which families practiced skills they learned in the separate sessions. Adolescents self-reported conduct problem behaviors, substance use, substance use problems, and depressive symptoms at ages 16 years (pretest) and 17 years 10 months (long-term assessment). Adolescents who participated in SAAF-T evinced lower increases in conduct problem behavior, substance use, substance use problems, and depressive symptom frequencies than did adolescents in the attention control condition across the 22 months between pretest and long-term assessment. This is the first study to demonstrate efficacy in a prevention program designed to deter conduct problems, substance use, substance use problems, and depressive symptoms among rural black adolescents. Because SAAF-T is a manualized, structured program, it can be easily disseminated to public health agencies, schools, churches, boys' and girls' clubs, and other community organizations.

**Randomized Trial of Standard Methadone Treatment Compared to Initiating Methadone without Counseling: 12-Month Findings.**

Schwartz R, Kelly S, O'Grady K, Gandhi D, Jaffe J. *Addiction*. 2012; 107 (5): 943-952.

This study aimed to determine the relative effectiveness of 12 months of interim methadone (IM; supervised methadone with emergency counseling only for the first 4 months of treatment), standard methadone treatment (SM; with routine counseling) and restored methadone treatment (RM: routine counseling with smaller case-loads). A randomized controlled trial was conducted comparing IM, SM and RM treatment. IM lasted for 4 months, after which participants were transferred to SM. The study was conducted in two methadone treatment programs in Baltimore, MD, USA. The study included 230 adult methadone patients newly admitted through waiting-lists. The authors administered the Addiction Severity Index and a supplemental questionnaire at baseline, 4 and 12 months post-baseline. Measurements included retention in treatment, self-reported days of heroin and cocaine use, criminal behavior and arrests and urine tests for heroin and cocaine metabolites. At 12 months, on an intent-to-treat basis, there were no significant differences in retention in treatment among the IM, SM and RM groups (60.6%, 54.8% and 37.0%, respectively). Positive urine tests for the three groups declined significantly from baseline (Ps < 0.001 and 0.003, for heroin and cocaine metabolites, respectively) but there were no significant group x time interactions for these measures. At least one arrest was reported by 30.6% of the sample during the year, but there were no significant between-group effects. Limited availability of drug counseling services should not be a barrier to providing supervised methadone to adults dependent on heroin—at least for the first 4 months of treatment.

**A Randomized Controlled Trial of a Brief Intervention for Illicit Drugs Linked to the Alcohol, Smoking and Substance Involvement Screening Test in Primary Health-Care Settings in Four Countries.**

Humeniuk R, Ali R, Babor T, Souza-Formigoni MO, Boengen de Lacerda R, Ling W, McRee B, Newcombe D, Pal H, Poznyak V, Simon S, Vendetti J. *Addiction*. 2012; 107 (5): 957-966.

This study evaluated the effectiveness of a brief intervention (BI) for illicit drugs (cannabis, cocaine, amphetamine-type stimulants and opioids) linked to the World Health Organization (WHO) Alcohol, Smoking and Substance Involvement Screening Test (ASSIST). The ASSIST screens for problem or risky use of 10 psychoactive substances, producing a score for each substance that falls into either a low-, moderate- or high-risk category. The study design was a prospective, randomized controlled trial in which participants were either assigned to a 3-month waiting-list control condition or received brief motivational counseling lasting an average of 13.8 minutes for the drug receiving the highest ASSIST score. The study was conducted in primary health-care settings in four countries: Australia, Brazil, India, and the United States. Participants comprised a total of 731 males and females scoring within the moderate-risk range of the ASSIST for cannabis, cocaine, amphetamine-type stimulants or opioids. Measurements collected were ASSIST-specific substance involvement scores for cannabis, stimulants or opioids and ASSIST total illicit substance involvement score at baseline and 3 months post-randomization. Omnibus analyses indicated that those receiving the BI had significantly reduced scores for all measures, compared with control participants. Country-specific analyses showed that, with the exception of the site in the United States, BI participants had significantly lower ASSIST total illicit substance involvement scores at follow-up compared with the control participants. The sites in India and Brazil demonstrated a very strong brief intervention effect for cannabis scores (P < 0.005 for both sites), as did the sites in Australia (P < 0.005) and Brazil (P < 0.01) for stimulant scores and the Indian site for opioid scores (P < 0.01). The Alcohol, Smoking and Substance Involvement Screening Test-linked brief intervention aimed at reducing illicit substance use and related risks is effective, at least in the short term, and the effect generalizes across countries.

**Life Stress, the Dopamine Receptor Gene, and Emerging Adult Drug Use Trajectories: A Longitudinal, Multilevel, Mediated Moderation Analysis.**

Brody GH, Chen Y\_F, Yu T, Beach SRH, Kogan SM, Simons RL, Windle M, Philibert RA. *Development and Psychopathology*. 2012; 24: 941–951

This study was designed to examine the prospective relations of life stress and genetic status with increases in drug use. African Americans (N = 399) in rural Georgia (Wave 1 mean age = 17 years) provided three waves of data across 27.5 months and a saliva sample from which the dopamine receptor D4 (DRD4) gene was genotyped. Multilevel growth curve modeling analysis indicated that emerging adults manifested the highest escalations in drug use when they reported high life stress and carried an allele of DRD4 with 7 or more repeats (7 + R allele). In addition, emerging adults who reported high life stress and carried the 7 + R allele evinced the largest increases in two proximal risk factors for drug use: affiliations with drug-using companions and drug use vulnerability cognitions. Furthermore, when the Gene x Environment interaction effects on the increases in affiliations with drug-using companions and vulnerability cognitions were entered into the model forecasting drug use, the Life Stress x DRD4



Status interaction on drug use became nonsignificant in the presence of the risk mechanisms. This finding provides an example of “second generation” Gene x Environment interaction research in which the interaction’s effects on proximal risk mechanisms account for its effects on outcomes.

**[AAVrh.10-Mediated Expression Of An Anti-Cocaine Antibody Mediates Persistent Passive Immunization That Suppresses Cocaine-Induced Behavior.](#)** Rosenberg JB, Hicks MJ, De BP, Pagovich O, Frenk E, Janda KD, Wee S, Koob GF, Hackett NR, Kaminsky SM, Worgall S, Tignor N, Mezey JG, Crystal RG. Mediated expression of an anti-cocaine antibody mediates persistent passive immunization that suppresses cocaine-induced behavior. *Hum Gene Ther.* 2012 May; 23(5): 451-459.

Cocaine addiction is a major problem affecting all societal and economic classes for which there is no effective therapy. The authors hypothesized an effective anti-cocaine vaccine could be developed by using an adeno-associated virus (AAV) gene transfer vector as the delivery vehicle to persistently express an anti-cocaine monoclonal antibody in vivo, which would sequester cocaine in the blood, preventing access to cognate receptors in the brain. To accomplish this, they constructed AAVrh.10antiCoc.Mab, an AAVrh.10 gene transfer vector expressing the heavy and light chains of the high affinity anti-cocaine monoclonal antibody GNC92H2. Intravenous administration of AAVrh.10antiCoc.Mab to mice mediated high, persistent serum levels of high-affinity, cocaine specific antibodies that sequestered intravenously administered cocaine in the blood. With repeated intravenous cocaine challenge, naive mice exhibited hyperactivity, while the AAVrh.10antiCoc.Mab-vaccinated mice were completely resistant to the cocaine. These observations demonstrate a novel strategy for cocaine addiction by requiring only a single administration of an AAV vector mediating persistent, systemic anti-cocaine passive immunity.

**[A Double-Blind Randomized Controlled Trial Of N-Acetylcysteine In Cannabis-Dependent Adolescents.](#)** Gray KM, Carpenter MJ, Baker NL, Desantis SM, Kryway E, Hartwell KJ, McRae-Clark AL, Brady KT. *Am J Psychiatry.* E-pub 2012 Jun 15.

Preclinical findings suggest that the over-the-counter supplement N-acetylcysteine (NAC), via glutamate modulation in the nucleus accumbens, holds promise as a pharmacotherapy for substance dependence. The authors investigated NAC as a novel cannabis cessation treatment in adolescents, a vulnerable group for whom existing treatments have shown limited efficacy. In an 8-week double-blind randomized placebo-controlled trial, treatment-seeking cannabis-dependent adolescents (ages 15-21 years; N=116) received NAC (1200 mg) or placebo twice daily as well as a contingency management intervention and brief (<10 minutes) weekly cessation counseling. The primary efficacy measure was the odds of negative weekly urine cannabinoid test results during treatment among participants receiving NAC compared with those receiving placebo, in an intent-to-treat analysis. The primary tolerability measure was frequency of adverse events, compared by treatment group. Participants receiving NAC had more than twice the odds, compared with those receiving placebo, of having negative urine cannabinoid test results during treatment (odds ratio=2.4, 95% CI=1.1-5.2). Exploratory secondary abstinence outcomes favored NAC but were not statistically significant. NAC was well tolerated, with minimal adverse events. This is the first randomized controlled trial of pharmacotherapy for cannabis dependence in any age group to yield a positive primary cessation outcome in an intent-to-treat analysis. Findings support NAC as a pharmacotherapy to complement psychosocial treatment for cannabis dependence in adolescents.

**[A Proof-Of-Concept Randomized Controlled Study Of Gabapentin: Effects On Cannabis Use Withdrawal and Executive Function Deficits In Cannabis-Dependent Adults.](#)** Mason BJ, Crean R, Goodell V, Light JM, Quello S, Shadan F, Buffkins K, Kyle M, Adusumalli M, Begovic A, Rao S. *Neuropsychopharmacology* 2012 Jun, (7): 1689-1698.

There are no FDA-approved pharmacotherapies for cannabis dependence. Cannabis is the most widely used illicit drug in the world and patients seeking treatment for primary cannabis dependence represent 25% of all substance use admissions. The authors conducted a phase IIa proof-of-concept pilot study to examine the safety and efficacy of a calcium channel/GABA modulating drug gabapentin for the treatment of cannabis dependence. A 12-week randomized double-blind placebo-controlled clinical trial was conducted in 50 unpaid treatment-seeking male and female outpatients aged 18-65 years diagnosed with current cannabis dependence. Subjects received either gabapentin (1200 mg/day) or matched placebo. Manual-guided abstinence-oriented individual counseling was provided weekly to all participants. Cannabis use was measured by weekly urine toxicology and by self-report using the Timeline Followback Interview. Cannabis withdrawal symptoms were assessed using the Marijuana withdrawal Checklist. Executive function was measured using subtests from the Delis-Kaplan Executive Function System.

Relative to placebo gabapentin significantly reduced cannabis use as measured both by urine toxicology ( $p=0.001$ ) and by the Timeline Followback Interview ( $p=0.004$ ) and significantly decreased withdrawal symptoms as measured by the Marijuana withdrawal Checklist ( $p<0.001$ ). Gabapentin was also associated with significantly greater improvement in overall performance on tests of executive function ( $p=0.029$ ). This POC pilot study provides preliminary support for the safety and efficacy of gabapentin for treatment of cannabis dependence that merits further study and provides an alternative conceptual framework for treatment of addiction aimed at restoring homeostasis in brain stress systems that are dysregulated in drug dependence and withdrawal.

**[Enhanced Attenuation Of Nicotine Discrimination In Rats By Combining Nicotine-Specific Antibodies With A Nicotinic Receptor Antagonist.](#)** LeSage MG, Shelley D, Pravetoni M, Pentel PR. *Pharmacol Biochem Behav.* 2012 Jul; 102(1): 157-162. E-pub 2012 Apr 4.

Tobacco addiction requires activation by nicotine of a variety of central nicotinic acetylcholine receptors (nAChRs). In animals, both nAChR antagonists and immunization against nicotine can reduce nAChR activation by nicotine and block a variety of addiction-relevant behaviors. However, clinical use of nAChR antagonists for smoking cessation is limited by dose-related side effects, and immunization does not reliably produce sufficient antibody levels in smokers to enhance smoking cessation rates. Combining these approaches may be one way of addressing the limitations of each while enhancing overall efficacy. This study examined the individual and combined effects of passive immunization with the monoclonal nicotine-specific antibody Nic311 and the nicotinic receptor antagonist mecamylamine (MEC) on nicotine's discriminative stimulus effects. Rats were trained to discriminate 0.4 mg/kg of nicotine from saline using a two-lever operant discrimination procedure. Antagonism of nicotine discrimination by Nic311 (160 mg/kg i.v.) and ascending doses of MEC (0.03, 0.1, 0.3, and 1.0 mg/kg s.c.) was assessed across four consecutive daily 2-min extinction test sessions using a 2x2 design. Nic311 alone produced a 24-48% reduction in %NLR across all four test sessions. MEC produced a dose-dependent decrease in %NLR, with no effect at the two lowest doses and 80-93% attenuation at the two highest doses. Nic311 combined with MEC significantly suppressed %NLR at every MEC dose (85-92% reduction across all four test sessions). Very low doses of MEC that were ineffective alone completely blocked nicotine discrimination when combined with Nic311. These data demonstrate that nicotine-specific antibodies and MEC can work synergistically to suppress the subjective effects of nicotine and suggest that low doses of MEC may significantly enhance the efficacy of immunotherapy.

**[Implementing Rapid HIV Testing With Or Without Risk-Reduction Counseling In Drug Treatment Centers: Results Of A Randomized Trial.](#)** Metsch LR, Feaster DJ, Gooden L, Matheson T, Mandler RN, Haynes L, Tross S, Kyle T, Gallup D, Kosinski AS, Douaihy A, Schackman BR, Das M, Lindblad R, Erickson S, Korhuis PT, Martino S, Sorensen JL, Szapocznik J, Walensky R, Branson B, Colfax GN. *Am J Public Health.* 2012 Jun;102(6):1160-7. E-pub 2012 Apr 19.

The authors examined the effectiveness of risk reduction counseling and the role of on-site HIV testing in drug treatment. Between January and May 2009, they randomized 1,281 HIV-negative (or status unknown) adults who reported no past-year HIV testing to (1) referral for off-site HIV testing, (2) HIV risk-reduction counseling with on-site rapid HIV testing, or (3) verbal information about testing only with on-site rapid HIV testing. The authors defined 2 primary self-reported outcomes a priori: receipt of HIV test results and unprotected anal or vaginal intercourse episodes at 6-month follow-up. The combined on-site rapid testing participants received more HIV test results than off-site testing referral participants ( $P<.001$ ; Mantel-Haenszel risk ratio=4.52; 97.5% confidence interval [CI] =3.57, 5.72). At 6 months, there were no significant differences in unprotected intercourse episodes between the combined on-site testing arms and the referral arm ( $P=.39$ ; incidence rate ratio [IRR]=1.04; 97.5% CI=0.95, 1.14) or the 2 on-site testing arms ( $P=.81$ ; IRR=1.03; 97.5% CI=0.84, 1.26). This study demonstrated on-site rapid HIV testing's value in drug treatment centers and found no additional benefit from HIV sexual risk-reduction counseling.

**[Stimulant Abuser Groups To Engage In 12-Step: A Multisite Trial In The National Institute On Drug Abuse Clinical Trials Network.](#)** Donovan DM, Daley DC, Brigham GS, Hodgkins CC, Perl HI, Garrett SB, Doyle SR, Floyd AS, Knox PC, Botero C, Kelly TM, Killeen TK, Hayes C, Kau'ibbaumhofer N, Seamans C, Zammarelli L. *J Subst Abuse Treat.* E-pub 2012 May 30.

The study evaluated the effectiveness of an 8-week combined group plus individual 12-step facilitative intervention on stimulant drug use and 12-step meeting attendance and service. The study design was a multisite randomized controlled trial, with assessments at baseline, mid-treatment, end of treatment, and 3- and 6-month post-

randomization follow-ups (FUs) conducted in intensive outpatient substance treatment programs. Individuals with stimulant use disorders (n=471) were randomly assigned to treatment as usual (TAU) or TAU into which the Stimulant Abuser Groups to Engage in 12-Step (STAGE-12) intervention was integrated. Urinalysis and self-reports of substance use and 12-step attendance and activities were collected. Group sessions focused on increasing acceptance of 12-step principles; individual sessions incorporated an intensive referral procedure connecting participants to 12-step volunteers. Compared with TAU, STAGE-12 participants had significantly greater odds of self-reported stimulant abstinence during the active 8-week treatment phase; however, among those who had not achieved abstinence during this period, STAGE-12 participants had more days of use. STAGE-12 participants had lower Addiction Severity Index Drug Composite scores at and a significant reduction from baseline to the 3-month FU, attended 12-step meetings on a greater number of days during the early phase of active treatment, engaged in more other types of 12-step activities throughout the active treatment phase and the entire FU period, and had more days of self-reported service at meetings from mid-treatment through the 6-month FU. The present findings are mixed with respect to the impact of integrating the STAGE-12 intervention into intensive outpatient drug treatment compared with TAU on stimulant drug use. However, the results more clearly indicate that individuals in STAGE-12 had higher rates of 12-step meeting attendance and were engaged in more related activities throughout both the active treatment phase and the entire 6-month FU period than did those in TAU.

**Frontal Systems Deficits In Stimulant-Dependent Patients: Evidence Of Pre-Illness Dysfunction and Relationship To Treatment Response.** Winhusen TM, Somoza EC, Lewis DF, Kropp FB, Horigian VE, Adinoff B. Drug Alcohol Depend. E-pub 2012 Jul 6.

Frontal systems dysfunction is present in stimulant-dependent patients. However, it is unclear whether this dysfunction is a pre-morbid risk factor or stimulant-induced, is severe enough to be clinically relevant, and if it is relevant to treatment response. These questions were addressed using the Frontal Systems Behavior Scale (FrSBe), a reliable and valid self-report assessment of three neurobehavioral domains associated with frontal systems functioning (Apathy, Disinhibition, and Executive Dysfunction, summed for a Total), that assesses both pre- and post-morbid functioning, and has a specific cutoff for defining clinically significant abnormalities. The study involved six sites evaluating 12-step facilitation for stimulant abusers obtained the FrSBe from 180 methamphetamine- and/or cocaine-dependent participants. Dichotomous treatment response measures included self-reported stimulant use, stimulant urine drug screens, and treatment completion. A substantial percentage of participants retrospectively reported clinically significant neurobehavioral abnormalities prior to lifetime stimulant abuse initiation (e.g., 67.5% on FrSBe-Total) with a significant increase in the proportion reporting such abnormalities for current functioning (86% on FrSBe-Total;  $p < 0.0001$ ). Treatment response was significantly worse for participants with, relative to those without, clinically significant Disinhibition as measured by treatment non-completion (31.6% vs. 15.6%, OR=2.51) and self-reported stimulant use during treatment (40.5% vs. 16.7%, OR=3.40). These findings suggest that frontal systems dysfunction is present prior to stimulant-abuse onset and worsens with stimulant use. Disinhibition may be a prime target for intervention in stimulant-dependent individuals.

**Medial Prefrontal Cortex Neuronal Activation and Synaptic Alterations After Stress-Induced Reinstatement of Palatable Food Seeking: A Study Using C-Fos-GFP Transgenic Female Rats.** Cifani C, Koya E, Navarre BM, Calu DJ, Baumann MH, Marchant NJ, Liu Q-R, Khuc T, Pickel J, Lupica CR, Shaham Y, Hope BT. (2012) Journal of Neuroscience 32(25):8480-8490.

Relapse to maladaptive eating habits during dieting is often provoked by stress and there is evidence for a role of ovarian hormones in stress responses and feeding. IRP scientists studied the role of these hormones in stress-induced reinstatement of food seeking and medial prefrontal cortex (mPFC) neuronal activation in *c-fos*-GFP transgenic female rats, which express green fluorescent protein (GFP) in strongly activated neurons. Food-restricted ovariectomized or sham-operated *c-fos*-GFP rats were trained to lever-press for palatable food pellets. Subsequently, lever-pressing was extinguished and reinstatement of food seeking and mPFC neuronal activation was assessed after injections of the pharmacological stressor yohimbine (0.5-2 mg/kg) or pellet priming (1-4 non-contingent pellets). Estrous cycle effects on reinstatement were also assessed in wild-type rats. Yohimbine- and pellet-priming-induced reinstatement was associated with Fos and GFP induction in mPFC; both reinstatement and neuronal activation were minimally affected by ovarian hormones in both *c-fos*-GFP and wild-type rats. *c-fos*-GFP transgenic rats were then used to assess glutamatergic synaptic alterations within activated GFP-positive and non-activated GFP-negative mPFC neurons following yohimbine-induced reinstatement of food seeking. This reinstatement was associated with reduced AMPAR/NMDAR current ratios and increased paired-pulse facilitation in activated GFP-positive but not GFP-

negative neurons. Together, while ovarian hormones do not appear to play a role in stress-induced relapse of food seeking in this rat model, this reinstatement was associated with unique synaptic alterations in strongly activated mPFC neurons. This paper introduces the *c-fos*-GFP transgenic rat as a new tool to study unique synaptic changes in activated neurons during behavior.

**Willingness To Wait and Altered Encoding of Time-Discounted Reward In the Orbitofrontal Cortex With Normal Aging.** Roesch MR, Bryden DW, Cerri DH, Haney ZR, Schoenbaum G. *J Neurosci.* 2012 Apr 18; 32(16):5525-5533.

Normal aging has been associated with cognitive changes, including shifts in responding for time-discounted rewards. The orbitofrontal cortex, an area previously associated with aging-related cognitive changes, is critical for normal discounting. Previously, IRP investigators have shown in a choice task that rats prefer immediate over delayed reward and that neural representations of delayed reward in orbitofrontal cortex were attenuated, whereas immediate reward elicited strong responses. Changes in choice performance were correlated with changes in firing rate in orbitofrontal neurons, suggesting that these reward representations were critical to the rats' ability to wait for reward. Here the authors asked whether age-dependent changes in discounting behavior were related to changes in the representation of delayed reward in the orbitofrontal cortex. Young (3-6 months) and aged (22-26 months) rats were trained on the same discounting paradigm used previously. They found that aged rats showed less sensitivity to increasing delay preceding reward delivery, shifting behavior away from the delayed reward more slowly than younger rats. This sensitivity was specific to delay, since choice performance did not differ between the two groups when delay was held constant and reward size varied. Aged rats exhibited a corresponding increase in the prevalence of neurons that fired more strongly for delayed reward. Again this change was specific to delay; there was no change in encoding of different-sized rewards. These results suggest that natural aging results in altered representations of reward in orbitofrontal cortex. These changes may relate to the increased ability to delay gratification and reduced impulsivity associated with aging.

**Molecular Determinants of Selectivity and Efficacy at the Dopamine D3 Receptor.** Newman AH, Beuming T, Banala AK, Donthamsetti P, Pongetti K, LaBounty A, Levy B, Cao J, Michino M, Luedtke RR, Javitch JA, Shi L. *J Med Chem* 2012, E-pub May 25, 2012.

The dopamine D3 receptor (D3R) has been implicated in substance abuse and other neuropsychiatric disorders. The high sequence homology between the D3R and D2R, especially within the orthosteric binding site (OBS) that binds dopamine, has made the development of D3R-selective compounds challenging. Here, IRP scientists deconstruct into pharmacophoric elements a series of D3R-selective substituted-4-phenylpiperazine compounds, and use computational simulations and binding and activation studies to dissect the structural bases for D3R selectivity and efficacy. They find that selectivity arises from divergent interactions within a second binding pocket (SBP) separate from the OBS, whereas efficacy depends on the binding mode in the OBS. These findings reveal structural features of the receptor that are critical to selectivity and efficacy that can be used to design highly D3R-selective ligands with targeted efficacies. These findings are generalizable to other GPCRs in which the SBP can be targeted by bitopic or allosteric ligands.

**R-Modafinil (Armodafinil): A Unique Dopamine Uptake Inhibitor and Potential Medication For Psychostimulant Abuse.** Loland CJ, Mereu M, Okunola OM, Cao J, Prisinzano TE, Mazier S, Kopajtic T, Shi L, Katz JL, Tanda G, Newman AH. *Biol. Psychiatry* 2012, E-pub April 24, 2012.

(±)-Modafinil has piqued interest as a treatment for ADHD and stimulant dependence. (±)-, R- and S-Modafinil bind to the DAT and inhibit dopamine uptake less potently than cocaine, with R-modafinil having ~3-fold higher affinity than its S-enantiomer. Molecular docking studies revealed subtle differences in binding modes for the enantiomers. R-modafinil was significantly less potent in the DAT Y156F mutant compared to wild-type DAT, whereas S-modafinil was affected less. Studies with the Y335A DAT mutant showed that the R- and S-enantiomers tolerated the inward facing conformation better than cocaine, which was further supported by MTSET reactivity on the DAT E2C I159C. Microdialysis studies demonstrated that both R- and S-modafinil produced increases in extracellular DA concentrations in the NAc shell less efficaciously than cocaine, and with a longer duration of action. Both enantiomers fully substituted in mice trained to discriminate cocaine from saline. R-modafinil displays an in vitro profile different from cocaine. Future trials with R-modafinil as a substitute therapy with the potential benefit of cognitive enhancement for psychostimulant addiction are warranted.

**Corticotropin-Releasing Factor Receptor-Dependent Effects of Repeated Stress on Tau Phosphorylation, Solubility, and Aggregation.** Rissman RA, Staup MA, Lee AR, Justice NJ, Rice KC, Vale W, Sawchenko PE. Proc Natl Acad Sci U S A. 2012 Apr 17;109(16):6277-82. E-pub 2012 Mar 26.

Exposure and/or sensitivity to stress have been implicated as conferring risk for development of Alzheimer's disease (AD). Although the basis for such a link remains unclear, the authors previously reported differential involvement of corticotropin-releasing factor receptor (CRFR) 1 and 2 in acute stress-induced tau phosphorylation (tau-P) and solubility in the hippocampus. Here IRP investigators examined the role of CRFRs in tau-P induced by repeated stress and the structural manifestations of altered tau solubility. Robust tau-P responses were seen in WT and CRFR2 null mice exposed to repeated stress, which were sustained at even 24 h after the final stress exposure. A portion of phosphorylated tau in these mice was sequestered in detergent-soluble cellular fractions. In contrast, CRFR1 and CRFR double-KO mice did not exhibit repeated stress-induced alterations in tau-P or solubility. Similarly, treatment with CRFR1 antagonist attenuated repeated stress-induced tau-P. Using histochemical approaches in a transgenic CRFR1 reporter mouse line, the authors found substantial overlap between hippocampal CRFR1 expression and cells positive for phosphorylated tau after exposure to repeated stress. Ultrastructural analysis of negatively stained extracts from WT and CRFR2 null mice identified globular aggregates that displayed positive immunogold labeling for tau-P, as well as conformational changes in tau (MC1) seen in early AD. Given that repeated stress exposure results in chronic increases in hippocampal tau-P and its sequestration in an insoluble (and potentially prepathogenic) form, these data may define a link between stress and an AD-related pathogenic mechanism.

## CONGRESSIONAL AFFAIRS

(Prepared August 8, 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**

**Women and Smoking.** On June 8, NIDA Director Dr. Nora Volkow participated in a Congressional briefing on women and smoking. The briefing was cosponsored and organized by Women's Policy, Inc., Legacy, and the Women's Health Task Force of the House Women's Caucus. Also presenting was Cheryl Heaton, Legacy's CEO. Dr. Volkow presented research on and discussed the unique and specific difficulties faced by women who try to quit smoking.

**HIV/AIDS.** On July 18, the Friends of the National Institute on Drug Abuse (NIDA) in conjunction with the Congressional Addiction, Treatment and Recovery Caucus hosted a congressional briefing titled "Treatment as Prevention: HIV/AIDS and Substance Abuse." The briefing, for which the American Psychological Association provided significant logistical support, featured presentations by NIDA director Dr. Nora Volkow and three other scientists whose research is funded by the institute. Dr. Nora Volkow presented on the shift in direction of HIV/AIDS research since the 2011 breakthrough discovery that early antiretroviral therapy prevented transmission, likely by suppressing HIV viral load. She presented statistics on the prevalence and the outcomes of treatment with highly active antiretroviral therapy (HAART), including that injection drug users are much less likely to receive the treatment than are other HIV positive patients.

## **Federal Regulations/Investigations/Reports Requested by Congress**

**Labor-HHS-Education Bill.** The Senate appropriations committee reported out its Labor-HHS-Education bill in June. That bill provides NIH with \$30,731,459,000, \$100 million over the President's request, including \$1.057 billion for NIDA. The House Labor-HHS-Education appropriations subcommittee reported out its bill in July. The bill provides NIH with \$30,631,459,000, equal to the FY 2012 level. This would include \$1.052 billion for NIDA. The full committee has yet to consider the bill. There have already been significant policy arguments about several sections of the bill. A [complete summary](#) is available for anyone interested in those details.

**Special Issue: Sequestration.** (Some text excerpted from Congressional Quarterly) On August 7, President Obama signed into law H.R. 5872, a bill that requires the administration to detail within 30 days how it would implement the looming spending cuts in domestic and defense programs. The "sequester transparency measure," as it is called, directs the White House to spell out what kinds of reductions at the "program, project and activity level" would result from allowing slated across-the-board cuts to take place. Leaders in both parties have said that federal law should be changed to derail these currently mandated \$109 billion cuts, known as the sequester, but Democrats and Republicans remain deeply split about how to find an alternative plan for deficit reduction.

**Special Issue: Continuing Resolution.** (text from NIH/OLPA) On July 31, 2012, Senator Harry Reid (D-NV), Senate Majority Leader, announced that he and Representative John Boehner (R-OH), Speaker of the House, had reached an agreement on a six-month Continuing Resolution for FY2013, which would be written during the August recess and voted on in September. Most continuing resolutions maintain flat-line funding from one fiscal year into the next. However, under this agreement, funding would be consistent with the \$1.047 trillion level for fiscal 2013 set forth in last year's Budget Control Act, and above the \$1.028 trillion called for by Representative Paul Ryan's (R-WI) budget proposal. It is also above the \$1.043 trillion level for the current fiscal year called for by the law.



## NIH/HHS POLICY UPDATES

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

- May 15** [Final Weeks to Register for NIH Regional Seminar on Program Funding and Grants Administration in Washington, D.C. - June 20-22, 2012](#)
- May 18** [Notice of NIH Piloting of Procedures for Special Council Review of Research Applications from PD\(s\)/PI\(s\) with More than \\$1.5 Million Total Annual NIH Support](#)
- May 23** [The Division of Receipt and Referral will use eRA Commons to Communicate with Applicants and Applicant Organizations](#)
- June 1** [Request for Information \(RFI\): Input on Proposed Modifications of the Biographical Sketch Used in NIH Grant Applications](#)
- June 1** [Clarification of Position Statements on Implementation of the Guide for the Care and Use of Laboratory Animals: Eighth Edition](#)
- June 11** [Notice of Impending Change in Peer Review Criteria and Submission Requirements for NIH Applications Involving Human Embryonic Stem Cells](#)
- June 15** [Clarification to the Interim Agency Policy, NIH Research Involving Chimpanzees](#)
- June 25** [NIH Announces Changes to the eRA Commons Financial Conflict of Interest \(FCOI\) Module to Accommodate Additional Reporting Requirements Required by the 2011 Revised FCOI Regulation](#)
- July 27** [NIH FCOI Reporting Requirements and Demo - Webinar, August 14, 2012 - Registration Open](#)
- August 1** [Clarification: Time Limit on NIH Resubmission Applications](#)
- August 2** [Guidance on Changes That Involve Human Subjects in Active Awards and That Will Require Prior NIH Approval](#)
- August 2** [Prior NIH Approval of Human Subjects Research in Active Awards Initially Submitted without Definitive Plans for Human Subjects Involvement \(Delayed Onset Awards\)](#)

## PROGRAM ACTIVITIES/FOAS

### New NIDA RFAs

On May 11, 2012, NIDA issued an RFA entitled **Identifying Health Outcomes Associated with Changes in Use of Illicit Drugs (R01)** [RFA-DA-13-007](#). The National Institute on Drug Abuse (NIDA) is soliciting grant (R01) applications to test the hypothesis that reductions in illicit drug use are associated with improved health outcomes in patients. This FOA will support both prospective and retrospective studies, which may include, but not limited to, identification and characterization of beneficial health outcomes that are associated with reduced levels of drug use. Such studies may focus on identification and or validation of strategies, methods, and tools (including biomarkers) that can assess the salutary consequences resulting from reduced use of a particular illicit drug. Open date: July 22, 2012. Application due date: August 22, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On May 15, 2012, NIDA issued an RFA entitled **Synthesis and Preclinical Evaluation of Medications to Treat Substance Use Disorders (SUDs) (R01)** [RFA-DA-13-004](#). The National Institute on Drug Abuse (NIDA) is soliciting grant (R01) applications to support the synthesis and preclinical evaluation of new molecular entities as potential treatments for Substance Use Disorders (SUDs). The goal is to identify candidate compounds and advance them towards Investigational New Drug (IND) submission. Open date: July 15, 2012. Application due date: August 15, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On June 14, 2012, NIDA issued an RFA entitled **Advancing Exceptional Research on HIV/AIDS (R01)** [RFA-DA-13-008](#). This FOA will support highly innovative R01 applications on HIV/AIDS and drug abuse and will complement the Avant-Garde program. This FOA focuses on innovative research projects that have the potential to open new areas of HIV/AIDS research and/or lead to new avenues for prevention and treatment of HIV/AIDS among drug abusers. Open date: November 17, 2012. Application due date: December 17, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On July 13, 2012, NIDA issued an RFA entitled **Translational Research on Interventions for Adolescents in the Legal System: TRIALS (U01)** [RFA-DA-13-009](#). The National Institute on Drug Abuse (NIDA) invites applications for cooperative agreement participants (multiple Research Centers and one coordinating center) to collaborate in developing and testing implementation strategies and associated measures to improve the continuum of substance abuse prevention and treatment services delivered to youth under juvenile justice supervision. Open date: October 28, 2012. Application due date: November 28, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On August 24, 2012, NIDA issued an RFA entitled **The Interplay of Substance Abuse and HIV-1 Infection on Glial Cell Function (R01)** [RFA-DA-13-010](#) (R21) [RFA-DA-13-011](#). This Funding Opportunity Announcement (FOA) issued by the National Institute on Drug Abuse (NIDA) solicits basic and pre-clinical research applications that study the combined and interactive effects of substance abuse and HIV-1 infection on glial cell biology. The goal of this FOA is to encourage research to determine the molecular and cellular consequences of substance abuse, HIV-1 infection, and their interactions on glial cells within the central nervous system (CNS). Open date: October 19, 2012. Application due date: November 19, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

### **New NIDA Program Announcements**

On June 8, 2012, NIDA issued a PAR entitled **Research Education Grants for Statistical and Computational Training in the Genetics of Addiction (R25)** [PAR-12-199](#). The purpose of this opportunity is to encourage applications focused on research education in statistical and computational models to address genetics-based problems in addiction. Open date: August 14, 2012. Application due date: September 14, 2012, August 21, 2013, August 21, 2014, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On June 29, 2012, NIDA issued a PAR entitled **Cohort Studies of HIV/AIDS and Substance Use (U01)** [PAR-12-222](#). This FOA will support the development and maintenance of new cohorts or the expansion of existing cohorts to address the natural and treated history of HIV infection in at-risk populations where substance use is a central factor. The intent of the FOA is to provide a strong resource platform for current and future collaborative efforts with other investigators to address emerging questions related to HIV infection, prevention, and treatment in the context of substance abuse, as well as to foster the creativity and efficiency of investigator-initiated research goals. Open date: November 11, 2012. Application due date(s): December 11, 2012, April 9, 2013, December 11, 2013, April 9, 2014, December 11, 2014, and April 9, 2015, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On July 6, 2012, NIDA issued a PA entitled **Development and Testing of Novel Interventions to Improve HIV Prevention, Care, and Program Implementation (R34)** [PA-12-231](#). This FOA is issued by the National Institute on Drug Abuse (NIDA), National Institutes of Health for R34 applications, and provides resources to support (a) pilot or feasibility studies of new or adapted interventions to prevent HIV infection among populations where substance use may be a contributing factor; (b) pilot or feasibility studies of new or adapted interventions to improve the care of HIV infection among populations where substance use is prevalent, including interventions that integrate treatment for substance use disorders and HIV infection; or (c) pilot or feasibility studies to increase the scale, uptake, delivery, and/or quality of HIV prevention or care interventions with established evidence of efficacy. Both primary and secondary prevention will be supported. Open date: August 7, 2012. Application due date(s): Not applicable. AIDS application due date: [Standard dates](#) apply, by 5:00 PM local time of applicant organization.



On July 16, 2012, NIDA issued a PAR entitled **Development of Minimally-Invasive Bioassays to Support Outpatient Clinical Trials of Therapeutics for Substance Use Disorders (R01) [PAR-12-239](#); (R21) [PAR-12-238](#)**. The announcement has two main aims. The first aim is to encourage the development of devices / techniques that will improve estimations of a subject's consumption of an abused drug (i.e. both quantity and frequency of consumption) during an outpatient clinical trial. The second aim of this FOA is to develop new, improved markers to evaluate a subject's adherence to the study medication. Open date: September 5, 2012 (R01); September 16, 2012 (R21). Application due date(s): [Standard dates](#) apply, by 5:00 PM local time of applicant organization. AIDS application due date: [Standard dates](#) apply, by 5:00 PM local time of applicant organization.

On July 26, 2012, NIDA issued a PAR entitled **Behavioral Science Track Award for Rapid Transition (B/START) (R03) [PAR-12-251](#)**. This FOA will use the NIH Small Research Grant (R03) award mechanism and seeks to facilitate the entry of beginning investigators into the field of behavioral science research related to drug abuse. To be appropriate for a B/START award, research must be primarily focused on behavioral processes and research questions. Open date: September 16, 2012. Application due date(s): Not applicable. AIDS application due date: [Standard dates](#) apply, by 5:00 PM local time of applicant organization.

### **New FOAs Issued by the NIH Roadmap**

On June 13, 2012, the NIH Common Fund issued a Roadmap RFA entitled **Human Heredity and Health in Africa (H3Africa): Ethical, Legal, and Societal Issues (ELSI) Research Program (U01) [RFA-RM-12-005](#)**. This Funding Opportunity Announcement (FOA) encourages applications to study the ethical, legal and societal issues (ELSI) of human genome research in African populations. Of particular interest are projects that propose focused bioethical, legal, and social science analyses of new or emerging issues. Open date: September 29, 2012. Application due date(s): October 29, 2012, by 5:00 PM local time of applicant organization. AIDS application due date(s): Not applicable.

On June 13, 2012, the NIH Common Fund issued a Roadmap RFA entitled **NIH Director's Transformative Research Awards (R01) [RFA-RM-12-017](#)**. The NIH Director's Transformative Research Awards complements NIH's traditional, investigator-initiated grant programs by supporting individual scientists or groups of scientists proposing groundbreaking, exceptionally innovative, original and/or unconventional research with the potential to create new scientific paradigms, establish entirely new and improved clinical approaches, or develop transformative technologies. Open date: August 21, 2012. Application due date(s): September 21, 2012, by 5:00 PM local time of applicant organization. AIDS application due date(s): Not applicable.

On June 13, 2012, the NIH Common Fund issued a Roadmap RFA entitled **NIH Director's Early Independence Awards (DP5) [RFA-RM-12-018](#)**. The NIH Director's Early Independence Award Program supports exceptional investigators who wish to pursue independent research directly after completion of their terminal doctoral/research degree or clinical residency, thereby forgoing the traditional post-doctoral training period. Open date: December 30, 2012. Application due date(s): January 30, 2013, by 5:00 PM local time of applicant organization. AIDS application due date(s): Not applicable.

On August 22, 2012, the NIH Common Fund issued a Roadmap RFA entitled **Human Heredity and Health in Africa (H3Africa): Collaborative Centers (U54) [RFA-RM-12-006](#)**. The purpose of this FOA is to call for applications for U54 Collaborative Centers that will provide funding to support multi-project research programs that address the goals of H3Africa and that involve two or more collaborations with investigators from outside the applicant institution. Application due date(s): October 29, 2012, by 5:00 PM local time of applicant organization. AIDS application due date(s): Not applicable.

On August 22, 2012, the NIH Common Fund issued a Roadmap RFA entitled **Human Heredity and Health in Africa (H3Africa): H3Africa Research Grants (U01) [RFA-RM-12-007](#)**. The purpose of this NIH FOA is to invite applications from Institutions in African countries for Research Projects (U01 cooperative agreements) that address one or more goals of the Human Heredity and Health in Africa (H3Africa) initiative. H3Africa is an NIH initiative in partnership with the Wellcome Trust with the goals of developing the study of genomic/genetic/environmental contributors to human

health and disease within Africa using cutting-edge genomic research tools, increasing capacity for biomedical research in Africa, in terms of building the infrastructure needed for genomic research (including data and research resources), and increasing the genomic proficiency of researchers and trainees in Africa. Open date: September 28, 2012. Application due date(s): October 29, 2012, by 5:00 PM local time of applicant organization. AIDS application due date(s): Not applicable.

On August 22, 2012, the NIH Common Fund issued a Roadmap RFA entitled **Human Heredity and Health in Africa (H3Africa): H3Africa Biorepository Grants. (UH2/UH3) [RFA-RM-12-008](#)**. The purpose of this FOA is to call for applications for UH2/UH3 cooperative agreements that will provide funding to develop plans for an H3Africa Biorepository, building upon existing infrastructure. Applications should include plans for initial two-year UH2 Phase I studies (Phase I) as well as plans for an additional four years of support for implementing a full-scale H3Africa Biorepository (Phase II), beginning in 2014. Open date: September 28, 2012. Application due date(s): October 29, 2012, by 5:00 PM local time of applicant organization. AIDS application due date(s): Not applicable.

#### **New Administrative Supplement Program Announcements Issued by NIH**

On June 13, 2012, NIDA, in collaboration with numerous other NIH components, issued an administrative supplement entitled **Establish Sharing of Human Brain Image Data Relevant to Drug Addiction (Admin Supp) [PAR-12-204](#)**. This program is intended to supplement NIDA funded projects to enable investigators to standardize and disseminate brain image data from patient (current or former drug abusers or subjects with risk factors) and/or healthy comparison subjects. Open date: July 9, 2012. Application due date(s): August 9, 2012, November 9, 2012, February 9, 2013, May 9, 2013, August 9, 2013, by 5:00 PM local time of applicant organization. AIDS application due date: not applicable.

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

On June 13, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **U.S. India Bilateral Collaborative Research Partnerships (CRP) on the Prevention of HIV/AIDS and Co-morbidities (R21) [RFA-AI-12-033](#)**. The U.S.-India Bilateral CRP Program is designed to develop collaborations between scientists and institutions in the U.S. and India to conduct high quality HIV/AIDS prevention research of mutual interest and benefit to both countries while developing the basis for future institutional and individual scientific collaborations. This FOA will utilize the research capacities of the institutions and scientists in both countries to advance the field of HIV/AIDS prevention and to develop preliminary data that may support a more extensive future research proposal to test an HIV/AIDS prevention program. Open date: August 4, 2012. Application due date(s): September 4, 2012, by 5:00 PM local time of applicant organization. AIDS application due date: September 4, 2012, by 5:00 PM local time of applicant organization.

On July 5, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **Advancing Community-level Approaches to Reduce HIV Infection in Highly Impacted Communities (R34) [RFA-MH-13-092](#); (R21) [RFA-MH-13-091](#); (R01) [RFA-MH-13-090](#)**. This Funding Opportunity Announcement (FOA) seeks research to advance our understanding of community-level HIV-prevention and care interventions within geographic locations and specific populations highly impacted by HIV. In targeting communities, this FOA invites applications to address the need for efficacious interventions that simultaneously impact a large number of individuals. Open date: December 11, 2012. Application due date(s): Not applicable. AIDS application due date: January 11, 2013, by 5:00 PM local time of applicant organization.

On August 23, 2012, NIDA, in collaboration with numerous other NIH components, issued an RFA entitled **TaRGET I: Chromatin Structure, Genomics, and Transcriptional Responses to the Environment (R01) [RFA-ES-12-008](#)**. The purpose of this FOA is to encourage research applications that will potentially move the field from descriptive and correlative studies to an enhanced mechanistic understanding of how environmental exposures affect the proteins and functional genomic elements involved in establishing and maintaining gene expression patterns and chromatin states. Open date: October 19, 2012. Application due date(s): November 19, 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 June 19, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **NIH Support for Conferences and Scientific Meetings (Parent R13/U13) [PA-12-212](#)**. The purpose of the NIH Research Conference (R13) Grant and NIH Research Conference Cooperative Agreement (U13) Programs is to support high quality conferences that are relevant to the public health and to the scientific mission of the participating Institutes and Centers. Open date: July 12, 2012. Application due date(s): [Standard dates](#) apply, by 5:00 PM local time of applicant organization. AIDS application due date: [Standard dates](#) apply, by 5:00 PM local time of applicant organization.

On June 21, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Women's Mental Health During Pregnancy and the Postpartum Period (R01) [PA-12-216](#); (R21) [PA-12-215](#)**. The purpose of this Funding Opportunity Announcement (FOA) is to outline priority areas for research related to women's mental health during pregnancy and the postpartum period. Priority areas include basic and clinical neuroscience, studies of clinical course, epidemiological factors and risk factors, as well as interventions and services research. Open date: September 5, 2012. Application due date(s): [Standard dates](#) apply, by 5:00 PM local time of applicant organization. AIDS application due date: [Standard dates](#) apply, by 5:00 PM local time of applicant organization.

On July 5, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Research on Children in Military Families: The Impact of Parental Military Deployment and Reintegration on Child and Family Functioning (R13) [PA-12-223](#)**. The purpose of this funding opportunity announcement (FOA) is to encourage interdisciplinary conferences and meetings to examine critical questions regarding the impact of parental military deployment, combat-related stress and reintegration with the family on child social and affective development outcomes as well as on family functioning. Open date: July 12, 2012. Application due date(s): [Standard dates](#) apply, by 5:00 PM local time of applicant organization. AIDS application due date: [Standard dates](#) apply, by 5:00 PM local time of applicant organization.

On July 24, 2012, NIDA, in collaboration with numerous other NIH components, issued a PAR entitled **Ethical Issues in Research on HIV/AIDS and its Co-morbidities (R01) [PAR-12-244](#); (R21) [PAR-12-243](#)**. This Funding Opportunity Announcement (FOA) invites applications addressing ethical issues in research relevant to populations with HIV and associated co-morbidities, and populations at high risk of HIV acquisition. Open date: November 7, 2012. Application due date(s): January 7, 2013; January 7, 2014; January 7, 2015, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

On August 24, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Successor-in-Interest (Type 6 Parent) [PA-12-269](#)**. The National Institutes of Health (NIH) hereby notify grantee organizations holding specific types of NIH research grants, listed in the full Funding Opportunity Announcement (FOA), that applications for change of grantee organization status, often referred to in this announcement as Successor-In-Interest, may be submitted in response to this FOA. Open date: August 24, 2012. Application due date(s): A successor-in-interest request must be made before the anticipated start date at the new organization and preferably several months in advance. AIDS application due date: Not applicable.

On August 24, 2012, NIDA, in collaboration with numerous other NIH components, issued a PA entitled **Change of Grantee Organization (Type 7 Parent) [PA-12-270](#)**. The National Institutes of Health (NIH) hereby notify grantee organizations holding specific types of NIH grants, listed in the full Funding Opportunity Announcement (FOA), that applications for change of grantee organization may be submitted in response to this FOA. Open date: August 24, 2012. Application due date(s): A change of grantee organization request must be made before the anticipated start date at the new organization and preferably several months in advance. AIDS application due date: Not applicable.

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

On July 10, 2012, NIDA, in collaboration with numerous other NIH components and with the FDA Center for Tobacco Products, issued a Request for Application entitled **Tobacco Centers of Regulatory Science for Research Relevant to the Family Smoking prevention and Tobacco Control Act (P50) [RFA-DA-13-003](#)**. The Tobacco Centers of Regulatory Science (TCORS) program objective is to conduct programs of multidisciplinary research that will inform tobacco product regulation and address the research priorities related to the regulatory authority of the Food and Drug Administration (FDA) Center for Tobacco Products (CTP). Application due date: November 14, 2012. Start Date: September 2013.

On August 23, 2012, NIDA, in collaboration with numerous other NIH components and with the FDA Center for Tobacco Products, issued a PAR entitled **Tobacco Control Regulatory Research (R21) [PAR-12-266](#); (R01) [PAR-12-267](#); (R03) [PAR-12-268](#)**. The purpose of this Funding Opportunity Announcement (FOA) is to encourage biomedical, behavioral, and social science research that will inform the development and evaluation of regulations on tobacco product manufacturing, distribution, and marketing. Open date: October 1, 2012. Application due date(s): November 1, 2012; January 16, 2013; June 18, 2013; January 15, 2014; June 17, 2014; January, 16, 2015, by 5:00 PM local time of applicant organization. AIDS application due date: Not applicable.

## **NIDA PUBLICATIONS**

### **Research Report Series: Drug Abuse and HIV**

**NIH Pub. No.: 12-5760**

This research report is designed to highlight the state of the science and to raise awareness of the link between HIV/AIDS and drug abuse – not just injection drug use but drug abuse in general. This update was released to coincide with the XIX International AIDS Conference held in Washington, DC in July 2012. The report details NIDA's multifaceted approach towards ending this disease and the ongoing research strategies to prevent and treat it. [En Español](#)

## **PRESS RELEASES**

**May 18, 2012** – [Optogenetics project takes top NIDA Addiction Science Award](#)

**May 21, 2012** – [NIH selects 11 Centers of Excellence in Pain Education](#)

**May 30, 2012** – [Odds of quitting smoking affected by genetics](#)

**June 28, 2012** – [NIDA Director Nora Volkow to speak at Aspen Ideas Festival July 2](#)

**July 18, 2012** – [NIDA Announces 2012 Avant-Garde Award for Medications Development](#)

**July 23, 2012** – [Prevention of HIV Spread Focus of NIDA's 2012 Avant-Garde Awards](#)

**July 25, 2012** – [NIDA supports development of combined anti-heroin and HIV vaccine](#)

**August 8, 2012** – [New dates. National Drug Facts Week begins Jan. 28, 2013](#)

## MEETINGS/CONFERENCES

### Select Meetings and Conferences in which NIDA played a significant role

On April 23-24, 2012, The "[NIDA Special Populations Research Development Seminar Series](#)" workshop was convened in Bethesda, Maryland for new investigators interested in becoming funded through NIDA and the NIH. Chaired by Flair Lindsey, Program Analyst, Special Populations Office, during the two-day session new investigators met with funded NIDA investigators and senior NIDA program officials, received feedback on research concept papers and learned about the NIH grants submission and review processes.

On May 3-4, 2012 in Rockville, MD, DCNBR hosted the [ACYF Neuroscience and Child Maltreatment Meeting](#), convened by Commissioner Bryan Samuels at the Administration on Children, Youth and Families (ACYF) DHHS in collaboration with NIDA, NICHD, and the Robert Wood Johnson Foundation.

On June 9-14, 2012 in Palm Springs, CA, the [75<sup>th</sup> Annual Meeting of the College on Problems of Drug Dependence \(CPDD\)](#) was held. As a part of this meeting OSPC coordinated a [NIDA Grant-Writing and Career Workshop](#), which included presentations on funding opportunities and career-building skills by Drs. David Shurtleff, Eliane Lazar-Wesley, Linda Cottler (University of Florida), and Frances Levin (Columbia University). NIDA program staff also organized multiple symposia on topics ranging from polydrug abuse in teens to medications development to novel uses of technology for the prevention and treatment of substance use disorders. In addition, NIDA and CPDD co-hosted a [Training Networking Event](#) to provide a forum for T-32 training directors, trainees, and NIDA staff to discuss the different training programs that NIDA supports, and for trainees to find employment and career development opportunities. Twenty [Director's Travel Awards](#) were given to National Research Service Award (NRSA) trainees and fellows and Diversity Supplement recipients at this year's meeting. In addition, NIDA's Women & Sex/Gender Differences Research Program gave twenty-eight [Women & Gender Junior Investigator Travel Awards](#) to first author junior investigators who made presentations on the topic of women and/or sex/gender differences.

On Thursday, June 21, 2012 at the Neuroscience Center in Rockville, Maryland, NIDA convened the inaugural "[NIDA SPO Translational Research Speaker Series: Promoting Diversity and Moving Toward Health Equity](#)," which was centered on the theme of HIV and Drug Abuse from Lab to Clinic. Dr. Benjamin Chen of Mount Sinai School of Medicine, the first guest speaker, presented on HIV and Drug Abuse from Lab to Clinic.

On July 16-19, 2012 in Atlanta, Georgia, NIDA's Special Populations Office convened its fifth annual "[Addiction Research Training Institute](#)," at the Morehouse School of Medicine. The four-day workshop provided grants writing, research training, and valuable mentoring to early career investigators interested in research careers in substance abuse and HIV/AIDS. Pamela Goodlow, Public Health Analyst, Special Populations Office and Dionne Jones, Deputy, Services Research Branch, DESPR, participated in the workshop.

On August 2-5, 2012 in Orlando, Florida, the National Institute on Drug Abuse (NIDA) organized a program at the [2012 American Psychological Association \(APA\) Annual Meeting](#). NIDA staff and grantees participated in a number of sessions on a wide range of topics related to addiction research, such as symposia on increasing implementation of evidence-based interventions by computerizing treatments and how innovations in neuroscience can spur innovations for adolescent drug abuse treatment. NIDA also co-sponsored an Early Career Investigator Poster Session with APA's Divisions 28 and 50 and the National Institute on Alcohol Abuse and Alcoholism (NIAAA) as part of the two Divisions' Social Hour.

## **Upcoming Conferences/Exhibits**

On Friday, October 12, 2012 in New Orleans, LA, the [NIDA Mini-Convention Frontiers in Addiction Research](#) will be held at the [Annual Meeting of the Society for Neuroscience \(SfN\)](#). Sessions to be included are:

- (1) Ghrelin, Leptin, and Insulin Modulated Reward
- (2) Jacob P. Waletzky Memorial Lecture
- (3) Role of Phagocytes in Synaptic Plasticity and Remodeling of Tissues in the Nervous System
- (4) Early Career Poster Session
- (5) Brain Energies and Neurotransmission: Fueling Neurons and Glia
- (6) Central Nervous System Immune Signaling and Addiction

### **Other tentatively scheduled NIDA-sponsored activities during the SfN meeting include:**

Toni S. Shippenberg Memorial Symposium - Friday, October 12, 2012, 6:00 p.m. – 7:30 p.m.

Julius Axelrod Lecture and Poster Session - Sunday, October 14, 2012, 6:30 p.m. – 9:30 p.m.

NIDA/INSERM Workshop on US-France Collaboration on Drug Abuse and Addiction Research - Monday, October 15, 2012, 6:30 p.m. – 9:00 p.m.

NIH Grant Workshop for Early Career Investigators - Tuesday, October 16, 2012, 6:30 p.m. – 9:00 p.m.

## **COMMUNITY AND PRESS EVENTS**

### **NIH K-12 Lessons About Biology (LAB) Challenge**

March 1, 2012, Winners announced. NIDA participated in the first LAB Challenge, a national call-to-action asking individuals, groups, organizations, and scientists to submit procedures for engaging, hands-on health- and life-science-related experiments for grades K-12. Activities needed to: (1) be geared towards grades K-12; (2) use safe, easily available, and inexpensive materials; (3) take 90 minutes (or less) of in-class time; (4) have at least one clear learning objective; and (5) be related to the NIH mission. An overwhelming number of engaging submissions were received. These submissions were carefully reviewed and the experiments were tried by NIH staff and in classrooms. The phase 1 winners were announced March 1, and the phase two winners announced May 1. Winners receive an official, electronic NIH Challenge badge to display online.

### **NIDA Director Dr. Nora Volkow Featured on 60 Minutes**

April 29, 2012, aired on *60 Minutes*. A piece profiling Dr. Nora Volkow titled “Hooked: Why Bad Habits are Hard to Break”. *60 Minutes Overtime* also produced four accompanying stories titled *Are You Addicted to Food?; A Cure for Addiction?; Addiction, Ethnicity and Environment; and The Most Dangerous Drugs?* The following evening, *60 Minutes* conducted its first ever Live Facebook Chat featuring Dr. Volkow. As a result of the promotion surrounding the profile piece and chat, NIDA reached over 12 million people on Twitter and nearly 5 million people on Facebook.

### **Aspen Ideas Festival**

Dr. Volkow participated in the Aspen Ideas Festival July 2 in Aspen, Colorado. The festival, in its eighth year, is presented by the Aspen Institute and *The Atlantic*, and is a gathering of the world’s foremost thought leaders.

### **Addiction Inc.**

NIDA hosted a screening of *Addiction Inc.*, a major motion picture about Dr. Victor DeNoble, a former research scientist for Philip Morris who testified before Congress in 1994 about his research showing the addictive nature of nicotine. More than 300 NIH staff attended the showing and panel discussion which featured Dr. Volkow, Dr. DeNoble, Charles Evans Jr., the film’s producer/director, and Dr. Paul Mele, a researcher also in the film.

### **Spanish Language Videos**

NIDA is creating a resource teachers can use to educate Spanish-speaking teens on drug abuse and addiction. Portions from Dr. Volkow’s meeting with high school students in Mexico and an instructional kit will be available online in time for [National Drug Facts Week](#) in January 2013.



## STAFF HONORS AND AWARDS

### 2012 NIH Director's Awards

#### **The NIDA Population Assessment of Tobacco and Health (PATH) Study Team**

For outstanding contributions to science and public health in launching a landmark longitudinal study on tobacco and health with FDA's Center for Tobacco Products.

Wilson Compton, M.D., M.P.E.

Kevin Conway, Ph.D.

Kathy Etz, Ph.D.

John Hamill

Donna M. Jones

Elizabeth Y. Lambert, M.Sc.

Marsha Lopez, Ph.D., M.H.S.

Brian H. O'Laughlin, M.P.A.

James L. Quinn, III, J.D.

Kay Wanke, Ph.D., M.P.H.

#### **Surgeon General Call to Action on Prescription Drug Abuse Among Youth**

In recognition of exceptional and sustained leadership to produce a U.S. Surgeon General's Call to Action to Prevent Prescription Drug Abuse Among Youth.

Jessica Cotto, M.P.H.

Gaya Dowling, Ph.D.

Jennifer Elcano, M.A.

Carol M. Krause

Anna Staton, M.P.A.

Isabelle Thibau

Eric Wargo, Ph.D.

Susan Weiss, Ph.D.

**Elizabeth Y. Lambert, M.Sc.** and **Tisha Wiley Ph.D.**, DESPR, received an NIH Director's Award for their participation in the NIH Lesbian, Gay, Bisexual, and Transgender Research Coordinating Committee.

### 2012 NIDA Director's Award

**Mark Fleming**, OSPC, received the NIDA Director's Innovator Award in recognition for his innovative redesign of NIDA's Web site to provide complete mobile access, thus broadening public access to scientific information about drug abuse and addiction.

### Other Staff Honors and Awards

**Dr. Rao Rapaka**, DBNBR, received the ICRS 2012 Lifetime Achievement Award from ATA for achievements in the area of Biomedical Science, Freiburg, Germany.

NIDA's **Easy-To-Read website** (<http://easyread.drugabuse.gov/>) has won several awards including a ClearMark Award of Distinction from the Center for Plain Language and a Merit Award from "Web Health Awards: Honoring the Best Digital Health Resources." In addition, an abstract entitled "NIDA's 'Easy-to-Read Drug Facts' website: Reaching low-literacy audiences online" was selected for a poster presentation at the 2012 APHA Annual Meeting.

## **Grantee Honors and Awards**

**Dr. Krista M. Lisdahl**, a Principal Investigator at the University of Wisconsin-Milwaukee, was selected as one of 20 NIH recipients of the 2011 Presidential Early Career Award in Science and Engineering (PECASE).

## **STAFF CHANGES**

### **New Employees**

**Ms. Jessica Henry** has joined the CCTN for a summer internship. Jessica is currently a doctoral student in Clinical/Community Psychology at The George Washington University in Washington, D.C. In 2006, she received her M.A. in Clinical Psychology from Teachers College, Columbia University, and she graduated summa cum laude from Howard University in 2005, with a B.S. in Psychology. Jessica already has an extensive list of publications, honors, awards, and clinical and research experience that includes work with Trauma-Focused CBT; Research Assistant positions with NIMH, Howard, Yale, Columbia, and Brown; and internships with NIDA in OSPC and DESPR.

### **New Roles within NIDA**

**Dr. Susan Weiss** has accepted a new position in the OD as Associate Director for Scientific Affairs, where she will serve as a senior advisor to the NIDA Director. Previously at NIDA, Dr. Weiss served as the Chief of the Science Policy Branch, overseeing the preparation of scientific and policy communications for members of Congress and the public, along with outreach and education programs for elementary school students and training of young scientists and new grantees. Most recently, she served as the Acting Director, Office of Science Policy and Communications, providing leadership and oversight for all of NIDA's interactions with its diverse stakeholders. Before coming to NIDA, Dr. Weiss supervised a research program in the Biological Psychiatry Branch of the National Institute of Mental Health (NIMH) focused on characterizing the evolving nature of psychiatric and neurologic illnesses to inform the development of novel treatment options for patients with affective, anxiety, and substance use disorders.

**Anita LoMonico**, IRMB, OM has been selected to serve as NIDA's Acting CIO. Anita has served as the Deputy CIO since her arrival at NIDA in January of 2010 and brings almost 30 years of NIH lab, IT and administrative experience to the position.

**Janet Linton**, OSPC, became a Federal employee in June of 2012 and serves as an IT Specialist with NIDA's web team. She has been working in OSPC under a contract since 2009 and will continue to provide web support to the NIDA Webmaster. Her primary tasks include, but are not limited to: ensuring NIDA's content is compliant with section 508, overseeing NIDA's Facebook, Flickr, and YouTube accounts, and maintaining the National Drug Facts Week site. Prior to joining OSPC, Ms. Linton was a lead operator, training and overseeing employees, at the PNC Bank Operation Center in Baltimore, MD. She holds a Bachelor of Science degree in Animation from Westwood College.

### **New Appointments**

**Jack B. Stein, MSW, Ph.D.** joined NIDA in August 2012 as the new Director of the NIDA Office of Science Policy and Communications (OSPC). Jack has over two decades of professional experience in leading national drug and HIV-related research, practice, and policy initiatives for NIDA, the Substance Abuse and Mental Health Services Administration (SAMHSA) and the Office of National Drug Control Policy (ONDCP) where, before coming back to NIDA, he served as the Chief of the Prevention Branch.



## **Departures**

**Christine Colvis, Ph.D.**, Director of Program Integration, left NIDA in July 2012 to accept a position at the newly formed National Center for Advancing Translational Sciences (NCATS), where she is leading their Therapeutics Discovery Program. Christine joined the Genetics and Molecular Neurobiology Research Branch in DNBDR in 2001. She had been doing proteomics research as a Research Fellow at the National Eye Institute prior to coming to NIDA. At NIDA, she helped build the proteomics portfolio, and when she saw the potential importance that epigenetics could play in addiction, she began building the epigenetics portfolio as well. Christine was well-known to the staff of the Common Fund serving on several project teams for Roadmap and the Common Fund since the program began. In 2008, Christine moved to the OD in NIDA where she worked with the Director on a number of projects including ARRA.

**Jan Leahey**, Deputy Director of NIDA's Office of Acquisitions (OA), has recently accepted a Program Analyst position in the Food and Drug Administration (FDA), Center for Tobacco Products. Ms. Leahey has worked as both an acquisition official and program manager in various Agencies throughout Health and Human Services (HHS) during her federal career. In 2001, Ms. Leahey joined the National Institute of Child Health and Human Development (NICHD), where she participated in the start-up of the National Children's Study (NCS). In addition to supporting the NCS, Ms. Leahey worked as a Program Manager in the Obstetric and Pediatric Pharmacology Branch, where she supported the Best Pharmaceuticals for Children Act. Ms. Leahey has worked in NIDA since 2010, providing support to acquisition and program staff, as they implemented various government initiatives, including the most recent Efficient Spending Policy.

**Jeff Weiner**, NIDA's Chief Information Officer left in July to help oversee business improvement processes at the Center for Tobacco Products at FDA. Jeff has been at NIH for twenty years and joined NIDA three years ago to help the IRMB group and NIDA improve our IT programs. His efforts helped solidify our IT operations, mature our IT portfolio activities, and expand community involvement in NIDA IT decisions. His commitment to enhancing the NIH and NIDA missions through IT services is greatly appreciated.

## **Retirements**

**Dr. Bennett Fletcher** retired on September 1, 2012. Bennett joined NIDA in 1987. His first assignment was to establish a study to evaluate treatment outcomes, which became the Drug Abuse Treatment Outcome Studies (DATOS), a prospective follow-up study of patients in drug abuse treatment that ran from 1989 until about 2008. DATOS resulted in more than 85 peer-reviewed publications and laid the groundwork for research on the organization and delivery of treatment services. Dr. Fletcher was also instrumental in building a research demonstration program on drug abuse treatment to prevent HIV/AIDS, which produced a number of important studies, including DATAR (PI D. Dwayne Simpson, TCU) and the Key-Crest studies (PI James Inciardi, U. Delaware). From 1996 until 2004, Dr. Fletcher was chief of the Services Research Branch. He expanded the role of health services research to include studies of the economics and financing of treatment, research on special populations, and research on the organization and management of treatment services. In 1992, he established a research program focused on the interaction between drug abuse treatment and the criminal justice system. This program led to the Criminal Justice – Drug Abuse Treatment Studies (CJ-DATS), an ongoing cooperative research program that has expanded NIDA's focus on the science of implementation and how evidence-based treatment is effectively integrated into the criminal justice system. Dr. Fletcher worked with NIDA colleagues to produce and publish several widely-distributed publications, including the Principles of Drug Addiction Treatment and the Principles of Drug Abuse Treatment for Criminal Justice Populations. From 2002 until his retirement, Dr. Fletcher was the Science Officer on CJ-DATS.

**Karen Skinner, Ph.D.**, retired from NIDA on June 30<sup>th</sup>, 2012. Prior to her retirement, Dr. Karen Skinner had been with NIH, including NIDA, for over 36 years, and her many contributions have been far reaching; from developing novel, high impact programs to bringing high caliber investigators to the area of drug abuse and addiction. Her commitment and dedication to our shared mission, and her love of science have inspired us. Karen sees the big picture and the molecular details as well – after all she is a chemist. As a carryover from her days as a writer at

Chemistry and Engineering News, she encouraged plain language and clear writing for all NIDA documents before it became fashionable at the NIH and government-wide levels. Her ability to see the connections among otherwise disparate programs and her intuition for where the science is going next are among her strengths. Karen has always had her hand on the pulse of technology and has always been “out front” to lead us into the areas that are barely visible on the horizon. Her discerning eye and her breadth of knowledge have identified key developments in areas of science that could be made relevant to the mission of NIDA. Karen was instrumental in establishing the Basic Neuroscience Program at NIDA. She was the first to recognize the importance of the BMAP—Brain Molecular Anatomy Project—which later morphed into the Neuroscience Blueprint. She envisioned the Neuroscience Information Framework (NIF), created the initiative and shepherded it through its gestation. Karen created the NIDA Neuroscience Consortium and, while Acting Director of DBNBR, instituted DBNBR’s Science Friday meetings and facilitated the incorporation of the internet into the everyday activities of NIDA/DBNBR staff. She insisted on having an on-site library or reading room, which became the Roger Brown room, named in memory of a DBNBR program officer. She would cold-call researchers (often prominent prize winners) to get them to apply to NIDA - Sidney Brenner, Paul Greengard, David Brecht, Richard Zare and Mark Wightman to name a few. She hired and mentored Jonathan Pollock and selected David Shurtleff as her deputy. Her impact on NIDA has been significant and her imprint is still evident in all we do. As is typical of Karen’s character—she will be serving as a volunteer at NIDA to continue to have her fingerprints on a variety of programs that are of interest to her.

## IN MEMORIAM

**Dr. Toni Shippenberg**, a highly respected IRP scientist, died after a long struggle with cancer. She was recognized both within NIDA and throughout the larger scientific community as an influential and distinguished researcher in the field of neuroscience, focusing for the past 2 decades on addiction research in support of NIDA’s mission. Toni elegantly combined genetics with behavioral pharmacology to uncover the pivotal role that the dynorphin/k-opioid receptor system plays in the control of dopamine dynamics in the brain’s reward center and, secondarily, in mood, motivation, and cognitive functions. Thanks to her seminal discoveries, the kappa opioid system has become a major focus of translational research that could lead to the development of novel medications for the treatment of several psychiatric disorders, including addiction. Her contributions to neuroscience and neuropharmacology leave an outstanding legacy that extends throughout this country and internationally, with achievements that include appointments to the prestigious Senior Biomedical Research Service (SBRS) and an honorary professorship at the University of Queensland in Australia. Dr. Shippenberg received numerous awards throughout her career and will be remembered not only as a distinguished neuroscientist, but also as a devoted mentor to many in our community who have gone on to achieve successful scientific careers of their own, all over the world.

