1. The Federal Communications Commission's (FCC) Wireless Telecommunications Bureau (Bureau) will be conducting a license audit of the operational status of all site-specific licenses operating under part 22, Paging and Radiotelephone Service, subpart E, with a ''CD'' radio service code and all site-specific licenses operating in the 929–930 MHz band on the exclusive channels with a ''GS'' radio service code, wherein all licensees listed, except for licensees in the ''GS'' service operating on shared channels, must respond to the audit letter and certify that its authorized station(s) has not permanently discontinued operations from the date of initial construction and operation. The audit is being performed to promote intensive use of the radio spectrum by updating and increasing the accuracy of the Commission's licensing database.

2. To prepare for the audit, the Bureau strongly encourages licensees in these radio services to verify the mailing address for each license held prior to September 25, 2004. Licensees can verify the accuracy of the Commission's information by accessing the License Search function in the Universal Licensing System (ULS) at *http://wireless.fcc.gov/uls.* If the information is incorrect, the licensee should use ULS to electronically file an Update application.

3. Another important step a licensee should take to prepare for the audit is to ensure that it has registered in CORES, received an FRN, and associated the FRN with all licenses held. This should be done by September 25, 2004.

4. The Bureau will send letters to all licensees operating in the ''CD'' and ''GS'' (exclusive channels) radio services inquiring about the operational status of each license held. The letters will be mailed during the week of September 27, 2004. Each letter will include the call signs of the licensee's authorizations involved in this audit and will be directed to each licensee at its address of record in ULS. A licensee will receive only one audit letter if the licensee has, by September 25, 2004, verified the address is listed correctly in ULS, obtained its FRN, and associated its call signs with the FRN. If the licensee has not performed these activities by September 25, 2004, the Bureau will attempt to include all of a licensee's call signs subject to this audit in one letter, but may issue more than one letter for an entity due to slight variations in licensee name or address in the Commission's licensing records. If a licensee receives multiple letters, the licensee must respond to each letter in order to account for all its call signs that are part of this audit. If a licensee holds authorizations in one of these radio services (''CD'' and ''GS'' exclusive channels) and does not receive an audit letter, the licensee must still respond to the audit. In order to determine if a particular license is a part of the audit, licensees should use Audit Search at *http://wireless.fcc.goc/licensing/audits after the audit letters have been mailed* (scheduled for the week of September 27, 2004). If the search shows an audit letter was mailed, the licensee is required to respond to the audit using the audit reference number. For instructions on how to proceed in this instance, licensees can call the Commission at 717–338–2888 or 888–CALLFCC (888–225–5322) and select option 2.

5. A response to the audit letter is mandatory. The process for responding to the audit and the internet site will be included in the audit letter. Each licensee is required to submit its response electronically within forty-five (45) calendar days of the date on the audit letter. Failure to provide a timely response may result in the Commission presuming that the station(s) has permanently discontinued operations pursuant to 47 CFR 22.317, and thus the license may be presumed to have automatically cancelled. Failure to provide a timely response may also result in a enforcement action, including monetary forfeiture, pursuant to section 503(b)(1)(B) of the Communications Act and 47 CFR 1.80(a)(2).

Federal Communications Commission.

Marlene H. Dortch,

Secretary.

[FR Doc. 04–20361 Filed 9–7–04; 8:45 am]

BILLING CODE 6712–01–P

FEDERAL TRADE COMMISSION

Sunshine Act Meeting

AGENCY: Federal Trade Commission.

TIME AND DATE: 10 a.m., Thursday, December 9, 2004.

PLACE: Federal Trade Commission Building, Room 532, 600 Pennsylvania Avenue, NW., Washington, DC 20580.

STATUS: Part of this meeting will be open to the public. The rest of the meeting will be closed to the public.

MATTERS TO BE CONSIDERED: Portion Open to Public: (1) Oral Argument in the matter of Rambus Incorporated, Docket 9302.

Portion Closed to the Public: (2) Executive Session to follow Oral Argument in Rambus Incorporated, Docket 9302.

CONTACT PERSON FOR MORE INFORMATION: Mitch Katz.

Office of Public Affairs: (202) 326–2180.

Recorded Message: (202) 326–2711.

Donald S. Clark,

Secretary.

[FR Doc. 04–20403 Filed 9–3–04; 12:10 pm]

BILLING CODE 6750–01–M

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

Renovations in the Division of Dockets Management

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice.

SUMMARY: The Food and Drug Administration (FDA) is announcing the partial closing of the Division of Dockets Management (DDM) on September 9, 2004, to September 14, 2004. During the renovations in DDM, it is necessary to partially close the office to allow the staff and others to store and dismantle furniture and other items. The purpose of this document is to inform the public in advance to avoid confusion in carrying out DDM's functions.

DATES: On September 10, 2004, the office and open space areas of DDM will be painted and the carpet replaced. Therefore, from September 9, 2004, to September 14, 2004, DDM will be partially closed. During this time, the public reading room will be open from 9 a.m. to 4 p.m., normal business hours, to accept hand-delivered documents, but will not provide other services.

FOR FURTHER INFORMATION CONTACT: Jennie C. Butler, Division of Dockets Management (HFA–305), Food and Drug Administration, 5630 Fishers Lane, rm. 1061, Rockville, MD 20852, 301–827–6880, e-mail: *jbutler1@oc.fda.gov.*

ADDRESSES: Anyone wishing to hand deliver documents to DDM should go to 5630 Fishers Lane, rm. 1061, Rockville, MD 20852. Normal operations of DDM will resume on September 15, 2004. Please note: The telephones will be unavailable from 1 p.m. on September 10, 2004, through 12 noon on September 13, 2004.

SUPPLEMENTARY INFORMATION:

I. Background

DDM, which is part of the Office of Management, is responsible for many activities under 21 CFR 10.20. The

major functions of DDM include the following: (1) Serving as the entry point for citizen petitions, comments, hearing requests, and other documents related to FDA's rulemaking and administrative activities; (2) maintaining a public reading room, in which documents are available for public inspections; (3) providing copies of official records maintained in accordance with the Freedom of Information Act; and (4) providing advice and guidance regarding filing requirements pertaining to FDA's rulemaking or administrative activities.

Dated: September 2, 2004.

William K. Hubbard,

Associate Commissioner for Policy and Planning.

[FR Doc. 04–20394 Filed 9–3–04; 11:49 am]

BILLING CODE 4160–01–S

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Opportunities for Cooperative Research and Development Agreements (CRADAs) To Undertake Research and Development of Compounds From Specific Categories for the Treatment of Drug Dependence

AGENCY: National Institutes of Health, PHS, DHHS.

ACTION: Notice.

SUMMARY: The National Institute on Drug Abuse (NIDA), a component of the National Institutes of Health, is seeking proposals from potential collaborators for one or more Cooperative Research and Development Agreements (CRADAs) to test, by scientific means meeting U.S. Food and Drug Administration (FDA) standards, the hypothesis that compounds representative of the following classes (numbered 1–8 below) may be useful in the treatment of drug dependence:

1. CRF–1 antagonists.
2. Cannabinoid-1 antagonists.
3. MGluR5 antagonists.
4. AMPA antagonists.
5. Selective, high affinity dopamine D3 agonists and antagonists.
6. Selective, high affinity dopamine D1 full or partial agonists.
7. Kappa opioid antagonists.
8. Compounds from classes not named in 1–7 above, but for which compelling rationales are provided by potential collaborators.

DATES: NIDA will consider all proposals received within 60 days of the date of the publication of this notice. This notice is active until November 8, 2004.

ADDRESSES: Questions and expressions of interest concerning this notice may be addressed to Dr. Frank Vocci (301–443–2711; e-mail: *fv6k@nih.gov*) or Mr. Lee Cummings (301–443–1143; e-mail: *lc65i@nih.gov*) or at the following address: Division of Pharmacotherapies and Medical Consequences of Drug Abuse, National Institute on Drug Abuse, 6001 Executive Boulevard, MSC 955, Room 4123, Bethesda, Maryland 20892–9551.

SUPPLEMENTARY INFORMATION:

Rationale for CRF–1 Antagonists

Evidence suggests that withdrawal syndromes associated with chronic use of drugs of abuse results in elevations of CRF levels. Stress has been shown to modify the intake of drugs of abuse in preclinical studies of drug self-administration. The effects of stress can increase drug intake and can be mimicked by CRF administration. CRF antagonists have a robust inhibitory effect on stress-induced increases in drug taking behavior.

Rationale for Cannabinoid-1 Antagonists

Cannabinoid-1 antagonists (CB–1) block the cell surface receptors activated by marijuana, but have been reported to be involved in effects of other abused substances. A CB–1 receptor antagonist has been shown to reduce nicotine self-administration and nicotine-induced dopamine release in the nucleus accumbens, reduce heroin self-administration in rats, and reduce amphetamine self-administration in rats. Further, CB–1 antagonists may also prevent relapse to cocaine or heroin by blocking rats' responses to both cocaine and heroin priming, and to cues associated with cocaine. Finally, mice lacking the CB–1 receptor do not show stress-induced increases in alcohol consumption, suggesting that the receptor may also contribute to stress-induced drinking. Taken together, results suggest a role for the cannabinoid system in abuse of several classes of drugs.

Rationale for mGluR5 Antagonists

Drugs of abuse increase glutamatergic neurotransmission in the nucleus accumbens, and metabotropic glutamate receptors located there may modulate release of glutamate and dopamine. Since gene knockout studies reported in 2001 showed that mice lacking the mGluR5 receptor show decreased locomotor stimulant effects of cocaine and fail to develop cocaine self-administration behavior, a number of studies have examined the effects of mGluR5 antagonist on behaviors related to drug abuse. Interestingly, mGluR5 antagonists have been reported to decrease cocaine and nicotine self-administration in rodents, decrease amphetamine-stimulated locomotor activity, and to attenuate relapse to alcohol, suggesting a role in abuse of more than one drug.

Rationale for AMPA Antagonists

AMPA antagonists may be useful in the treatment of cocaine addiction because AMPA antagonists have been shown to affect three cocaine-induced processes thought to be important in the development of cocaine addiction for: (1) Prevention of locomotor sensitization, (2) Blockade of cocaine-cue induced drug seeking behavior, and (3) blocking cocaine-primed reinstatement in an animal model of cocaine self-administration.

Rationale for D3 Partial Agonists and Antagonists

Dopamine D3 receptors are localized in areas of the brain that are involved in drug abuse, and have been reported to be up-regulated in the brains of cocaine overdose fatalities. The potency of compounds that activate D3 receptors is related to their ability to decrease cocaine self-administration in rats, suggesting the involvement of these receptor types in cocaine drug-taking. Dopamine D3 partial agonists have been shown to block the behaviorally activating effects of cues that have been paired with cocaine in rats, suggesting potential usefulness in blocking relapse following contact with environmental cues associated with drug use. Dopamine D3 antagonists have recently been reported to block nicotine-primed nicotine seeking behavior in rats as well as cocaine-primed cocaine seeking in rats, suggesting a potential role in preventing relapse. Further, a D3 antagonist has been reported to block both the acquisition and expression of heroin conditioned place preference in rats, suggesting, overall, that both dopamine D3 partial agonists and D3 antagonists may be useful treatments for more than one drug of abuse.

Rationale for D1 Agonists

There is evidence that dopamine D1 receptors are down-regulated in rats and monkeys following exposure to cocaine using in vitro measures. In addition, D1 agonists have been shown to lack priming effects in rats trained to self-administer cocaine, and are able to block the effects of a priming dose of cocaine in this model. Other cocaine-self administration models indicate that D1 agonists can reduce the self-administration of both low and high