



MEDIVATION PRESENTS POSITIVE NEW DATA ON DIMEBON'S LONG-TERM EFFICACY AND NOVEL MECHANISM OF ACTION AT THE INTERNATIONAL CONFERENCE ON ALZHEIMER'S DISEASE

SAN FRANCISCO (July 30, 2008) – Medivation, Inc. (NASDAQ: MDVN) today announced new data showing that its investigational drug Dimebon continues to produce broad, clinically meaningful benefits in Alzheimer's disease patients after long-term dosing, and appears to operate through a novel mechanism of action. These data were presented today in a podium session and two poster sessions at the 2008 Alzheimer's Association International Conference on Alzheimer's Disease (ICAD) in Chicago. The presentations are highlighted below.

Dimebon Preserves All Key Functions in Alzheimer's Patients for 18 Months in Open-Label Extension of First Pivotal Trial

New data from a six-month open-label extension of the 12-month placebo-controlled study of Dimebon in patients with mild-to-moderate Alzheimer's disease demonstrated that Dimebon continued to improve the clinical course of the disease. After 18 months of treatment, Dimebon preserved function in patients at or near their original levels upon entering the trial across all key aspects of Alzheimer's disease, specifically memory and thinking, behavior, activities of daily living and overall function. These results are noteworthy as untreated Alzheimer's patients progressively deteriorate over time in these areas. Dimebon remained well tolerated throughout the 18-month treatment period.

The open-label extension data were presented in a poster session by Jeffrey Cummings, M.D., Director of the Mary S. Easton Center for Alzheimer's Disease Research at UCLA. "To my knowledge, no other approved or investigational treatment has stabilized function across all facets of Alzheimer's disease for this length of time," said Dr. Cummings. "These data suggest that Dimebon may provide long-term benefits to Alzheimer's patients and provide further support for its potential as a promising therapeutic to treat this devastating disease."

Patients originally on placebo for 12 months who were then crossed over to Dimebon in the open-label extension phase stabilized across all key measures tested. Since these patients had declined over the previous 12 months while on placebo, they generally stabilized at a lower level of function than those treated with Dimebon for the full 18 months, suggesting a benefit of earlier treatment.

Dimebon Benefits Both Mild and Moderate Patients in 12-month Subgroup Analyses

New data from subgroup analyses by disease severity of the Dimebon double-blind placebo-controlled trial showed that Dimebon benefited both mild and moderate patients. In both mild and moderate patients, Dimebon treatment resulted in significant benefit on the study's primary endpoint, the Alzheimer's Disease Assessment Scale–cognition subscale, or ADAS-cog. The benefit in the moderate subpopulation was particularly robust, with a 9.7 point drug-placebo difference on the ADAS-cog ($p < 0.0001$) after 12 months of treatment.

The subgroup analyses were presented in a separate poster presentation at ICAD 2008 by Rachele Doody, M.D., Ph.D., the Effie Marie Cain Chair in Alzheimer's Disease Research at the Alzheimer's Disease and Memory Disorders Center, Baylor College of Medicine in Houston. "A nearly 10-point improvement over placebo in moderate patients on the ADAS-cog, a well-validated cognition scale in Alzheimer's disease, is unquestionably of clinical significance, especially in light of a clinical effect seen on the clinician's assessment of global function," said Dr. Doody. "If the results we saw for both the mild and moderate patients can be replicated, I believe that Dimebon will be an important advance in the treatment of Alzheimer's disease, regardless of stage."

Dimebon's Novel Mechanism of Action

In a podium presentation at ICAD 2008, Medivation presented new data on Dimebon's novel mitochondrial mechanism of action. Mitochondria generate energy for cells and play important roles in mediating cell function and survival. Mitochondrial dysfunction has been linked in the published literature to both Alzheimer's and Huntington's diseases. Preclinical data presented showed that Dimebon improves mitochondrial function in the setting of cellular stress with very high potency. For example, Dimebon treatment improved mitochondrial function and increased the number of surviving cells after treatment with a cell toxin known as ionomycin in a dose-dependent fashion. The effect of Dimebon to improve mitochondrial dysfunction has been confirmed in the independent laboratory of Maria Ankarcrona, Ph.D., Associate Professor at the Karolinska Institutet in

Sweden.

"All of the approved Alzheimer's disease drugs operate by one of two mechanisms – cholinesterase inhibition or NMDA-receptor antagonism," noted Bengt Winblad, M.D., Ph.D., Head of the Karolinska Institutet's Alzheimer's Disease Research Center. "The body of preclinical and clinical data generated thus far convinces me that Dimebon is exerting its effects through a different mechanism. The data presented today support the hypothesis that Dimebon improves mitochondrial dysfunction. This is a novel mechanism that may, in part, explain the clinical benefits seen in Alzheimer's patients treated with Dimebon."

About the Pivotal Study Dimebon's first pivotal Alzheimer's trial was a randomized, double-blind, placebo-controlled study of 183 patients with mild to moderate Alzheimer's disease. In this study, patients treated with Dimebon experienced statistically significant improvements compared to placebo in all the key aspects of the disease: memory and thinking, activities of daily living, behavior and overall function – after both six months and a full year of treatment. Dimebon's benefit over placebo continued to increase throughout the 12-month treatment period. At the end of 12 months, Dimebon-treated patients preserved their starting level of function on each measure of Alzheimer's disease. Results of the pivotal study were published in the July 19, 2008 issue of *The Lancet*.

Earlier this year, the U.S. Food and Drug Administration (FDA) informed Medivation that this study can be used as one of the pivotal studies required to support the approval of Dimebon to treat mild-to-moderate Alzheimer's disease, as long as a significant proportion of the sites in the confirmatory Phase 3 trial are located in the United States. The Company recently began a confirmatory pivotal Phase 3 trial of Dimebon in Alzheimer's disease known as the CONNECTION study. Patients and caregivers can learn more about the study by visiting www.connectionstudy.com or by calling 1-877-888-6386.

About the Open-Label Extension All patients who completed 12-months of dosing in the first pivotal trial were eligible to enroll in an open-label extension. All participants in the open-label extension received Dimebon, including patients who had previously received placebo during the prior 12 months of the trial. Because there was no placebo-control in the open-label extension, direct comparisons versus placebo cannot be made.

About Dimebon Dimebon is an orally available small molecule that has been shown to inhibit brain cell death in preclinical models relevant to Alzheimer's and Huntington's diseases, making it a potential treatment for these and other neurodegenerative diseases. Preclinical data generated to date suggest that Dimebon operates through a novel mitochondrial mechanism of action.

On July 7, 2008, Medivation announced positive safety and efficacy results from its Phase 2 trial of Dimebon for the treatment of Huntington's disease, which was conducted in collaboration with the Huntington Study Group. The study met its primary endpoint of safety and tolerability; in addition, Dimebon showed statistically significant benefit versus placebo in cognition as measured by the Mini-Mental State Examination, a secondary endpoint in the study. Huntington's disease is a progressive neurodegenerative disease characterized by the gradual development of involuntary muscle movement, progressive deterioration of cognitive processes and memory and severe behavioral disturbances. There are currently no approved drugs in the United States to treat this uniformly fatal genetic disorder.

About Medivation Medivation, Inc. is a biopharmaceutical company focused on the rapid development of novel small molecule drugs to treat serious diseases for which there are limited treatment options. Medivation aims to transform the treatment of these diseases and offer hope to critically ill patients and their caregivers. The Company's current clinical development program includes a pivotal and confirmatory Phase 3 trial of Dimebon in Alzheimer's disease and a Phase 1-2 clinical trial of MDV3100 in patients with castration-resistant (also known as hormone-refractory) prostate cancer. Medivation recently announced that it plans to continue further development of Dimebon in patients with mild-to-moderate Huntington's disease based on the positive results seen in its Phase 2 trial. For more information, please visit us at www.medivation.com.

This press release contains forward-looking statements, including statements regarding future clinical development plans, which are made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. Forward-looking statements involve risks and uncertainties that could cause actual results to differ significantly from those projected. You are cautioned not to place undue reliance on the forward-looking statements, which speak only as of the date of this release. None of the Company's product candidates has been approved for sale, significant additional animal and human testing is required in order to seek marketing approval for any of its product candidates, and Medivation cannot assure you that marketing approval can be obtained for any of its product candidates. Furthermore, as is typically the case at this stage of the regulatory review process, the FDA has not yet performed an in-depth review of Medivation's preclinical and clinical data, so its views remain subject to change. Medivation's filings with the Securities and Exchange Commission, including its current report on Form 8-K filed on June 23, 2008, include information about additional factors that could affect the Company's financial and operating results.