COMPANY CONTACT: **Vion Pharmaceuticals, Inc.**
Alan Kessman, Chief Executive Officer
Howard B. Johnson, President & CFO
(203) 498-4210 phone

Vion Presents Preclinical Data on Cloretazine® (VNP40101M), a Hypoxia-Selective Compound (VNP40541) and Hydrazones at AACR Meeting

NEW HAVEN, CT, April 4, 2006 - VION PHARMACEUTICALS, INC. (NASDAQ NATIONAL MARKET: VION) announced today that preclinical data of its lead anticancer agent Cloretazine® (VNP40101M), its preclinical anticancer agent VNP40541 (formerly KS119W), and hydrazone compounds, were presented in five poster sessions at the 97th Annual Meeting of the American Association of Cancer Research (AACR) in Washington, D.C. on April 2nd-4th.

Cloretazine® (VNP40101M) is a novel alkylating agent which is being evaluated in a pivotal Phase III trial in combination with cytarabine (Ara-C) for the treatment of relapsed acute myelogenous leukemia (AML). Vion also recently announced that it plans to conduct a pivotal Phase II trial in patients with previously untreated elderly *de novo* poor-risk AML. This additional pivotal trial is expected to commence in the second quarter of this year.

Abstract 4729 describes two experiments, both conducted by Vion, which examine the anti-tumor efficacy of Cloretazine® (VNP40101M) alone and in combination with cytarabine (Ara-C) and fludarabine (Ara-A) in murine tumor models. In one experiment, the combination therapy of 10 mg/kg of Cloretazine® (VNP40101M) plus 50 mg/kg Ara-C achieved 100% survival by day 68 post-implantation in CDF1 or BDF1 mice intraperitoneally implanted with the L1210 murine leukemia, compared to 90% for Cloretazine® (VNP40101M) monotherapy and 0% for Ara-C monotherapy. In a separate experiment of 10 mg/kg Cloretazine® (VNP40101M) and five doses of 70 mg/kg Ara-A every other day, at day 65 post-implantation, the combination therapy yielded a 90% survival rate, compared to 40% for Cloretazine® alone and 0% for Ara-A alone.

Dr. Ivan King, Vice President, Research & Development, said, “In preclinical models, Cloretazine® (VNP40101M), either alone or in combination with Ara-C or Ara-A, significantly reduced tumors and improved survival, while limiting short-term toxicity. This data supports our clinical effort, where Cloretazine® (VNP40101M) is being evaluated in AML as a single agent and in combination with Ara-C.”

VNP40541 (formerly KS119W) is a hypoxia-selective compound which releases the same active agent as Cloretazine® (VNP40101M). In experiments conducted by Vion described in abstracts 4717 and 4736, VNP40541 was evaluated both as a single agent and in combination with conventional anticancer agents such as cyclophosphamide, gemcitabine, paclitaxel, etoposide and cisplatin in murine tumor models. Two enantiomers (R-KS119W and S-KS119W) were evaluated and R-KS119W (VNP40541) was chosen for further development based on a better toxicity profile. Both enantiomers demonstrated tumor inhibition ranging from 52-85% in various tumor models. VNP40541 was also evaluated in combination regimens in order to complement its activity against hypoxic tumor cells with agents that preferentially attack the well-oxygenated, actively growing cell populations within tumors. All the different combination therapies were more efficacious than VNP40541 or any other agent alone. Tumor growth inhibition ranged from 55% to 98%. No unexpected severe toxicities were observed during the studies.

Dr. King stated, “This new preclinical data for VNP40541 continues to demonstrate that the compound is active in preclinical tumor models. We look forward to filing an Investigational New Drug (IND) Application for VNP40541 in the first half of 2006. Clinical trials of this compound should commence this year.”

Vion is also evaluating certain hydrazone compounds in preclinical studies. In 2005, the Company entered into an exclusive license for these anticancer compounds with several Austrian inventors and an Austrian development bank. In experiments conducted by Vion in abstract 4732, 13 analogues from this class were evaluated in *in vitro* and *in vivo* studies. The inhibitory effects on the growth of solid tumors varied from compound to compound, ranging from 40% to 80%. In order to understand the mechanism of action, three compounds were investigated more extensively in cellular and biochemical assays. Although the target has not been fully identified, it is possible that these compounds inhibit cell proliferation by blocking cell growth in the G0/G1 growth phase and leading to cell death by apoptosis. Results demonstrated that the heterocyclic hydrazones also caused a reduction in CDK4 expression. Further confirmation of the activity of certain of these compounds is reported in abstract 566 from experiments conducted by Austrian researchers, Vion and researchers at the Baylor College of Medicine.

Dr. King commented, “We continue to evaluate the hydrazones in preclinical studies. Our goal is to identify a lead compound from this series and to complete the studies necessary to file an IND.”

Vion Pharmaceuticals, Inc. is developing cancer therapeutics. Vion has two agents in clinical trials: Cloretazine® (VNP40101M), a unique alkylating agent, is being evaluated in a Phase III trial in combination with cytarabine in relapsed acute myelogenous leukemia. Trials of Cloretazine® (VNP40101M) as a single agent in previously untreated elderly acute myelogenous leukemia and high-risk myelodysplastic syndrome, adult and pediatric brain tumors, small cell lung cancer and chronic lymphocytic leukemia, and in combination with temozolomide in hematologic malignancies, are also underway. Triapine®, a potent inhibitor of a key step in DNA synthesis, is being evaluated in trials sponsored by the National Cancer Institute. In preclinical studies, Vion is also evaluating VNP40541, a hypoxia-selective compound, and hydrazone compounds. The Company also is seeking development partners for TAPET®, its modified *Salmonella* vector used to deliver anticancer agents directly to tumors. For additional information on Vion and its product development programs, visit the Company’s Internet web site at www.vionpharm.com.

This news release contains forward-looking statements. Such statements are subject to certain risk factors which may cause Vion's plans to differ or results to vary from those expected, including Vion’s ability to secure external sources of funding to continue its operations, the inability to access capital and funding on favorable terms, continued operating losses and the

inability to continue operations as a result, its dependence on regulatory approval for its products, delayed or unfavorable results of drug trials, the possibility that favorable results of earlier clinical trials are not predictive of safety and efficacy results in later clinical trials, the need for additional research and testing, and a variety of other risks set forth from time to time in Vion's filings with the Securities and Exchange Commission, including but not limited to the risks discussed in Vion's Annual Report on Form 10-K for the year ended December 31, 2005. Except in special circumstances in which a duty to update arises under law when prior disclosure becomes materially misleading in light of subsequent events, Vion does not intend to update any of these forward-looking statements to reflect events or circumstances after the date hereof or to reflect the occurrence of unanticipated events.

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