AFFIDAVIT OF NATHANIEL E FRANK-WHITE

1. I am a Records Request Processor at the Internet Archive, located in San Francisco, California. I make this declaration of my own personal knowledge.

2. The Internet Archive is a website that provides access to a digital library of Internet sites and other cultural artifacts in digital form. Like a paper library, we provide free access to researchers, historians, scholars, and the general public. The Internet Archive has partnered with and receives support from various institutions, including the Library of Congress.

3. The Internet Archive has created a service known as the Wayback Machine. The Wayback Machine makes it possible to browse more than 450 billion pages stored in the Internet Archive's web archive. Visitors to the Wayback Machine can search archives by URL (i.e., a website address). If archived records for a URL are available, the visitor will be presented with a display of available dates. The visitor may select one of those dates, and begin browsing an archived version of the Web. Links on archived files in the Wayback Machine point to other archived files (whether HTML pages or other file types), if any are found for the URL indicated by a given link. For instance, the Wayback Machine is designed such that when a visitor clicks on a hyperlink on an archived page that points to another URL, the visitor will be served the archived file found for the hyperlink's URL with the closest available date to the initial file containing the hyperlink.

4. The archived data made viewable and browsable by the Wayback Machine is obtained by use of web archiving software that automatically stores copies of files available via the Internet, each file preserved as it existed at a particular point in time.

5. The Internet Archive assigns a URL on its site to the archived files in the format http://web.archive.org/web/[Year in yyyy][Month in mm][Day in dd][Time code in hh:mm:ss]/[Archived URL] aka an "extended URL". Thus, the extended URL http://web.archive.org/web/19970126045828/http://www.archive.org/ would be the URL for the record of the Internet Archive home page HTML file (http://www.archive.org/) archived on January 26, 1997 at 4:58 a.m. and 28 seconds (1997/01/26 at 04:58:28). The date indicated by an extended URL applies to a preserved instance of a file for a given URL, but not necessarily to any other files linked therein. Thus, in the case of a page constituted by a primary HTML file and other separate files (e.g., files with images, audio, multimedia, design elements, or other embedded content) linked within that primary HTML file, the primary HTML file and the other files will each have their own respective extended URLs and may not have been archived on the same dates.

6. Attached hereto as Exhibit A are true and accurate copies of screenshots of the Internet Archive's records of the archived files for the URLs and the dates specified in the attached coversheet of each printout.

7. I declare under penalty of perjury that the foregoing is true and correct.

DATE: 04/01/2022

Nathaniel E Frank-White

EXHIBIT A

https://web.archive.org/web/20070709161226/http:/investor.pharmion.com/phoenix.zhtml?c=142045&p=irol-newsArticle&ID=1010376&highlight=

About Pharmion
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Clinical Data Presented on Pilot Pharmacokinetic Study of Oral Azacitidine

Bioavailability Data from First Oral Demethylating Agent in Clinical Trials

Presented at the American Society of Clinical Oncology 43rd Annual Meeting

CHICAGO, June 2 /PRNewswire-FirstCall/ -- Pharmion Corporation (Nasdaq: PHRM) today reported data from a pilot study that demonstrated the bioavailability of the Company's oral dosage formulation of Azacitidine. These data were presented at the 43rd Annual Meeting of the American Society of Clinical Oncology (ASCO) in Chicago. (June 1-5, 2007). Pharmion's epigenetic anti-cancer products, which include Vidaza(R) (azacitidine for injection), MCGD0103 and oral Azacitidine, are being investigated in the treatment of hematologic malignancies and solid tumors and are the subject of poster and oral presentations at the Meeting.

An oral dosage formulation of Azacitidine would provide a more desirable and convenient route of administration for patients and clinical staff and eliminate injection-site reactions. In addition, it would enable the evaluation of a low-dose regimen that could maximize demethylation and gene re-expression, as well as the evaluation of long-term or maintenance therapy.

The study was designed to assess the safety, tolerability, and pharmacokinetics of escalating single doses of orally administered Azacitidine in patients with Myelodysplastic Syndromes (MDS), acute myeloid leukemia (AML) or other solid tumors. A total of four patients were enrolled and received study drug. One patient received a 60 mg dose and three patients received a single 80 mg dose. In each case, Azacitidine was well-tolerated and quantifiable in plasma. Following an 80mg dose the bioavailability of the oral formulation of Azacitidine was 18 percent relative to subcutaneous (SC) administered Vidaza. Having established that acceptable plasma concentrations of azacitidine can be achieved with single oral dosing, this pilot study has concluded. A multi-dose escalating study of oral azacitidine is now underway.

"Oral azacitidine represents an extremely exciting opportunity," said Dr. Guillermo Garcia-Manero, Associate Professor of Medicine and Chief, Section of Myelodysplastic Syndromes, Department of Leukemia, M.D. Anderson Cancer Center. "The potential for a chronic oral demethylating agent will not only apply to MDS but to many other tumor types as well. This is a very important step forward for epigenetic therapies."

Data Presented Saturday, June 2

Phase I/II study of a novel oral isotype-selective histone deacetylase (HDAC) inhibitor MGCD0103 in combination with azacitidine in patients (pts) with high-risk Myelodysplastic Syndrome (MDS) or Acute Myelogenous Leukemia (AML) - G. Garcia-Manero, MD, MD Anderson Cancer Center; Abstract #7062; June 2, 2007; 8:00-12:00 pm; McCormick Convention Center, S Hall A2

The combination of 5-azacytidine and valproic acid is safe and active in advanced solid tumors - A.O. Soriano, MD Anderson Cancer Center; Abstract #3547; June 2, 2007; 2:00-6:00pm; McCormick Place Convention Center, S102a

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About Vidaza

Vidaza was the first drug approved for the treatment of all five subtypes of myelodysplastic syndromes (MDS), including refractory anemia (RA), refractory anemia with ringed sideroblasts (RARS) (if accompanied by neutropenia or thrombocytopenia or requiring transfusions), refractory anemia with excess blasts (RAEB), refractory anemia with excess blasts in transformation (RAEB-T), and chronic myelomonocytic leukemia (CMMoL).

Vidaza is believed to exert its antineoplastic effects by causing hypomethylation of DNA and direct cytotoxicity on abnormal hematopoietic cells in the bone marrow. The concentration of azacitidine required for maximum inhibition of DNA methylation in vitro does not cause major suppression of DNA synthesis. Hypomethylation may restore normal function to genes that are critical for differentiation and proliferation. The cytotoxic effects of azacitidine cause the death of rapidly dividing cells, including cancer cells that are no longer responsive to normal growth control mechanisms. Non- proliferating cells are relatively insensitive to Vidaza.

Important Safety Information

Vidaza is contraindicated in patients with a known hypersensitivity to azacitidine or mannitol and in patients with advanced malignant hepatic tumors. In clinical studies, the most commonly occurring adverse reactions were nausea (70.5%), anemia (69.5%), thrombocytopenia (65.5%), vomiting (54.1%), pyrexia (51.8%), leukopenia (48.2%), diarrhea (36.4%), fatigue (35.9%), injection site erythema (35.0%), constipation (33.6%), neutropenia (32.3%) and ecchymosis (30.5%). Other adverse reactions included dizziness (18.6%), chest pain (16.4%), febrile neutropenia(16.4%), myalgia (15.9%), injection site reaction (13.6%), aggravated

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