Enzon Presents Data at 2010 AACR Meeting

Novel pipeline continues to advance and show promise

BRIDGEWATER, N.J., Apr 21, 2010 (BUSINESS WIRE) -- Enzon Pharmaceuticals, Inc. (Nasdaq: ENZN) presented data from its pipeline programs at the 2010 AACR (American Association for Cancer Research) annual meeting in Washington, DC.

"We are pleased to present new data from our novel pipeline programs. Our Customized PEG linkers continue to improve the delivery of cytotoxic molecules," said Dr. Ivan Horak, President of Research and Development and Chief Scientific Officer of Enzon. "Additionally, our novel locked nucleic acid (LNA)-based mRNA antagonists continue to show promise for inhibition of tumor growth in a variety of cancers."

The Posters and Abstracts that were presented included:

Customized PEG Linkers Improve the Pharmaceutical Properties of Cytotoxic Small Molecules (Poster 2645)

A series of releasable customized PEG linkers have been developed to improve the delivery of cytotoxic molecules. PEG-conjugates were synthesized with improved solubility and in general, increased the exposure time of the parent molecule.

- PEG-SN38 showed markedly improved solubility leading to significantly enhanced therapeutic efficacy in various xenograft models (including tumors refractory to CPT-11). These encouraging results led to the clinical evaluation of PEG-SN38 (currently in Phase II program).
- The releasable PEG-BE linker system, used in PEG-Daunorubicin conjugate, is an excellent example of releasable PEG-linker Technology whereby small molecules can be released in controlled fashion.
- In case of PEG-Ara-C, optimal pharmacokinetic properties are achieved by applying releasable Customized Linker Technology(TM). Certain PEG-Ara-C conjugates showed remarkable activity against solid tumors as well as ascites model consistent with improved bioavailability of native Ara-C.

LNA Antagonists (GIL2, Beta-Catenin, Androgen Receptor (AR))

The mRNA antagonists are being developed by Enzon under a license with Santaris Pharma A/S.

Targeting the hedgehog pathway by LNA (locked nucleic acid) oligonucleotide based-GLI2 RNA antagonists, EZN-4482 and EZN-4496, *in vitro* and *in vivo* (Abstract 600)

The Hedgehog (Hh) signaling pathway mediates cell growth and differentiation in the embryonic development and in some adult cells. Aberrant activation of signaling has been described as causal factors in a wide variety of human cancers as well as chemotherapy resistance. EZN-4482 and EZN-4496 are two LNA-based GLI2 mRNA antagonists. Conclusions from the data reported included:

- EZN-4482 and EZN-4496 were demonstrated to silence GLI2 expression with or without transfection in vitro.
- The LNA based GLI2 antagonists potently and specifically inhibited GLI2 mRNA expression and tumor growth in two tumor models.
- Specific silence of GLI2 expression EZN-4482 in vivo can lead to inactivation of Hh signaling pathway as indicated by down-modulation of GLI1 and Ptch1 expression
- The efficacy observed with EZN-4482 in vivo may be due, in part, to silence of in mouse stromal GLI2 mRNA.
- GLI2 RNA antagonist may be an effective therapy to treat a broad spectrum of cancers including ones that fail treatment with a small molecule inhibitor.

***In vitro* and *in vivo* characterization of two novel B-catenin RNA antagonists, EZN-3889 and EZN-3892 (Abstract 601)**

EZN-3889 and EZN-3892 are able to potently inhibit B-catenin mRNA and protein *in vitro* and inhibit the growth of numerous cancer cell lines. Inhibition of B catenin using these LNAs results in the inhibition of spheroid formation in the SW480 B-cell line. Both LNAs are well tolerated *in vivo* with >90% knockdown of B-catenin mRNA B observed in mouse liver. Excitingly, treatment of

mice bearing Colon-EZN tumors intravenously with EZN-3892 results in significant inhibition of tumor growth. A wide therapeutic window exits for EZN-3892 making the therapeutic development of these LNAs highly desirable.

Silencing of the androgen receptor (AR) with a novel mRNA antisense oligonucleotide causes antitumor effects in xenograft models of prostate cancer (Abstract 602)

The AR is a ligand-activated transcriptional factor that plays an important role in prostate cancer. While androgen-deprivation therapies typically inhibit tumor growth, tumor recurrence due to the castration resistant form of the disease frequently occurs and leads to mortality.

- AR antagonist, EZN-4176, inhibits the growth of CWR22 tumors and down-regulates mRNA of AR and its regulated genes such as PSA and TMPRSS2.
- The antagonist also down-regulates AR protein level in CWR22 tumors.
- The compound distributes to multiple organs including prostate and tumors.
- Results demonstrate the down regulation of mRNA and AR protein level in C4-2B tumors resulting in apoptosis.
- EZN-4176 inhibits transcription activity of AR in C4-2B tumors and inhibits growth of the C4-2B tumors.

The abstracts and posters can be found on the Company's website at www.enzon.com.

About Enzon

Enzon Pharmaceuticals, Inc. is a biopharmaceutical company dedicated to the discovery and development of innovative medicines for patients with cancer. Enzon's drug development programs utilize several cutting-edge approaches, including its industry-leading PEGylation technology platform, Customized Linker technology(TM) and mRNA antagonists using the Locked Nucleic Acid(TM) (LNA) technology. Enzon's receives a royalty revenue stream from licensing partnerships for other products developed using the proprietary PEGylation technology. Further information about Enzon and this press release can be found on the Company's web site at www.enzon.com.

Forward Looking Statements

There are forward-looking statements contained herein, which can be identified by the use of forward-looking terminology such as the words "believes," "expects," "may," "will," "should," "potential," "anticipates," "plans," or "intends" and similar expressions. Such forward-looking statements involve known and unknown risks, uncertainties and other factors that may cause actual results, events or developments to be materially different from the future results, events or developments indicated in such forward-looking statements. Such factors include, but are not limited to the timing, success and cost of clinical studies; the ability to obtain regulatory approval of products, market acceptance of, and continuing demand for, Enzon's products and the impact of competitive products and pricing. A more detailed discussion of these and other factors that could affect results is contained in our filings with the U.S. Securities and Exchange Commission, including our annual report on Form 10-K for the period ended December 31, 2009. These factors should be considered carefully and readers are cautioned not to place undue reliance on such forward-looking statements. No assurance can be given that the future results covered by the forward-looking statements will be achieved. All information in this press release is as of the date of this press release and Enzon does not intend to update this information.

SOURCE: Enzon Pharmaceuticals, Inc.

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