FORM 6-K SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

Report of Foreign Private Issuer

Pursuant to rule 13a-16 or 15d-16 of the Securities Exchange Act of 1934 for the month of September 2008

<u>Compugen Ltd.</u> (Translation of registrant's name in English)

72 Pinchas Rosen Street, Tel-Aviv 69512, Israel (Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover Form 20-F or Form 40-F.

Form 20-F <u>X</u> Form 40-F ____

On September 16, 2008 Compugen Ltd. (the "Registrant") issued a Press Release, filed as Exhibit 1 to this Report on Form 6-K, which is hereby incorporated by reference herein.

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

Compugen Ltd. (Registrant)

By: /s/ Dikla Czaczkes Axselbard Title: Chief Financial Officer Date: September 16, 2008



Compugen Discovers Peptide Drug Candidate for Treatment of Solid Tumor Cancers and Announces Positive In Vivo Results

Second Discovery of Compounds Enhancing Responsiveness to Frequently Used Cancer Drugs

Tel Aviv, Israel, September 16, 2008 – Compugen Ltd. (NASDAQ: CGEN) today announced that its proprietary DAC Blockers Discovery Platform has led to the discovery of CGEN-25008, a novel peptide antagonist of the Clusterin protein. Compugen also announced that recently analyzed *in vitro* and initial *in vivo* results from cell-based assays and a lung cancer mouse model indicate that CGEN-25008 reduces the growth rate of several cancer cell lines and significantly enhances the anti-cancer activity of Taxol[™], a frequently used cancer chemotherapeutic drug.

Compugen's DAC Blockers Platform was designed to predict peptides that can block proteins of interest from achieving certain disease-associated three-dimensional conformations. This discovery platform, announced in March 2008, is one of nine proprietary discovery platforms, to date, that have been developed and validated by Compugen for predictive discovery of drug and diagnostic product candidates.

Alex Kotzer, president and CEO of Compugen said, "Following our long term investment in establishing unique research capabilities, we are extremely pleased by our growing inventory of validated field-focused discovery platforms and the resulting accelerating pace of product candidate discoveries. Not surprisingly, these achievements are leading to substantially increased interest by leading biopharma and diagnostic companies to learn more about our unique capabilities and to explore potential collaborations and licensing agreements."

Today's announcement follows the July 2008 announcement that *in vitro* and *in vivo* validation studies have shown that co-administration of CGEN-50001 significantly increases the effect of Tamoxifen, which like Taxol, is a frequently used drug for cancer therapy. However, unlike the novel peptide CGEN-25008, CGEN-50001 is an existing drug used in Europe for central nervous system (CNS) disorders. The potential use of this known drug in cancer therapeutics was initially predicted *in silico* (by computer) by Compugen's New Indications Platform, designed to find new therapeutic uses for existing drugs. Patents have been filed by the company for both CGEN-25008 and the use of CGEN-50001 in cancer therapy.

The novel peptide CGEN-25008 being disclosed today has been shown to slow the growth of human non-small cell lung cancer cells and other malignant cell lines including breast, prostate, colon and melanoma cancers when tested directly on such cancer cells. In addition, administration of CGEN-25008 in remarkably low doses (1 nano Molar) in combination with Taxol was shown to increase the sensitivity of the cancer cells to Taxol, allowing a ten-fold reduction in the concentration of Taxol while maintaining the same anti-cancer effect. In addition a combination of a higher dose of CGEN-25008 (80 nano Molar) with Taxol was shown to result in a 40 percent increase over the maximal anti-cancer effect achieved by Taxol alone.

Yossi Cohen, M.D., vice president of Research and Development at Compugen said, "In a mouse model of lung cancer, the tumor size decreased significantly more when Taxol was given in combination with

CGEN-25008, as compared to when mice were treated with Taxol alone. This may ultimately translate to lower required doses of chemotherapy, with reduced side effects, while maintaining its comparable therapeutic benefits or potentially strengthening the maximal anti-cancer effects achieved."

About Clusterin

Clusterin is a stress-associated cytoprotective extracellular chaperone protein that is up regulated by various apoptotic triggers and confers treatment resistance in various cancers. Among the stress signals that cause up-regulation of Clusterin are chemotherapy, irradiation and hormone ablation. The function of Clusterin in the acquisition of chemotherapy resistance is currently an area of interest in both academic research and within the biopharma industry. A Clusterin-inhibiting antisense oligonucleotide is currently in clinical development by another company as a potential treatment for increasing the susceptibility of solid tumors to conventional cancer therapies.

About the DAC Blockers Discovery Platform

The Blockers of Disease-Associated Conformation (DAC Blockers) platform is a discovery platform designed for the prediction and selection of peptides that block proteins from adopting their disease-associated conformations. This is accomplished through the use of a series of proprietary algorithms to identify segments in proteins of interest that, if introduced as synthetic peptides, would prevent the proteins from adopting disease-associated conformations and related activities and thus could have therapeutic benefits. In addition, a key capability of the platform is that the prediction and selection capability enables proteome-wide searches for such peptides in proteins of interest within human, viral and bacterial proteomes.

About Compugen

Compugen's mission is to be the world leader in the discovery and licensing of product candidates to the drug and diagnostic industries under milestone and revenue sharing agreements. The Company's increasing inventory of powerful and proprietary discovery platforms enable unprecedented computer-based, predictive biological discoveries, field after field, of numerous therapeutic and diagnostic product candidates. The discovery platforms are based on the Company's decade-long focus on the predictive understanding of important biological phenomena at the molecular level. Compugen's current collaborations include Biosite, Medarex, Inc., Merck & Co., Inc., Ortho-Clinical Diagnostics (a Johnson & Johnson company), Roche, Siemens Healthcare Diagnostics, Inc., and Teva Pharmaceutical Industries. In 2002, Compugen established an affiliate, Evogene Ltd. (TASE: EVGN.TA), to utilize the Company's in-silico (computerized) predictive discovery capabilities in the agricultural biotechnology field. For additional information, please visit Compugen's corporate Website at www.evogene.com.

This press release may contain "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. These statements include words such as "may", "expects", "anticipates", "believes", and "intends", and describe opinions about future events. These forward-looking statements involve known and unknown risks and uncertainties that may cause the actual results, performance or achievements of Compugen to be materially different from any future results, performance or achievements expressed or implied by such forward-looking statements. Some of these risks are: changes in relationships with collaborators; the impact of competitive products and technological changes; risks relating to the development of new products; and the ability to implement technological improvements. These and other factors are identified and more fully explained under the heading "Risk Factors" in Compugen's annual reports filed with the Securities and Exchange Commission.

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