

**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 January 2007

Compugen Ltd.  
(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  X       Form 40-F    

On January 30, 2007 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/ Nurit Benjamini  
Title: Chief Financial Officer  
Date: January 30, 2007

## Exhibit 1



### **Compugen to Present Experimental Data of its MCP-1 Antagonist Candidate at GTCBio's Cytokines and Inflammation Conference**

Tel Aviv, Israel – January 29, 2007 - Compugen Ltd. (NASDAQ: [CGEN](#)) announced today that it will present experimental results for its previously disclosed CGEN-54, an MCP-1 antagonist therapeutic candidate, at GTCBio's 5<sup>th</sup> Cytokines and Inflammation Conference (January 29-30, 2007, Breckenridge, Colorado).

Compugen's CGEN-54 is a truncated variant of MCP-1 that is encoded by a novel splice variant, and the results to be presented at the Conference demonstrate the molecule's ability to inhibit the MCP-1/CCR2 pathway both *in-vitro* and *in-vivo*. In cell culture, CGEN-54 strongly inhibited MCP-1-induced monocytes migration, whereas *in-vivo*, CGEN-54 was effective in reducing macrophage infiltration in a mouse model of peritoneal inflammation.

CGEN-54 was one of three therapeutic candidates for which Compugen announced in late 2006 the discovery and demonstration of functional activity. This molecule, and splice variants of c-Met receptor and ANP hormone, had initially been predicted *in silico* utilizing the Company's first therapeutics discovery engine, based on Compugen's long-term leadership in the field of alternative splicing.

MCP-1 (Monocyte Chemoattractant Protein 1, also named CCL2) belongs to the CC chemokines family and is induced in response to various inflammatory stimuli. Binding of MCP-1 to its cognate receptor, CCR2, leads to recruitment of specialized immune cells into the site of inflammation, resulting in tissue destruction. The inhibition of the MCP-1/CCR2 pathway represents a promising target to effectively modulate disease progression in chronic inflammatory diseases, such as multiple sclerosis.

#### **About Compugen**

Compugen's mission is to be the world leader in the discovery and licensing of product candidates to the drug and diagnostic industry. The Company's powerful discovery engines enable the predictive discovery of numerous potential therapeutics and diagnostic biomarkers. This capability results from the Company's decade-long pioneering efforts in the deeper understanding of important biological phenomena at the molecular level through the incorporation of ideas and methods from mathematics, computer science and physics into biology, chemistry and medicine. To date, Compugen's diagnostic and therapeutic product discovery efforts and its initial discovery engines have focused mainly within the areas of cancer, immune-related and cardiovascular diseases. (should anything be added due to Medarex) The Company's primary commercialization pathway for its therapeutic and diagnostic product candidates is to enter into milestone and revenue sharing out-licensing and joint development agreements with leading companies. Compugen has established an agricultural biotechnology affiliate – Evogene, and a small-molecule drug discovery affiliate – Keddem Bioscience. For additional information, please visit Compugen's corporate Website at [www.cgen.com](http://www.cgen.com).

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