COMPUGEN LTD
Form 6-K
July 19, 2004

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 February 2004

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 July 19th, 2004 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/ Mor Amitai

Title: President & CEO

Date: July 19th, 2004

Exhibit 1

Compugen Announces Discovery of Abundant RNA Editing Sites in the Human Transcriptome

- Findings published in Nature Biotechnology -

Tel Aviv, Israel- July 19, 2004- Compugen Ltd. (Nasdaq: CGEN) announced today the systematic identification of adenosine to inosine (A to I) RNA editing sites in the human transcriptome, increasing the number of known A to I editing sites from approximately 100 to more than 10,000. The discovery is being published in Nature Biotechnology (Levanon et al., electronic publication ahead of print: http://dx.doi.org/10.1038/nbt996).

RNA editing is a type of RNA modification in which small nucleotide changes occur after DNA has been transcribed into RNA. Although it is known that RNA editing is an essential factor for mammalian development and recent evidence has suggested that it may be a fairly common phenomenon, prior to today`s announcement very few RNA editing sites had been discovered, and it was generally believed to be impossible to systematically discover such sites with current experimental and computational procedures and tools.

In A to I RNA editing there is a site-specific conversion of adenosine to inosine. The deficiency or misregulation in this type of editing has been associated with a number of neurological diseases such as amyotrophic lateral sclerosis (ALS or "Lou Gehrig`s disease"), malignant gliomas, epilepsy, and depression. While many of the effects of RNA editing are still unknown, it is believed that it could significantly affect gene expression and regulation, and may provide a mechanism for controlling RNAi.

The results being announced today involve the discovery by Compugen`s scientists of 12,723 A to I editing sites in 1637 genes. Examples of predicted editing sites in selected genes were experimentally validated by Compugen and in collaboration with Dr. Michael Jantsch`s laboratories in the Department of Cell Biology and Genetics, University of Vienna, Austria. The research was done in cooperation with Professor Gidi Rechavi, Head of the Cancer Research Center of Sheba Medical Center, Tel Aviv University, Israel.

"Today`s announcement is further evidence of the uniqueness of Compugen`s predictive approach and the value of incorporating ideas and methods from advanced mathematics, computer science and physics into life science,"

stated Mor Amitai, Ph.D., Compugen`s President and Chief Executive Officer. "Compugen`s pioneering research has already led to better understandings of important biological phenomena such as alternative splicing - the expression of multiple transcripts from individual genes; naturally occurring sense/antisense pairs - the expression from both strands of DNA at the same location on the genome; and now, RNA editing, which is known to be an important natural mechanism for modifying transcripts after expression. These deeper understandings provide Compugen with the opportunity to make novel putative therapeutic and diagnostic discoveries, and form the basis for the continued improvement of our predictive models and iterative research efforts, thereby increasing the probability of future discoveries through these efforts," Dr. Amitai concluded.

About LEADS

The results being announced today regarding Compugen`s ability to systematically quantify the abundance of A to I RNA editing sites and identify their specific locations in the human genome relied heavily on the use of the Company`s proprietary LEADS computational biology platform. The LEADS platform uses advanced proprietary algorithms to create a predictive view of the complete transcriptome of complex organisms. LEADS models important biological phenomena and experimental artifacts, such as alternative splicing, antisense genes, SNPs, chimeric sequences, cross-homology, and multiple polyadenylation. Modeling of these phenomena and accounting for artifacts in the data provide a detailed and accurate representation of gene structures. LEADS has been demonstrated to accelerate the identification and prioritization of drug targets and biological products in Compugen's internal drug discovery efforts and for the Company`s biopharma clients.

About Compugen

Compugen, a genomics-based drug and diagnostic discovery company, increases the probability of successful development of novel drug and diagnostic products by incorporating ideas and methods from mathematics, computer science, and physics into biology, chemistry and medicine. This unique capability results in powerful predictive models and discovery engines, which are both advancing the understanding of important biological phenomena and enabling the discovery of numerous potential therapeutic products and diagnostic markers. The Company has an early stage in-house pipeline consisting of selected therapeutic protein candidates discovered by the Company; additional discoveries have been out-licensed for development. Among Compugen`s customers and partners are leading pharmaceutical and diagnostic companies, such as Abbott Laboratories, Diagnostic Products Corporation, Novartis, and Pfizer. Compugen has established a small-molecule drug discovery subsidiary - Keddem Bioscience, and an agricultural biotechnology subsidiary - Evogene. For additional information, please visit Compugen's corporate Website at www.cgen.com.

This press release contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. These statements include words like "may," "expects," "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; the ability to implement technological

improvements; the ability of Compugen to obtain and retain customers. These and other factors are identified and more fully explained under the heading "Risk Factors" in Compugen's annual reports filed on form 20F that are filed with the Securities and Exchange Commission.

Notes for Editors:

What is RNA editing?

RNA editing is a type of RNA modification in which small nucleotide changes occur after DNA has been transcribed into RNA

DNA is transcribed to RNA in the nucleus of a cell, and is typically spliced to form mature messenger RNA (mRNA). The mature mRNA is transported to the cell`s cytoplasm, where translation to protein occurs. However, it is known that in some cases RNA is edited prior to translation. Single or multiple nucleotides may be edited before the mature mRNA moves into the cytoplasm, leading ultimately to the production of a protein that does not fully reflect the original genetic instructions in the DNA. In A to I RNA editing, there is a site-specific conversion of adenosine to inosine in precursor mRNAs.

The enzymes responsible for A to I editing, the adenosine deaminases acting on RNA (ADARs), specifically recognize partially double-stranded (ds) RNA structures where they modify individual adenosines to inosine depending on the local structure and sequence environment. Since inosine (I) is read as guanosine (G) by the protein translation machinery, A to I editing often leads to protein sequence changes that result in the alteration of protein structure or function. It can also create or destroy pre-mRNA splice signals or lead to alterations in RNA secondary structure. If editing occurs in non-coding sequences, gene regulation may be affected.

The deficiency or misregulation of A to I RNA editing has been implicated in the etiology of neurological diseases, such as epilepsy, amyotrophic lateral sclerosis (ALS), and depression in mammals, and it has been shown that a loss of A to I editing following the genetic inactivation of ADARs in mammals, as well as flies and the worm, results in behavioral or neurological dysfunctions or death at the embryo stage.