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 2012

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, 2012, 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: Ms. Dikla Czaczkes Axselbrad
Title: Chief Financial Officer
Date: January 30, 2012

Compugen Presents Three Product Candidates for Treatment of Autoimmune Diseases at the Immunotherapeutics & Immunomonitoring Conference

Presentation demonstrates the therapeutic effects of CGEN-15001, CGEN-15021 and CGEN-15091 in disease animal models of rheumatoid arthritis and multiple sclerosis

San Diego, California, January 30, 2012 --- Speaking today at the 4th Immunotherapeutics & Immunomonitoring Conference in San Diego, Dr. Galit Rotman, Chief Scientist of Therapeutics at Compugen Ltd. (NASDAQ: CGEN), presented data demonstrating the therapeutic efficacy of CGEN-15001, CGEN-15021 and CGEN-15091 in animal models of multiple sclerosis (MS), and the therapeutic efficacy of CGEN-15001 and CGEN-15021 in animal models of rheumatoid arthritis (RA). CGEN-15001, CGEN-15021 and CGEN-15091 are predicted B7/CD28-like proteins discovered using Compugen's Protein Family Members Discovery Platform.

Dr. Rotman reported that in the collagen induced arthritis model of RA, treatments with either CGEN-15001 or CGEN-15021 in animals with established disease resulted in dramatic amelioration of clinical symptoms. Also, both treatments resulted in reduced damage to the joints, as evidenced by a histological analysis that supports the disease modifying potential of these treatments. In the experimental autoimmune encephalitis mouse model of MS, short term treatments with CGEN-15001, CGEN-15021 or CGEN-15091 all resulted in a long term dramatic improvement of disease symptoms. Specifically for CGEN-15001, results demonstrated inhibition of pathological immune responses and of epitope spreading, which underlie the relapsing remitting nature of the disease. Overall, these results indicate that CGEN-15001 may prevent disease progression by immune tolerance induction, a process whereby the immune system no longer attacks the self-antigens that cause the disease. Modifying such diseases through immune tolerance induction is a promising mode of action that may result in effective drugs for autoimmune diseases.

In her talk, Dr. Rotman presented data demonstrating that the effects of CGEN-15001 include modulating the activity of a sub-group of white blood cells called T helper cells, which are known to provide signals for orchestrating the immune response. CGEN-15001 has been shown to inhibit the pro-inflammatory T helper cells, Th1 and Th17, while at the same time promoting anti-inflammatory Th2 responses, a phenomenon known as Th1/Th2 shift. T cell modulation can be therapeutically beneficial in the treatment of T cell mediated autoimmune diseases such as MS, RA, diabetes type 1, psoriasis and others. These encouraging results were demonstrated both by *in vitro* and *in vivo* based test systems. The research involving CGEN-15001 in MS animal models suggests that it exerts its beneficial therapeutic effect by modulating the immune system through the Th1/Th2 shift, inhibiting epitope spreading and preventing infiltration of reactive immune T cells into the central nervous system."

Dr. Rotman concluded, "An efficient treatment for autoimmune diseases with minimal side effects is a major therapeutic need. Currently, many of the approved drugs act via global immune suppression, which involves multiple, potentially serious side effects and expose the body to opportunistic pathogenic attacks. CGEN-15001, CGEN-15021 and CGEN-15091 offer the opportunity to regulate the immune response in a specific manner potentially providing significant therapeutic benefits with fewer side effects."

Dr. Anat Cohen-Dayag, Compugen's president and CEO added, "The CGEN-15001, CGEN-15021 and CGEN-15091 product candidates are based on three of the nine novel B7/CD28-like proteins predicted and selected *in silico* using our Protein Family Members Discovery Platform. This platform, which utilizes the integration of multiple data sources and algorithms modeling biological phenomena, was designed to predict and select unknown members of protein families of high industry interest, such as the B7/CD28 family. Members of this protein family are important regulators of the immune system and thus can be targets for treatment of autoimmune diseases as well as cancer immunotherapy."

Dr. Cohen-Dayag concluded, "These and other previously disclosed preclinical results for our novel B7/CD28-like proteins, the first protein family upon which we have chosen to focus our Protein Family Members Discovery Platform, highlight the potential of this unique platform, a potential we are only beginning to tap."

About CGEN-15001, CGEN-15021, CGEN-15091 and the B7/CD28 protein family
Members of the B7/CD28 protein family have been intensively studied over the past decade as positive and negative regulators of the immune response. A growing body of evidence indicates that dysfunction of immune regulation contributes to the development of autoimmune diseases.

Positive and negative co-stimulatory pathways play critical roles in immune regulation and are considered potential targets for modulating chronic inflammation in autoimmune diseases. To date, one soluble recombinant fusion protein that selectively blocks the co-stimulatory signal mediated by the prototype B7/CD28 pathway has been cleared for marketing in the U.S. for the treatment of moderate to severe rheumatoid arthritis, and is in clinical trials for other autoimmune indications. In addition, a number of clinical and preclinical studies for therapeutic agents targeting these protein families are underway at various companies.

CGEN-15001 is a novel protein drug candidate consisting of the extracellular region of CGEN-15001T, a previously unknown membrane protein predicted by Compugen to be a member of the B7/CD28 family, fused to a mouse antibody Fc domain. CGEN-15001T was discovered using Compugen's Protein Family Members Discovery Platform, and was predicted to have an immunomodulatory function based on its bioinformatic characteristics. To date, utilization of this predictive platform by Compugen has resulted in the discovery of nine proteins predicted to serve as novel members of this family, including CGEN-15001T and the two proteins that are the basis of CGEN-15021 and CGEN-15091.

CGEN-15021 and CGEN-15091 are also soluble fusion proteins, each combining the extracellular domain of one of the new B7/CD28-like proteins discovered by Compugen, and an Fc antibody fragment. The therapeutic potential of CGEN-15021 was recently validated in animal disease models of both multiple sclerosis and rheumatoid arthritis, and that of CGEN-15091 in an animal disease model of multiple sclerosis. In each of these disease models, the Compugen fusion proteins demonstrated dramatic therapeutic effects in ameliorating disease symptoms. In addition, in earlier *in vitro* experiments, CGEN-15021 and CGEN-15091 exhibited inhibition of T cell activation, confirming their predicted role in the modulation of the immune system.

About Multiple Sclerosis
Multiple sclerosis (MS) is an autoimmune disease that affects the central nervous system, and is caused by damage to the myelin sheath, the protective covering that surrounds nerve cells. When this nerve covering is damaged, nerve impulses are slowed down or stopped. The nerve damage is caused by inflammation, which occurs when the body's own immune cells attack the nervous system. In MS, the immune response is primarily mediated by T cells, that gain entry into the brain via the blood–brain barrier, but the trigger to this inflammatory process remains unknown. It is common for the disease to return (relapse). However, the disease may continue to get worse without periods of remission. Currently there is no cure for MS, but several drugs are used for controlling and managing the disease.

About Rheumatoid Arthritis
Rheumatoid arthritis (RA) is a chronic, systemic inflammatory disorder that affects about 1% of the world population, and is three times more prevalent in women compared with men. The disease affects mainly joints, but may also affect other tissues and organs. Although the cause of rheumatoid arthritis is unknown, autoimmunity plays a pivotal role in both its chronicity and progression, and RA is considered a systemic autoimmune disease. RA can be a disabling and painful condition, which can lead to substantial loss of functioning and mobility if not adequately treated. Currently available pharmacological treatments include anti-inflammatory drugs, such as steroids and disease-modifying anti-rheumatic drugs (DMARDs). Due to side effects associated with current therapies, efforts are being made to develop a newer group of biologics to increase treatment options.

About Compugen
Compugen is a leading therapeutic product discovery company focused on therapeutic proteins and monoclonal antibodies to address important unmet needs in the fields of immunology and oncology, either for Compugen or its partners. Unlike traditional high throughput trial and error experimental based drug candidate discovery, Compugen's discovery efforts are based on systematic and continuously improving *in silico* (by computer) product candidate prediction and selection followed by experimental validation, with selected product candidates being advanced in its Pipeline Program to the pre-IND stage. Compugen's *in silico* predictive models utilize a broad and continuously growing infrastructure of proprietary scientific understandings and predictive platforms, algorithms, machine learning systems and other computational biology capabilities. The Company's business model primarily involves collaborations covering the further development and commercialization of Compugen-discovered product candidates and various forms of "discovery on demand" arrangements, in both cases providing Compugen with potential milestone payments and royalties on product sales or other forms of revenue sharing. In 2002, Compugen established an affiliate, Evogene Ltd. (www.evogene.com) (TASE: EVGN.TA), to utilize certain of the Company's *in silico* predictive discovery capabilities in agricultural biotechnology. For additional information, please visit Compugen's corporate website at www.cgen.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 discussed in the "Risk Factors" section of Compugen's Annual Report on Form 20-F for the year ended December 31, 2010 as filed with the Securities and Exchange Commission. In addition, any forward-looking statements represent Compugen's views only as of the date of this release and should not be relied upon as representing its views as of any subsequent date. Compugen does not assume any obligation to update any forward-looking statements unless required by law.

Company contact:
Tsipi Haitovsky
Global Media Liaison
Compugen Ltd.
Email: tsipih@netvision.net.il
Tel: +972-52-598-9892