CEL SCI CORP
Form 10-K
January 17, 2007

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ON JANUARY 17, 2007 PURSUANT TO A RULE 201 TEMPORARY HARDSHIP EXEMPTION

FORM 10-K

SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549
(Mark One)

(X) ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934

For the fiscal year ended September 30, 2006.

OR

( ) TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934

For the transition period from _____ to _____.

Commission file number 1-11889

CEL-SCI CORPORATION

(Exact name of registrant as specified in its charter)

COLORADO	84-0916344
(State or other jurisdiction of incorporation or organization)	(I.R.S.Employer Identification No.)
8229 Boone Blvd., Suite 802 Vienna, Virginia	22182
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code: (703) 506-9460 Securities registered pursuant to Section 12(b) of the Act: None Securities registered pursuant to Section 12(g) of the Act:

Common Stock, $.01 par value

(Title of Class)

Indicate by check mark if the registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act. [ ]

Indicate by check mark if the registrant is not required to file reports pursuant to Section 13 or Section 15(d) of the Act. [ ]

Indicate by check mark whether the registrant (1) has filed all reports to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days. Yes [X] No [ ]

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405

of Regulation S-K is not contained herein, and will not be contained, to the best of Registrant's knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. [X]

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer, or a non-accelerated filer. (Check one):

Large accelerated filer [ ] Accelerated filer [ ] Non-accelerated filer [X]

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-25 of the Exchange Act): [ ] Yes [X] No

The aggregate market value of the voting stock held by non-affiliates of the Registrant, based upon the closing sale price of the common stock on March 31, 2006, as quoted on the American Stock Exchange, was approximately $54,485,000.

As of January 12, 2007, the Registrant had 83,291,941 issued and outstanding shares of common stock.

Documents Incorporated by Reference: None

PART I

ITEM 1. BUSINESS

CEL-SCI Corporation was formed as a Colorado corporation in 1983. CEL-SCI's principal office is located at 8229 Boone Boulevard, Suite 802, Vienna, VA 22182. CEL-SCI's telephone number is 703-506-9460 and its web site is www.cel-sci.com. CEL-SCI makes its electronic filings with the Securities and Exchange Commission (SEC), including its annual reports on Form 10-K, quarterly reports on Form 10-Q, current reports on Form 8-K and amendments to these reports available on its website free of charge as soon as practicable after they are filed or furnished to the SEC.

OVERVIEW

CEL-SCI's lead product, Multikine(R), is being developed for the treatment of cancer. Multikine is a patented immunotherapeutic agent consisting of a mixture of naturally occurring cytokines, including interleukins, interferons, chemokines and colony-stimulating factors, currently being developed for the treatment of cancer. Multikine is designed to target the tumor micro-metastases that are mostly responsible for treatment failure. The basic concept is to add Multikine to the current cancer treatments with the goal of making the overall cancer treatment more successful. Phase II data indicated that Multikine treatment resulted in a substantial increase in the survival of patients. The lead indication is advanced primary head & neck cancer (500,000 new cases per annum). Since Multikine is not tumor specific, it may also be applicable in many other solid tumors.

In January 2007 the US Food and Drug Administration (FDA) concurrd with the initiation of global Phase III clinical trial in head and neck cancer patients using Multikine. The Canadian regulatory agency, the Biologics and Genetic Therapies Directorate, had previously concurred with the initiation of a global Phase III clinical trial in head and neck cancer patients using Multikine.

The protocol is designed to develop conclusive evidence of the efficacy of Multikine in the treatment of advanced primary squamous cell carcinoma of the oral cavity (head and neck cancer). A successful outcome from this trial should enable CEL-SCI to apply for a Biologics License to market Multikine for the treatment of this patient population.

The trial will test the hypothesis that Multikine treatment administered prior to the current standard therapy for head and neck cancer patients (surgical resection of the tumor and involved lymph nodes followed by radiotherapy or radiotherapy and concurrent chemotherapy) will extend the overall survival, enhance the local/regional control of the disease and reduce the rate of disease progression in patients with advanced oral squamous cell carcinoma.

Clinical trials in over 200 patients have been completed with Multikine with the following results:

1) It has been demonstrated to be safe and non-toxic.

2) It has been shown to render cancer cells much more susceptible to radiation therapy (The Laryngoscope, December 2003, Vol.113 Issue 12).

3) A publication in the Journal of Clinical Oncology (Timar et al, JCO, 23(15): May 2005), revealed the following:

(i) Multikine induced anti-tumor immune responses through the combined activity of the different cytokines present in Multikine following local administration of Multikine for only three weeks.

(ii) The combination of the different cytokines caused the induction, recruitment into the tumor bed, and proliferation of anti-tumor T-cells and other anti-tumor inflammatory cells, leading to a massive anti-tumor immune response.

(iii) Multikine induced a reversal of the CD4/CD8 ratio in the tumor infiltrating cells, leading to a marked increase of CD4 T-cells in the tumor, which resulted in the prolongation of the anti-tumor immune response and tumor cell destruction.

(iv) The anti-tumor immune-mediated processes continued long after the cessation of Multikine administration.

(v) A three-week Multikine treatment of patients with advanced primary oral squamous cell carcinoma resulted in an overall response rate of 42% prior to standard therapy, with 12% of the patients having a complete response.

(vi) A histopathology study showed that the tumor load in Multikine treated patients was reduced by nearly 50% as compared to tumors from control patients in the same pathology study.

(vii) The tumors of all of the patients in this Phase II trial who responded to Multikine treatment were devoid of the cell surface marker for HLA Class II. This finding, if confirmed in this global Phase III clinical trial, may lead to the establishment of a

marker for selecting the patient population best suited for treatment with Multikine.

(viii) In a Phase II study, using the same drug regimen as will be used in the Phase III study, the addition of Multikine as first-line treatment prior to the standard of care treatment resulted in a 33-40% improvement in the median survival at 3 1/2 years post-surgery, when compared to the results of 39 OSCC clinical trials published in the scientific literature between 1987 and 2004.

CEL-SCI also owns a pre-clinical technology called L.E.A.P.S.TM (Ligand Epitope Antigen Presentation System). The lead product derived from this technology is the CEL-1000 peptide which has shown protection in animals against herpes, malaria, viral encephalitis and cancer.

MULTIKINE

Multikine has been tested in over 200 patients in clinical trials conducted in the U.S., Canada, Europe and Israel. Most of these patients were head and neck cancer patients, but some studies were also conducted in prostate

cancer patients, HIV-infected patients and HIV-infected women with Human Papilloma Virus ("HPV")-induced cervical dysplasia, the precursor stage before the development of cervical cancer. The safety profile was found to be very good and CEL-SCI believes that the clinical data suggests that further studies are warranted.

The function of the immunological system is to protect the body against infectious agents, including viruses, bacteria, parasites and malignant (cancer) cells. An individual's ability to respond to infectious agents and to other substances (antigens) recognized as foreign by the body's immune system is critical to health and survival. When the immune response is adequate, infection is usually combated effectively and recovery follows. Severe infection can occur when the immune response is inadequate. Such immune deficiency can be present from birth but, in adult life, it is frequently acquired as a result of intense sickness or as a result of the administration of chemotherapeutic drugs and/or radiation. It is also recognized that, as people reach middle age and thereafter, the immune system grows weaker.

Two classes of white blood cells, macrophages and lymphocytes, are believed to be primarily responsible for immunity. Macrophages are large cells whose principal immune activity is to digest and destroy infectious agents. Lymphocytes are divided into two sub-classes. One sub-class of lymphocytes, B-cells, produces antibodies in response to antigens. Antibodies have unique combining sites (specificities) that recognize the shape of particular antigens and bind with them. The combination of an antibody with an antigen sets in motion a chain of events which may neutralize the effects of the foreign substance. The other sub-class of lymphocytes, T-cells, regulates immune responses. T-cells, for example, amplify or suppress antibody formation by B-cells, and can also directly destroy "foreign" cells by activating "killer cells."

It is generally recognized that the interplay among T-cells, B-cells and the macrophages determines the strength and breadth of the body's response to infection. It is believed that the activities of T-cells, B-cells and macrophages are controlled, to a large extent, by a specific group of hormones called cytokines. Cytokines regulate and modify the various functions of both

T-cells and B-cells. There are many cytokines, each of which is thought to have distinctive chemical and functional properties. IL-2 is but one of these cytokines and it is on IL-2 and its synergy with other cytokines that CEL-SCI has focused its attention. Scientific and medical investigation has established that IL-2 enhances immune responses by causing activated T-cells to proliferate. Without such proliferation no immune response can be mounted. Other cytokines support T-cell and B-cell proliferation. However, IL-2 is the only known cytokine which causes the proliferation of T-cells. IL-2 is also known to activate B-cells in the absence of B-cell growth factors.

Although IL-2 is one of the best characterized cytokines with anticancer potential, CEL-SCI is of the opinion that to have optimum therapeutic value, IL-2 should be administered not as a single substance but rather as a mixture of IL-2 and certain cytokines, i.e. as a "cocktail". This approach, which was pioneered by CEL-SCI, makes use of the synergism between these cytokines. It should be noted, however, that neither the Food and Drug Administration (FDA) nor any other agency has determined that CEL-SCI's Multikine product will be effective against any form of cancer.

Research and human clinical trials sponsored by CEL-SCI have indicated a correlation between administration of Multikine to cancer patients and immunological responses. On the basis of these experimental results, CEL-SCI believes that Multikine may have application for the treatment of solid tumors in humans.

Between 1985 and 1988 Multikine was tested at St. Thomas Hospital in London, UK in forty-eight patients with various types of cancers. Multikine was shown to be safe when used by these patients.

In November 1990, the Florida Department of Health and Rehabilitative Services ("DHRS") gave the physicians at a southern Florida medical institution approval to start a clinical cancer trial in Florida using CEL-SCI's Multikine product. The focus of the trial was unresectable head and neck cancer.

In 1991, four patients with regionally advanced squamous cell cancer of the head and neck were treated with CEL-SCI's Multikine product. The patients had previously received radical surgery followed by radiation therapy but developed recurrent tumors at multiple sites in the neck and were diagnosed with terminal cancer.

Significant tumor reduction occurred in three of the four patients as a result of the treatment with Multikine. Negligible side effects, such as injection site soreness and headaches, were observed and the patients were treated as outpatients. Notwithstanding the above, it should be noted that these trials were only preliminary and were only conducted on a small number of patients. It remains to be seen if Multikine will be effective in treating any form of cancer.

These results caused CEL-SCI to embark on a major manufacturing program for Multikine with the goal of being able to produce a drug that would meet the stringent regulatory requirements for advanced human studies. This program included building a pilot scale manufacturing facility.

The objective of CEL-SCI scientists is to use Multikine as an adjunct (additive) therapy to the existing treatment of previously untreated head & neck cancer patients with the goal of reducing cancer recurrence and ultimately increasing survival. However, pursuant to FDA regulations, CEL-SCI was required to test the drug first for safety in locally recurrent, locally metastatic head

and neck cancer patients who had failed other cancer therapies. This dose escalation study was started in 1995 at several centers in Canada and the US where 16 patients were enrolled at 4 different dosage levels. The study ended in 1998 and showed Multikine to be safe and well tolerated at all dose levels.

Because CEL-SCI scientists have determined that patients with previously untreated disease would most likely benefit more from Multikine treatment, CEL-SCI started a safety trial in Canada in 1997 in advanced primary head & neck cancer patients who had just recently been diagnosed with head & neck cancer. This study ultimately enrolled 28 patients, also at 4 different dosage levels, and ended in late 1999. Halfway through this study, CEL-SCI launched a number of phase II studies in advanced primary head & neck cancer to determine the best dosage, best route of administration and best frequency of administration of Multikine. Those studies involved 19 patients in Israel (1997 - 2000), 30

patients in Poland and the Czech Republic (1999 - 2000), and 94 patients (half treated with Multikine and the other half disease-matched cancer patients served as control) in Hungary (1999 - 2003). The Hungarian trial compared the control group (receiving only conventional cancer therapy) to the Multikine treated patients (receiving Multikine prior to conventional therapy) by histopathology and immunohistochemistry. The results of these studies were published in peer-reviewed scientific journals and/or presented at scientific meetings. The studies that have not yet been published were conducted in support of Multikine's safety and clinical utility.

The above studies, which are all completed, indicate that Multikine was safe and well tolerated at all dose levels investigated. The studies also showed partial and complete tumor responses following Multikine treatment at the best treatment regimen combinations as well as tumor necrosis (destruction) and fibrosis (as determined by histopathology).

While CEL-SCI scientists believe partial and complete tumor responses to be very important, they also believe that other findings with Multikine in these studies are equally important since they may serve to enhance existing cancer therapies, thereby affecting the clinical outcome of the cancer patient's treatment.

The initial results of the Hungarian study were published in December 2003. Data from a Phase I/II clinical trial in fifty-four (54) advanced primary head and neck cancer patients (half treated, half control), the first part of the Hungarian study, were published in The Laryngoscope, December 2003, Vol.113 (12). The title of the article is "The Effect of Leukocyte Interleukin Injection (MULTIKINE) on the Peritumoral and Intratumoral Subpopulation of Mononuclear Cells and on Tumor Epithelia: A Possible New Approach to Augmenting Sensitivity to Radiation Therapy and Chemotherapy in Oral Cancer - A Multi Center Phase I/II Clinical Trial".

The data demonstrates that treatment with Multikine rendered a high proportion of the tumor cell population highly susceptible to radiation therapy. This finding represents a major advance in the treatment of cancer since, under current standard therapy, only about 5%-10% of the cancer cells are thought to be susceptible to radiation therapy at any one point in time.

The increased sensitivity of the Multikine-treated tumors to radiation was derived from a dramatic increase in the number of proliferating (those that are in cell cycle) cancer cells. Following Multikine treatment, the great majority of the tumor cells were in a proliferative state, as measured by the well-established cell proliferation marker Ki67. The control patients (not

treated with Multikine) had only low expression (near background) of the same proliferation marker (Ki67) in this study. These findings were statistically significant (p