UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 10-QSB

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

For quarter Ended March 31, 1996
Commission File Number 1-12584

SHEFFIELD MEDICAL TECHNOLOGIES INC.

DELAWARE	13-3808303
(State or Other Jurisdiction of Incorporation or organization)	(I.R.S. Employer Identification Number)

30 Rockefeller Plaza, Suite 4515 New York, New York (Address of Principal Executive Offices)	10112 (Zip Code)

Registrant's telephone number, including area code: (212) 957-6600

Check whether the issuer: (1) filed all reports required to be filed by Section 13 or 15(d) of the Exchange Act during the past 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 / /

The number of shares outstanding of the Issuer's Common Stock is 10,067,397 shares of Common Stock as of March 31, 1996.

Transitional Small Business Disclosure Format:

Yes / / No /X/

SHEFFIELD MEDICAL TECHNOLOGIES INC.
(A Development Stage Enterprise)

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SHEFFIELD MEDICAL TECHNOLOGIES INC. AND SUBSIDIARIES
(A DEVELOPMENT STAGE ENTERPRISE)
CONSOLIDATED BALANCE SHEET
MARCH 31, 1996
(UNAUDITED)

ASSETS

<TABLE>
<CAPTION>

<S>	<C>
Current Assets:	
Cash and cash equivalents	$ 2,042,759
Prepaid expenses and other current assets	62,101
Total current assets	2,104,860
Property and Equipment:	
Laboratory equipment	185,852
Office equipment	82,198
Leasehold improvements	61,390
	329,440
Less accumulated depreciation	108,892
Net property and equipment	220,548
Other Assets	201,090
Total assets	$ 2,526,498

LIABILITIES AND STOCKHOLDERS' EQUITY

Current liabilities:	
Accounts payable and accrued liabilities	$ 174,709
Sponsored research payable	244,939
Capital lease obligation-current portion	24,422
Total current liabilities	444,070
Capital lease obligation - non-current portion	42,540
Stockholders' equity	
Preferred stock, $.01 par value. Authorized, 3,000,000 shares; none issued	--
Common stock, $.01 par value. Authorized, 20,000,000 shares; issued and outstanding, 10,067,397 shares	100,674
Additional paid-in capital	23,174,630
Deficit accumulated during development stage	(21,235,416)
	2,039,888
Total liabilities and stockholders' equity	$ 2,526,498

</TABLE>

See accompanying notes to unaudited consolidated financial statements.

SHEFFIELD MEDICAL TECHNOLOGIES INC. AND SUBSIDIARIES
(A DEVELOPMENT STAGE ENTERPRISE)
CONSOLIDATED STATEMENTS OF OPERATIONS
FOR THE THREE MONTHS ENDED MARCH 31, 1996 AND 1995 AND FOR THE PERIOD FROM OCTOBER 17, 1986 (INCEPTION) TO MARCH 31, 1996
(UNAUDITED)

<TABLE>
<CAPTION>

	Three months ended March 31,		October 17, 1986 (inception) to March 31,
	1996	1995	1996
<S>	<C>	<C>	<C>
Interest Income	$ 16,515	$ 1,619	$ 249,764
Expenses:			
Research and development	1,239,791	885,999	12,921,170
General and administrative	430,548	541,884	8,494,036
Interest	1,829	4,595	112,761
Loss before extraordinary item	1,655,653	1,430,859	21,278,203
Extraordinary item	--	--	42,787
Net Loss	$ 1,655,653	$ 1,430,859	$21,235,416
Loss per share of common stock:			
Loss before extraordinary item	$ 0.17	$ 0.21	$ 5.74
Extraordinary item	--	--	0.01
Net Loss	$ 0.17	$ 0.21	$ 5.73
Weighted average common shares outstanding	9,656,540	6,795,995	3,706,141

</TABLE>

See accompanying notes to unaudited consolidated financial statements.

SHEFFIELD MEDICAL TECHNOLOGIES INC. AND SUBSIDIARIES
(A DEVELOPMENT STAGE ENTERPRISE)
CONSOLIDATED STATEMENTS OF CASH FLOWS
FOR THE THREE MONTHS ENDED MARCH 31, 1996 AND 1995 AND FOR THE PERIOD FROM OCTOBER 17, 1986 (INCEPTION) TO MARCH 31, 1996

<TABLE>
<CAPTION>

	Three months ended March 31		October 17, 1986 (inception) to March 31
	1996	1995	1996
<S>	<C>	<C>	<C>
Cash outflows from development stage activities and extraordinary gain:			
Loss before extraordinary item	($ 1,655,653)	($ 1,430,859)	($21,278,203)
Extraordinary gain on extinguishment of debt	--	--	42,787
Net loss	(1,655,653)	(1,430,859)	(21,235,416)
Adjustments to reconcile net loss to net cash used by development stage activities:			
Issuance of common stock, stock options and warrants for services	--	--	900,241
Non-cash interest expense	--	--	50,000
Issuance of common stock for license	--	--	5,216
Issuance of common stock for intellectual property rights	--	--	866,250
Amortization of organizational and debt issuance costs	--	--	77,834

Depreciation	18,537	12,022	108,892
Increase in debt issuance and organizational costs	--	--	(77,834)
Decrease (increase) in prepaid expenses and other current assets	91,684	(34,166)	(121,142)
(Increase) decrease in other assets	(116,720)	49,941	(142,049)
(Decrease) in accounts payable, accrued liabilities	(26,576)	(287,793)	(402,361)
Increase in sponsored research payable	17,537	523,048	822,009
Net cash used by development stage activities	(1,671,191)	(1,167,807)	(19,148,360)
Cash flows from investing activities:			
Acquisition of laboratory and office equipment	(44,314)	(8,543)	(256,987)
Net cash used by investing activities	(44,314)	(8,543)	(256,987)
Cash flows from financing activities:			
Principal payments under capital lease	(5,491)	--	(5,491)
Conversion of convertible, subordinated notes	--	--	749,976
Proceeds from issuance of debt	--	550,000	550,000
Proceeds from issuance of common stock	--	3,233,571	13,268,035
Proceeds from exercise of stock options	137,175	--	1,003,302
Proceeds from exercise of warrants	1,766,003	--	5,881,200
Net cash and cash equivalents provided by financing activities	1,897,687	3,783,571	21,447,022
Net increase (decrease) in cash and cash equivalents	182,182	2,607,221	2,041,675
Cash and cash equivalents at beginning of period	1,860,577	80,130	1,084
Cash and cash equivalents at end of period	$ 2,042,759	$ 2,687,351	$ 2,042,759
Noncash investing and financing activities:			
Common stock, stock options and warrants issued for services	$ --	--	$ 900,241
Common stock issued for license	--	--	5,216
Common stock issued for intellectual property rights	--	--	866,250
Common stock issued to retire debt	--	--	600,000
Equipment acquired under capital lease	72,453	--	72,453
Notes payable converted to common stock	--	--	749,976
Supplemental disclosure of cash flow information:			
Interest paid	$ 1,829	$ 4,595	$ 112,761

</TABLE>

See accompanying notes to unaudited consolidated financial statements.

SHEFFIELD MEDICAL TECHNOLOGIES INC. AND SUBSIDIARIES
(A DEVELOPMENT STAGE ENTERPRISE)

NOTES TO CONSOLIDATED FINANCIAL STATEMENTS
MARCH 31, 1996
(UNAUDITED)

1. CONSOLIDATED FINANCIAL STATEMENTS

The accompanying consolidated balance sheet as of March 31, 1996 and the accompanying consolidated statements of operations and cash flows for the three months ended March 31, 1996 and 1995 and for the period from October 17, 1986 (inception) to March 31, 1996, have been prepared by Sheffield Medical Technologies Inc. (the "Company"), without audit. In the opinion of management, all adjustments (consisting only of normal recurring accruals) necessary to present fairly the financial position, results of operations, and changes in cash flows at March 31, 1996, and for all periods presented have been made.

Certain information and footnote disclosures normally included in financial statements prepared in accordance with generally accepted accounting principles have been condensed or omitted. It is suggested that these consolidated financial statements be read in conjunction with the financial statements and notes thereto included in the Company's annual report on Form 10-KSB for the year ended December 31, 1995. The results of operations for the three months ended March 31, 1996 and 1995 are not necessarily indicative of the operating results for the full years.

Sheffield Medical Technologies Inc. ("Sheffield") was incorporated on October 17, 1986, under the Canada Business Corporations Act. The Company's wholly-owned subsidiary, U-Tech Medical Corporation ("U-Tech") was incorporated in the state of Texas on January 13, 1992. On January 10, 1996, Ion Pharmaceuticals, Inc., a Delaware corporation ("Ion"), was formed as a wholly-owned subsidiary of the Company. At that time, Ion acquired the Company's rights with respect to the anti-proliferative technology. Unless the context requires otherwise, Sheffield, U-Tech and Ion are referred to as "the Company". The Company commenced its biotechnology operations in the United States in January 1992 under new management and Sheffield became domesticated as a Wyoming corporation in May 1992. At the Annual Meeting of shareholders of the Company held on January 26, 1995, the Company's shareholders approved the proposal to reincorporate the Company in Delaware, which was effected on June 13, 1995. All significant intercompany transactions are eliminated in consolidation.

The Company is in the development stage and as such has been principally engaged in licensing and research efforts. The Company has not generated any operating revenue and requires additional capital, which it intends to obtain through equity and debt offerings to continue to operate its business. The likelihood of the success of the Company must be considered in light of the expenses, difficulties and delays frequently encountered in emerging technology-related businesses, particularly since the Company will focus on research, development and unproven technologies which may require a lengthy period of time and substantial expenditures to complete. Even if the Company is able to successfully develop new products or technologies, there can be no assurance that the Company will generate sufficient revenues from the sale or licensing of such products and technologies to be profitable.

2. CAPITAL STOCK TRANSACTIONS

The following table represents the issuance of common stock since the Company's incorporation:

	Number of common shares issued
Date of incorporation	900,000
Issued during year ended December 31, 1986	990,000
Issued during year ended December 31, 1991	412,500
Issued during year ended December 31, 1992	850,000
Issued during year ended December 31, 1993	2,509,171
Issued during year ended December 31, 1994	1,134,324
Issued during year ended December 31, 1995	2,765,651
Issued during three months ended March 31, 1996	505,751
Balance outstanding at March 31, 1996	10,067,397

On March 15, 1996, the Company offered holders of warrants issued in private placements completed in 1995 the opportunity to exercise such warrants at up to a 12 1/2 % discount from the actual exercise prices of such warrants. This warrant discount offer expired on April 30, 1996. In the first quarter, a total of $1,458,500 was received from the exercise of

362,750 of the Company's stock purchase warrants under the warrant discount program. In addition to proceeds received from the warrant discount program, $677,981 was received from the exercise of 143,001 of the Company's stock purchase warrants and options.

3. STATUS OF RESEARCH AND DEVELOPMENT ACTIVITIES

RBC-CD4 ELECTROINSERTION TECHNOLOGY

BACKGROUND. The Company is the worldwide licensee of certain technology (the "RBC-CD4 Electroinsertion Technology") relating to the electroinsertion of full-length CD4 protein into the red blood cell membrane ("RBC-CD4") for use as a potential therapeutic in the treatment of human immunodeficiency virus ("HIV") that leads to Acquired Immune Deficiency Syndrome ("AIDS"). The electroinsertion process inserts CD4, the protein that serves as the binding site of the HIV virus, into a red blood cell. This altered cell complex acts as a decoy and is designed to cleanse the blood of infection by binding to and removing the HIV virus from circulation before it can infect other cells in the human immune system.

TECHNOLOGY. A number of AIDS research projects have studied CD4, which is a glycoprotein found on the surface of T4 lymphocytes. T4 lymphocytes are helper cells that mediate antigen presentation of the immune system. CD4 attaches to a glycoprotein on the surface of HIV known as gp120. HIV binds the CD4 glycoprotein, which enables it to enter the T4 cells, where it can replicate. By this process, HIV attacks T4 cells and, as a result, debilitates the immune system by rendering the immune system incapable of neutralizing HIV. Eventually, the number of T4 cells decrease and the level of HIV in the blood increases. This typically leads to the development of AIDS which is characterized by the ultimate collapse of the immune system. Once the immune system is destroyed, other germs and viruses that ordinarily

would be successfully neutralized by the immune system lead to opportunistic diseases. These opportunistic diseases are ultimately the cause of death in AIDS patients.

A number of approaches have been used in the search for a treatment for AIDS. Scientific efforts have focused principally on the use of compounds or vaccines with the ability to stop the multiplication or replication of HIV. The four principal compounds that have been approved by the FDA to date are AZT, ddI, ddC and d4T.

The use of CD4 as a potential treatment for AIDS is not new. Previous research by many others focused on the soluble form of CD4. This technique has proved ineffective because: (i) the half-life of soluble CD4 or hybrid molecules such as CD4-IgG is short in blood circulation; (ii) the binding of soluble CD4 to HIV appears to tear some of the viral envelope glycoprotein without reducing infectivity; and (iii) the amounts of soluble CD4 needed to establish therapeutic concentrations are very large.

The Company's RBC-CD4 Electroinsertion Technology differs from the traditional focus on compounds and vaccines that inhibit the replication of HIV. RBC-CD4 Electroinsertion Technology has its basis in studies that indicate that HIV will bind to red blood cells ("RBC") containing CD4 in its membrane and that once so internalized into the RBC, may disintegrate. In simplest terms, the technology focuses on incorporating the full-length CD4 into the RBC membrane. The technology is intended to slow the spread of HIV in the body of an infected patient and diminish or eliminate the possibility of HIV infection being spread to others by contact with the infected person, and to help eliminate cells that produce HIV from circulation. Because the technology may slow or eliminate the advancement of HIV infection to AIDS, it is a potential therapeutic, but may not be a cure.

The Company's RBC-CD4 Electroinsertion Technology was originated in 1987 by Dr. Y. Claude Nicolau and other scientists then associated with The Texas

A&M University System ("TAMUS"). Dr. Nicolau is the principal investigator for the RBC-CD4 Electroinsertion Technology research sponsored by the Company. RBC-CD4 Electroinsertion Technology exposes RBC to a pulsed electric field that allows the incorporation of certain proteins into the cell membrane. Many types of proteins can be used as therapeutics. Proteins which contain a sequence called a "hydrophobic membrane spanning sequence" can be attached to RBC by the electroinsertion technique. The hydrophobic membrane spanning sequence is a portion of the protein that is not water soluble. This is critical in order for the protein to immerse itself into the membrane during the electroinsertion procedure. The electroinsertion process causes a temporary disordering of the cell membrane lipid bilayer. When this disordering of the membrane occurs in the presence of a protein with a hydrophobic sequence, the hydrophobic portion of the protein immerses itself into the membrane at the point of disordering, resulting in a cell with the protein inserted in the membrane. One such protein that contains a hydrophobic sequence is "full-length" CD4. Significantly, full-length CD4 consists of the hydrophobic portion and a soluble extracellular domain and a cytoplasmic domain. When the hydrophobic sequence is deleted, CD4 is secreted as a soluble protein which, as described below, is the protein that has been unsuccessful in research for the development of HIV/AIDS therapeutics. The Company's licensed technology is for insertion of the potentially more effective "full-length" CD4 into red blood cells for use as a therapeutic for the treatment of HIV/AIDS. In the research funded by the Company, Dr. Nicolau has successfully electroinserted full-length CD4 into rabbit, mouse, pig and human red blood cell membranes to determine the affinity and binding strength of the RBC-CD4 with the HIV virus. These tests have shown that RBC-CD4 may overcome the problems associated with soluble CD4, including: (i) RBC-CD4 has shown no immune response in animals or humans; (ii) RBC-CD4 remains in the body for almost the normal half life span of a RBC, which is 60 days; and (iii) RBC-CD4 has shown a significantly improved binding affinity and indicates the capacity to inhibit HIV infection of susceptible cells.

Because infection also occurs in the lymph nodes, the Company is developing a companion technology, Liposome-CD4, to address the elimination of HIV in the lymphatic system. In addition, the Company is developing an AIDS Vaccine for preventing HIV infection.

Progress of Research and Development. The IND and test protocols were submitted in 1991 and were approved by the FDA in 1992. Phase I Clinical Trials with HIV-infected patients began in February 1992 on four patients. Researchers affiliated with TAMUS, the Center for Blood Research Laboratories, Inc. ("CBRL"), a wholly owned subsidiary of The Center for Blood Research, Inc. (an affiliate of Harvard Medical School), and Baylor College of Medicine, in conjunction with the Veterans Affairs Medical Center, completed these Phase I Clinical Trials in Houston, Texas, in April 1992. The 60-day trial included meeting three criteria: (i) adequate residence time in the blood stream by RBC-CD4 (the red blood cells into which the CD4 protein has been inserted that act as the binding site for HIV) to permit the HIV virus to bind with the cells and potentially be eliminated from the circulation; (ii) no reduction in the normal functioning of the red blood cell; and (iii) no adverse immune response or toxicity.

The completion of Phase I Clinical Trials essentially confirmed that there are no significant adverse human responses to the process at sub-therapeutic doses. Results indicated that (i) the red blood cell's normal functioning is not altered by the electroinsertion procedure; (ii) the life span of the RBC-CD4 is equal to the life span of normal red blood cells; (iii) the majority of the electroinserted CD4 remains on the red blood cell surface for the entire life span and little shedding of CD4 occurs, if any; and (iv) no side effects or immune responses were observed. The companion studies demonstrated that RBC-CD4 reproducibly inhibits the transmission of primary "wild type" HIV strains cultured from HIV-infected patients, or cell-to-cell transmission of the virus, up to nearly 100

percent. IN VITRO studies also have shown that the RBC-CD4 loaded with HIV virus does not infect macrophages during phagocytosis, the process of normally removing foreign particles and red cells at the end of their life span (approximately 120 days). Phase I Clinical Trials did not confirm anti-viral activity in humans, which is the purpose of additional trials.

The IND for Phase I/IIA Clinical Trials was submitted by the Company to the FDA on August 18, 1994 for approval to conduct Phase I/II human clinical studies at the Johns Hopkins University Schools of Public Health and Medicine ("Johns Hopkins") to test the product's safety and anti-viral activity at various doses, and the Company received approval from the FDA to commence the trial on July 17, 1995. The Phase I/IIA Clinical Trial consists of a safety study with two patients at the lowest dose of RBC-CD4 and a safety and activity study with two parts: (1) five patients being dosed with a middle dose of RBC-CD4, one of which receives placebo; and (2) 12 patients being dosed at the highest dose of RBC-CD4, two of which receive placebo.

RECENT DEVELOPMENTS. The first patient under the Phase I/IIA Clinical Trials was dosed on August 8, 1995, the first patient to be dosed with the middle dose of RBC-CD4 was dosed on November 16, 1995, and the first patient to be dosed at the highest dose of RBC-CD4 was dosed on January 29, 1996. No significant adverse events have been reported to date, and the last portion of the trial with patients receiving the highest dose of RBC-CD4 is ongoing. The Company is currently participating in discussions with certain third parties regarding the possibility of partnering or licensing this technology.

LIPOSOME-CD4 TECHNOLOGY

BACKGROUND. The Company is the worldwide licensee of certain technology (the "Liposome-CD4 Technology") relating to the incorporation of CD4 antigens into liposome bilayers and their use as a potential therapeutic agent in the treatment of HIV/AIDS. While RBC-CD4 Electroinsertion Technology is being developed by the Company to target HIV and HIV-infected cells in the blood, Liposome-CD4 Technology is being developed by the Company to target infections in the human lymphatic system, a major reservoir for infection not directly reached by blood circulation.

TECHNOLOGY. CD4 is a glycoprotein found on the surface of T4 lymphocytes, which are helper cells that mediate antigen presentation of the immune system. CD4 also acts as the receptor for a glycoprotein on the surface of the human immunodeficiency virus (HIV) known as gp120. HIV binds to the CD4 glycoprotein which enables the virus to enter the T4 cells where it can replicate. By this process, HIV attacks T4 cells and debilitates the immune system, which typically leads to the development of AIDS. Once the immune system is destroyed, other germs and viruses that would ordinarily be successfully neutralized by the immune system lead to opportunistic diseases, which ultimately cause death to AIDS patients.

Lipids consist of two layers (bilayers) of fatty acids surrounded by water; such bilayers are fluid and very flexible. Liposomes can be formed by agitating phospholipids in water suspensions at high frequencies to form a closed vesicle surrounded by a continuous lipid bilayer. Liposomes have properties that are very similar to those of natural membranes and have been studied for carrying, in their interior, specific drugs for the purpose of increasing their potency and safety. Liposomes are eventually broken down and metabolized by the body, or fuse with their target, at which time the content of the liposome is released. The Company is researching the use of liposomes in treating HIV/AIDS because the virus is not only found in the circulatory system, but the lymphatic system as well, which is an area that liposomes can reach. It is believed that the lymph nodes, which are a reservoir of HIV infection, could be targeted for removal of HIV and HIV-infected cells. Liposomes inserted with CD4 ("Liposome-CD4") would be used in conjunction with the Company's RBC-CD4 Electroinsertion Technology which targets the circulatory system, thereby providing a treatment package for both the blood stream and the lymph nodes.

The strategy of Liposome-CD4 is to incorporate CD4 in the bilayer of the liposomes, providing a specific target (I.E., HIV and HIV-infected cells) for liposome fusion. The Liposome-CD4 may also be loaded with cytotoxic agents, or agents that will kill the target cell. When the free-floating HIV comes in contact with Liposome-CD4, the virus fuses with Liposome-CD4 and is inactivated. The remains of the killed infected T4 cell and inactivated virus fused with Liposome-CD4 would then be removed by macrophages (white blood cells). The therapeutic aim, as with RBC-CD4, is to reduce HIV infectivity and slow or eliminate the advancement of HIV infection to AIDS.

PROGRESS OF RESEARCH AND DEVELOPMENT. The first milestone of the Liposome-CD4 research, which included IN VITRO studies of Liposome-CD4 interaction with HIV from patient (and simian immunodeficiency virus ("SIV") from M. Rhesus monkeys) isolate studies with Liposome-CD4 encapsulating a cytotoxic agent, was completed in August 1994 with the IN VITRO studies demonstrating promising anti-viral activity.

RECENT DEVELOPMENTS. IN VITRO HIV inactivation results have shown favorable viral inhibition against HIV patient isolates and a new SHIV (hybrid virus of SIV containing the HIV envelope) isolate. Further studies are underway to optimize the liposome formulation and test viral inhibition against patients isolates of HIV using the optimized formulation. In addition, studies are underway to compare the effectiveness of

Liposome-CD4 loaded with a cytotoxic agent with Liposome-CD4 without a cytotoxic agent. The Company is currently participating in discussions with certain third parties regarding the possibility of partnering or licensing this technology.

HIV/AIDS VACCINE

BACKGROUND. The Company holds an option to acquire an exclusive worldwide license to a potential HIV/AIDS vaccine (the "HIV/AIDS Vaccine") under development at the French National Institute of Health and Medical Research ("INSERM"). This research project is headed by Professor Jean-Claude Chermann, one of the original Pasteur Institute discoverers of the HIV virus. The vaccine concept developed by Professor Chermann utilizes a portion of b2 microglobulin (the epitope), a cellular antigen, that is presented on the HIV viral coating after the HIV virus has reproduced in a human cell. This cellular antigen does not appear to vary across the various strains of the virus and may provide a stable target to develop antibodies that can prevent infection. The Company believes this approach may also protect against both blood-born and sexual transmission of HIV. The Company's goal is to develop an oral formulation that would make the vaccine potentially less costly and easier to distribute to a broad population.

TECHNOLOGY. When the HIV virus infects a cell, it replicates and then it buds from the infected cell's surface. A protein which is present on the cell's surface then becomes incorporated in HIV's viral envelope as it leaves the infected cell. The classical path of vaccine development to date has been one of raising antibodies against a viral protein in an attempt to neutralize the pathogen. All these attempts have been largely unsuccessful. The HIV/AIDS Vaccine encompasses a new and different approach directed toward immunization against HIV/AIDS. The HIV/Vaccine is designed to be different than previous attempts for two basic reasons: (i) it would use a cellular versus a viral antigenic approach and is therefore, common to all strains of HIV, and (ii) it would utilize a delivery system that would offer both humoral (blood transmission) and mucosal (sexual transmission) protection, as opposed to other vaccines now being investigated as therapeutics for preventing cell to cell transmission of the virus.

PROGRESS OF RESEARCH AND DEVELOPMENT. Research has been directed toward HIV/AIDS prevention following isolation of the virus in 1983. Research began in 1988 in this area and in the use of a cellular antigenic approach

directed toward conquering the disease. Preclinical research has demonstrated neutralization of HIV IN VITRO. The peptide sequence that encodes this portion of a cellular protein has been identified and sequenced and will be incorporated in a vaccine to test for production of antibodies against the epitope. The Company plans to produce a vaccine for humans that will elicit mucosal as well as humoral immunity and that can be delivered orally. Upon the successful completion of pre-clinical animal studies, the Company plans to submit an IND for conducting Human Clinical Trials.

RECENT DEVELOPMENTS. The Company entered into an agreement with an unaffiliated consulting firm in December of 1995 to develop a commercial diagnostic assay for detection of the monoclonal antibody. This assay would be used in animal and human clinical studies for the vaccine and could be sold for research purposes prior to receiving approval from the FDA. Upon approval from the FDA, the assay could be sold to physicians and clinical laboratories. The Company will be working over the next few months to refine this assay.

UGIF TECHNOLOGY - PROSTATE CANCER AND BENIGN PROSTATIC HYPERPLASIA THERAPY

BACKGROUND. The Company holds an option to acquire an exclusive worldwide license for a growth regulator factor, termed Urogenital Sinus Derived Growth Inhibitory Factor ("UGIF/ps20"), which could serve as a potential prostate cancer therapy (the "UGIF Technology").

TECHNOLOGY. Based on studies at Baylor College of Medicine directed at understanding how one particular tissue type influences the growth of an adjacent tissue in the development of the prostate gland, UGIF/ps20 was identified. Specifically, UGIF/ps20 has been isolated and purified from rat fetal urogenital sinus tissue which differentiates into the mature prostate gland as a result of tissue-tissue interactions. Since UGIF/ps20 was demonstrated to be active in human cells, it was believed that UGIF/ps20 isolated from the rat would be essentially identical to human UGIF/ps20. Commercial application and economic feasibility of UGIF/ps20 is not dependent upon the availability of either rat or human fetal urogenital sinus tissue, but rather the successful cloning, expression and testing of recombinant UGIF/ps20.

The discovery of UGIF/ps20 indicates that urogenital sinus tissue, and more specifically UGIF/ps20, may possibly be effective in altering the phenotype (state of cell differentiation) of cells that affect the secretion of newly synthesized proteins. UGIF/ps20 has shown inhibition of the growth of transformed cells and tumors in culture including human prostate cancer cells with non-toxic and reversible effects. In addition to the treatment of cancer, there exists a potential use of UGIF/ps20 or its analogues in the treatment of other diseases or conditions dealing with abnormalities of the genitourinary system. For example, since UGIF/ps20 induces changes in the state of cellular differentiation to that more suggestive of what should be normal, UGIF/ps20 may be effective in treating diseases that are manifested by the loss or change in normal tissue or normal cell differentiation.

Dr. David R. Rowley is the principal investigator for the UGIF Technology project. Dr. Rowley is Assistant Professor in the Department of Cell Biology at the Baylor College of Medicine

PROGRESS OF RESEARCH AND DEVELOPMENT. A method for successfully purifying UGIF/ps20 was identified in April 1992 by Dr. David R. Rowley and biological activity of the factor was demonstrated in mice in May 1992. Research to date has shown that UGIF/ps20 inhibits the growth of transformed cells and tumors in culture including human prostate cancer cells with non-toxic and reversible effects. In addition, in preliminary animal studies, UGIF/ps20 has shown an ability to inhibit DNA synthesis and cell proliferation of human prostatic carcinoma cells. Results confirmed that there is a human form of UGIF/ps20 and that it is a growth factor associated with the prostate gland.

RECENT DEVELOPMENTS. The rat and human genes for UGIF/ps20 were sequenced in late 1995. The rat gene has been incorporated into an expression system and recombinant rat UGIF/ps20 is currently being produced. The human gene is currently being incorporated into an expression system for production of recombinant human UGIF/ps20. Once sufficient quantifies of recombinant UGIF/ps20 are produced and purified, the activity of the UGIF/ps20 protein will be tested for verification in IN VITRO and IN VIVO studies. It is anticipated that additional animal studies will be conducted to determine the modes of delivery and biological effects of recombinant UGIF/ps20 on prostate cancer in "nude" mice. In the event that recombinant UGIF/ps20 is verified in these studies, additional preclinical studies with a delivery system, and toxicity tests, will be conducted prior to commencement of human Clinical Trials.

ANTI-PROLIFERATIVE THERAPIES

BACKGROUND. The Company, through its wholly-owned subsidiary, Ion Pharmaceuticals, Inc. ("Ion Pharmaceuticals"), is the worldwide licensee of certain proprietary uses of certain anti-proliferative compounds (the "Anti-Proliferatives"). Such compounds have demonstrated promise in therapeutic applications for treating a number of conditions characterized by abnormal cell proliferation, such as cancer and certain proliferative dermatological conditions. The Company also has a collaborative program with an unaffiliated company to develop proprietary new chemical entities related to the Anti-Proliferatives.

TECHNOLOGY. The Anti-Proliferatives are active through ion transport modulation and may be applicable for treating, either by topical or oral administration, a number of diseases and conditions. Through research conducted by Dr. Jose Halperin at Harvard Medical School, new potential uses for the Anti-Proliferatives have been identified based on inhibition of cell proliferation, including the use of such Anti-Proliferative compounds in treating cancer, proliferative dermatological conditions, cardiovascular disorders, such as arteriosclerotic conditions, and diseases caused by neovascularization, such as diabetic retinopathy. In addition to the compounds ability to inhibit cell proliferation, the Anti-Proliferative compounds have also been shown to inhibit the Ca++-activated K+ channel in the human red blood cell membrane. Dr. Carlo Brugnara at Children's Hospital in Boston has studied and is continuing to study the effects of such compounds in blocking this channel, one of the erythrocyte's principal dehydration pathways, to prevent the sickling tendency of erythrocytes. Such an approach could potentially be used in the treatment of sickle cell anemia.

It is anticipated that the Anti-Proliferative compounds would be formulated in two new formulations 3/4 an oral formulation and a topical formulation. The new oral formulation will be used in the study and potential treatment of cancers, including colon, lung and breast, sickle cell anemia, atherosclerotic conditions, including restenosis after balloon angioplasty. The new topical formulation will be used by the Company in the study and potential treatment of proliferative dermatological conditions, such as actinic keratosis, certain cancers, such as basal cell carcinoma and Kaposi's sarcoma, and, possibly, other dermatological conditions.

PROGRESS OF RESEARCH AND DEVELOPMENT. An initial human efficacy study with a preliminary topical formulation of one of the Anti-Proliferative compounds at a low concentration in comparison with a placebo was conducted by the Company in Kaposi's sarcoma patients which led to inconclusive results. Results showed that the topical formulation was not optimized. The Company has recently entered into an agreement with an unaffiliated company to develop an optimal topical formulation at a higher concentration of drug for use in additional clinical trials for actinic keratosis and Kaposi's sarcoma.

Dr. Halperin has demonstrated that IN VITRO proliferation of three human

cancer cell lines (I.E.,a melanoma, a lung adenocarcinoma, and a colon adenocarcinoma) were strongly inhibited by one of the Anti-Proliferatives in a dose-dependent manner. Dr. Halperin's group has also studied the effect of certain of the Anti-Proliferatives in an experimental model for lung metastasis induced by injection of a human melanoma cell line in mice. After 10 weeks, all control animals developed numerous lung micrometastases; in contrast, half of the treated animals were free of metastases and two did not demonstrate any evidence of disease. No evidence of toxicity was seen based on clinical observations and animal weights. In another animal study, the induction of skin tumors by a known carcinogen, with and without the Anti-Proliferatives treatment, was evaluated with favorable results.

For the sickle cell application, in vitro studies performed by Dr. Brugnara have demonstrated that the Anti-Proliferative compounds blocked ion transport in homozygous sickle cells, and studies in a transgenic

mouse model for sickle cells have demonstrated that the compound given orally produced inhibition of the red cell Gardos channel, increased red cell potassium content, and decreased mean corpuscular hemoglobin concentration. A pilot Phase I clinical trial has been completed in which four normal subjects were given the Anti-Proliferative compound orally and the peak inhibition of the Gardos channel was measured.

RECENT DEVELOPMENTS. The Company is currently negotiating a sponsored research and license option agreement with an option to license the use of the Anti-Proliferatives in treating secretory diarrhea.

A topical formulation of one of the Anti-Proliferatives has been developed for the Company pursuant to an agreement with an unaffiliated company. The lead topical formulation is currently being tested for long-term stability and other testing needed to meet FDA requirements. Clinical trial material is also being manufactured for use in the Company's Phase I/II Clinical Trial for the treatment of actinic keratosis. Two clinical sites in Israel have been chosen and the Company anticipates this Phase I/II Clinical Trial will begin the second quarter of 1996. Upon successful results of this study, the Company plans to file an IND application with the FDA for conducting a clinical trial in the U.S. for the treatment of actinic keratosis.

Additional animal tumor model studies are underway to test the effects of the Anti-Proliferative compounds in the treatment of certain cancers.

A Phase II Clinical Trial supported by the National Institutes of Health and the FDA is currently underway in which five sickle cell anemia patients are being given an Anti-Proliferative compound orally. For this clinical trial, short-term results show that the administration of the compound results in an increase in cell potassium content, a reduction in the number of dense cells, a significant reduction in plasma indirect bilirubin levels, and an increase in hemoglobin levels, indicating that the drug decreases the tendency of the red cells to sickle. The next phase of the ongoing Phase II clinical trial will assess the survival of red blood cells and hemoglobin levels over a longer-term period. The Company plans to conduct an additional clinical study to assess long-term efficacy of a new oral formulation of the Anti-Proliferative compound.

4. SUBSEQUENT EVENT

On April 30, 1996, the Company completed its warrant discount program, whereby, the Company offered holders of warrants issued in private placements completed in 1995 the opportunity to exercise such warrants at up to a 12 1/2% discount from the actual exercise prices of such warrants. At the expiration of the discount program, on April 30, 1996, a total of $5.6 million was received from the exercise of 1,360,750 of the Company's stock purchase warrants.

Item 2:

MANAGEMENT'S DISCUSSION AND ANALYSIS OR PLAN OF OPERATION

PLAN OF OPERATIONS

The Company, being a development enterprise, has incurred a net loss in each of the fiscal years since its inception and has had to rely on outside sources of funds to maintain its liquidity. Substantial operating losses are expected to be incurred for the next several years as the Company expends its resources for product acquisition, sponsored research and development and preclinical and clinical testing.

As a development stage company without revenues, the Company has financed its technology development activities and operations primarily through public and private offerings of securities. In connection with this, the Company completed two private offerings in 1995, raising total gross proceeds of $8.8 million. On March 15, 1996, the Company offered holders of warrants issued in private placements completed in 1995 the opportunity to exercise such warrants at up to a 12 1/2% discount from the actual exercise prices of such warrants. As of the close of business on April 30, 1996, the expiration date of the warrant discount program, a total of $5.6 million was received. Management estimates that based on its successful history of raising capital, its plans to seek additional funds through planned offerings, the results of the warrant discount program, and the continued focus on selling, licensing and commercialization of its technologies, the Company will have sufficient resources to fund its activities for at least the next twelve months. There can be no assurance that planned offerings will be completed or, if not completed as planned, that other sources of capital can be obtained in amounts and upon terms acceptable to the Company during the twelve month plan period. In the event that such funds are not available when needed, the Company would be required to reduce or delay its funding of current research projects and delay making commitments for future research projects. The Company's operating results have fluctuated significantly during each quarter since its reorganization, and the Company anticipates that such fluctuations, largely attributable to varying sponsored research and development commitments and expenditures, will continue into the foreseeable future.

The Company continues to conduct scientific research, clinical trials, development, and intellectual property protection. During the three months ended March 31, 1996, the Company paid $1.2 million for research and development on its projects. During the succeeding 12-month period, approximately $2.8 million in additional funding is projected to be spent on clinical and laboratory research and development.

Of the $2.8 million estimated to be spent on clinical and laboratory research and development during the next 12 months, approximately $100,000 is expected to be applied to RBC-CD4 Technology, $800,000 to the HIV/AIDS project, $200,000 to the UGIF Technology, and $2,100,000 to the Anti-Proliferative Therapies.

In addition to clinical and laboratory research development, the Company expects to incur ongoing costs in connection with its intellectual property protection and patent prosecution, which costs are expected to approximate $100,000 over the next 12 months.

REVENUES AND EXPENSES

Interest Income:

From inception through the period ended March 31, 1996, the Company has earned interest income of $249,764 and an extraordinary item from gain on early extinguishment of debt of $42,787. The Company's ability to generate material revenues is contingent on the successful commercialization of the RBC-CD4 Electroinsertion Technology, the Liposome-CD4 Technology, the HIV/AIDS Vaccine, UGIF Technology, Anti-Proliferative Therapies and other technologies which it

may acquire, followed by the successful marketing and commercialization of such technologies through licenses, joint ventures or other arrangements.

Income for the three months ended March 31, 1996 was $16,515 compared to $1,619 for the same period ended March 31, 1995. The increase in interest earned is attributable to a higher amount of cash applied to short-term investments In each period interest income represented all of the Company's income.

Operating Expenses:

From inception through the period ended March 31, 1996, the Company incurred $21,527,967 of operating expenses. Sixty percent (60%) or $12,921,170 of the total operating expenses for that period were costs of research and development for the Company's technologies. The remainder of expenses for the same period were incurred principally as consulting costs, costs of management, legal and other professional support for the Company's technologies, and for its completed and proposed financing plans. Research and development costs will remain high as the Company implements later-stage research projects of its technologies and such costs will continue to be expensed for financial reporting purposes.

Operating expenses for the three months ended March 31, 1996, were $1,653,653 compared to $1,430,859 for the same period ended March 31, 1995. The increase was due primarily to the advancement of projects to clinical trials; general and administrative costs decreased for the period. Research and development costs were $1,239,791, or $353,792 higher. The increase was attributable primarily to three factors: (1) increased sponsored research payments for the Company's RBC-CD4 project as it entered into its Phase I/II Human Clinical Trials, (2) the completion of the second milestone of its Liposome-CD4 project and the subsequent advancement of the project to its next stage of development (3) increased costs of production of CD4 for both those projects and (4) continued funding of the HIV/AIDS Vaccine project and the Anti-Proliferative therapies.

LIQUIDITY AND CAPITAL RESOURCES

In February 1993, the Company conducted its initial United States public offering of 833,334 Units, each Unit consisting of two shares of Common Stock and one Redeemable Common Stock Purchase Warrant exercisable for one share of Common Stock at a price of $3.75, subject to adjustment in certain circumstances, at any time until February 10, 1998 (the "public offering"). The net proceeds of the public offering to the Company, after payment of Underwriter's discounts and commissions, non-accountable expenses and reimbursable expenses, and other expenses of the offering, were approximately $4,190,000. Also, during fiscal year 1993, the Company received $762,833 in total proceeds from the exercise of warrants. In March 1994 a total of $3,121,164 was received from the exercise of 832,324 of the Company's Redeemable Stock Purchase Warrants. Each warrant was exercisable for one share of Sheffield's Common Stock at an exercise price of $3.75.

In April 1995, the Company completed a private placement of 410,075 units to accredited investors at a price of $8.00 per unit for gross proceeds of $3,280,600. Each unit consists of two shares of the Company's common stock and a warrant to purchase one share of common stock at a price of $5.00 at any time to and including February 10, 2000. The warrants are redeemable by the Company under certain circumstances. Proceeds will be used for

funding research and development for projects and licensing arrangements, patent prosecution and working capital and general corporate purposes.

In July 1995, the Company completed a second private placement of 1,375,000 units at $4.00 per unit, which grossed $5,500,000. Each unit consists of one share of the Company's common stock and one warrant to purchase one share of common stock at a price of $4.50 at any time from March 1, 1996 to and including February 10, 2000. The warrants are subject to redemption under certain conditions.

On April 30, 1996, the Company completed its warrant discount program through which the Company offered holders of warrants issued in private placements

completed in 1995 the opportunity to exercise such warrants at up to a 12 1/2% discount from the actual exercise prices of such warrants. At the expiration of the discount program, on April 30, 1996, a total of $5.6 million was received from the exercise of such warrants and the related issuance of 1,360,750 shares of common stock.

In addition to the potential commercialization of its technologies, the Company plans to seek additional funds through exercise of outstanding warrants and options, financings and/or public grants, joint ventures or other commercial arrangements to obtain necessary working capital. It is not uncommon, for instance, for a third-party commercial partner to enter into a license agreement with a development company, on the merits of successful research relating to a given technology, which would yield up-front royalty advances to such company before market-ready products are developed. It is also not uncommon for a third-party commercial partner to enter into an agreement with a development company whereby a third party will contribute funds in support of the research and operating needs of such development companies in consideration for rights related to the technologies.

At March 31, 1996, the Company's assets were $2.5 million of which $2.0 million was cash and cash equivalents.

PART II: OTHER INFORMATION

Item 6. Exhibits and Reports on Form 8-K.
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No reports on Form 8-K were filed by the Company during the quarter ended March 31, 1996.

In accordance with the requirements of the Exchange Act, the registrant caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

SHEFFIELD MEDICAL TECHNOLOGIES INC.

Dated: May 9, 1996 /s/ Douglas R. Eger

Douglas R. Eger
Chairman & Chief Executive Officer

Dated: May 9, 1996 /s/ George Lombardi

George Lombardi
Vice President & Chief Financial Officer
(PRINCIPAL FINANCIAL AND ACCOUNTING OFFICER)

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This Schedule contains summary financial information extracted from the condensed financial statements for the first quarter ended March 31, 1996 and is qualified in its entirety by reference to such statements.
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