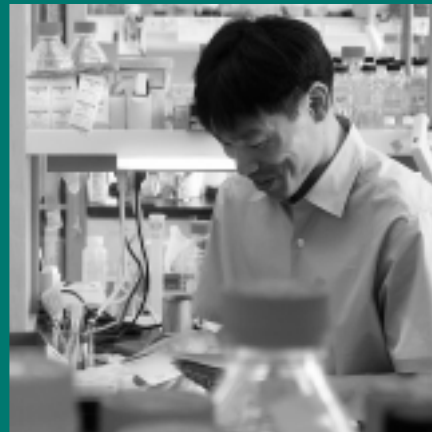


2002 - 2003

United
Mitochondrial
Disease
Foundation

Annual
Report





In 2002-03, the United Mitochondrial Disease Foundation continued its rapid expansion, doubling our grant awards to \$500,000, increasing our membership, and developing new groups.

Our proudest achievement was awarding \$500,108 to six researchers. Four are looking at how mitochondria work. Two are investigating potential therapies. This shows progress toward research that will have a direct impact on patients. The grant program is drawing new people into the field, including 2003 grant recipient Matthew Freeman, PhD, from the prestigious Medical Research Council in Cambridge, England.

Here's a list of some of UMDF's other accomplishments this year:

Research

- UMDF reviewed 27 full grant proposals, doubling the size of the process from the previous year.
- Six researchers joined the Scientific Advisory Board, increasing its membership to 27.
- UMDF and the Mitochondrial Medicine Society started developing the World Patient Registry — web-based to allow doctors from around the world to enter data and positioned to support research at every level.

Chapters, Groups, and Members

- Six healthy chapters and 20 operating groups.
- New support groups in Indianapolis and Chicago.
- Two support groups (Kansas City and Indiana) working toward chapter status.
- Cincinnati-based group revitalized.

Communications

- *Mitochondrial News* redesigned and produced quarterly.
- New brochure of *Ask the Mito Doc* questions and answers.
- New UMDF folder featuring faces of affected adults and children.

Development

- First grant to UMDF for a post-doctoral fellowship (\$50,000 for three years) from the Sage Foundation.
- First 5K run/walk in Pittsburgh on May 31 (the same day the Ohio chapter held its second 5K event) providing expertise that can be shared with other chapters and groups.
- UMDF coin collection program in KFC restaurants in the Cleveland and Pittsburgh areas—a program that we hope to expand to additional cities.

UMDF Office

- Moved the UMDF database from Access software to an SQL server to accommodate data growth and more sophisticated analysis.
- Started a monthly e-mail update to chapter and group leaders.
- Hired a bookkeeping service to handle accounting and provide detailed reports.
- Expanded into additional office space to provide workspace for staff, interns, and volunteers — furnishings provided by the Pinnacle Corporation and landlord Gordon Kidd.

The UMDF Office continues to receive an increasing number of phone calls, e-mail, checks, and good ideas that we need to pursue. UMDF is definitely on the move—thanks to the efforts of its members and supporters.

Yours toward a cure,

Christopher J. Rice
Executive Director

About the United Mitochondrial Disease Foundation

The United Mitochondrial Disease Foundation promotes research for cures and treatments of mitochondrial disorders and provides support to affected individuals and families.

UMDF continues to validate its mission through grants awarded annually, dedicated to the research of mitochondrial mechanisms and diseases. UMDF developed a grants award process based upon the funding awards process used by the National Institutes of Health. A tremendous amount of basic science continues to be done, with the hope that unlocking the secrets of the mitochondria will lead to treatments and a cure of mitochondrial diseases.

UMDF established a Scientific Advisory Board which includes 27 researchers, scientists, and doctors - all leaders in their respective fields - to evaluate and choose the recipients for each year's awards. UMDF first awarded \$30,000 in 1997. To date, UMDF has awarded more than \$1 million towards research. In addition to the six researchers funded in 2003, UMDF funds a Worldwide Mitochondrial Disease Registry and a Tissue Exchange Program. The Registry and the Exchange Program supply researchers and clinicians with the standards and samples they require in order to succeed in their search for treatments and a cure for mitochondrial diseases.



UMDF Staff

Programs for People

The second part of UMDF's mission is realized through outreach to those touched in some way by the disease. The UMDF provides support and information to thousands of patients, family members, donors, researchers, doctors and other allied health professionals. UMDF continues to educate the general public, the medical and the scientific communities about mitochondrial diseases as another component of its outreach programming. Public awareness and outreach fulfill a significant obligation that UMDF has to its mission.

UMDF continues to serve patients and families with a myriad of programs which include: newsletters and mailings, support groups and chapters, a member service phone line for information and referrals, networking opportunities, access to a reference library with up-to-date information on mitochondrial diseases, a website, and an annual symposium focused on mitochondrial diseases where members have the opportunity to meet clinicians and other members faced with battling the effects of the same disease.

UMDF chapters and groups provide members with opportunities for mutual support and productive action. Some groups form around the need to talk with others faced with the same concerns. Other groups form to raise funds for research or to raise awareness and educate physicians. UMDF provides groups and chapters with guidance on operations, assistance in communicating with local members, materials for public awareness and physician education, and advice on fundraising. Fundraising events continue to be a major source of funds for the foundation, as well as an excellent way to raise awareness about the disease. UMDF fundraising events run the gamut from golf outings and 5K walks and runs to elegant wine tasting receptions and dinner dances.



UMDF Chapters and Groups

The following were UMDF chapters, groups and leaders from January 1, 2002 through June 30, 2003:

ARIZONA

Arizona Chapter
Phoenix, AZ
President: Karen Lipps
Members: 100+

CALIFORNIA

Southern California Chapter
Los Angeles, CA
President: Sharon Shaw
Members: 350+

FLORIDA

Florida Rays of Hope
Melbourne, FL
Contacts: Christine Golden
Carrie Waters
Members: 50+

INDIANA

Indiana Support Group
Indianapolis, IN
Contact: Sue Ann Bube
Members: 80+



ILLINOIS

Chicago Area Support Group
Chicago, IL
Contacts: Gail Wehling
Karen Lewis
Members: 105+

KENTUCKY

Kentucky Area Support Group
Georgetown, KY
Contact: Melissa Tritsch
Members: 25+

MARYLAND

Tri State Area (DC area) Support Group
Bethesda, MD
Contact: Andrea Gropman, M.D.
Members: 30+

MASSACHUSETTS

New England Chapter
Boston, MA
President: Justine Fargo
Members: 150+

MICHIGAN

Detroit, Michigan
Contact: Ann Clark
Members: 150+

MISSOURI / KANSAS

Kansas City Area Support Group
Kansas City, MO
President: Heidi Harmon
Members: 45+

NEW MEXICO

New Mexico Support Group
Albuquerque, NM
Contact: Laura Owen
Members: 30+

NEW YORK

New York Metro Chapter
Long Island, NY
President: Joe Rice
Members: 325+

Western New York Support Group
Buffalo, NY
Contact: Angela Geising
Members: 30+

Southern New York Support Group
New Paltz, NY
Contact: Beth and James DeArce
Members: 125+

OHIO

Ohio Chapter
Cleveland, OH
President: Jennifer Lyman
Members: 300+

Columbus Support Group
Columbus, OH
Contact: Shawna Steele
Members: 50+

Cincinnati Support Group
Cincinnati, Ohio
Contact: Jennifer Neal (Cooper)
Members: 45+

OREGON

Pacific Northwest Support Group
Portland, OR
Contact: Cathy Akins
Members: 80+

PENNSYLVANIA

Delaware Valley Chapter
Philadelphia, PA
President: Maripat Shelly
Members: 250+

Western PA Support Group
Pittsburgh, PA
Contact: Karen Wilson
Members: 150+

SOUTH CAROLINA

Columbia, S.C.
Contact: Karis Mott
Members: 50+

VIRGINIA

Virginia Support Group
Williamsburg, VA
Contact: Shelby Hawthorne
Members: 50+

OUTSIDE OF THE UNITED STATES

AUSTRALIA

Australia Support Group
Queensland, Australia
(Mainly meets via internet)
Contact: Tara Collyer
Members: 30+

CANADA / ONTARIO

Canada-Ontario Support Group
Toronto, Ontario Canada
Contact: Valerie McGarry
Members: 20+

Research Grant Projects Funded in 2003

*Principal Investigator: Mikhail Alexeyev, Ph.D.
Institution: University of South Alabama,
Mobile, AL*

Amount of Award: \$100,000

Award Date: 2003

Project Title: Selective Elimination of Defective Mitochondrial Genomes as an Approach to the Reversal of NARP and MILS Syndromes, Heritable Mitochondrial Disorders.

Research Assistant Professor Mikhail F. Alexeyev, PhD, will investigate a treatment for two diseases caused by a mutation in mitochondria by destroying the mutant — but not the normal — mitochondrial DNA within cells. He hypothesizes this can eliminate symptoms of the NARP and MILS syndromes

Alexeyev has identified two bacteria producing enzymes that can selectively recognize and destroy 99.5% of the mutant mitochondrial genomes in a test tube without affecting normal ones. "Our calculations indicate that even if bacterial enzymes will be only 1% as efficient in mitochondria as they are in a test tube, they still should be able to bring mutant DNA content down below the 60% threshold in a patient with 93% mutant mtDNA," says Alexeyev.

*Principal Investigator: Immo Scheffler, Ph.D.
Institution: University of California, San Diego
Amount of Award: \$100,000*

Award Date: 2003

Project Title: Application of RNA interference in the study of NADH-ubiquinone oxidoreductase (complex I) assembly in mammalian mitochondria.

The Sage Fellowship, a grant award of \$100,000 over two years, was presented to Professor Immo E. Scheffler, PhD. Professor Scheffler plans to study the workings of the first

step (performed by Complex I) in the five-step process by which mitochondria produce energy for the cell. In bacteria, there are 14 proteins coded in the bacterial DNA for Complex I. However, in mammals, the nuclear DNA has blueprints for 39 protein units involved in Complex I, and an additional 7 are encoded by mitochondrial DNA.

He will learn what each of these proteins does by preventing their production through the use of "small interfering RNA." Blocking production of one protein at a time, Professor Scheffler will learn about its role in the assembly, stability, activity, and regulation of Complex I.

"The insights we gain," says Dr. Scheffler, "can be applied to the diagnosis and understanding of mitochondrial diseases, particularly the growing class of such diseases resulting from a partial Complex I deficiency." This project focuses on animal cells, but it can lead to similar experiments in whole animals, such as a mouse, to study the broader physiological and pathological aspects of mitochondrial diseases.

*Principal Investigator: Matthew Freeman, Ph.D.
Institution: Laboratory of Molecular Biology —
M.R.C., Cambridge, UK*

Amount of Award: \$90,000

Award Date: 2003

Project Title: Role of Rhomboid Proteolysis in Optic Atrophy.

Matthew Freeman, PhD, plans to study rhomboid proteolysis in an eye disease called dominant optic atrophy that causes some children to go blind.

Dr. Freeman will study model organisms including mice, flies, and even yeast to determine the link between the protein OPA1 and a Rhomboid protein in the regulation of mitochondrial function and human optic atrophy. Dominant optic atrophy is a genetic disease affecting one in 10,000 that causes early childhood blindness. Freeman believes the role of the Rhomboid protein may be an important mechanism in OPA1 defects that cause the eye disease.



"OPA1 is a protein responsible for the proper structure and function of the mitochondria, a vital part of every cell," explains Freeman. "For OPA1 to function properly, it needs to be cut at the correct place and time by another protein."

"We use model organisms to accelerate our understanding of the fundamental biology underlying disease - we could not move as fast if we only worked on humans," explains Freeman.

*Principal Investigator: Koji Okamoto, Ph.D.
Institution: University of Utah, Salt Lake City, UT
Amount of Award: \$83,400*

Award Date: 2003

Project Title: Molecular Basis of Mitochondrial Membrane Dynamics: a New Paradigm of Human Disease

Koji Okamoto, PhD, will study the mechanism of mitochondrial fusion. Understanding the membrane dynamics of mitochondria may lead to understanding the causes of certain neurological diseases and cancers.

Professor Okamoto plans to identify a molecule that helps regulate the transmembrane GTPase Fzo1, a protein that is essential for mitochondrial fusion in both yeasts and humans. Studying these regulatory molecules for mitochondrial fusion can help researchers



understand the pathogenesis leading to certain human diseases.

Understanding how the Fzo1 GTPase cycle controls mitochondrial function will allow researchers to investigate the role of regulated fusion and mitochondrial fragmentation during programmed cell death, called apoptosis. Better understanding of mitochondrial fusion could contribute to the development of gene therapies based on mitochondrial complementation. The study also will promote establishment of animal models for understanding pathogenesis of human diseases associated with mitochondrial fusion defects.

Working in yeast, Okamoto wants to identify the GTPase-activating protein (GAP) that stimulates GTP hydrolysis by Fzo1. His team will then analyze the GAP by a combination of biochemical, cell biological, and genetic approaches, as well as in vitro investigation of GAP-mediated stimulation of the Fzo1 GAPase activity.

Since Fzo1 has a human homologue, it is likely that the yeast GAP also does, so this should lead to a better understanding of the mitochondrial fusion mechanism in humans.

Mitochondria are dynamic cell organelles that change size and shape (morphology) to optimize their energy production for cell function. Loss of normal mitochondrial morphology can result in a variety of human diseases, including neurological disorders and certain cancers. It is also linked to programmed cell death and aging in humans.

*Principal Investigator: Bernard Lemire, Ph.D.
Institution: University of Alberta, Edmonton,
Alberta, Canada*

Amount of Award: \$76,780

Award Date: 2003

*Project Title: The Use of the Yeast CYB2 Gene as
Therapy for Complex I
Mutations in a C. elegans
Model System.*

Bernard D. Lemire, PhD, will study gene therapy for mutations affecting the Complex I metabolic process in mitochondria. One goal of his study is to better understand the biological processes surrounding mitochondrial energy production in normal and disease states. The results will address the contribution of lactic acidosis to mitochondrial diseases and may lead to the development of a new therapy.

“The mitochondrial respiratory chain (MRC) is the major source of energy for most cells in the human body,” explains Lemire. “It captures energy from the food we eat by catalyzing the transfer of electrons from NADH — which is derived from the food — to the oxygen we breathe.”

When the respiratory chain doesn't function properly, the NADH accumulates and is diverted to form lactic acid, which can cause weakness, exercise intolerance, and vomiting, according to Lemire. It may also contribute to developmental delays and long-term progression of mitochondrial diseases by affecting the expression of genes related to energy metabolism.

“We propose to investigate the use of a yeast enzyme called cytochrome *b2* that will act directly to reduce the levels of lactic acid and NADH.” Lemire has identified mutations in a round worm, or nematode, that mimics human mutations in Complex I of the mitochondrial respiratory chain.

“We will introduce the DNA encoding cytochrome *b2* into each of the mutants and evaluate how the yeast protein affects the round worms' fitness by measuring fertility, motility, lifespan, and levels of lactic acid,” explains Lemire.

*Principal Investigator: Giovanni Manfredi
M.D., Ph.D.*

*Institution: Weill Medical College of Cornell
University, New York, NY*

Amount of Award: \$50,000

Award Date: 2003

Dr. Giovanni Manfredi's goal is to better understand the mechanisms of mitochondrial DNA complementation because this can contribute to the identification of novel tools for the treatment of mitochondrial diseases. He has generated a hybrid cell culture model from the fusion of two human cell lines with identical nuclear DNA but each with a distinct mutation in the mtDNA. This model allows his research team to study complementation among mutated mtDNAs in a controlled system.

Mutations in the mitochondrial DNA (mtDNA) cause mitochondrial diseases. Because mtDNA is maternally inherited, mutations in the mtDNA typically result in family pedigrees exhibiting maternal inheritance, ie, the disease should pass only through the females, and essentially all the children inherit the mtDNA mutation.

However, despite this simple way of inheritance, the manifestation of mtDNA-related diseases may be very variable even within the same family. Often, in patients with mitochondrial diseases associated with mtDNA mutations, normal and mutated mtDNAs may coexist within cells and tissues, a condition known as heteroplasmy.

If mutant mtDNA molecules function as independent units, unable to interact across different organelles (parts of the cell), the protective effect of normal molecules coexisting with the mutated ones (heteroplasmy) would be rather limited.

The consequences of randomly occurring new mtDNA mutations, for example acquired during aging, would be more severe in the absence of efficient complementation between normal and mutated mtDNA molecules. This is especially relevant for patients that already harbor an inherited pathogenic mtDNA mutation, in whom the occurrence of acquired mutations may precipitate the clinical phenotype.

INDEPENDENT AUDITOR'S REPORT

To the Board of Trustees of the
United Mitochondrial Disease Foundation, Inc.

We have audited the accompanying statement of financial position of the United Mitochondrial Disease Foundation, Inc. ("the Foundation") as of June 30, 2003, and the related statements of activities, functional expenses and cash flows for the year then ended. These financial statements are the responsibility of the Foundation's management. Our responsibility is to express an opinion on these financial statements based on our audit.

We conducted our audits in accordance with auditing standards generally accepted in the United States of America. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audit provides a reasonable basis for our opinion.

In our opinion, the financial statements referred to above present fairly, in all material respects, the financial position of the United Mitochondrial Disease Foundation, Inc. as of June 30, 2003, and the changes in its net assets and its cash flows for the year then ended in conformity with accounting principles generally accepted in the United States of America.

STELMACK DOBRANSKY & EANNACE, LLC
Pittsburgh, Pennsylvania
August 19, 2003

BALANCE SHEET

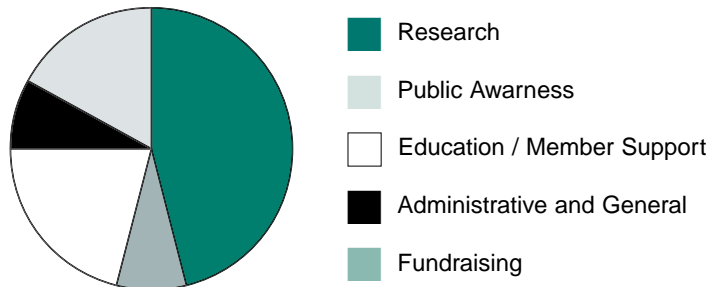
STATEMENTS OF FINANCIAL POSITION
June 30, 2003 and 2002

	2003	2002
ASSETS		
Cash and cash equivalents	\$ 973,683	\$1,113,247
Contributions receivable (Note 2)	99,020	11,250
Interest receivable	702	8,667
Investments (Note 3)	642,050	654,603
Prepaid expenses	7,445	1,935
Fixed assets - net (Note 4)	56,735	45,794
Total Assets	\$1,779,635	\$1,835,496
LIABILITIES		
Accounts payable	47,769	\$ 183,997
Grants payable (Note 5)	382,599	266,500
Deferred revenue	71,204	19,040
Total liabilities	501,572	469,537
NET ASSETS		
Unrestricted	1,102,269	1,352,367
Temporarily restricted (Note 6)	175,794	13,592
Total net assets	1,278,063	1,365,959
TOTAL LIABILITIES AND NET ASSETS	\$1,779,635	\$1,835,496

See Independent Auditor's Report and Notes to the Financial Statements

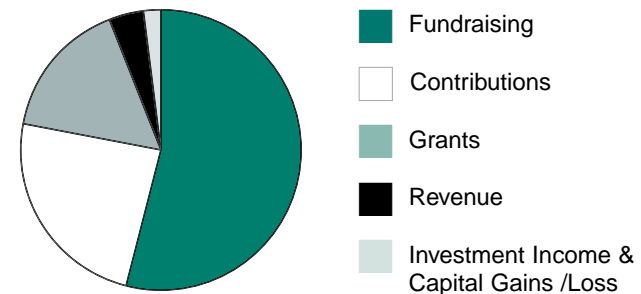
2003 EXPENSES

Research	\$535,989	46%
Public Awareness	\$96,065	8%
Education / Member Support	\$245,368	21%
Administrative and General	\$100,850	8%
Fundraising	\$197,242	17%
Total Expense	\$1,175,514	



2003 REVENUE SOURCES

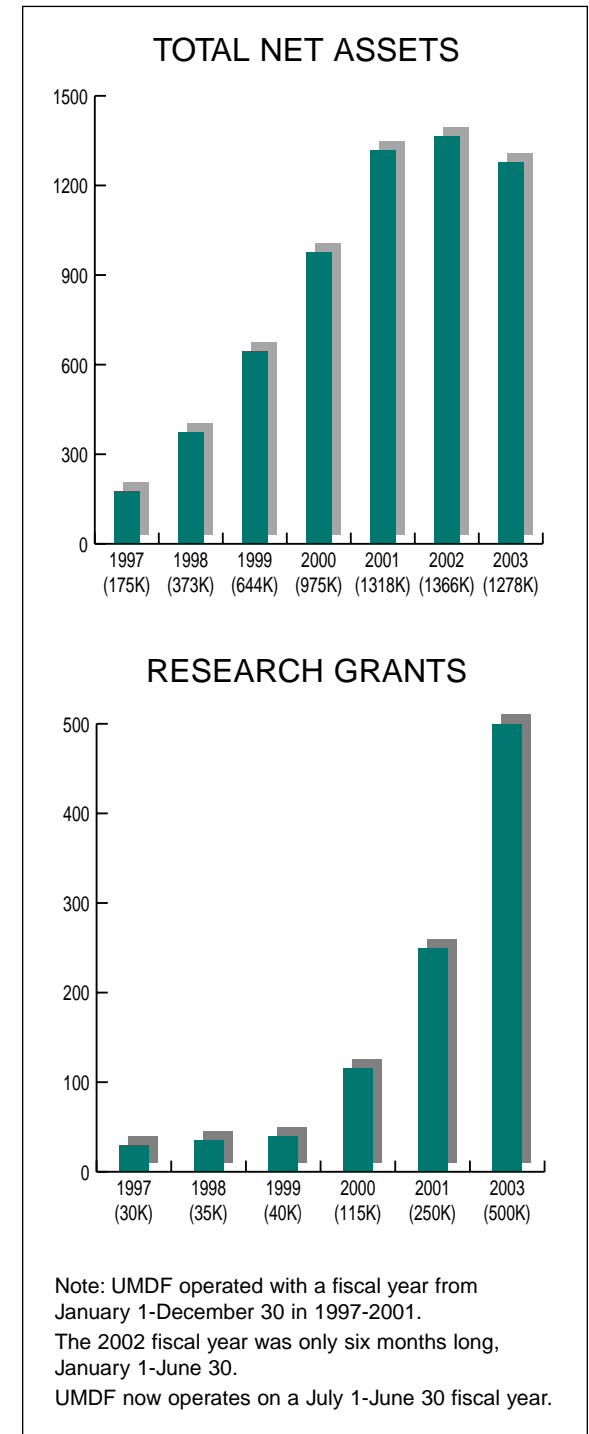
Fundraising	\$589,699	54%
Contributions	\$262,942	24%
Grants	\$173,170	16%
Revenue	\$ 42,544	4%
Investment Income & Capital Gain/Loss	\$ 19,263	2%
Total Public Support and Revenue	\$1,087,618	



Statement of Activities and Changes in Net Assets
For the years ended June 30, 2003 and 2002

	2003			2002
	Unrestricted	Temporarily Restricted	Total	Total
SUPPORT AND REVENUE				
Support:				
Fundraising	\$ 589,699	\$ 0	\$ 589,699	\$ 729,650
Contributions	24,967	12,414	37,381	48,802
In honor of	114,335	0	114,335	174,278
In kind	17,262	0	17,262	20,999
In memory of	93,964	0	93,964	118,404
Grants	0	173,170	173,170	279
Total support	840,227	185,584	1,025,811	1,092,412
Revenue:				
Symposium and seminars	5,230	0	5,230	280,104
Membership	34,859	0	34,859	20,401
Miscellaneous	2,455	0	2,455	1,063
Total revenue	42,544	0	42,544	301,568
Loss on disposal of fixed assets	0	0	0	(39,850)
Investment income	35,958	0	35,958	37,426
Net unrealized gain (loss) on investments	42,274	0	42,274	(89,826)
Net realized gain (loss) on investments	(58,969)	0	(58,969)	0
Net assets released from program restrictions	23,382	(23,382)	0	0
Total public support and revenue	925,416	162,202	1,087,618	1,301,730
FUNCTIONAL EXPENSES				
Program services:				
Research	535,989	0	535,989	268,465
Public awareness	96,065	0	96,065	163,891
Education/member support	245,368	0	245,368	430,249
Total program services	877,422	0	877,422	862,605
Supporting expenses:				
Administrative and general	100,850	0	100,850	84,338
Fundraising	197,242	0	197,242	120,366
Total supporting services	298,092	0	298,092	204,704
Total expense	1,175,514	0	1,175,514	1,067,309
CHANGES IN NET ASSETS				
Changes in net assets	(250,098)	162,202	(87,896)	234,421
Net assets – Beginning of year	1,352,367	13,592	1,365,959	1,131,538
Net assets – End of year	\$1,102,269	\$175,794	\$1,278,063	\$1,365,959

See Independent Auditor's Report and Notes to the Financial Statements



Statement of Cash Flows
For the years ended June 30, 2003 and 2002

	2003	2002
CASH FLOWS FROM OPERATING ACTIVITIES		
Change in net assets	\$ (87,896)	\$ 234,421
Adjustments to reconcile change in net assets provided by (used in) operating activities:		
Depreciation	13,952	11,922
Loss on disposal of asset	0	39,850
Realized (gain) loss on sale of investments	58,969	0
Unrealized (gain) loss on investment	(42,274)	(89,826)
Changes in assets (increase)/decrease:		
Contribution receivable	(87,770)	(11,250)
Interest receivable	7,965	(8,667)
Prepaid expenses	(5,510)	(1,935)
Changes in liabilities (decrease)/increase:		
Accounts payable and accrued expenses	(136,228)	122,036
Grants payable	116,099	266,500
Deferred revenue	52,164	19,040
Net cash provided by (used in) operating activities	<u>(110,529)</u>	<u>582,091</u>
CASH FLOWS FROM INVESTING ACTIVITIES		
Purchase of equipment	(24,893)	(16,048)
Purchase of investments	(416,732)	(125,121)
Proceeds on sale of investments	412,590	0
Net cash provided by (used in) investing activities	<u>(29,035)</u>	<u>(141,169)</u>
CASH AND CASH EQUIVALENTS		
Net increase (decrease) in cash and cash equivalents	(139,564)	440,922
Cash and cash equivalents – Beginning of year	1,113,247	672,325
Cash and cash equivalents – End of year	<u>\$ 973,683</u>	<u>\$1,113,247</u>
SUPPLEMENTAL INFORMATION		
Interest paid	\$ 0	\$ 0
Income taxes paid on unrelated business income	\$ 0	\$ 0

See Independent Auditor's Report and Notes to the Financial Statements

Notes to the Financial Statements
For the years ended June 30, 2003 and 2002

1. SUMMARY OF SIGNIFICANT ACCOUNTING POLICIES

Organization and Nature of Activities

The United Mitochondrial Disease Foundation, Inc. ("the Foundation") was organized on April 28, 1995, and is the result of a merger between a number of specific Mitochondrial disease organizations to form a larger, more cohesive united foundation representing all mitochondrial diseases and all sufferers, adult and children alike. The Foundation's mission is to promote research for cures and treatments of mitochondrial disorders and to provide support to affected individuals and families.

Basis of Accounting

The accompanying financial statements are prepared on the accrual basis of accounting, and accordingly, reflect all significant receivables, payables, and other liabilities.

Change in Year End

Effective January 1, 2002, the Foundation has changed its fiscal year end from December 31 to June 30.

Comparative Financial Information

Due to the change in fiscal year end and in order to present comparative financial statements, the prior year information in the statements of activities and changes in net assets was comprised of six months ended December 31, 2001 and six months ended June 30, 2002. Accordingly, we have not expressed an opinion on the prior year financial statements.

Financial Statement Presentation

The Foundation's financial statements are prepared in accordance with Statement of Financial Accounting Standards (SFAS) No. 117, "Financial Statements of Not-for-Profit Organizations." Under SFAS No. 117, the Foundation is required to report information regarding its financial position and activities according to three classes of net assets: unrestricted net assets, temporarily restricted net assets and permanently restricted net assets.

Contributions

The Foundation records contributions and grants in accordance with SFAS No. 116, "Accounting for Contributions Received and Contributions Made." Under SFAS No. 116, contributions received are recorded as unrestricted, temporarily restricted, or permanently restricted support depending on the existence or nature of any donor restrictions. Contributions that are required to be reported as temporarily restricted support are then reclassified to unrestricted net assets upon expiration/satisfaction of the donor restrictions.

Cash and Cash Equivalents

For purposes of the Statement of Cash Flows, the Foundation considers all highly liquid investments with an initial maturity of three months or less to be cash equivalents. For the years ended June 30, 2003 and 2002, the Foundation had no noncash investing or financing activities for cash flow purposes.

Estimates

Management uses estimates and assumptions in preparing financial statements. Those estimates and assumptions affect the reported amounts of assets and liabilities, the disclosure of contingent assets and liabilities and the reported revenues and expenses. Actual results could differ from those estimates.

Investments

The Foundation records investments in accordance with SFAS No. 124, "Accounting for Certain Investments Held by Not-for-Profit Organizations" which established standards of financial accounting, reporting and disclosures for certain financial securities held by not-for-profit organizations.

Under SFAS No. 124, investments are presented in these financial statements at their current market value. These current market values are established using published market prices.

Fixed Assets

Fixed assets are recorded at cost and depreciated using the straight-line method over estimated useful lives of 5 to 7 years. Depreciation expense, totaling \$13,952 and \$11,922 for the years ended June 30, 2003 and 2002, respectively, is allocated to the various activities based on usage.

Revenue and Expense Recognition

Income from membership dues and program service fees are deferred and recognized over the periods to which the specific types of income relate. Costs and expenses related to such activities are also deferred as prepaid expenses and recognized in the period when the programs are held.

Functional Allocation of Expenses

The costs of providing the various programs and other activities have been summarized on a functional basis in the statement of activities and changes in net assets. Accordingly, certain costs have been allocated among the programs and supporting services benefited.

Income Tax Status

The Foundation is exempt from federal income tax under Section 501(c)(3) of the Internal Revenue Code. However, income from certain activities not directly related to the Foundation's tax-exempt purpose is subject to taxation as unrelated business income. For the years ended June 30, 2003 and 2002, the Foundation had no such income. In addition, the Foundation qualifies for the charitable contribution deduction under Section 170(b)(1)(A) and has been classified as an organization that is not a private foundation under Section 509(a)(1).

Concentration of Credit Risk

Financial instruments which potentially subject the organization to a concentration of credit risk, as defined by FASB Statement No. 105, consist principally of cash, temporary cash investments and marketable securities. The cash, temporary cash investments and marketable security accounts of the organization are maintained at high quality financial institutions. At times such accounts may be in excess of FDIC insurance limits, but pose no significant concentration of credit risk.

2. CONTRIBUTIONS RECEIVABLE (PROMISES TO GIVE)

Pledges of contributions (or promises to give) have been classified as unconditional or conditional. Unconditional promises to give at June 30 are as follows:

	2003	2002
Receivable in less than one year	\$ 50,000	\$ 11,250
Receivable in one to five years	50,000	0
Total unconditional promises to give	100,000	11,250
Less discounts to net present value	980	0
Net unconditional promises to give	<u>\$ 99,020</u>	<u>\$ 11,250</u>

The discount rate used on long-term promises to give was 2%.

Management has deemed these promises to give to be fully collectible, and thus, no allowance for uncollectible pledges receivable has been recorded.

There were no conditional promises to give at June 30, 2003 and 2002, respectively.

3. INVESTMENTS

At June 30, investments were as listed below:

	2003		2002	
	Cost	Market	Cost	Market
Common stocks	\$ 33,418	\$ 6,647	\$ 33,418	\$ 31,058
Mutual funds	647,961	635,403	702,476	623,545
Total investments	<u>\$681,379</u>	<u>\$642,050</u>	<u>\$735,894</u>	<u>\$654,603</u>

4. FIXED ASSETS

Fixed assets are summarized as follows at June 30:

	2003	2002
Furniture and fixtures	\$39,146	\$23,652
Computer equipment	47,740	38,341
Total fixed assets	86,886	61,993
Accumulated depreciation	30,151	16,199
Fixed assets – net	<u>\$56,735</u>	<u>\$45,794</u>

5. GRANTS PAYABLE

Grants authorized but unpaid at year end are reported as liabilities in accordance with SFAS No. 116, "Accounting for Contributions Received and Contributions Made." On March 1, 2001, the Board of Trustees approved future research grants of \$250,000 to be paid to qualified recipients during the year 2002. Of the \$250,000 grant payable at June 30 2002, \$130,559 remains unpaid at June 30, 2003.

In addition, on June 17, 2003, the Board of Trustees approved future research grants totaling \$500,180 to be paid to qualified recipients during the year 2003 and 2004. As of June 30, 2003, \$252,040 was unpaid.

6. NET ASSETS

Temporarily restricted net assets at June 30, 2003 and 2002 are available for the following purposes:

	2003	2002
2004 Symposium	\$ 11,000	\$0
Equipment	5,000	0
Restricted as to time	99,020	11,250
Scholarships	1,000	0
Family support	264	579
Support groups	9,510	0
Fellowship	50,000	1,763
Total	<u>\$175,794</u>	<u>\$13,592</u>

7. OPERATING LEASE

The Foundation leases office space under an operating lease agreement that expires June 30, 2006. The Foundation also leases a copier under an operating lease agreement that expires December 31, 2008. The future minimum rental payments required under these lease agreements are:

Year Ended December 31	Amount
2004	\$ 50,000
2005	51,200
2006	51,600
2007	3,996
2008	3,996
Thereafter	1,998
Total	<u>\$162,790</u>

Rental expense amounted to \$38,154 and \$23,395 for the years ended June 30, 2003 and 2002, respectively.

8. CONTRIBUTED SERVICES

SFAS No. 116 requires contributed services to be recognized if the services received create or enhance nonfinancial assets or require specialized skills, are provided by individuals possessing those skills, and would typically need to be purchased if not provided by volunteers. The Foundation receives such services from community members who volunteer to provide video production, website development and accounting services. The value of these services was calculated as \$17,262 and \$20,999 for the years ended June 30, 2003 and 2002, respectively, and is included in the accompanying Statements of Activities as revenue and expense.

9. CHAPTERS

In addition to the national office of the United Mitochondrial Disease Foundation, Inc., local chapters have also been formed throughout the United States. These chapters include:

- Ohio Chapter
- New England Chapter
- Southern California Chapter
- Delaware Valley Chapter
- Arizona Chapter
- Wisconsin Chapter
- New Mexico Chapter
- New York Metro Chapter

Each chapter is required to file an application for their own employer identification number, abide by their signed chapter affiliation agreement and by-laws and to provide the national office their monthly chapter finance report. The Foundation has received a group exemption under 501(c)(3) of the Internal Revenue Code, and accordingly, will file a group tax return for the chapters. The primary purpose of the chapters is to provide a support network and conduct charitable fundraising activities for the Foundation. The chapters meet the requirements for consolidation and accordingly, their balances are included in the accompanying financial statements. As of June 30, 2003, the Wisconsin and New Mexico Chapters have disbanded their Chapters.

The Statements of Financial Condition include the cash balances of each chapter as of June 30 as follows:

	2003	2002
Ohio Chapter	\$ 5,230	\$ 160
New England Chapter	414	4,970
Southern California Chapter	773	7,430
Delaware Valley Chapter	21,083	686
Arizona Chapter	685	650
Wisconsin Chapter	0	973
New Mexico Chapter	0	566
New York Metro Chapter	0	0

9. CHAPTERS

The Statements of Activities and Change in Net Assets for the year ended June 30, includes the activity for each chapter as follows:

	2003	
	Revenue	Expenses
Ohio Chapter	\$194,386	\$59,388
New England Chapter	44,282	1,425
Southern California Chapter	7,274	1,664
Delaware Valley Chapter	65,330	9,313
Arizona Chapter	2,364	57
Wisconsin Chapter	7,930	959
New Mexico Chapter	0	335
New York Metro Chapter	33,620	4,244
Total	<u>\$355,186</u>	<u>\$77,385</u>



UNITED MITOCHONDRIAL DISEASE FOUNDATION

8085 Saltsburg Road, Suite 201
Pittsburgh, PA 15239
Phone 412-793-8077
Fax 412-793-6477
email: info@umdf.org
<http://www.umdf.org>

UMDF MISSION

***To promote research for cures and treatments of
mitochondrial disorders and to provide
support to affected individuals and families.***