

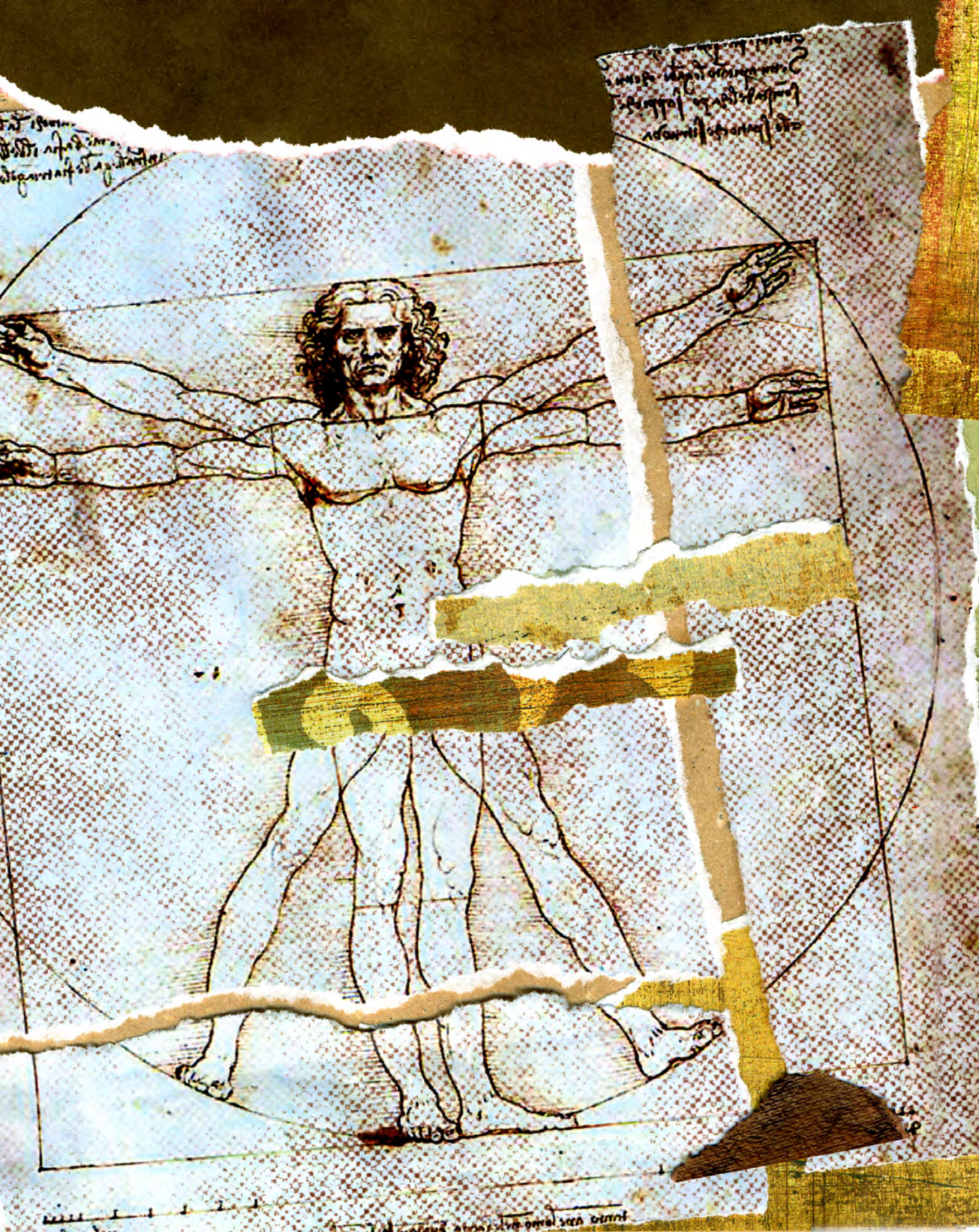
OMRF

2008 Annual Report

2008

Head to Toe

Oklahoma Medical Research Foundation



Head. Neck. Torso. Two arms. Two legs. Simple enough?

Okay, let's try a slightly different formulation: One hundred trillion cells. And that, in a nutshell, captures the paradox that is the human body, something at once elegantly straightforward and staggeringly complex.

When Leonardo da Vinci sketched his Vitruvian man in 1487, physicians believed that the body was composed of four humors: black bile, yellow bile, blood and phlegm. Sickened, they theorized, occurred due to an imbalance in the humors. Thus, when a patient fell ill, the physician would prescribe a regimen of therapy designed to restore the equilibrium.

Five hundred twenty-one years later, da Vinci's drawing still stands as the model of the human form. Its observations about ideal human proportions (the armspan equals the height, the distance from the hairline to the eyebrows is one-third of the length of the face) have largely withstood the test of time. Meanwhile, you'd be hard-pressed to find a modern physician who treats feverish patients with blood-letting and purging.

Thanks to advances in medical research, we now know that the body of an average adult contains about 6.7×10^{27} atoms and is composed of 60 different chemical elements. And that non-steroidal anti-inflammatories are more likely to control a fever than a good leeching.

**Still, even today, what we understand
about the human body is dwarfed by
what we still haven't figured out.**

At the Oklahoma Medical Research Foundation, we work each day to unlock those mysteries. In these pages, you'll learn about some of the remarkable progress we made in 2008.

At OMRF, scientists employ technology—DNA sequencing, cell sorting, nuclear magnetic resonance spectroscopy—that would boggle the mind of a 15th-century physician. But our mission remains one with his: helping people live longer, healthier lives.



Stephen M. Prescott, M.D.
President



BRAIN

When doctors diagnosed Sen. Edward Kennedy with a malignant brain tumor in the spring of 2008, the outlook was bleak.

The average prognosis for the most aggressive form of this tumor, known as a glioma, is approximately 15 months, while those suffering from slower growing tumors might expect to live two to four years.

At OMRF, two scientists are exploring a promising new therapy that could one day change those grim statistics. Working with an experimental compound, Drs. Rheal Towner and Robert Floyd found that, in rodents, the drug significantly shrinks the tumors.

"We've seen dramatic effects on the same kind of tumor that Senator Kennedy has," says Floyd. "If the drug worked the same way in humans, it would, at a minimum, extend lives. And if it worked really well, it might suppress the tumors indefinitely."

The compound has already been tested for safety in humans in large-scale clinical trials, and it was found to be safe. The next step, which the scientists hope to take in 2009, will be to initiate human trials to study the drug's efficacy in treating gliomas, the same form of brain cancer.

According to the American Cancer Society, more than 21,000 Americans will develop brain and nervous system cancers in 2009, and about 13,000 people will die from these conditions this year. "Brain cancers are a devastating medical problem," says Towner. "Although there's still a long road ahead, this research holds the potential to change lives."



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EXPLOSIONS.
JET
ENGINES.
BURSTS
OF
GUNFIRE.

IN TODAY'S MILITARY, SOLDIERS' EARS ARE UNDER CONSTANT ATTACK. Even with external hearing protection, the sounds of warfare can damage the sensitive inner ear, or cochlea, and severely reduce hearing.

But a new drug combination, developed through a collaboration between OMRF and the Hough Ear Institute, has shown promise in reducing hearing loss. The treatment could have both military and civilian applications.

OMRF's Dr. Robert Floyd, working with Hough CEO Dr. Richard Kopke, found that a combination of two compounds—4-OHPBN nitron and the drug n-acetyl-cysteine—could stop damage to the inner ear caused by acute acoustic trauma.

"This is a very exciting finding," says Floyd. "The research is still at a pre-clinical stage, but we're hopeful that we soon can begin testing in humans."

Without the medication, prolonged exposure to loud noise can damage and kill hair cells that register sound, causing hearing loss. But in laboratory animals, the hearing loss was almost completely prevented if the drug combination was given within four hours of exposure to noise levels that would otherwise cause acoustic trauma. Significant decreases were also seen if the combination was administered within 24 hours of exposure.

"If this therapy ultimately proves effective," says Floyd, "it could also have many civilian applications, including combating age-related hearing loss."

HEARING LOSS IS THE SECOND MOST COMMON TYPE OF DISABILITY AMONG VETERANS, ACCOUNTING FOR MORE THAN 75,000 CASES. IT IS ALSO THE MOST COMMON INJURY FOR WHICH PEOPLE ARE EVACUATED FROM A WAR ZONE.

H E A R T

ward R Polley

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Finding new recipes for heart health

A 2008 study by scientists at OMRF and Penn State University found that eating modest amounts of pistachios helped decrease cellular inflammation, cholesterol levels and risk of heart disease. The findings appeared in *The American Journal of Clinical Nutrition*.

With OMRF's Dr. Petar Alaupovic performing lipid measurements for the study, the researchers found that daily intake—one to two handfuls—of pistachios reduced risk for cardiovascular disease by significantly reducing levels of low density lipoprotein (LDL or “bad”) cholesterol.

The researchers studied 28 men and women whose average bad cholesterol level was “borderline high.” They ate a diet rich in cheese, oil and butter before they switched to low-fat diets. After the switch, they incorporated pistachios into their meals and found that bad cholesterol levels dropped by 12 percent when two daily servings of pistachios were eaten. Good cholesterol levels did not change.

“It appears from this study that nuts in general, and pistachios in this case, help bad cholesterol,” Alaupovic says. “So they’re not just pleasant to eat, but they’re also beneficial for your health.”


**Just because they
taste good doesn't mean
they're bad for you.**



LUNGS



**IN THE
FIGHT
AGAINST
INFECTION,
THE HUMAN
IMMUNE
SYSTEM
ISN'T
READY FOR
A WAR.**



ALTHOUGH VACCINES PUSH THE IMMUNE SYSTEM TO CREATE DEFENSES AGAINST ILLNESS, THEY TAKE TIME TO WORK.

But a new process developed by scientists at OMRF stands to revolutionize the process, creating the potential to quickly and effectively create new treatments for influenza and a variety of other communicable diseases that strike the **LUNGS**.

OMRF scientists unveiled their discovery in a 2008 paper in the journal *Nature*. The new process develops a “smart bomb” for the immune system, fighting infection without doing any harm to the body. While the research is aimed at combating influenza, it can be used to create treatments for any condition—such as anthrax or pneumococcal pneumonia—for which there is already a vaccine.

“Vaccines can activate the immune system, but they need time to take effect, and many offer less than 100 percent protection and carry risks of side effects,” says OMRF President Stephen Prescott. “With further research and testing, this new method might allow a nurse going into the center of an outbreak to receive a shot to keep her safe from infection. Soldiers in the field could keep a shot of anti-anthrax in their packs in case of a biological attack.”

“WE NOW HAVE AN OUTSTANDING OPPORTUNITY TO CREATE TREATMENTS FOR A HOST OF DISEASES,” SAYS DR. JUDITH JAMES, WHO IS LEADING EFFORTS AT OMRF TO DEVELOP DISEASE-FIGHTING THERAPEUTICS. “THIS DISCOVERY HAS GREAT CLINICAL POTENTIAL.”

JO



INTS

LUPUS is a devastating illness that affects as many as 2 million Americans and 15 million worldwide.

For nearly everyone who suffers from lupus, each day brings a constant struggle with joint pain, stiffness and arthritis.

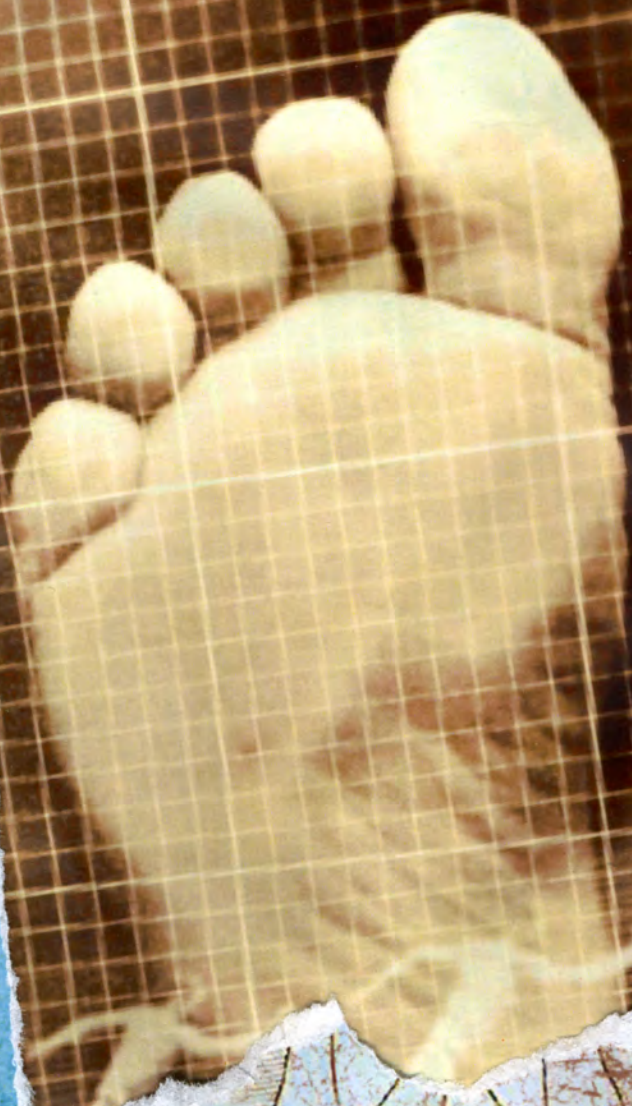
With mis-wired immune systems, those with lupus are subject to constant attacks on the joints in their hands, wrists and feet. Everyday tasks as simple as buttoning a shirt or chopping vegetables become excruciating. As the disease progresses, the attacks can also extend to the shoulders, knees and ankles. "Too often, living with lupus means living with chronic pain," says Dr. John Harley, who chairs OMRF's Arthritis and Immunology Research Program.

***The findings represent the culmination of a massive research effort spearheaded by OMRF; the project involved 7,000 research volunteers and 150 scientists and staff at more than a dozen institutions across the U.S. and Europe.**

In 2008, Harley and his OMRF colleagues made major strides toward unmasking the genetic roots of lupus. In a trio of watershed studies in the journal *Nature Genetics*, Harley and Drs. Swapan Nath, Patrick Gaffney and Kathy Moser identified 14 genes associated with lupus. Those genes included four previously thought unconnected to the disease and a region once believed to be "junk DNA," a piece of genetic material without any known function.

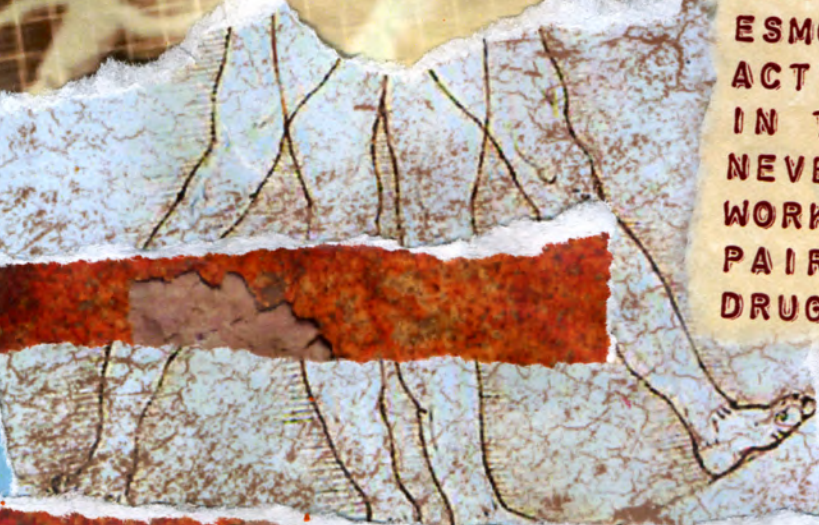
"As clinical investigators, our goal is to reduce the burden of suffering caused by this disease," says Harley. "These new findings promise to transform our understanding of lupus and to accelerate the day when safe and effective therapies are available."

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FEET

WHEN DR. CHARLES
ESMON BEGAN STUDYING
ACTIVATED PROTEINIC
IN THE 1970S, HE
NEVER IMAGINED HIS
WORK WOULD LEAD TO A
PAIR OF LIFE-SAVING
DRUGS.



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"Back then, we didn't even know what activated protein C did in the body," says the OMRF researcher. "Our goal was to figure it out."

Figure it out he did. Indeed, Esmon's research on the naturally occurring protein helped create two FDA-approved drugs: one for children suffering from life-threatening protein C deficiencies and another for a blood infection that kills more than 200,000 Americans each year.

And now, working with collaborators around the globe, Esmon is exploring a host of new disease applications for activated protein C.

ONE OF THE DISEASES IN ESMON'S CROSSHAIRS IS **DIABETES**, WHICH AFFECTS AN ESTIMATED 23 MILLION AMERICANS AND IS THE LEADING CAUSE OF FOOT AND LOWER-LIMB AMPUTATIONS IN THE U.S.

390

"What we found with sepsis is that activated protein C was protective in situations where you had inflammation, vascular injury and organ damage as part of the disease process," says Esmon. "So now we're looking at conditions like diabetes, where blocking inflammatory response and coagulation and protecting organs could be beneficial." Promising results for the prevention of kidney damage in diabetic mice were published in 2008 in the journal *Nature Medicine*. "If activated protein C has broader implications for people suffering from diabetes and other illnesses," says Esmon, "that would be wonderful."

WONDERFUL.

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Between July 1, 2007, and June 30, 2008, more than 8,000 individuals, corporations, foundations and organizations made gifts to OMRF. Each one of those donations made a difference. In this Honor Roll, we have recognized gifts of \$500 and above. Pledged amounts are listed in full the year they are made; subsequent payments on those pledges will not be listed in future annual reports.

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 Mr. and Mrs. John A. Shelley
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 Mrs. Barbara J. Smith
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 Mr. and Mrs. Kenneth A. Stanley
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 Miss Joy Sullivan
 Superstition Sunrise RV Resort
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DISCOVERIES CAMPAIGN

OMRF is poised to play a key role in the revolution in biomedical science that tomorrow will bring. With our staff of world-renowned scientists, we will perform the research that drives the next generation of life-changing discoveries. To this end, OMRF has launched the most ambitious expansion in our 63-year history—the Discoveries Campaign.

The centerpiece of this effort will be a new “green” research tower, the first lab building in North America to tap both wind and solar energy as power sources. This new structure will add 180,000 square feet of state-of-the-art scientific and administrative space, and it will serve as a home to the most precious resource in biomedicine: human capital.

We have set the total fundraising goal for the Discoveries Campaign at \$125 million: \$85 million to construct and furnish the building and \$40 million to recruit 30 world-class scientists and equip their labs. Achieving this goal will ensure that Oklahoma stays on the leading edge of biomedical research. And that OMRF scientists continue making discoveries that make a difference.

Leadership Gifts and Pledges

\$1,000,000 and above

J.A. and Leta Chapman Trusts
 Mary K. Chapman Foundation
 David Chernicky
 Hocker Foundation
 Aubrey and Katie McClendon
 Samuel Roberts Noble Foundation Inc.
 Presbyterian Health Foundation
 The Rainbolt Family
 State of Oklahoma Opportunity Fund

\$500,000 – \$999,999

John and Virginia Groendyke
 Inasmuch Foundation
 McCasland Foundation
 Sarkeys Foundation

\$250,000 – \$499,999

Ann Simmons Alspaugh
 William Randolph Hearst Foundation
 Puterbaugh Foundation
 Dr. John H. Saxon III

Total gifts/pledges less than \$ 250,000 \$ 1,559,230

Campaign total as of December 31, 2008 \$47,708,230

SELECTED 2008 PUBLICATIONS

Arthritis and Immunology Research Program

Carreras E, Turner S, Paharkova-Vatchkova V, Mao A, Dascher C, Kovats S. Estradiol acts directly on bone marrow myeloid progenitors to differentially regulate GM-CSF or Flt3 ligand-mediated dendritic cell differentiation. *J Immunol* 180:727-738, 2008.

Dozmorov MG, Kyker KD, Hauser PJ, Saban R, Bueth DD, Dozmorov I, Centola MB, Culkun DJ, Hurst RE. From microarray to biology: an integrated experimental, statistical and in silico analysis of how the extracellular matrix modulates the phenotype of cancer cells. *BMC Bioinformatics* 9 Suppl 9:S4, 2008.

Graham RR, Cotsapas C, Davies L, Hackett R, Lessard CJ, Leon JM, Burtt NP, Guiducci C, Parkin M, Gates C, Plenge RM, Behrens TW, Wither JE, Rioux JD, Fortin PR, Graham DC, Wong AK, Vyse TJ, Daly MJ, Altschuler D, Moser KL, Gaffney PM. Genetic variants near TNFAIP3 on 6q23 are associated with systemic lupus erythematosus. *Nat Genet* 40:1059-1061, 2008.

International Consortium for Systemic Lupus Erythematosus Genetics (SLEGEN), Harley JB, Alarcón-Riquelme ME, Criswell LA, Jacob CO, Kimberly RP, Moser KL, Tsao BP, Vyse TJ, Langefeld CD, Nath SK, Guthridge JM, Cobb BL, Mirel DB, Marion MC, Williams AH, Divers J, Wang W, Frank SG, Namjou B, Gabriel SB, Lee AT, Gregersen PK, Behrens TW, Taylor KE, Fernando M, Zidovetzki R, Gaffney PM, Edberg JC, Rioux JD, Ojwang JO, James JA, Merrill JT, Gilkeson GS, Seldin MF, Yin H, Baechler EC, Li QZ, Wakeland EK, Bruner GR, Kaufman KM, Kelly JA. Genome-wide association scan in women with systemic lupus erythematosus identifies susceptibility variants in ITGAM, PXX, KIAA1542 and other loci. *Nat Genet* 40:204-210, 2008.

Nath SK, Han S, Kim-Howard X, Kelly JA, Viswanathan P, Gilkeson GS, Chen W, Zhu C, McEver RP, Kimberly RP, Alarcón-Riquelme ME, Vyse TJ, Li QZ, Wakeland EK, Merrill JT, James JA, Kaufman KM, Guthridge JM, Harley JB. A nonsynonymous functional variant in integrin- α (M) (encoded by ITGAM) is associated with systemic lupus erythematosus. *Nat Genet* 40:152-154, 2008.

Sawalha AH, Kaufman KM, Kelly JA, Adler AJ, Aberle T, Kilpatrick J, Wakeland EK, Li QZ, Wandstrat AE, Karp DS, James JA, Merrill JT, Lipsky P, Harley JB. Genetic association of interleukin-21 polymorphisms with systemic lupus erythematosus. *Ann Rheum Dis* 67:458-461, 2008.

Wrammert J, Smith K, Miller J, Langley WA, Kokko K, Larsen C, Zheng NY, Mays I, Garman L, Helms C, James J, Air GM, Capra JD, Ahmed R, Wilson PC. Rapid cloning of high-affinity human monoclonal antibodies against influenza virus. *Nature* 453:667-671, 2008.

Wren JD. URL decay in MEDLINE—a 4-year follow-up study. *Bioinformatics* 24:1381-1385, 2008.

Cardiovascular Biology Research Program

Fu J, Gerhardt H, McDaniel JM, Xia B, Liu X, Ivanciu L, Ny A, Hermans K, Silasi-Mansat R, McGee S, Nye E, Ju T, Ramirez MI, Carmeliet P, Cummings RD, Lupu F, Xia L. Endothelial cell O-glycan deficiency causes blood/lymphatic misconnections and consequent fatty liver disease in mice. *J Clin Invest* 118:3725-3737, 2008.

Johnson CM, Chichili GR, Rodgers W. Compartmentalization of phosphatidylinositol 4,5-bisphosphate signaling evidenced using targeted phosphatases. *J Biol Chem* 283:29920-29928, 2008.

Miner JJ, Xia L, Yago T, Kappelmayer J, Liu Z, Klopocki AG, Shao B, McDaniel JM, Setiadi H, Schmidtke DW, McEver RP. Separable requirements for cytoplasmic domain of PSGL-1 in leukocyte rolling and signaling under flow. *Blood* 112:2035-2045, 2008.

Yago T, Lou J, Wu T, Yang J, Miner JJ, Coburn L, Lopez JA, Cruz MA, Dong JF, McIntire LV, McEver RP, Zhu C. Platelet glycoprotein Ibalph forms catch bonds with human WT vWF but not with type 2B von Willebrand disease vWF. *J Clin Invest* 118:3195-3207, 2008.

Cell Cycle and Cancer Biology Research Program

Conrad MN, Lee CY, Chao G, Shinohara M, Kosaka H, Shinohara A, Conchello JA, Dresser ME. Rapid telomere movement in meiotic prophase is promoted by NDJ1, MPS3, and CSM4 and is modulated by recombination. *Cell* 133:1175-1187, 2008.

Vorozhko VV, Emanuele MJ, Kallio MJ, Stukenberg PT, Gorbsky GJ. Multiple mechanisms of chromosome movement in vertebrate cells mediated through the Ndc80 complex and dynein/dynactin. *Chromosoma* 117:169-179, 2008.

Free Radical Biology and Aging Research Program

Applegate MA, Humphries KM, Szveda LI. Reversible inhibition of alpha-ketoglutarate dehydrogenase by hydrogen peroxide: glutathionylation and protection of lipoic acid. *Biochemistry* 47:473-478, 2008.

D'Souza A, Kurien BT, Rodgers R, Shenoi J, Kurono S, Matsumoto H, Hensley K, Nath SK, Scofield RH. Detection of catalase as a major protein target of the lipid peroxidation product 4-HNE and the lack of its genetic association as a risk factor in SLE. *BMC Med Genet* 9:62, 2008.

Genetic Models of Disease Research Program

Clarke M, Maddera L, Harris RL, Silverman PM. F-pili dynamics by live-cell imaging. *Proc Natl Acad Sci USA* 105:17978-17981, 2008.

Edwards SL, Charlie NK, Milfort MC, Brown BS, Gravlin CN, Knecht JE, Miller KG. A novel molecular solution for ultraviolet light detection in *Caenorhabditis elegans*. *PLoS Biol* 6:e198, 2008.

Immunobiology and Cancer Research Program

Chen X, Esplin BL, Garrett KP, Welner RS, Webb CF, Kincaid PW. Retinoids accelerate B lineage lymphoid differentiation. *J Immunol* 180:138-145, 2008.

Joachims ML, Marble PA, Laurent AB, Pastuszko P, Paliotta M, Blackburn MR, Thompson LF. Restoration of adenosine deaminase-deficient human thymocyte development in vitro by inhibition of deoxynucleoside kinases. *J Immunol* 181:8153-8161, 2008.

Langer M, Malykhin A, Maeda K, Chakrabarty K, Williamson KS, Feasley CL, West CM, Metcalf JP, Coggeshall KM. *Bacillus anthracis* peptidoglycan stimulates an inflammatory response in monocytes through the p38 mitogen-activated protein kinase pathway. *PLoS ONE* 3:e3706, 2008.

Malhotra S, Baba Y, Garrett KP, Staal FJ, Gerstein R, Kincaid PW. Contrasting responses of lymphoid progenitors to canonical and noncanonical wnt signals. *J Immunol* 181:3955-3964, 2008.

Nie L, Perry SS, Zhao Y, Huang J, Kincaid PW, Farrar MA, Sun XH. Regulation of lymphocyte development by cell type-specific interpretation of notch signals. *Mol Cell Biol* 28:2078-2090, 2008.

Nixon JC, Ferrell S, Miner C, Oldham AL, Hochgeschwender U, Webb CF. Transgenic mice expressing dominant-negative Bright exhibit defects in B1 B cells. *J Immunol* 181:6913-6922, 2008.

Takedachi M, Qu D, Ebisuno Y, Oohara H, Joachims ML, McGee ST, Maeda E, McEver RP, Tanaka T, Miyasaka M, Murakami S, Krahn T, Blackburn MR, Thompson LF. CD73-generated adenosine restricts lymphocyte migration into draining lymph nodes. *J Immunol* 180:6288-6296, 2008.

SELECTED FINANCIAL INFORMATION

Operating Fund July 1, 2007 - June 30, 2008

OPERATING REVENUE

Competitive research grants

National Institutes of Health grants	\$ 24,431,808
Other competitive research grants	<u>7,987,712</u>
Total grants	32,419,520

Private contributions

Income and gifts from trusts	7,056,362
Gifts & bequests	285,328
Contributions	1,237,247
Memorials	<u>733,643</u>
Total private contributions	9,312,580

Other revenue

Interest and investment income	3,995,103
Mineral income	2,037,916
Royalties and licensing income	10,080,640
Rent, analytical lab and miscellaneous	<u>1,376,223</u>
Total other revenue	17,489,882

Total revenue 59,221,982

Operating revenue from wills, pledges and other restricted gifts
recorded in prior years

1,334,142

Total operating revenue 60,556,124

OPERATING EXPENSES

Program services - research	47,239,713
Support services - general and administrative	5,248,979
Total operating expenses	52,488,692

Excess of revenues over expenses \$ 8,067,432

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Since 1946, OMRF has been dedicated to understanding and developing more effective treatments for human disease. Our scientists focus on such critical research areas as heart disease, cancer, lupus and Alzheimer's disease. Discoveries at OMRF have helped create three FDA-approved drugs, including first and only treatment for a deadly blood infection that kills 200,000 Americans each year. OMRF researchers have also identified the enzyme believed responsible for Alzheimer's disease, and an experimental drug based on that discovery is now progressing through human clinical trials. *Learn more at OMRF.org/discoveries.*

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