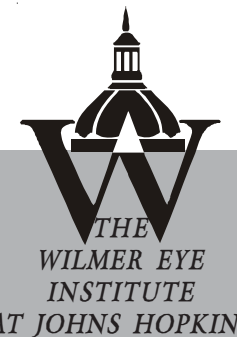


The Wilmer Macular Degeneration Center

Johns Hopkins Hospital
Maumenee 702
600 N. Wolfe Street
Baltimore, MD 21287-9228

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The Newsletter of the Wilmer Macular Degeneration Center

MACFACTS

Volume 8, Issue 1
December 2008

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Progress Report on the Genetics of Macular Degeneration Study at the Wilmer Eye Institute

It is our pleasure to share with study participants, family members and interested individuals, the status of this research project; specifically, where we are, how we got

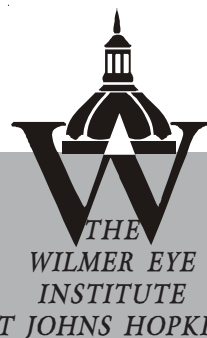
“This is a very exciting time for genetics research. New discoveries are being unveiled at record pace throughout the world.”

here, and what is in sight for the future. This is a very exciting time for genetics research. New discoveries are being unveiled at a record pace throughout the world, thus expanding the breadth and depth of knowledge needed to answer critical basic questions on the causes of this sight-threatening disorder. These new theories are needed to advance investigations that will bring the scientific community closer to piecing the age-related macular degeneration puzzle together.

Overview of Age-related Macular Degeneration (AMD)

AMD is the most common cause of severe, irreversible vision loss among people over the age of 60 in the United States. Since effective treatment is available only for the early stages of the disease, early detection is vital if vision loss from AMD is to be reduced. It is important for

Continued on page 2 . . .



The Newsletter of the Wilmer Macular Degeneration Center

MACFACTS

Building for the Future . . .



The Wilmer Eye Institute, 2010



*The Wilmer Eye Institute
1925*



Overview of AMD . . . continued from page 1

those at risk from AMD to be familiar with its symptoms.

The macula

The macula is located in the central back portion of the retina and is the light-sensitive nerve layer that lines the inner surface of the eye. Light rays from objects we look at are focused on the retina and converted into electrical impulses by the nerve cells in the retina. These electrical impulses are then conducted through the optic nerve to the brain where they are interpreted as visual images. The macula is responsible for sharp central (straight-ahead) vision. Central vision is important for reading, driving a car, and recognizing faces.

Macular degeneration

The macula can degenerate and cease to function properly in a number of eye conditions. This deterioration of the macula is called *macular degeneration*. It is thought that macular degeneration is caused by the interaction of a multitude of factors, broadly grouped into two categories:

- ◆ **Individual characteristics and exposures.** These factors include advanced age, smoking history, high body mass, diet, and sunlight exposure.
- ◆ **Genetic.** The genes that make up an individual can increase the risk for AMD.

When family members have AMD, one's chance of having inherited similar genes is increased. If one has a high-risk genetic makeup, or a genetic predisposition, the risk is increased further by the individual's characteristics and exposures.

There are two forms of AMD: *dry* and *wet*. Dry AMD is also known as non-exudative or non-neovascular AMD and wet AMD is also termed exudative or neovascular AMD.

Dry AMD

Dry AMD is more common than wet AMD, accounting for about 90% of all cases. It develops slowly and is characterized by the death of critical cells in the macula. Dry AMD often causes mild vision loss, described by some affected individuals as a dimming of their vision when they read. The condition may not affect the two eyes equally and some people may only notice decreased vision in the more severely affected eye initially.

Wet AMD

Wet AMD is less common, accounting for about 10% of all cases. However, it is a much greater threat to central vision, and accounts for about 90% of severe vision loss from AMD. In the early stages, abnormal new blood vessels grow underneath the macula (subretinal or choroidal neovascularization), leaking fluid and blood, causing disturbance of central vision. Later, the light-sensitive retinal cells



person is deceased.

To help focus the importance of family history gathering, the U.S. Surgeon General, along with several other organizations, have developed a family health initiative as a national public health campaign. As part of this action, a free website was developed to assist families in drawing their own family trees that include medical information. This user-

“ . . . a free website was developed to assist families in drawing their own family trees that include medical information. This user-friendly site can be found on the internet at <https://familyhistory.hhs.gov/>.”

friendly site can be found on the internet at <https://familyhistory.hhs.gov/>. But drawing your own can also be very easy and straight forward. An example of one pedigree can be seen to the left.

So--go ahead--and somewhere between the turkey and the pumpkin pie, (but before the egnog) start the medical history talking this holiday season. Put it down on paper or computer and then share it with your health care providers as well as your family. Who knows, maybe this activity will help you figure out a great and healthful New Year's resolution!!

-- Kitty Sackett, R.N., C.A.N.P
& Betsy Campochiaro, R.N., M.S.N

Coming To Terms with Terms

Want to know more about some of the terms mentioned in our newsletter? See below for specific definitions and websites you can visit for further information.

◆ **Amsler Grid.** The Amsler Grid is a diagnostic line grid used by eye doctors to detect vision changes caused by damage to the macula. If the straight lines on the grid appear distorted, it may be an indication that the macula has developed irregularities. See <http://www.allaboutvision.com/conditions/amsler-grid.htm> for more details.

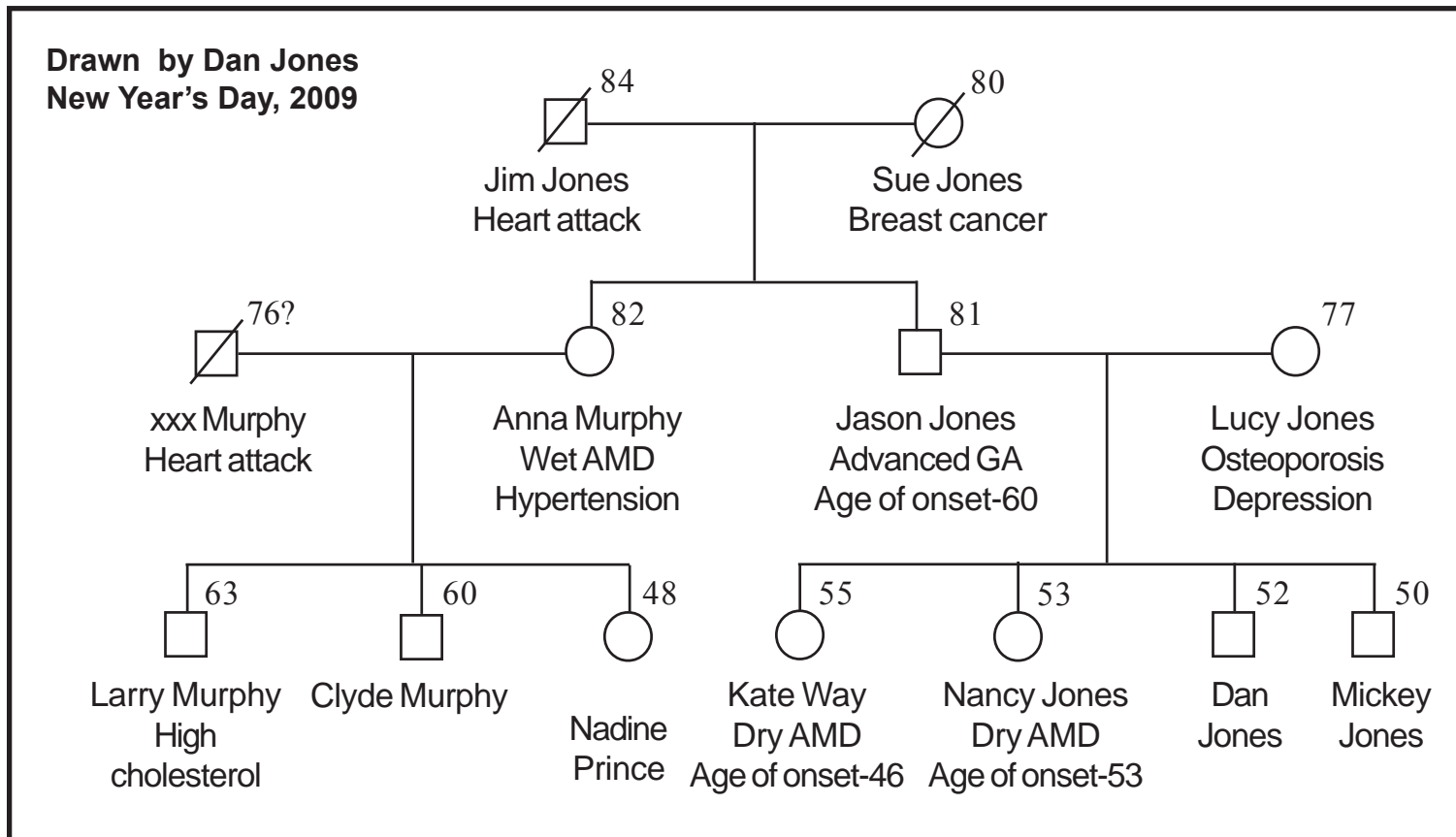
◆ **Gene mutations.** Gene mutations are permanent changes in the make up of a gene that are either random, hereditary, or acquired. Not all mutations are bad; for example, genetic mutations drive evolution. The National Library of Medicine has a comprehensive handbook online to help you better understand the language and concepts of genetics at <http://ghr.nlm.nih.gov/handbook>.

◆ **Stem cells.** Stem cells are found in almost every multi-cellular organism. They are unique in that they have the ability to transform into almost any type of cell in the body. For more information, see the National Institutes of Health Stem Cell Information page at <http://stemcells.nih.gov/info>.

For a selected list of terms that pertain to the anatomy of the eye and AMD, please see the Macular Degeneration website at <http://www.hopkinsmedicine.org/wilmer/services/mdc/terms.html>.



Know Your Family History . . . continued from page 13



This is an example of a family tree. The names are fictitious

she will use it to identify your susceptibility to disorders and diseases.

Ideally, information is gathered from three generations of family members. The best way to record this information is through a family tree, also called a pedigree drawing. This drawing helps to visualize patterns of disease within a family. A sample pedigree is shown above.

Information to record about each family member are: country of origin; age, and if deceased, then age at death (guessing about

age is ok); medical conditions; vision loss and eye diseases if known; and age of onset of conditions. Individuals can be identified with initials. The father is typically drawn on the left and the mother on the right. Children are drawn from oldest to youngest. Males are drawn as a square and females are circles. If you don't know the sex of a family member, such as a distant cousin's child, draw him or her as a diamond. A diagonal line through the shape indicates that



(photoreceptors) degenerate, and scar tissue forms in the macula. This causes a blind spot in the center of the vision.

Symptoms

AMD affects mainly the central vision and the effect can range from mild vision loss to central blindness. It does not cause pain or redness of the eye.

The most common symptom of dry AMD is slightly blurred vision or a blurred spot in the center of the vision. Over time, this blurred spot may become larger and blurrier. Affected individuals may need more light for reading and other tasks.

Wet AMD may also cause blurred or fuzzy vision. In addition, straight lines, such as the edge of this page, telephone poles, or the sides of buildings may appear wavy. A grid pattern, such as the tiles in your bathroom or an Amsler grid, may appear distorted. This happens because of leakage of fluid and blood from the abnormal blood vessels under the macula. The onset of symptoms in wet AMD is often sudden, in contrast to dry AMD, which tends to be more gradual. Over time, a dark or "empty" patch may appear in the center of the vision as photoreceptor degeneration and scarring occurs.

Diagnosis

An ophthalmologist can usually diagnose AMD during an eye exam. The ophthalmologist will be looking for several characteristics of AMD found in the retina:

◆ **Drusen.** Drusen are tiny white or yellow deposits found below the surface of the retina that can be the first signs of disease. Their shape, consistency, and number can help the doctor determine prognosis. Normally, few drusen that are small, round and hard, can be found in the aging eye. Multiple drusen appearing larger and confluent is an indication of macular degeneration. Often individuals do not know they have these deposits because they do not cause any visual changes. The only way to detect this sign is with an exam by a skilled ophthalmologist.

◆ **Pigmentary changes--either light or dark spots--often occur in the macula.** This is due to damage to the retinal pigment epithelial cells. These special cells are located just below the retina layer and they are responsible for nourishing the retinal visual cells. In the early stages of AMD, these cells may appear either faded or more pronounced, depending on the amount of pigment in the cells. As this process progresses, the cells die causing patchy areas of irregular pigment.

◆ **Blood or fluid beneath or within the macula which is an indication of abnormal blood vessel growth.** As mentioned above, the growth of these blood vessels occurs in only approximately 10% of those affected with AMD, but they cause the most vision loss from AMD. The most

Continued on page 4 . . .



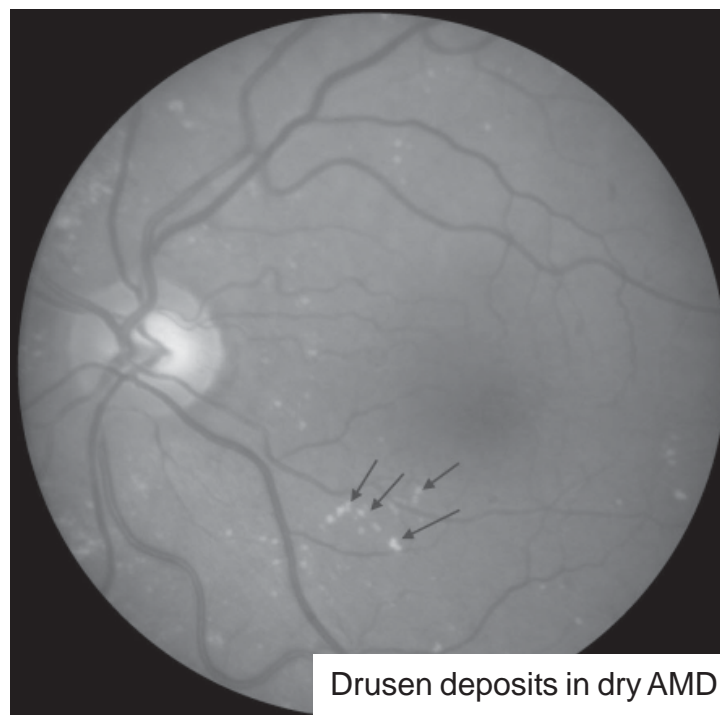
Overview of AMD . . . continued from page 3

sensitive way to detect abnormal blood vessels is with a fluorescein angiogram. This test is usually done in the clinic and involves injection of a yellow dye (fluorescein) into a vein, followed by a rapid sequence of flash pictures of the eyes. Patients usually notice blurred or distorted vision when they have abnormal blood vessels under the macula. See photo insert 1 and 2 (right) for examples of how the retina looks when one has dry AMD and wet AMD.

Treatment

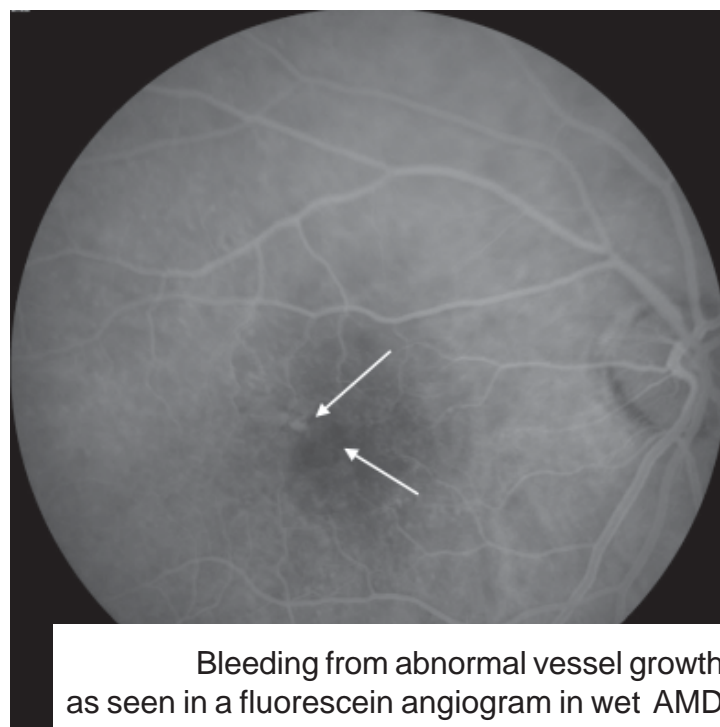
As a result of the Age Related Eye Disease Study (AREDS), vitamin therapy has been found to affect the progression of AMD. It was found that individuals at high risk of progressing to the advanced forms of AMD (either advanced wet or dry) could lower their risk of developing the advanced stages of AMD by 25% over 5 years by using vitamin supplements. The vitamin compound tested in the AREDS study is marketed as Ocuville Preservision and is recommended to patients only after a consultation with their retina specialist.

Currently, the first choice of treatment for the wet type of AMD is drugs that are injected into the eye. These drugs block the factor which stimulates abnormal blood vessels to grow. This factor is called vascular endothelial growth factor or VEGF. There are two drugs that block VEGF-



Drusen deposits in dry AMD

Photo insert 1



Bleeding from abnormal vessel growth as seen in a fluorescein angiogram in wet AMD

Photo insert 2



Know Your Family History

Although a crazy, hectic time of the year is fast approaching, the holidays also provide us with wonderful opportunities to renew and strengthen our family ties. How

“The health of your family, your brothers and sisters, parents, and grandparents can provide insight into health and susceptibility to diseases.”

often have we sat around the holiday dinner table recalling and recounting family legends and remembering those who have come before us? Have you ever considered that these times may offer clues that will offer us and our children a chance to stay healthy?

The health of your family, your brothers and sisters, parents, and grandparents can provide insight into health and susceptibility to diseases. Just as eye and hair color, height, and weight tendencies are inherited from parents, many diseases can also pass from one generation to the next. Macular degeneration is one such disease. If a first degree relative has or had macular degeneration, then this increases the risk that you will get this disease but it does not guarantee that you will develop AMD.

Genetic predisposition, the

environment, and our life style or habits can sometimes collide and create the conditions that can cause disease. Your nutritional status, exposure to cigarette smoking, weight, and having other diseases such as heart disease or hypertension, can increase the chance of getting AMD, but no one factor can cause this disease. Can you affect whether or not you get AMD? While you can't change your genes, you can change your lifestyle to include healthy behaviors. Increasing your physical activity, eating a balanced diet, and not smoking can reduce your chances of getting AMD.

Family gatherings at holiday times provide an excellent opportunity to share health information and update family records. The knowledge gained through

“Family gatherings at holiday times provide an excellent opportunity to share health information and update family records.”

this process may influence you to adopt a healthier life style and also promote regular sharing of disease information among your family members.

An accurate recording of your family history is also an important tool to show your doctor when getting annual exams. He or

Continued on page 14 . . .



This Research Is Made Possible By . . .

. . . the generous support of the Foundation Fighting Blindness, a nonprofit organization that supports leading edge research aimed at prevention, treatment, and cures for retinal degenerative diseases, such as age related macular degeneration. We have enjoyed 11 years of partial funding for this project from the foundation. Their support made it possible to launch and continue our investigations, but the costs of long term genetic studies are too great for one organization to fully fund. Many of you have responded generously to our request for support and we would like to personally thank those who have donated to this project.

- ◆ Mr. Richard Albright
- ◆ Mr. Nezhat Aliabadi
- ◆ Dr. & Mrs. William Binder
- ◆ Mrs. Rosemarie Breczinski
- ◆ Mrs. Carmella Carnibucci
- ◆ Mr. Eugene Caruso
- ◆ Dr. & Mrs. Paul Cline
- ◆ Mr. John Crisp
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- ◆ Mr. Richard Lansburgh

- ◆ Mr. Richard Leahy
- ◆ Sgt. Earl Lipscomb
- ◆ Mrs. Nicole Logan
- ◆ Mr. Raymond Maher
- ◆ Mrs. Rosalyn Mansouri
- ◆ Mrs. Cornelia Merwin
- ◆ Mr. William Murray
- ◆ Mr. & Mrs. William Parker
- ◆ Mrs. Penelope Partlow
- ◆ Mr. William Patterson
- ◆ Mr. Norman Richter
- ◆ Mr. Randy Sigman
- ◆ Mrs. Jean Smith
- ◆ Mr. Frank Susino
- ◆ Mrs. Ernestine Triner
- ◆ Mrs. Ruth Velker
- ◆ Mrs. Gene White
- ◆ The Barth Foundation
- ◆ The Burlington Foundation
- ◆ The Hultquist Foundation
- ◆ And many others who wish to remain anonymous.

We urge you to become a part of this campaign to end vision loss from macular degeneration. Your contributions, large or small, can make an enormous difference to the outcome of this research.

For more information, please contact:
 The Development Office of the Wilmer Eye Institute
 Macular Degeneration Genetics Research
 Attention: Susan Williams
 The Johns Hopkins Medical Institutions
 600 N. Wolfe Street, Wilmer 112
 Baltimore, MD 21287-9015
 (410) 955-2020; email: swilli91@jhmi.edu



Lucentis and Avastin. Usually multiple injections every six to eight weeks are necessary to stop the growth of blood vessels. These drugs have revolutionized the treatment of wet AMD by helping thousands of patients avoid severe loss of vision.

Some patients (about 15%) with early wet AMD can be treated with conventional “hot” laser surgery. This involves directing a very intense light beam (laser) at the abnormal blood vessels to destroy them. Unfortunately, the laser beam also destroys the retina overlying the abnormal blood vessels. If the abnormal blood vessels are outside the center of the macula, good central vision can be preserved because the laser does not affect the center of the macula. On the other hand, if the abnormal blood vessels are in the center of the macula, laser treatment will affect the central vision.

Photodynamic therapy (PDT) with verteporfin (VISUDYNE) is another laser option for treating certain patterns of abnormal blood vessel growth in the wet form of AMD. This treatment destroys abnormal blood vessels with less damage to the macula. But some damage may still occur, which can decrease vision.

Prognosis

Dry AMD usually progresses slowly and can actually come to a point where no more damage occurs to the macula, thus stabilizing vision. Or dry AMD can progress to a more severe form so that the

patient has poor or no central vision. This is referred to as *advanced geographic atrophy*. Wet AMD, if found early and treatment is given, can also stabilize. Or it can cause rapid loss of central vision. Although the advanced forms of AMD can

“The success of past research and treatments now provides great anticipation and hope where little hope previously existed.”

cause poor central vision, the peripheral retina is usually spared and side vision is retained. Affected individuals therefore do not become completely blind. Low vision aids can help those affected make the most of their remaining vision. Many patients continue to enjoy many of their favorite activities and live independently.

Although a great many people with wet AMD have been helped with new drugs, this group represents only a small percentage of those with AMD. There is much work to be done before prevention or cures will be available. Wilmer scientists are meeting this challenge by conducting much research in laboratories and in clinical trials where new treatments are being tested. The success of past research and treatments now provides great anticipation and hope where little hope previously existed.

--Peter Campochiaro, M.D.
& Betsy Campochiaro, R.N., M.S.N



**Volunteers with a Vision:
Who We Need & Why They Join**

The age related macular degeneration (AMD) genetics project was initiated at the Wilmer Eye Institute in 1998. At that time, there were few alternatives for treating patients with AMD. The causes of AMD were little known but the scientific community suspected that genetics played a critical role. The work began at Wilmer to identify the genetic causes of macular degeneration, with the ultimate goal of developing new approaches for the diagnosis and treatment of AMD. This disease often runs in families, and it is through long-term family studies that important discoveries have been made. During the past several years, investigators from around the country have identified several genes that put individuals at increased risk for developing AMD. Knowing these specific genetic risk factors helps the scientists further explore the interactions between these genes and other unknown genes to figure out the disease process. Wilmer initiated this investigation because of the important role genetics will play in preventing and curing this disease in the future.

Successful gene studies on diseases such as AMD require the participation of a large number of patients and family members. We have had the good fortune of working with many dedicated individuals. As of this date, we have recruited and enrolled

over 1600 individuals for the AMD genetics study. This represents over 1000 families.

Study subject recruitment began in the clinics at Wilmer and quickly spread to the entire country. This was due in large part to the networking of family members. The first person in the family came to Baltimore to be seen by a Wilmer doctor and after he or she enrolled in the study, other family member(s) were contacted and enrolled regardless of where they lived. Many people were able to join the study through the mail. As a result of this networking, word quickly spread. Many people continue to enthusiastically volunteer unsolicited.

Participants in the study have given us a small sample of their blood, photos of their retina, and permission to study their genes for factors that might affect whether a person gets AMD. As you will read in the Research Update sections, pages 8 - 10, discoveries have been made due to this terrific and generous response from many people.

In the initial recruitment phase, families with multiple members with the disease were asked to join the study. Individuals within the same family have many genes in common. We look at their genes for changes or mutations in the genes that are suspected to be involved in the disease process. As explained in the article entitled "New Insights into the Genetics of Dry AMD" (page 10), some significant changes were found, but in order to fully understand the meaning and



**The Research Team
of the Macular Degeneration Genetics Study
at the Wilmer Eye Institute**



Donald J. Zack, M.D., Ph.D.
Principal Investigator, Professor of
Ophthalmology, Molecular Biology,
Genetics, & Neuroscience



Peter Campochiaro, M.D.
Professor of Ophthalmology &
Neuroscience



Nicholas Katsanis, Ph.D.
Associate Professor of
Ophthalmology, Molecular
Biology, & Genetics



Betsy Campochiaro, R.N.,
M.S.N.
Research Coordinator



Carol Kelly
Assistant Research
Coordinator



Research Update

New Insights into the Genetics of Dry AMD



You may not have thought that a gene mutation could be good for you, as typically we think of mutations as contributing to the development of disease. Not always, as proven by the collaboration of three groups of scientists - Johns Hopkins University, University of Kentucky, and University of California at San Diego.

This landmark study was published in the *New England Journal of Medicine*, August 27, 2008. Some gene variation can protect you from getting a disease. A gene called toll-like receptor 3 (TLR3) was demonstrated to suppress the death of retinal cells, thus protecting against an advanced stage of dry macular degeneration known as advanced geographic atrophy.

This gene was looked at carefully for several reasons, including prior studies that suggested that, at least in some cases, AMD could be initiated by inflammation, possibly caused by an immune reaction to an invading virus. The TLR3 gene makes a protein that helps the immune system act against bacteria and viruses. After looking at this gene in the DNA of many individuals, comparing DNA sequences between individuals with and without AMD, the investigators concluded that when the TLR3 protein has low activity, it prevents the degeneration of the macula, but when it has

high activity, it can cause cell death, which leads to the central vision loss associated with advanced AMD.

These findings have potential implications for treatment of the dry form of AMD. If a way could be found to prevent the activity of this protein, or to turn off its activity, it might be possible to slow down the death of the cells and thereby stop the disease from progressing to an advanced stage. This would mean help for the one in ten people who have AMD and go on to develop advanced geographic atrophy. Currently, AMD is the leading causes of vision loss in individuals over the age of 60, and 90% of those individuals have dry AMD. Its prevalence is expected to double by the year 2020. This discovery unlocks some of the mystery of this disease, and provides promise and raises hope for future prevention and new treatment strategies.

--Betsy Campochiaro, R.N., M.S.N



We wish to thank all the generous patients who participated in and/or helped to fund the Wilmer AMD genetics study. Your participation helps make this scientific advance possible, and we and patients around the world appreciate it greatly. Hopefully, 2009 will bring even more advances in the genetics of AMD, which together will accelerate the development of newer and more effective strategies for the diagnosis and treatment of AMD.

--The Research Team



implications of these changes scientists need to learn if these changes exist in people without AMD and if so, how the functions are different.

In the current recruitment phase, we are enrolling individuals who are over the age of 70 and who do not think they have AMD. These people are called controls. We will look for differences in genes both in the structure of the DNA and in its function between those with the disease and those without the disease. We are trying to answer many questions such as: How is the normal functioning of the macular cells interrupted? What genes are turned on or off in those with AMD? Do the control participants have a gene that protects the macula? Comparisons between these two groups is so important that recruitment of controls will continue until we have the same number in the study as those with AMD.

In the process of recruiting control subjects, we find some individuals unknowingly have the early stages of AMD. Examination of the eye photos taken at the time of enrollment show changes typical of AMD. While this comes as an unpleasant surprise to the volunteer, these findings are expected in some of our subjects because AMD occurs so frequently in seniors. When this situation happens, we encourage the volunteer to follow up with

their ophthalmologist. Having AMD and not knowing underlines the importance of annual eye exams by an ophthalmologist.

So why would anyone volunteer to help us? It may seem that asking seniors to participate in a research study that may not

“It is due to the efforts of the volunteers that our scientists will be able to pursue better treatments and hopefully a cure for AMD.”

benefit them personally is a great challenge. But that is not the case. We find that many individuals volunteer their time out of concern for others. They want to help friends, families, and future generations. They want to learn more about the disease and to be screened for AMD. We have many who happily reply “Why not?”

It is with great respect, gratitude, and thanks that we continue recruiting many more such generous and committed people to this study. It is due to the efforts of the volunteers that our scientists will be able to pursue better treatments and hopefully find a cure for this disease.

--Betsy Campochiaro, R.N., M.S.N.



Research Update

Development of Neuroprotective and Stem Cell Based Therapies for AMD



The dry form of age-related macular degeneration (AMD) is a disease in which there is generally slow but progressive loss of photoreceptors, the

“There is . . . much excitement about the potential of neuroprotective and stem cell technologies, but at the moment, neither is yet ready for clinical application.”

cells in the eye that sense light and act like the film (or digital chip) in a camera. Development of appropriate treatment for patients with dry AMD will depend on the stage of a patient’s disease. In early stages of the disease, in which there are still many photoreceptors left, treatment strategies can aim at promoting the health and function of these remaining photoreceptors. At later stages of the disease, such “neuroprotective” strategies will have limited benefit since few viable photoreceptors remain. In such cases, cell-based strategies such as the transplantation into the eye of progenitor (or stem) cells that can develop into functioning photoreceptor cells may

some day provide an effective approach for treatment. There is thus much excitement about the potential of neuroprotective and stem cell technologies, but at the moment, neither is yet ready for clinical application.

In studies funded by a Wynn-Gund Translational Research Grant from the Foundation Fighting Blindness, Donald J. Zack, M.D., Ph.D., and his colleagues in the Guerrieri Center for Genetic Engineering and Molecular Ophthalmology at Wilmer are working hard to bring these technologies closer to the clinic. Using an automated robotic microscope system, they are searching thorough libraries containing thousands of molecules for compounds that can:

- 1) protect and increase the survival of cultured photoreceptor cells (relevant to the neuroprotective approach) and
- 2) encourage progenitor and stem cells to develop into photoreceptor cells (relevant to the cell replacement approach).

As part of these studies, techniques have been developed to efficiently introduce DNA into photoreceptor cells that are grown in tissue culture plates (see figure, above right). This new ability to “electroporate” photoreceptor cells with DNA allows the investigators to determine how various genes influence the development, survival, and function of photoreceptor cells. Dr. Zack’s group has already successfully used approaches similar to those described here to identify



**MACFACTS
Volume 8, Issue 1
December 2008**

Editor: Betsy Campochiaro, R.N., M.S.N.
Co-editor: Kitty Sackett, R.N., C.A.N.P.
Layout & design: Anne Meltzer

This newsletter is available on the Macular Degeneration Center website:

<http://www.hopkinsmedicine.org/wilmer/services/mdc/>

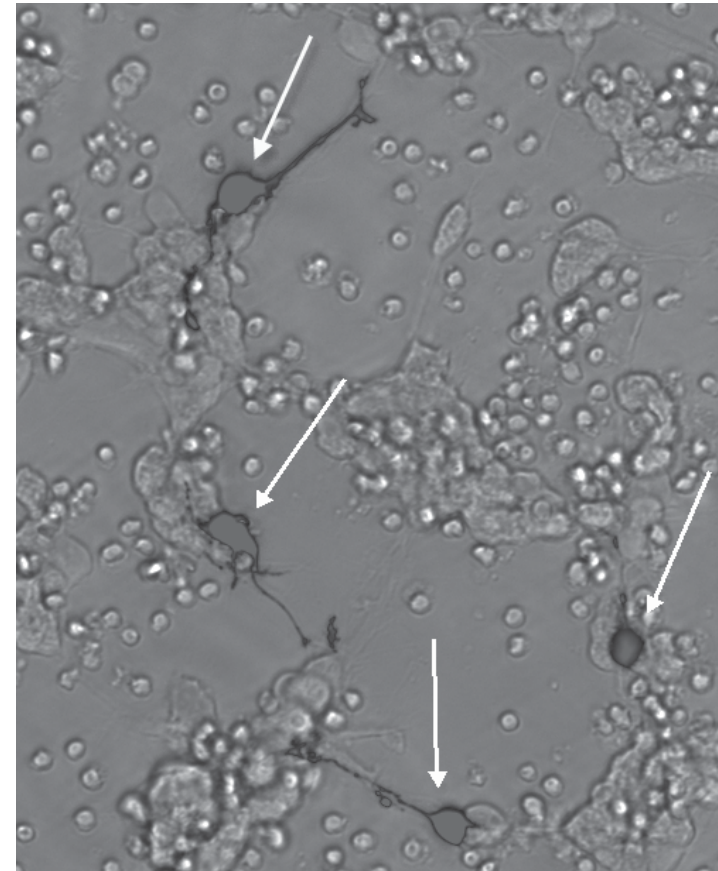
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The Wilmer Macular Degeneration Center
Maumenee 702
600 N. Wolfe Street
Baltimore, MD 21287-9228

Email: mdc@jhmi.edu

General information & referrals:
410-955-5080



Photoreceptor cells in tissue culture

molecules that promote the survival of retinal ganglion cells, the cells that die in glaucoma. We are hopeful that these new studies on photoreceptor cells will also be successful and thereby help in the development of new and more effective treatments for AMD.

--Donald J. Zack, M.D., Ph.D.