PXE Awareness

National Association for Pseudoxanthoma Elasticum (NAPE, Inc.)

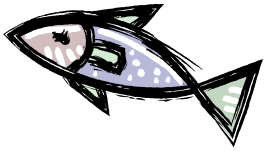
Volume 13, Issue 1 April 2007

PXE and Genetics

Gregor Mendel

…see page 8

Charles Darwin



2007 NAPE Conference…
see pages 24-29

National Association for Pseudoxanthoma Elasticum (NAPE, Inc.)

8760 Manchester Road, St. Louis, MO 63144-2724
Voice & Fax: 314-962-0100
Email: napestlouis@sbcglobal.net Web: www.napxe.org

NAPE, a non-profit 501(c)(3) support group whose mission is to provide education and support for PXE-affected persons, publishes *PXE Awareness*. Articles in this newsletter are provided for information only and are not a substitute for professional medical advice. You should not use information in this newsletter to diagnose or treat medical or health conditions. Please consult your healthcare provider before beginning or changing any course of treatment.

Board of Directors

Medical Advisors

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President's Message

Dear NAPE Friends,

Please read about our 2007 conference in this issue. We believe it will provide an excellent opportunity to gather useful information and to share experiences with others who have PXE.

Important to our discussion will be the drug Avastin. Dr. Chris Bergstrom, MD, of Emory University will explain how it is achieving success in stabilizing leaky subretinal blood vessels and in improving vision. PXE patients report they have benefited greatly from Avastin injections. Retina specialists report optimism for this new treatment. We ask for reports from anyone who has been treated. Please contact me at the NAPE office. We are eager to learn as much as possible about the impact of Avastin on PXE. We are told that an upcoming issue of the medical journal *Retina* will praise Avastin as revolutionary in the treatment of AMD vision loss.

Some doctors are using Lucentis with results similar to those with Avastin, and we want to know that experience also. VEGF-TRAP, now in second phase trials, is being touted as a major breakthrough in improving AMD vision loss. We want to hear from anyone in those trials.

Certain questions recur often in our patient contacts. Perhaps most often are those about the possibility of passing on PXE to the next generation. I have been looking for some time for a biology teacher to provide a basic explanation of such inheritance. Imagine my delight when I accidentally met Ed Ruppert on a flight from Baltimore to St. Louis. We learned that we share admiration for writings of evolutionary biologists. Ed introduced himself as a recently retired high school biology teacher; it didn't take me long to ask him to write "Basic Genetics and PXE" for this issue. I am grateful for Ed's generosity. He and I hope his article will help NAPE readers understand how our inherited recessive disorder will or will not be passed to those yet to be born. I think readers will agree that Ed is an excellent teacher. Thank you, Ed Ruppert!

I am so very pleased that Kattesh V. Katti, PhD, will participate with us in Atlanta where he will share his nanomedical research on AMD. Dr. Katti is interested in PXE and will share his ideas about research using nanomedical techniques and products in treating PXE vision loss.

Have a wonderful Spring.

Fran Benham

Honorary Board Associate

NAPE's Board of Directors, in its Spring Teleconference Board Meeting, established a lifetime Honorary Board Associate membership to recognize those who have made exemplary contributions to NAPE and to our mission of education and patient support for those who have PXE and their families. The Board selected for this award **Elspeth Lax**, former NAPE Board member and President of PiXiE, the first PXE patient advocacy group.

Elspeth, diagnosed with PXE as a young woman, quickly realized a grave lack of knowledge about PXE in the medical community. Her response was to build a network of support in the early 1980's which she continues to lead today. PiXiE served as a model for NAPE which was started in New York State a few years later. Elspeth also helped support groups to organize in Europe. She has participated in many NAPE activities over the years, and in turn has included NAPE leaders in PiXiE activities in Great Britain. Those wishing to learn more about Elspeth's work can tap into PiXiE on the web at www.pxe.org.uk.

Congratulations, and thank you, Elspeth Lax.

Honorary Board Associate

NAPE's Board of Directors, in its Spring Teleconference Board Meeting established a lifetime Honorary Board Associate membership to recognize those who have made exemplary

contributions to NAPE and to our mission of education and patient support for those who have PXE and their families. The Board elected **Jane Tipton** as an Honorary Board Associate. Diagnosed with PXE, Jane became an early member of NAPE as it organized in Denver. She became Treasurer in 1994 until she stepped down from this post and from the Board this year.

A career business woman, Jane brought excellent financial and organizational guidance to NAPE's management. She played a major role in the move of our office to St. Louis. She has offered to provide financial consultation as needed into the future. The Board is pleased to accept her gracious offer. Over the years Jane has been actively involved in the development of NAPE in all its varied activities and in providing support for others who have been diagnosed with PXE.

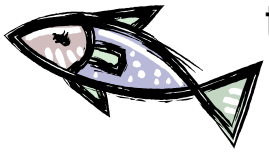
Thank you, Jane Tipton, for your many years of service and friendship for NAPE.

Basic Genetics and PXE

By Ed Ruppert

One cannot discuss this topic without including a respectful nod to Charles Darwin and Gregor Mendel. Although Mendel knew of Darwin's work, Darwin and the scientific community in general did not recognize the importance of the work of Gregor Mendel - an Austrian monk living in a monastery and working with pea plants.

With his collections of fossils and species of animal life, Darwin was able to argue and show that life on Earth had changed over time, and he believed that his concepts of natural selection and survival of the fittest were responsible - that the great variation of life on Earth had somehow come about through natural processes. By noting the variation that he saw within species, and the fact that man had been successfully breeding animals for many years for desired traits, Darwin realized that change did occur over time and that it occurred through the process of sexual reproduction. What Charles Darwin could not explain was the basis for this change. What was it that caused traits that the environment acted upon? Some traits were successful and were passed on through sexual reproduction to the next generation, while other traits disappeared.



The concept of genes, or factors as Gregor Mendel called them, was unknown at that time. Even

though Mendel (1822-1884), and Darwin (1809-1882) were contemporaries, Darwin was not aware of the significance of Mendel's work. It was left to this monk working alone in a small garden to explain how traits in plants and also in animals were passed on to offspring in a predictable mathematical way through sexual reproduction.

Gregor Mendel is the Father of Genetics - he introduced the world to the concept of the gene and showed how these factors (invisible at the time) are passed on to future generations. Genetics is the science that studies how genes work and how they are passed on to our children and from our children to their offspring and into the future.

We have learned much since the time of Mendel and Darwin. Genes are carried on chromosomes and each species, of plants and animals alike, has its own distinctive number of chromosomes. The human species has 46, and these chromosomes and the genes on them occur in pairs - thus we have 23 pairs of chromosomes and perhaps as many as 30,000 genes on the 46 chromosomes.

Almost every cell in our bodies has 46 chromosomes in its nucleus. Almost - because there are a couple of exceptions. Our mature red blood cells do not have a nucleus or chromosomes, and more importantly, our sex cells also known as gametes (also called sperm and eggs) contain only half the 46 number and thus half the usual number

of genes that all of the other body cells contain. We will see in a moment why this is so important.

The letter N is used to indicate one set of chromosomes and the notation 2N refers to two sets. All the somatic (body) cells have the 2N number or two sets whereas the reproductive cells have the N number or one set. For example, skin cells, liver cells, etc., are 2N whereas sperm and eggs are the only cells that are N. When humans reproduce, the mother's egg (N) and the father's sperm (N) unite to make an embryo which is (2N) and all the cells produced as the embryo grows will have the 2 sets of chromosomes (2N) because as cells divide to produce new cells - the two new cells will be exact copies of the original cell. All of this cell division is responsible for growth. The scientific term for this division of cells is MITOSIS.

When a child reaches sexual maturity and starts producing sperm or eggs, a new type of cell division begins. The 2N cells in the testis and ovary begin their own special type of division - this type of cell division is called MEIOSIS and reduces the chromosome number from 2N to N. The result of this is gametes (sperm and eggs) that are now N and have only one set of chromosomes. This reduction of chromosome number maintains the distinctive number of chromosomes for our species. When our children are produced (the sperm's N set combining with the egg's N set forms the 2N condition in the embryo) they will have the same number of chromosomes as their parents -

maintaining the constant number of chromosomes for our species - 46.

So we see that our cells receive half their genes from our fathers and half their genes from our mothers. These genes are responsible for our traits: height, eye color, blood type, etc. Sometimes one pair of genes (one gene from dad and one gene from mom) will determine a trait - say blood type.

Let's follow blood type as an example: say your mother has type A blood and she passes the A gene on to you through her egg, and say your father has type O blood and he passes the O gene on through his sperm. You do not end up with two blood types - you will only have one - either A or O. In this example the A gene is the DOMINANT gene and will hide (mask) the O gene and you will have type A blood. The O gene has not disappeared; it is still in your cells and thus will be in some of your gametes, and even though you have type A blood you may one day produce a child with type O blood because you are also a carrier of the O gene although it is RECESSIVE and is hidden by the dominant A gene. So genes are either dominant or recessive. The recessive genes will be hidden by dominant genes but if there is no dominant gene in the pair the two recessive genes will then make up the pair and the recessive trait will appear in the offspring.

Frequently in genetic disorders the mother and father are normal (they will not have the genetic

problem) because they have the dominant normal gene that is hiding the other abnormal gene of the pair that causes the problem (the recessive one). If both the father's sperm and mother's egg carry the recessive gene when they meet and unite at fertilization, the baby will have two recessive genes making up the pair and there will not be a dominant gene to hide the problem gene. These recessive genes will be expressed and the child will have the genetic problem.

A simple array of four squares can be used to examine more closely the genetics that can occur in an embryo that is formed at fertilization. Let's use the blood type example again. There are four different blood types for this trait: A, B, AB and O. The A and B genes are both dominant genes, and the O is a recessive gene. Let's look at a couple of examples.

Example 1: One parent has AB blood and the other parent has O blood. Their pairs of genes can be written as: AB and OO (this is before meiosis occurs to make the sperm and eggs which will reduce the chromosome number to N). The AB parent can produce either A gametes or B gametes and the other parent can only produce O gametes during meiosis. By placing the possible gametes of one parent along the top of the array and the possible gametes that the other parent can produce along the side of the array as seen below, one can

see not only the possible results of fertilization but also the probabilities for those results.

AB x OO

	A	B
O	AO	BO
O	AO	BO

Remember that the O gene is recessive and is hidden. The four children in the inside boxes in this example will have either blood type A or blood type B. They have a 2 in 4 or 50% chance of having type A and the same odds for type B blood. These parents cannot produce a child with type O blood even though one parent has type O blood.

Example 2: Say the parents have these genes for this blood trait: AO and BO:

AO x BO

	A	O
B	AB	BO
O	AO	OO

In this example these parents can produce children with any blood type: A, B, AB or O. There is a 1 in 4 or a 25% chance that a child will be one blood type or one of the other three blood types.

Example 3: Now let's use PXE in this example. We assume that PXE is carried by a recessive gene and will show up as a genetic disorder in a child only when each parent is a carrier of the recessive gene and each passes the recessive gene to the child via their gametes. The two recessive genes unite at fertilization (when the sperm meets the egg). Let's use the following letters to follow this through: N = the normal gene, n = the PXE gene. Let's use an example where both parents do not have the disorder but are both carrying the recessive gene for PXE. Their pairs of genes will be: Nn and Nn.

Nn x Nn

	N	n
N	NN	Nn
n	Nn	nn

There is a 3 in 4 (75%) chance that any pregnancy will produce a normal child (NN, Nn, Nn) and a 1 in 4 (25%) chance that any pregnancy will produce a child with PXE (nn). There is a 2 in 4 (50%) chance that a child born to them will not have PXE but will

be a carrier of the recessive gene (Nn) for PXE. There is a 1 in 4 (25%) chance that any pregnancy will produce a child who does not have PXE and is not a PXE carrier.

More examples of the presence or absence of the PXE gene in the sperm and/or egg at fertilization follow:

1. One parent has PXE - this parent will have two recessive genes making up the pair of genes for this trait and will be (nn). 100% of the gametes of this parent (sperm or eggs) will carry the recessive gene for PXE and pass the gene on to 100% of his/her children. The other parent is perfectly normal - his/her two genes for the trait will be (NN). This parent will pass the normal, dominant gene on to all of the children guaranteeing that every pregnancy will produce a normal child without PXE (Nn).

nn x NN

	n	n
N	Nn	Nn
N	Nn	Nn

The children in this example will all have (Nn). In other words they will all have the dominant gene (N) that will mask the recessive gene (n) and there

will be no chance that their children will have PXE (0%). On the other hand 100% of their offspring will be carriers of the recessive gene for PXE (Nn) and will be able to pass this gene on to their children (the grandchildren of the original parents).

2. If one parent is normal but is a carrier of the recessive PXE gene (Nn) and the other parent is perfectly normal and does not have the PXE gene (NN) the following diagram represents this situation.

NN x Nn

	N	N
N	NN	NN
n	Nn	Nn

In this example 100% of the children will be normal - every pregnancy will produce a child without PXE. There is a 50% chance though that each pregnancy will produce a child who will be a carrier of the PXE gene (Nn) but will not have PXE.

3. In this example let's assume that one parent is a carrier of the PXE gene (n) but has the dominant normal gene (N) to mask the recessive PXE gene (n) and thus will be (Nn). The other parent will have the PXE trait (nn) and all of his/her gametes will carry the PXE gene.

Nn x nn

	N	n
n	Nn	nn
n	Nn	nn

There is a 2 in 4, (50%) chance that each pregnancy will produce a child with the PXE trait (nn) and a 2 in 4, (50%) chance that each pregnancy will produce a child who does not have PXE but is a carrier for the trait (Nn).

4. If both parents have PXE they will both have the double recessive pair of genes for this trait (nn) and 100% of their sperm and eggs will carry the PXE gene (n) on to their children. This will be very unusual because PXE is rare and for two individuals with PXE to meet and have children will seldom happen.

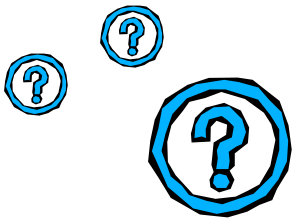
nn x nn

	n	n
n	nn	nn
n	nn	nn

If it did occur though, all of the sperm and eggs of these two parents will carry the PXE gene(n) and at fertilization every pregnancy will result in a child with two recessive genes (nn) - with no dominant gene present - 100% of their children will have PXE. As noted above though - this will be very rare.

Many PXE patients ask if there is a simple inexpensive test to determine if their mate is a PXE carrier. According to Dr. Berthold Struk, there is no such test. He notes that the only meaningful way to determine this is through a family mutation analysis in ABCC6, which if done completely costs about $3000. One could have a skin biopsy of nonlesional skin. If it is positive, one is likely a PXE carrier. If it is negative, it tells you nothing. Thus, the best approach is to draw blood for DNA extraction and mutation analysis. A geneticist can determine the need for a complete mutation analysis for a couple regarding the relative risk of each being a carrier. The geneticist will look at family history, geographic location, etc. In the end, the couple will have to decide their own fear of having a child who has PXE.

NAPE Q & A



Q My sister has PXE and very low vision. She has a lot of joint pain and has been prescribed cortisone injections. She is worried that they might cause bleeding in her eyes.

A Cortisone, administered by any route, has been used for over forty years and is very effective for a variety of medical conditions. The most important thing is not to use it for long periods of time because side effects begin to appear after about 4-6 weeks of continual use, so for most conditions its use should be limited to about six weeks.

- Kenneth Neldner, MD, Dermatologist

Q Do all who have PXE end up having cardiovascular problems?

A This is not a “must,” but is likely. In genetic and medical terms “cardiovascular problems” are considered to be a cardiovascular phenotype, i.e., a cardiovascular disease manifestation of some kind.

This can be coronary artery disease that eventually may lead to a heart attack if not appropriately treated. It can be peripheral artery disease that can cause blockage of an artery of a leg, arm or of the carotid artery that supplies the brain.

These disease phenotypes are called complex genetic phenotypes (traits). This means that they are usually determined by many different interacting genes. The PXE gene, ABCC6, is one of those genes that contributes to cardiovascular phenotypes in patients with PXE because these patients carry two defective copies of this gene that eliminate the normal function of the gene. Whether or not – and if at what age – PXE patients develop a cardiovascular phenotype depends not only on the defective ABCC6 but also on additional risk factors (risk genes) that determine predisposition to diabetes, high lipids, high blood pressure, adipositas, etc. In case anyone has all these polygenic risk factors in addition to suffering from PXE, then this person is likely to develop cardiovascular disease very early on, approximately 10-20 years earlier than a person with all these risk factors but without PXE.

Assuming that a person has PXE and no other cardiovascular risk factors and perhaps a certain number of genetic factors that protect this person from susceptibility to cardiovascular disease, this person may develop cardiovascular symptoms

very late in life or, if lucky, never and may die of a different cause than cardiovascular disease.

- Berthold Struk, MD, Cardiologist

Q Until recently PXE patients understood that angioid streaks play an important role in loss of vision when subretinal bleeding occurs. Now we are told that vascular endothelial growth factor (VEGF) plays a significant role in causing the growth of subretinal leaky blood vessels when VEGF increases in the eye. What is the role of angioid streaks in PXE, either as an indicator of the likelihood of bleeding or as a more direct cause in the development of subretinal leaky blood vessels?

A This is a great question and the person who discovers the answer may earn the Nobel Prize.

Angioid streaks continue to play a role as they represent a break (or discontinuity) in Bruch's membrane, which separates the choroid (vascular layer) from the retina. The real question is why choroidal neovascularization (leaky fragile new blood vessels) do not grow through these streaks for many years, and many times never grow through the angioid streaks at all.

There are many factors implicated in the cascade of events leading to the growth of these visually

threatening blood vessels. For example, inflammation may play a role and certainly vascular endothelial growth factor (VEGF) is increased in eyes developing neovascularization in age related macular degeneration (ARMD). Inhibitors of VEGF are a powerful new tool in treating patients with choroidal neovascularization in ARMD, and I have personal experience using these agents in PXE and ARMD with great success.

In summary, whatever the mechanism is that begins the neovascular process, the angioid streak or break in Bruch's membrane is probably a prerequisite. Of course, it is the fragile leaky blood vessels that may ultimately lead to a hemorrhage, scarring and possible loss of central vision. Even in ARMD, a microscopic defect in Bruch's membrane is probably required for the neovascular lesion to grow from the choroid to under the retina.

A final reminder is that patients with angioid streaks should wear protective eye wear when engaging in contact or racket sports as mild trauma can lead to a hemorrhage even in the absence of choroidal neovascularization. Strenuous exercise like heavy weightlifting, which can turn the face red (Valsalva maneuver), can also lead to spontaneous hemorrhage in the presence of angioid streaks and should be avoided.

- Wayne Fuchs, MD, Retinologist

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Mark your calendar!

2007 NAPE Annual Conference
September 28 and 29

The conference will be held in Atlanta, Georgia, at the Holiday Inn Select near the Atlanta International Airport south of the city center. Free airport shuttle, free on-site parking available.

More information will be mailed separately to NAPE members who register for the conference by September 7. The conference registration form is at the end of this issue.

2007 Conference Information

When: Friday afternoon, September 28 through Saturday afternoon, September 29. Schedule included in this issue and posted on the NAPE website. It will also be mailed to those who register by September 7.

Where: Holiday Inn Select – Atlanta Airport South
4669 Airport Blvd, College Park, Georgia
Room rate (single and double) $85 if reserved by September 7, otherwise you will be charged the going rate. Call 1-404-763-8800 or 1-800-448-2296 and state that you are a NAPE conference registrant.

Meals: Conference registration fee of $45 per person includes Friday evening dinner, Saturday early lunch, plus Saturday mid-afternoon break. You are responsible for your own breakfast on Saturday.

Air-travel: The Holiday Inn Select is near the Atlanta International Airport (ATL) south of the city center. A free shuttle is available to the hotel from the airport. Free shuttle from MARTA Atlanta Airport station also.

Driving directions: From Interstate 85, take exit 71 and go east on Riverdale, then turn south on Airport Blvd to the hotel.

2007 NAPE CONFERENCE SCHEDULE

Holiday Inn Select – Atlanta Airport South
Atlanta, Georgia

Friday, September 28, 2007
First Session

2:00-4:30pm NAPE Board Meeting (observers welcome)

4:45-5:00pm Conference Convenes
Welcome by Dr. Fran Benham, NAPE President

5:00-7:00pm PXE 101 - Dr. Kenneth Neldner

7:00-7:15pm Break

7:15-10:00pm Buffet Dinner (included in your registration)
Enjoy a leisurely meal and visit with conference peers

Saturday, September 29, 2007
Second Session

8:00-8:10am Opening remarks by Dr. Fran Benham
(breakfast on your own before this session)

8:10-10:45am PXE Cardiology Issues, Inheritance and Difficult Choices - Dr. Berthold Struk

10:45-11:00am Break

11:00am-12:30pm Lunch (included in your registration)

Third Session

12:30-2:30pm	Nanomedicine and PXE – Dr. Kattesh Katti
2:30-2:45pm	Break (refreshments available)
2:45-4:15pm	Research Update on AMD (and PXE) Vision Loss – Dr. Chris Bergstrom

Fourth Session

4:15-5:00pm	NAPE Business Meeting (this is an opportunity for NAPE members to ask questions and to provide information/suggestions to NAPE Board members)
5:00pm	Adjournment

Atlanta Conference Presenters

Dr. Kenneth Neldner, Dermatologist, is a long term NAPE leader. His 1988 monograph described the first extensive PXE research. *Clinics for Dermatology: Pseudoxanthoma Elasticum* remains a basic text for researchers interested in PXE. Dr. Neldner will set the stage on Friday, preparing for more fruitful discoveries and understanding of Saturday's program.

Dr. Berthold Struk, Cardiologist, Max-Delbruck Center for Molecular Medicine and Franz-Volhard Clinic for Cardiovascular Disease in Berlin, Germany, will present information about the impact of PXE on the cardiovascular system, basic care procedures to be followed by patients and their physicians, the inheritance of PXE and difficult treatment choices PXE patients may face.

Dr. Kattesh Katti, PhD, Department of Radiology, University of Missouri-Columbia, enjoys a worldwide reputation among scientists for his discoveries which have helped to usher in nanomedicine, as part of the nanotechnology revolution in science and industry. Dr. Katti, holder of such prestigious awards as the 2006 Gottingen Professorship and the St. Louis Academy of Science 2007 Scientist of the Year, will explain basic nanomedicine and how he has used it to diagnose and treat cancer and AMD.

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Dr. Chris Bergstrom, Senior Retina Fellow, Emory Eye Center, Emory University, Atlanta, Georgia, will provide a research update on retinal treatments including vascular endothelial growth factor (VEGF) inhibitors such as Avastin, Lucentis and VEGF-Trap. He will also provide an update on stem cell research efforts related to the retina – both adult and embryonic.

NAPxE – National Association for Pseudoxanthoma Elasticum

8760 Manchester Road
St. Louis, MO 63144-2724
Telephone: 314-962-0100
Website: www.napxe.org Email: napestlouis@sbcglobal.net

REGISTRATION FORM – 2007 ANNUAL MEETING

Friday, September 28 – Saturday, September 29, 2007

Registration fee of $45 per person includes all programs, Friday evening's reception/supper , Q & A sessions, Saturday lunch and refreshments.

NAME _____ **PHONE** _____

ADDRESS _____ **FAX** _____

CITY _____ **EMAIL** _____

STATE _____ **ZIP** _____ **COUNTRY** _____

ARRIVAL DATE _____

NUMBER ATTENDING MEETING _____ x $45.00 = AMOUNT ENCLOSED $_____

NAME(S) OF GUEST(S) ATTENDING WITH YOU:

You are responsible for making your own hotel reservations. Please call the **Holiday Inn Select, Atlanta Airport South in Atlanta, Georgia, at 1-404-763-8800 or 1-877-843-3621**. Be sure to **call by September 7, 2007**, and say you are with NAPE to get the group rate of $85 per night (single or double) plus tax. Parking onsite is free and hotel-airport shuttle is available free.

Payment of the registration fee must accompany this form. Please make your check payable to NAPE, Inc., in U.S. currency. We cannot accept credit card payments. Mail your registration and check to NAPE at the address shown above. We will send you a confirmation packet if registration is received by September 7.

If you require special assistance to participate fully, please provide a written description of your needs. Vegetarian meals can be accommodated.

SIGNATURE _____ **DATE** _____

Please mail this form to NAPE with payment by September 7, 2007
CANCELLATIONS ARE NOT REFUNDABLE AFTER SEPTEMBER 7, 2007

National Association for Pseudoxanthoma Elasticum

8760 Manchester Rd., St. Louis, MO 63144-2724

Donations - Membership

No membership fee is required, athough donations are appreciated and needed to pay operational expenses, including telephone, fax, email, website and newsletter services.

Donations can be made in Honor or Memory of a loved one, for the Research Fund and/or for the Low-Vision Fund. All donations are tax deductible in the USA.

❒**Operations** ❒**Honor** ❒**Memory** ❒**Low-Vision** ❒**Research**

Name of Loved One: _____
Address for Acknowledgement:

PLEASE COMPLETE THE SECTION BELOW IF YOU HAVE PXE, THINK YOU HAVE PXE, OR ARE FILLING THIS OUT FOR SOMEONE ELSE

Name: _____ **Phone:** _____

Email: _____ **Fax:** _____

Address: _____

City: _____ **State:** _____ **Zip:** _____ **Country:** _____

Male ❒ **Female** ❒ **Birthdate:** _____ **Age:** _____

I am diagnosed with PXE ❒**Yes** ❒ **No** **Occupation:** _____

Are you legally blind? ❒ **Yes** ❒ **No** **Request Newsletter:** ❒ **Printed** ❒ **CD**

Do others in your family have PXE? ❒ **Yes** ❒ **No** **If so, who? (Mother, Father, Sibling, etc. & Name)** _____

_____

Please list any medical problem(s) you are experiencing: e.g., eye involvement, skin lesions, heart problems, gastric bleeding, etc., and comments/questions (use another page if required):

_____

_____

_____

Are you willing to be contacted by another who wishes to talk with someone else who has PXE? ❒ **Yes** ❒ **No**

Have You Changed Your Address?

Please help by letting us know. Please be sure to print your new zip code number, including the extra four digits, if possible. When we use the full zip code, our costs of mailing in the United States are lower. Please help.

New Address
Name: _____

Street: _____

City, State, Zip _____

Old Address
Name, if different: _____

Street: _____

City, State, Zip _____

PLEASE PRINT NEATLY

National Association for Pseudoxanthoma Elasticum
NAPE, Inc.
8760 Manchester Road
St. Louis, MO 63144-2724

ADDRESS SERVICE REQUESTED