

# Alpha-1 Nor'Easter a Support Group Newsletter (February 2010)

*Serving the North Eastern US for the most part!*

## UPCOMING ALPHA EVENTS

(see "SUPPORT GROUP NEWS " for more info -Page 2)

There are some exciting upcoming events.

1. The Alpha-1 Foundation will be hosting a three day, training session for Building Friends for a Cure. They will pay your hotel and travel expenses! (3/11-13)
2. Celebrate St. Patrick's Day at the Celtic Connection. Enjoy the company of other Alpha Families while helping to find a cure! (3/13)
3. The Annual George Washington Bridge Walk, Saturday, May 8. Have fun and help raise money to find a cure.
4. The 19th Annual National Education Conference will be held on June 11-13, 2010 in Orlando, FL at the Hilton Walt Disney Resort. Take a vacation, bring the family it will be an awesome experience! I plan on attending

A	B	C	D	E
DATE	TOPIC	PLACE	CONTACT	
3/11-13	A1F Fundraising Training	Sheraton Needham Hotel, Needham, MA	Angela McBride	1 888 825-7421 X233
Sat 3/13	The Celtic Connection	Sheraton Needham Hotel, Needham, MA	Bob Healy	1 (718) 447-1447
Sat 4/24		Connecticut	Jo Anne Brailey	1 (860) 739-4331
Sat 5/8	A1F Fund Raiser	3rd Annual George Washington Bridge Walk	Lori Tartell	1 (212) 523-5471
6/11-13	19th Annual National Education Conf	Hilton Walt Disney Resort, Orlando, FL	Alpha-1 Assoc	1 (800) 521-3025
Sat 7/17		Connecticut	Jo Anne Brailey	1 (860) 739-4331
Sun 9/26	Educational Brunch	Metro NY	Lori Tartell	1 (212) 523-5471
Sat 10/16		Connecticut	Jo Anne Brailey	1 (860) 739-4331
Tues 12/7	Holiday Dinner	Metro NY	Lori Tartell	1 (212) 523-5471

We would hope and recommend that everyone receiving this notice is a member of the Alpha-1 Association (A1A) and has enrolled in the Alpha-1 Foundation (A1F) Registry. Likewise, if you don't receive the A1F Research Registry Update you are NOT part of the cure for Alpha-1 (shame! shame!). Even if you receive a copy of the Alpha-1 News you are NOT necessarily a member of A1A. Call them just to be sure. 1 (800) 521-3025 Another great publication is the Alpha1 to One magazine contact the Alpha-1 Foundation to get a copy. If interested contact me (Joe) as shown at the end or look at our phone list.

### IMPORTANT PHONE NUMBERS: if all these numbers confuse you give me a call 201-444-7839

Alpha-1 Association (A1A)	1 (800) 521-3025	www.alpha1.org	Support
A1A Genetic Counselor	1 (800) 785-3177	courtesy of A1A	Family Concerns
Alpha-1 Foundation	1 (888) 825-7421	www.alphaOne.org	Research
Alpha-1 Coded Testing (ACT)	1 (877) 886-2383	AlphaOne@musc.edu	Free coded (anonymous) testing
Alpha-1 Registry	1 (877) 886-2383	http://www.musc.edu/	Be part of the cure
Baxter Healthcare	1 (800) 423-2090	www.Baxter.com/ Aralast.com	Aralast
Talecris Biotherapeutics	1 (800) 243-4153	www.Prolastin.com	Prolastin
CSL Behring	1-866-936-2472	www.Zemaira.com	Zemaira
Accredo	1 (866) 625-7421	www.Accredotx.com	Aralast/ Zemaira
AlphaNet	1 (800) 577-2638	www.AlphaNet.org	Prolastin/ Zemaira
Prolastin Direct	1 (800) 305-7881	www.ProlastinDirect.com/	Prolastin
Coram Healthcare	1 (866) 367-2174	www.CoramHealthcare.com	Aralast

## WORLD WIDE WEB

Most of the following information was gathered at the ALPHA-1 MAILING LISTS. As with everything else in this meeting notice take it with a grain of salt. Remember We do edit some of these E-mail posts and We select which to print. There is much more on the lists. Some of which is very interesting. If you have any questions contact me <JoeReidy@Verizon.net >. Alpha-1 International Support Groups are email networks which Alphas use to rapidly exchange information, support, suggestions, questions, plans, activities; nearly anything that comes to our minds. Many of us have had to reduce our activities and contacts; the Alpha1 contacts are a great way to maintain contact with others. It is also a big psychological boost to know that we are not alone. Here is a list of the E-mail groups to which I (Joe) subscribe:

#	Mailing List	enrollment	owner(s)	TO JOIN: E-mail to:
1	Alpha-1 International	open to most interested in Alpha-1	Connie Storey	ALPHA-1-REQUEST@home.ease.lsoft.com
2	Alpha-1 LIVER	restricted mostly liver affected Alphas & family	Nancy Cropper	ALPHA-LIVER-REQUEST@home.ease.lsoft.com
3	Alpha-1 Lungs & Life	open to all interested in Alpha-1	Sally Turner	Alpha1_Lungs_and_Life-subscribe@yahooogroups.com

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# Support Group News

## NY/NJ Alpha-1 Support Group Meeting

As we told you last month, here are the Support Group plans for 2010.

**GWB Walk** Saturday, May 8th details will be forth coming.

We considered a change the venue to the Brooklyn Bridge.

Apparently there is parking on the Brooklyn side but no nearby restaurants. On the Manhattan side there are restaurants but it is quite an uphill hike to the bridge.

**Sunday Brunch and Educational Meeting** Tentatively 9/26/10

Plans are sketchy at this time but we will keep you informed.

**Holiday Dinner:** Tentatively 12/7/10

## Massachusetts Support Group Meeting

**DATE:** Usually 2nd Thursday of even numbered months

**PLACE:** Wingate Nursing Home

589 Highland Ave.

Needham, MA

**more info:** Susan Binnall 617-916-9805 sbinnall@comcast.net

## Alpha Nutmeggers (CT) Support Group

Below is the schedule for Connecticut Nutmeggers Support Group meetings for 2010. They are all Saturdays.

April 24th

July 17th

October 16th

**more info:** Jo Anne Brailey 1 (860) 739-4331 iluv2sew@snet.net

## Alpha-1 Foundation Events

**The Celtic Connection** Save the Date and Join the Party Celebrate Our Irish Heritage by Observing St. Patrick's Day with Friends and Family.

- Enjoy a Traditional Dinner with Live Music by "Hogan's Goat"
- Irish Step Dancers and More Music for You to Dance to.

Our Celtic Connection Will Help Raise Funds to Benefit Alpha-1 Research Programs.

**Date:** Saturday, March 13 (the Luck O' the Irish)

**Time:** From 7:00 to 11:00 PM

**Place:** Sheraton Needham Hotel

100 Cabot Street, Needham, MA 02494

**Tickets:** \$40 per Person; \$75 per couple; \$350 per table for 10

**Contact:** Bob Healy at : 1 (718) 447-1447 or

email : BobHealy125@msn.com

(Silent Auction and Pot-O'-Gold 50/50 raffle)

## Building Friends for a Cure Training Session

### Program Details

• The Alpha-1 Foundation will be hosting a Building Friends for a Cure Training on **Friday, March 12th in Needham, MA.**

• Attendees will learn how easy it is to become more involved with raising awareness for Alpha-1 and walk away with the tools you need to raise funds for Alpha-1 research in their community.

- Training to include: how to organize
- Themed Dinner
- Golf Tournament
- Walk
- Bowling event
- Media training
- Empowerment

### We offer the following:

- Air/land transportation to Boston
- Three night hotel accommodation – Thursday, March 11th Friday, the 12th and Saturday, March 13th
- Opportunity to attend the Celtic Connection event on Saturday March 13th where you will receive hands on training on how they put together their event. Space is limited

### Criteria

- We are looking for individuals who are willing to commit to planning an event in the next 12 months.

### Contact

For more information, please contact **Angela McBride** at 888-825-7421 ext. 233 or amcbride@alphaone.org.

The Alpha-1 Foundation's Building Friends for a Cure program is centered on facilitating and empowering Alphas to increase funding to support our research mission. It is up to the Foundation to give them the tools they need to ensure that their fundraising events are a success. In order to do so we must provide training in event management, building committees, volunteer management and securing sponsors for their events. When Alphas

commit to hosting an event, we ensure that they understand budgeting for the event and show them how to make their fundraiser profitable. This type of training ranges from negotiating venue costs to organizing profit centers such as silent auctions and raffles. Providing the tools to increase awareness and updating Alphas in the community about research are important parts of the program as well.

## Alpha-1 Association News

### \$12,500 in Educational Scholarships to be awarded to Alphas and their families!

The Association is pleased to announce that it will award seven educational scholarships totaling \$12,500. The recipients will be announced on May 15th. We gratefully acknowledge CSL Behring and Talecris Biotherapeutics Center for Science and Education for providing the funds to substantially increase the Peter Smith Memorial Scholarships. In addition, the Association has established an educational scholarship in memory of Jack Walsh, III.

A total of seven (7) scholarships will be awarded based on eligibility:

- John "Jack" Walsh III Memorial Scholarship \$2,500
- 4 Peter Smith Scholarships each \$2,000
- 2 Peter Smith Scholarships each \$1,000

These scholarships are open to Alphas and their immediate family members who demonstrate financial need and have been accepted for study at an approved institution, i.e., an accredited university, community college or technical institute. Applications are now being accepted and are due to the Scholarship Committee by April 1, 2010. For more information on how to apply, please click here.

**From:** Edward BRAILEY <nube@SNET.NET>

Last year I think 8 people applied for these scholarships. These are substantial amounts and can help alot. Please go to our web site Alpha1.org and look for applications and qualifications. Please take advantage of this and its not only for the younger students but the older ones like me that are going back to school.

If you have any questions please don't hesitate to contact me Nube@snet.net

## The 19th Annual National Education Conference

will be held on June 11-13, 2010 in Orlando, FL at the Hilton Walt Disney Resort. You can now make hotel reservations online. Registration forms for the conference will be available by the end of January. If you have any questions regarding scholarships to attend the conference, please contact Cathey Horsak at 877-346-3212 or via email at chorsak@alpha1.org.

## GENETIC SCREENING

**From:** Ava

**Date:** February 4, 2010 11:46:15 AM EST

**Subject:** genetic testing mandated by the gov. w/o parental consent

(CNN) -- When Annie Brown's daughter, Isabel, was a month old, her pediatrician asked Brown and her husband to sit down because he had some bad news to tell them: Isabel carried a gene that put her at risk for cystic fibrosis.

While grateful to have the information -- Isabel received further testing and she doesn't have the disease -- the Mankato, Minnesota, couple wondered how the doctor knew about Isabel's genes in the first place. After all, they'd never consented to genetic testing.

It's simple, the pediatrician answered: Newborn babies in the United States are routinely screened for a panel of genetic diseases. Since the testing is mandated by the government, it's often done without the parents' consent, according to Brad Therrell, director of the National Newborn Screening & Genetics Resource Center.

In many states, such as Florida, where Isabel was born, babies' DNA is stored indefinitely, according to the resource center.

Many parents don't realize their baby's DNA is being stored in a government lab, but sometimes when they find out, as the Browns did, they take action. Parents in Texas, and Minnesota have filed lawsuits, and these parents' concerns are sparking a new debate about whether it's appropriate for a baby's genetic blueprint to be in the government's possession.

"We were appalled when we found out," says Brown, who's a registered nurse. "Why do they need to store my baby's DNA indefinitely? Something on there could affect her ability to get a job later on, or get health insurance."

According to the state of Minnesota's Web site, samples are kept so that tests can be repeated, if necessary, and in case the DNA is ever needed to help parents identify a missing or deceased child. The samples are also used for medical research.

Art Caplan, a bioethicist at the University of Pennsylvania, says he understands why states don't first ask permission to screen babies for genetic diseases. "It's paternalistic, but the state has an overriding interest in protecting these babies," he says.

However, he added that storage of DNA for long periods of time is a different matter.

"I don't see any reason to do that kind of storage," Caplan says. "If it's anonymous, then I don't care. I don't have an issue with that. But if you keep names attached to those samples, that makes me nervous."

DNA given to outside researchers

Genetic testing for newborns started in the 1960s with testing for diseases and conditions that, if undetected, could kill a child or cause severe problems, such as mental retardation. Since then, the screening has helped save countless newborns.

Over the years, many other tests were added to the list. Now, states mandate that newborns be tested for anywhere between 28 and 54 different conditions, and the DNA samples are stored in state labs for anywhere from three months to indefinitely, depending on the state. (To find out how long your baby's DNA is stored, see this state-by-state list).

Brad Therrell, who runs the federally funded genetic resource consortium, says parents don't need to worry about the privacy of their babies' DNA.

"The states have in place very rigid controls on those specimens," Therrell says. "If my children's DNA were in one of these state labs, I wouldn't be worried a bit."

The specimens don't always stay in the state labs. They're often given to outside researchers -- sometimes with the baby's name attached.

According to a study done by the state of Minnesota, more than 20 scientific papers have been published in the United States since 2000 using newborn blood samples.

The researchers do not have to have parental consent to obtain samples as long as the baby's name is not attached, according to Amy Gaviglio, one of the authors of the Minnesota report. However, she says it's her understanding that if a researcher wants a sample with a baby's name attached, consent first must be obtained from the parents.

More Empowered Patient news and advice

Scientists have heralded this enormous collection of DNA samples as a "gold mine" for doing research, according to Gaviglio.

"This sample population would be virtually impossible to get otherwise," says Gaviglio, a genetic counselor for the Minnesota Department of Health. "Researchers go through a very stringent process to obtain the samples. States certainly don't provide samples to just anyone."

Brown says that even with these assurances, she still worries whether someone could gain access to her baby's DNA sample with Isabel's name attached.

"I know the government says my baby's data will be kept private, but I'm not so sure. I feel like my trust has been taken," she says.

Parents don't give consent to screening

Brown says she first lost trust when she learned that Isabel had received genetic testing in the first place without consent from her or her husband.

"I don't have a problem with the testing, but I wish they'd asked us first," she says.

Since health insurance paid for Isabel's genetic screening, her positive test for a cystic fibrosis gene is now on the record with her insurance company, and the Browns are concerned this could hurt her in the future.

"It's really a black mark against her, and there's nothing we can do to get it off there," Brown says. "And let's say in the future they can test for a gene for schizophrenia or manic-depression and your baby tests positive -- that would be on there, too."

Brown says if the hospital had first asked her permission to test Isabel, now 10 months old, she might have chosen to pay for it out of pocket so the results wouldn't be known to the insurance company.

Caplan says taking DNA samples without asking permission and then storing them "veers from the norm."

"In the military, for instance, they take and store DNA samples, but they tell you they're doing it, and you can choose not to join if you don't like it," he says.

What can parents do

In some states, including Minnesota and Texas, the states are required to destroy a baby's DNA sample if a parent requests it. Parents who want their baby's DNA destroyed are asked to fill out this form in Minnesota and this form in Texas.

Parents in other states have less recourse, says Therrell, who runs the genetic testing group. "You'd probably have to write a letter to the state saying, 'Please destroy my sample,'" he says.

He adds, however, that it's not clear whether a state would necessarily obey your wishes. "I suspect it would be very difficult to get those states to destroy your baby's sample," he says.

CNN's John Bonifield and Jennifer Bixler contributed to this report

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**From:** Joe Reidy <JoeReidy@Verizon.net>

**Date:** February 17, 2010 11:34:33 AM EST

**Subject:** [Lungs & Life] Genetic Diseases Less Prevalent

I wonder about the rate of Alpha-1?  
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Gene tests appear to reduce levels of some inherited diseases

By Marilyn Marchione (CP) ? 1 hour ago

Some of mankind's most devastating inherited diseases appear to be declining, and a few have nearly disappeared, because more people are using genetic testing to decide whether to have children. Births of babies with cystic fibrosis, Tay-Sachs and other less familiar disorders seem to have dropped since testing came into wider use, The Associated Press found from interviews with numerous geneticists and other experts and a review of the limited research available.

Many of these diseases are little known and few statistics are kept. But their effects - ranging from blood disorders to muscle decline - can be disabling and often fatal during childhood.

Now, more women are being tested as part of routine prenatal care, and many end pregnancies when diseases are found. One study in California found that prenatal screening reduced by half the number of babies born with the severest form of cystic fibrosis because many parents chose abortion.

More couples with no family history of inherited diseases are getting tested before starting families to see if they carry mutations that put a baby at risk. And a growing number are screening embryos and using only those without problem genes.

The cost of testing is falling, and the number of companies offering it is rising. A 2008 federal law banning gene-based discrimination by insurers and employers has eased fears.

Genetic testing pushes hot-button issues: abortion, embryo destruction and worries about eugenics - selective breeding to rid a population of unwanted traits. Yet it is touching a growing number of people:

-In suburban Cleveland, Beth and Thad Meese were stunned to learn during her second pregnancy that they carry genes that can cause cystic fibrosis. Tests show the baby won't have the disease, but they have decided against having a third child or to screen embryos if they do. "I feel like we got lucky" and should not tempt fate again, she said.

-In Boston, Harvard psychologist and author Steven Pinker and his wife, novelist Rebecca Goldstein, learned last year that they carry genes that cause a serious neurological disease, familial dysautonomia. Too old to have children, they shared the news with younger relatives, who are being tested to see if they, too, have the gene. "There's a tendency psychologically to think these are very rare and what are the chances that two people could both have rare genes," Pinker said. "Not only can it happen, but it happened to me."

-In the Canadian city of Vancouver, Jeff and Megan Carroll screened embryos to have two children free of the Huntington's disease gene. Jeff has. "I felt very strongly that I didn't want to pass on this," he said. Huntington's "is done killing people in my family when I am gone."

Although genetic testing can raise moral dilemmas, at least one conservative religious group - Orthodox Jews - has found ethically acceptable ways to use it to lessen diseases that have plagued its populations.

"I am a Holocaust survivor. I was born in the middle of the second World War. I hope that I am not a suspect for practicing eugenics. We are trying to have healthy children," said Rabbi Josef Ekstein of New York, who founded a group that tests couples and discourages matches when both carry problem genes.

Some diseases - sickle cell, cystic fibrosis, Tay-Sachs, thalassemia, spinal muscle atrophy - occur when people inherit two bad genes, one from each parent. The genes can pass quietly for generations until two carriers mate; then children have a one-in-four

chance of getting the disease.

(Down syndrome is the best known disorder for which prenatal testing has long been available, but it's caused by an extra chromosome during abnormal cell division - not genes inherited from the parents.) Statistics for inherited diseases are hard to come by - birth certificates often don't list them, and they sometimes aren't diagnosed for months or years after birth. Yet, there's little doubt that testing has put a dent in many.

"We're definitely seeing decreased rates of certain genetic disorders as a result of carrier screening," said Dr. Wendy Chung, clinical genetics chief at Columbia University. In five years, she has seen only one case of Tay-Sachs, a neurological disease that used to be more common in Ashkenazi, or Eastern European Jews. Children with the disease lack a key enzyme; they lose mental and physical abilities and usually die by age 4.

In the last decade, only about a dozen new cases of Tay-Sachs occurred each year in the United States, said Dr. Michael Kuback, a professor at the University of California at San Diego who tracks the disease.

Ekstein, the rabbi, lost four children to it before founding Dor Yeshorim, a Brooklyn-based group that recruits Jews to be tested. Using confidential PIN numbers, they call a hotline to see if a prospective mate would be a risky match. The group has 300,000 members and tests for nine diseases, including cystic fibrosis. "In the Orthodox Ashkenazi community around the world, we virtually have wiped out the diseases we screen for," said the group's development director, Allan Binder.

One is familial dysautonomia. Since 2004, only a few children worldwide have been born with it each year, and it soon may cease to exist because of genetic screening, said Dr. Barron Lerner, a Columbia University medical historian. The disease causes faulty nerve development, floppy muscles, digestive and other problems, and kills many by young adulthood.

Fragile X syndrome, the leading cause of mental impairment in boys, may decline because carrier testing for parents and prenatal testing of fetuses is now available for it, said Barbara Biesecker, director of the genetic counselling program at the National Institutes of Health. Lots of eyes are on cystic fibrosis, a disease that causes sticky mucus buildup in the lungs, digestive problems and death in young adulthood. More than 10 million Americans - one in 25 to 29 whites, who are more at risk for it than blacks - carry a gene mutation for it. In 2001, the American College of Obstetricians and Gynecologists and other groups recommended that white pregnant women be offered testing for mutations. Tests on partners and fetuses often followed, and an unknown number of abortions.

The impact showed up two years later in Massachusetts, one of the few states testing newborns for the disease at the time. Births of babies with cystic fibrosis dropped, from 29 in 2000 to only 10 in 2003, ticking up to 15 in 2006, said Dr. Richard Parad, a Brigham and Women's Hospital physician who helped set up the screening program. In California, Kaiser Permanente, a large health maintenance organization, offered prenatal screening. From 2006 through 2008, 87 couples with cystic fibrosis mutations agreed to have fetuses tested, and 23 were found to have the disease. Sixteen of the 17 fetuses projected to have the severest type of disease were aborted, as were four of the six fetuses projected to have less severe disease. Comparisons to couples not given prenatal screening suggested that screening had cut births of babies with severe disease in half, researchers reported at a genetics conference in 2008. Studies in Canada, Italy, Australia and in Europe also found that cases dropped after screening began.

The Cystic Fibrosis Foundation's registry, which tracks voluntarily reported cases, shows a steady rise in recent years. But that is because more states have started testing all newborns, discovering cases that previously went unreported, some researchers believe. In December, Texas became the final state to add such testing; the first reliable national estimate of cases is expected in a couple of years. Beth Meese, the Cleveland nurse who discovered from prenatal tests that she and her husband are carriers, wishes they had been screened before pregnancy. By the time they learned of their risk, they had seen an ultrasound and decided to have the baby no matter what its tests showed. "We saw the baby, saw it moving," she said. "It makes that decision that much more difficult to make."

Gene testing hasn't led to declines in all diseases. Sickle cell, a blood disorder that causes anemia and pain and raises the risk of stroke, has not dropped. It mostly afflicts blacks; gene carriers are said to have sickle cell "trait," which sounds harmless. "Now we're actually learning that it's not as benign as we thought it was," and

that carriers have higher risks for certain medical problems, said Dr. Lanetta Jordan, a Florida physician and chief medical officer of the Sickle Cell Disease Association of America. Newborn screening is finding more sickle cell carriers and cases, but this doesn't seem to affect parents' future family plans, Jordan said. Gene testing also has had little impact on Huntington's disease, a progressive, fatal neurological disorder. Unlike many other inherited diseases, only one bad copy of a gene is needed to cause Huntington's, and symptoms don't usually appear until middle age, after many have already had children. Fewer than 15 per cent of people in families with a history of it agree to be tested, said Kimberly Quaid, an Indiana University genetics researcher. "They just prefer to live their life and hope for the best," she said. Jeff Carroll, the Canadian who, with his wife, screened embryos because he carries the Huntington's gene, said it is "unconscionable" to procreate without taking steps to prevent passing on the disease. "Having my test result has immensely improved my life. I was able to make reproduction decisions that ended HD in my family," and to launch a career as a biologist researching the disease, he said. The number of fertility treatments that include embryo screening has been on the rise in recent years, with nearly 5,200 screenings in 2006, according to the Society for Assisted Reproductive Technology. Carrier testing also is rising. A California company, Counsyl, sells a \$349 saliva test for genes for more than 100 inherited disorders. Several thousand people used it over the last year, the company reports. Eliminating disease is a noble goal but also "should give us pause," Lerner, the Columbia historian, wrote recently in the *New England Journal of Medicine*.

"If a society is so willing to screen aggressively to find these genes and then to potentially have to abort the fetuses, what does that say about the value of the lives of those people living with the diseases?" he asked.

It's a touchy issue. The Cystic Fibrosis Foundation points out that the disease varies greatly in severity, and life expectancy with it is now 37 years.

Diseases like familial dysautonomia and Tay-Sachs, which kill before school age, are easier cases. If one of those vanishes, "thank God," said Rabbi Ekstein of the Jewish testing group. "It gives me a very good feeling that we are a part of such life-saving efforts." Copyright © 2010 The Canadian Press. All rights reserved.

## INHALED ALPHA-1

**Talecris gets FDA "orphan drug" designation for inhaled treatment for Alpha-1** The Medical News

Talecris Biotherapeutics has been granted orphan drug designation by the US Food and Drug Administration (FDA) for the development of an aerosol formulation of Alpha1-Proteinase Inhibitor to treat congenital alpha1-antitrypsin (AAT) deficiency.

Alpha-1 Antitrypsin Deficiency (Alpha-1) is a chronic, hereditary condition that increases the risk of lung and liver disease, especially emphysema, which typically emerges in the fourth decade of life.

Currently, there are no approved inhaled treatments available for the treatment of Alpha-1.

Orphan drug designation is granted to companies to encourage the development of treatments that prevent, diagnose or treat rare, life-threatening or chronic illnesses that affect fewer than 200,000 people per year in the US

The designation provides incentives such as tax credits and seven years of market exclusivity to companies willing to support the costly research and development programs associated with developing specialized drugs for a small population of individuals.

Talecris received a similar orphan drug designation for the aerosolized form of AAT from the European Commission in June '08.

**From:** Dutt's Logger <pdlogger@WINDSTREAM.NET>

**Date:** February 8, 2010 10:54:01 AM EST

**Subject:** [ALPHA-1] FYI: NO MORE STICKS 2/8/2010

Kamada Announces Enrollment of First Patient in Its Pivotal Study for Inhaled AAT in Europe

NESS ZIONA, Israel--(BUSINESS WIRE)-- Kamada, a biopharmaceutical company engaged in the development, manufacturing and marketing of specialty life-saving therapeutics, announced today that it has enrolled the first patient into its pivotal clinical trial with its new breakthrough compound of inhaled alpha-1 antitrypsin (AAT) delivered by an Investigational eFlow Nebulizer System (PARI Pharma GmbH), in patients with alpha-1 antitrypsin deficiency.

The Phase 2-3, multi-center, randomized, double-blind,

placebo-controlled and international study will evaluate the efficacy and safety of inhaled, human AAT in alpha-1 deficient patients with emphysema. The trial will be conducted across several European countries. The study protocol has been designed in agreement with the EMEA under the product's orphan drug designation status.

David Tsur, Chief Executive Officer of Kamada said, "We are very pleased with the advancement of the trial and hope that the rate of enrollment would reflect the excitement of the patients of this potential new treatment. We recognize the importance of bringing this product to this unique patient population for whom, at the moment, there is no therapeutic resolution.

## LUNG TRANSPLANT

**From:** Dutt's Logger <pdlogger@WINDSTREAM.NET>

**Date:** February 12, 2010 11:54:32 AM EST

**Subject:** FYI: IN THE EYE OF PREVENTING REJECTION 2/12/10

PARI Pharma Enrolls First Patient in Phase 2b Study of L-CsA  
PARI Pharma has enrolled the first patient in its Phase 2b clinical trial studying inhaled liposomal cyclosporine A (L-CsA) delivered via a customized Investigational eFlow Nebulizer System. The multinational study is investigating the safety and efficacy of PARI's L-CsA formulation. In previous clinical trials, reactions from physicians and lung transplant recipients to PARI's drug-device combination were encouraging.

"We are very pleased to move forward with this investigational treatment aimed at preventing bronchiolitis obliterans, which is an incurable small airway disease in lung transplant recipients. This study has been designed with advice from the European Medicines Agency under L-CsA's orphan drug designation status," said Manfred Keller, chief scientific officer and executive vice president of PARI Pharma.

PARI Pharma's Phase 2b trial is a multi-center, randomized, double-blind, placebo controlled, parallel group, dose-finding study to investigate the safety and efficacy of L-CsA in doses of 10mg/day and 20mg/day to prevent bronchiolitis obliterans in recipients of lung transplants.

"We are seeing early success from our L-CsA program. This underscores PARI Pharma's unique position to combine formulation expertise with our advanced aerosol delivery technology to develop best in class therapies for unmet medical needs," added Martin Knoch, president of PARI Pharma.

Positive data regarding human lung deposition and distribution of L-CsA was published in the Journal of Aerosol Medicine and Pulmonary Drug Delivery last year, and clinical as well as preclinical data will be presented in April at the Annual Meeting of the International Society of Heart and Lung Transplantation in Chicago.

### About Bronchiolitis Obliterans

According to the International Society of Heart and Lung Transplantation Registry, development of bronchiolitis obliterans is the single most important risk factor for mortality among lung transplant recipients. Bronchiolitis obliterans is an incurable small airways disease, which manifests as chronic allograft rejection and results in airflow obstruction. The disease is also a factor in other lung diseases such as collagen vascular diseases, inhalation of toxic fumes, and respiratory tract infections. Bronchiolitis obliterans affects approximately 60,000 patients worldwide, and once it develops, most patients die of respiratory failure within about 5 years.

Find this article at: <http://www.prnewswire.com/news-releases/pari-pharma-enrolls-first-patient-in-phase-2b-study-of-l-csa-84113542.html>

**From:** Dutt's Logger <pdlogger@WINDSTREAM.NET>

**Date:** February 1, 2010 3:54:24 PM EST

**Subject:** [ALPHA-1] FYI: YOU SWINE YOU 1/2/2010

Melbourne medical breakthrough means pig lungs could be transplanted into humans

PIG lungs could be transplanted into humans to overcome a shortage of donor organs after a Melbourne medical breakthrough.

Scientists from The Alfred hospital have kept pig lungs alive and functioning with human blood, paving the way for animal-human transplants in as little as five years.

The possibility of animal-to-human transplants - xenotransplantation - has divided the medical ethics community.

But with 200 Australians on transplant waiting lists dying in the past year, Dr Glenn Westall said the world-first discovery meant

pig-human lung transplants were a real prospect.

"Five to six hours into the experiment they seemed to be working as well as they were at the start," he said. "The blood went into the lungs without oxygen and came out with oxygen, which is the exact function of the lungs.

"It showed that these lungs were working perfectly well and doing as we were expecting them to do. This is a significant advance compared to experiments that have been performed over the past 20 years."

The breakthrough came after scientists at Melbourne's St Vincent's Hospital were able to remove a section of swine DNA called the Gal gene, which made the pig organs incompatible with human blood.

Prof Tony D'Apice - who has been breeding pigs for possible transplants since 1989 - said human DNA was added to the engineered animals to control blood clotting and rejection in humans.

His team at St Vincent's is trying insulin-producing islet transplants and kidney transplants in baboons as well as backing research in the US, Europe and Japan. He said the lung breakthrough at The Alfred was exciting.

The Alfred scientists removed the lungs and hooked them up to a machine mimicking the human circulation system under a process mirroring that used for traditional lung transplants.

The machine uses a ventilator to cause the lungs to "breathe" while a pump acting as a heart allows blood to flow through the lungs.

Previous attempts to combine unmodified pig lungs and human blood ended abruptly two years ago when blood clots began forming immediately, causing the organs to become so blocked no blood could pass through.

But when the genetically modified lungs were used at the end of last year the results were overwhelming, fuelling hopes of clinical trials in five to 10 years.

"Where before we saw the system crash and the lungs destroyed within 10 minutes, the lungs seemed to be working perfectly well at the end of our experiment after many hours," Dr Westall said. "This is a major advance but there remain significant hurdles."

The full results of the research are being guarded before being announced at a world transplant meeting in Vancouver in August.

The Federal Government is developing guidelines for xenotransplantation after a moratorium on the practice expired on December 31.

But medical ethicist Assoc Prof Nicholas Tonti-Filippini said such transplants had the potential to bring animal diseases into the human population. He said the creation of genetically modified pigs was not ethically acceptable.

"It is basically a human-pig, a hybrid, or whatever you want to call it. It is about whether the community is prepared to accept a part human, part animal."

## LUNG DISEASE NEWS

**From:** Dutt's Logger <pdlogger@WINDSTREAM.NET>

**Date:** February 22, 2010 3:41:39 PM EST

**Subject:** [ALPHA-1] FYI: THE INREACH SYSTEM 2/22/2010

LAFAYETTE — Donald Richard was hospitalized last fall at Lafayette General Medical Center with pneumonia and then faced uncertainty when scans showed a small lesion in his upper right lung.

More scans didn't rule out that the lesion was cancerous, and its location proved precarious for traditional biopsy methods. It would have been difficult to get to," said Richard, 71, of Rayne.

Months earlier, if biopsy attempts were unsuccessful, alternatives included surgical removal without certainty that it was necessary or to watch and wait — giving time for the tumor to spread if it were cancer.

Instead, Richard was Lafayette General's first patient treated with a new navigational scope technology, the inReach System.

The technology enables doctors to steer a scope and biopsy forceps into the periphery of the lung.

Without being able to steer, I never would have gotten near the lesion," said Dr. Gary Guidry, Richard's pulmonologist at Lafayette General.

The biopsy showed that Richard's lesion was benign.

It was scar tissue," said Richard, who quit smoking a year and a half ago and struggles with emphysema and COPD.

My breathing capacity is about 45 percent," he said. "I'm feeling as good as I'm going to get."

The technology, developed by superDimension, is designed to help doctors reach lesions in the periphery of the lungs that before

were unreachable with a traditional bronchoscope.

In the past, using traditional bronchoscopy, doctors would guide a scope using an active X-ray of the area. In some cases, the area could not be reached and alternative procedures, like a needle aspiration would follow.

The inReach system increases the chances of a diagnosis on the first attempt and is expected to prevent unnecessary surgeries, Guidry said.

Now with this system, our ability to make a diagnosis has gone from 30 percent to 74 percent. It's not perfect but a lot better," Guidry said.

The technology uses software that provides a map to doctors. The inReach software creates a 3-D image of the patient's CAT scan and allows the doctor to do a virtual bronchoscopy leaving virtual markers or registration points along the route to the tumor or lesion.

During the actual procedure, when these virtual markers are "touched" by the scope, the software matches the "live" image with the 3-D map.

Basically it was guess work to get into the periphery. This drives us. The tools help steer us," Guidry said.

Prior to the technology, a traditional bronchoscopy was guided with an active X-ray. An active X-ray is still used, but the inReach system offers a doctor an image inside the lungs.

The technology can also be used to tattoo lesions in the lungs for surgery and to place fiducials — or markers — inside the lungs for radiosurgical procedures at Lafayette General's CyberKnife Center of Louisiana.

Since early fall, 18 procedures have been performed at the hospital with the system.

In Louisiana, the inReach technology is in use in hospitals in the Greater New Orleans area and at Opelousas General Health System.

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**From:** Dutt's Logger <pdlogger@WINDSTREAM.NET>  
**Date:** February 22, 2010 4:42:21 PM EST  
**Subject:** [ALPHA-1] FDA Issues Warnings On Asthma Treatments

FDA Issues Warnings On Asthma Treatments

The Food and Drug Administration has issued a warning limiting several popular asthma drugs.

The FDA said long-acting drugs Serevent and Foradil should not be taken alone. It also moved to limit usage of Advair and Symbicort together.

All of the drugs contain long-acting beta agonists (LABAs), which can lead to a sudden, fatal asthma attack. The risk is most common in children, but adults are also warned by the FDA.

"The risks of hospitalization and poor outcomes are of particular concern for children; parents need to know that their child with asthma should not be on a LABA alone," said Dianne Murphy, director of the FDA's Office of Pediatric Therapeutics.

The FDA ruled patients should not use drugs containing LABAs alone and should use them for as short a time as possible. The FDA also said warnings need to be carried in the labels of the drugs.

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## GENE THERAPY

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**From:** Dutt's Logger <pdlogger@WINDSTREAM.NET>  
**Date:** February 6, 2010 1:09:07 PM EST  
**Subject:** FYI: PERHAPS - DOWN THE COPD ROAD 2/6/2010

Biomedical Company Investigates Stem Cell Therapy for COPD

According to the COPD Foundation, there are 24 million cases of COPD in the United States, and one COPD patient dies every 4 minutes. Now a biomedical company offers hope for a cure.

Entest Biomedical Inc, a subsidiary of Bio-Matrix Scientific Group Inc, has begun studies of the company's stem cell/laser regenerative therapy for COPD. According to an announcement, Entest has begun investigations into the effects of low energy near infrared radiation on cultured cells as a first step toward animal preclinical studies for the treatment of the disease.

The focus of these investigations, which are an extension of current Entest intellectual property covering an enhancement of stem cell growth and activity, will be on type II alveolar epithelial cells and related stem cells that reside in the lung.

In the announcement, Steve Josephs, PhD, co-principal investigator for Entest, stated that the company was pleased with the progress of its program, "which uses low level laser therapy for COPD as a means of inhibiting inflammation and stimulating lung

regeneration."

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## ALPHA-1 PRIMER

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**From:** Jules <julie.moore3@BIGPOND.COM>

**Date:** January 25, 2010 9:30:38 PM EST

**Subject:** [ALPHA-1] Forgotten etiology

### Background and history

Alpha-1 Antitrypsin deficiency is a genetic disorder characterized by low serum levels of AAT. Low plasma and alveolar concentrations of AAT in the human body predispose individuals to development of early-onset pulmonary disease, most commonly emphysema and COPD. Life-threatening liver disease is another possible consequence of AATD. Although it is one of the most common inherited conditions-it affects about 1 in 2000 to 5000 individuals-it is underrecognized.8 It is estimated that only about 5% of patients with AATD in the United States have been properly diagnosed; therefore, most affected patients are unaware that they could benefit from healthier lifestyle changes (such as smoking cessation) or from the specific therapy that is available.9

The understanding of AATD has evolved since it was initially discovered in the early 1960s by Laurell and Eriksson10 as the cause of familial emphysema. Alpha-1 Antitrypsin is synthesized primarily by the liver then released into the bloodstream, ultimately to protect the lungs by blocking the effects of neutrophil elastase (NE). Neutrophil elastase is secreted by neutrophils in response to infection or irritants in order to digest damaged tissue in the lungs. Alpha-1 Antitrypsin binds to the excess NE, which results in inactivation of the protease. Sometimes a gene mutation produces an abnormal form of the AAT protein that cannot be released from the liver, which means it cannot enter the bloodstream. Without the protection provided by this protein, the lungs are left vulnerable to attack by NE. Conversely, accumulation of excess AAT in the liver can lead to cellular congestion and destruction, with possible development of severe liver disease and organ failure.11

### Genetics and AAT plasma levels

More than 100 different genetic variants of AAT have been identified. Differences in the speed of migration on gel electrophoresis have been used to identify the protein variants. Normal individuals are homozygous for the M variant (Pi\*M/Pi\*M) and have AAT serum concentrations between 20 and 53  $\mu\text{mol/L}$ ; other genotypes lead to reduced levels of AAT. The S allele produces moderately low levels of this enzyme, and the Z allele produces very little AAT. Most individuals affected by a clinically significant deficiency have some combination of the 2 abnormal alleles. In individuals with Pi SS, Pi MZ, or Pi SZ phenotypes, blood levels of AAT are reduced to 40% to 60% of normal levels. The most common form of severe AAT deficiency occurs in those homozygous for the Z allele (Pi\*Z/Pi\*Z). Serum levels of AAT in these patients are about 3.4 to 7  $\mu\text{mol/L}$ -10% to 15% of normal serum levels. The protective threshold in serum levels has been identified as 11  $\mu\text{mol/L}$ . Emphysema develops in most (but not all) individuals with serum levels below 9  $\mu\text{mol/L}$ .12 Homozygotes for the Z allele account for approximately 95% of cases of clinically recognized AATD.13

### Epidemiology

Alpha-1 Antitrypsin deficiency is a common hereditary disorder affecting individuals in almost all regions of the world, including North America, Europe, the Middle East, Africa, Asia, and Australia. Alpha-1 Antitrypsin deficiency has been identified in all populations, but it is most common in individuals of Northern European and Iberian descent. Rates found among white people are similar worldwide, with an estimated 117 million carriers and 3.4 million affected individuals.14 If the estimated 19.3 million white COPD patients in the United States were tested for AATD, it is predicted that approximately 1.29 million new patients would be identified.14 This number includes severely deficient individuals in addition to carriers. Despite this prevalence, AATD is still largely undetected and underdiagnosed by health care providers.9 The results presented by de Serres and colleagues in 2006 suggest that targeted screening for AATD should be implemented in countries with large populations of white COPD patients.14 One of the main challenges for physicians is that AATD can cause similar respiratory symptoms to those seen in asthma or COPD. The earliest symptoms include shortness of breath, cough, excess sputum production, reduced ability to exercise, and wheezing. Symptoms can initially be sporadic; however, if wheezing is the main symptom, individuals are often diagnosed with asthma. Smoking or exposure to tobacco smoke accelerates the appearance of symptoms and damage to the lungs.

## Management of AATD

Although there is no cure for AATD, early diagnosis remains important because affected patients can modify their lifestyle choices to reduce the risk of emphysema and they can benefit from the effective therapy that is available. Treatment can include smoking cessation, inhaled bronchodilators, corticosteroids, and supplemental oxygen. The only specific therapy for AATD is augmentation therapy, which is administration of exogenous protease inhibitors in the form of pooled human plasma AAT. The aim of augmentation therapy is to restore the level of circulating AAT (ie, to raise and maintain serum levels above the protective threshold of 11  $\mu\text{mol/L}$ ) and thus arrest the course of the disease and halt any further damage to the lungs. There are currently 4 different commercially available purified pooled human plasma-derived AAT products that are licensed in several countries: Prolastin (Talecris Biotherapeutics), Aralast (Baxter), Zemaira (CSL Behring), and Trypsone (Probitas Pharma). Since 1988, only Prolastin has received regulatory approval in Canada.

Various studies have evaluated the effects of augmentation therapy, including several observational cohort studies with concurrent or historical controls<sup>1,2,4,5</sup> and, until recently, a single small randomized controlled trial<sup>3</sup> (Table 11-6).

The largest observational cohort study (level II evidence) was conducted by the US National Heart, Lung and Blood Institute (NHLBI).<sup>2</sup> In 1988 the NHLBI established a registry for individuals with severe deficiency of AAT as a means of collecting information on the natural history of AATD. In 1998 the NHLBI published data derived from 1048 patients who were followed for 3.5 to 7 years. Among all subjects receiving augmentation therapy (continuously or intermittently) no difference in FEV1 decline was detected; however, among patients with FEV1 values at 35% to 49% of the predicted values, FEV1 decline slowed significantly for subjects receiving therapy (mean difference 27 mL/year;  $P = .03$ ) compared with those not receiving therapy. Further, patients receiving augmentation therapy had a 36% reduction in mortality compared with patients not receiving therapy ( $P = .02$ ). The study was not randomized, so the differences favouring augmentation therapy might have been the result of other factors, such as smoking habits (more current smokers) and socioeconomic status (lower income and less insurance coverage), factors for which researchers could not control.

Lieberman addressed the question of clinical efficacy by conducting a Web-based survey of patients with the Pi ZZ phenotype who were not receiving therapy compared with patients who had received augmentation therapy for 1 to 10 years (level II evidence).<sup>4</sup> The results of this study, derived from the responses of 143 patients (96 receiving therapy; 47 not receiving therapy), suggest that augmentation therapy is associated with a significant reduction in the frequency and severity of lung infections in patients with AAT-related emphysema. Before starting augmentation therapy most patients reported a frequency of 3 to 5 infections per year; this number dropped to 0 to 1 infections per year while receiving therapy ( $P < .001$ ). Further, 55% of untreated patients experienced more than 2 infections per year compared with 18% of patients receiving therapy ( $P < .001$ ).

A more recent multicentre, retrospective cohort study evaluating augmentation therapy was conducted by Wencker and colleagues (level II evidence).<sup>5</sup> The progress of emphysema was evaluated in 96 patients with severe AATD before and after receiving weekly AAT augmentation therapy. Lung function tests were conducted for a minimum of 12 months, both before and after therapy. This was the first study comparing lung function before the introduction of augmentation therapy with lung function after treatment in the same individuals. The overall rate of FEV1 decline after initiation of augmentation therapy was significantly lower than the rate of decline before therapy (34.2 mL/year vs 49.2 mL/year;  $P = .019$ ). The highest reduction in the decline in FEV1 was observed in patients who had a rapid decline in FEV1 before initiation of augmentation therapy. Furthermore, even patients with FEV1 more than 65% of predicted values had a large and significant reduction in decline in FEV1 of 73.6 mL/year after augmentation therapy ( $P = .045$ ). The reported decline in FEV1 is similar to the reduction previously reported.<sup>1,2</sup>

The first randomized, parallel, double-blind, controlled trial comparing augmentation therapy to placebo in Pi ZZ phenotype ex-smokers with moderate emphysema was published in 1999 (level I evidence).<sup>3</sup> Twenty-six patients from the Danish Alpha-1-Antitrypsin Deficiency Registry and 32 patients from a similar Dutch registry participated. Patients were randomized to receive monthly infusions of either AAT therapy or albumin over a period of 3 years. Two Dutch subjects dropped out of the study during the first 2 years because they

resumed smoking; their data were omitted from further analyses. The rates of FEV1 decline using both laboratory-based spirometry and daily home spirometry measurements showed no difference between the treated and control groups. Unfortunately, this study did not have enough power to show a statistically significant effect of augmentation therapy on preventing the continuous decline of lung function compared with placebo. However, assessment of lung densitometry by chest computed tomography (CT) scans showed a nearly significant trend favouring a slower progression of emphysema in the augmentation therapy group ( $P = .07$ ).

Recently, a European randomized, multicentre, double-blind, placebo-controlled, parallel-group study was published (level I evidence).<sup>6</sup> This study investigated novel outcome measures to determine the effects of augmentation therapy on emphysema progression in AATD patients. A total of 77 patients with AATD were randomized to receive either weekly infusions (60 mg/kg) of AAT therapy or placebo (2% albumin) for 2 to 2.5 years. The primary end points were frequency of exacerbations and progression of emphysema as assessed by CT lung density measurement. The authors concluded that CT densitometry is a sensitive measure of the loss of lung tissue. Lung density decline was lower with augmentation therapy compared with placebo at all time points ( $P$  values for treatment difference ranged from .049 to .084), suggesting a beneficial treatment response to augmentation therapy.

Taken as a whole, the results of the published studies have shown that augmentation therapy is associated with impeded rates of decline of lung function and fewer lung infections.

## Guidelines

In 2001, the Canadian Thoracic Society published a position statement on AATD.<sup>15</sup> In view of the data available, and owing to the limited supply of AAT available for augmentation therapy, the authors recommended reserving AAT augmentation therapy for AAT-deficient patients who had FEV1 values at 35% to 50% of their predicted values, who had quit smoking, and who were taking optimal medical therapy yet continued to show rapid decline in FEV1. They also recommended that all AATD patients participate in the Canadian AAT Registry.

In 2005 the same authors revised their recommendations<sup>16</sup> because the limitations on the supply of AAT available had been alleviated as a result of increased production. Augmentation therapy is now recommended for individuals with FEV1 between 35% and 65% of predicted values, who have quit smoking and still have rapid decline in FEV1. Augmentation therapy is also recommended for treating the rare null homozygotes, because these individuals have no detectable AAT and are more likely to have accelerated decline in lung function than Pi ZZ patients are.

In 2003, joint guidelines for the diagnosis and management of individuals with AATD from the American Thoracic Society and the European Respiratory Society were published.<sup>12</sup> They recommended that a single quantitative test for AAT should be performed on all individuals with COPD who remained symptomatic despite bronchodilator therapy, individuals with emphysema, those who had asthma with airflow obstruction that was not completely reversible, and those with unexplained liver disease. They also recommended testing asymptomatic patients with both persistent airflow obstruction on pulmonary function tests and risk factors (eg, smoking) for the disorder (Box 112). Other advanced testing is usually not necessary.<sup>17</sup>

## Challenges

Despite these guideline recommendations there has been no improvement in the time taken to diagnose AATD; moreover, it appears that the disease is still largely undetected or misdiagnosed. Enhanced awareness of the disorder among health care providers should result in better management of AATD patients. Results of a patient survey showed that the average age of diagnosis for AATD patients was 45.5 years, and the average interval between onset of symptoms and diagnosis was 8.3 years.<sup>9</sup> Most unrecognized AATD occurs among those with COPD; as primary care physicians treat many of these patients, primary care physicians can play an important role in identifying and improving the management of AATD.

source: <http://www.cfp.ca/cgi/content/full/56/1/19>

Thank you,

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# Alpha-1 Nor'Easter

## Regional Support Group Newsletter (February 2010)

There are some exciting upcoming events. (more details S/G News - Page 2)

1. The Alpha-1 Foundation will be hosting a three day, training session for Building Friends for a Cure. They will pay your hotel and travel expenses! (3/11-13)
2. Celebrate St. Patrick's Day at the Celtic Connection. Enjoy the company of other Alpha Families while helping to find a cure! (3/13)
3. The Annual George Washington Bridge Walk, Saturday, May 8. Have fun and help raise money to find a cure.
4. The 19th Annual National Education Conference will be held on June 11-13, 2010 in Orlando, FL at the Hilton Walt Disney Resort. Take a vacation, bring the family it will be an awesome experience!  
I plan on attending

The NY/NJ Alpha-1 Support Group (not this newsletter) is endorsed by the:

# Alpha-1 Association



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