Energetic medicine for Retinitis Pigmentosa

Retinitis Pigmentosa

Classification and external resources

Fundus of patient with retinitis pigmentosa, mid stage (Bone spicule-shaped pigment deposits are present in the mid periphery along with retinal atrophy, while the macula is preserved although with a peripheral ring of depigmentation. Retinal vessels are attenuated.)

From a review by Christian Hamel, 2006.

ICD-10  H35.5

ICD-9  362.74

OMIM  268000

MedlinePlus  001029

MeSH  D012174

GeneReviews  Retinitis Pigmentosa Overview

Author - Editor: Professor of Medicine Desire’ Dubounet, D. Sc. L.P.C.C
Retinitis pigmentosa (RP) is an inherited, degenerative eye disease that causes severe vision impairment and often blindness. The progress of RP is not consistent. Some people will exhibit symptoms from infancy, others may not notice symptoms until later in life. Generally, the later the onset, the more rapid is the deterioration in sight. Those who do not have RP have 90 degree peripheral vision, while some people who have RP have less than 90 degrees.

A form of retinal dystrophy, RP is caused by abnormalities of the photoreceptors (rods and cones) or the retinal pigment epithelium (RPE) of the retina leading to progressive sight loss. Affected individuals may experience defective light to dark, dark to light adaptation or nyctalopia (night blindness), as the result of the degeneration of the peripheral visual field (known as tunnel vision). Sometimes, central vision is lost first causing the person to look sidelong at objects.

The effect of RP is best illustrated by comparison to a television or computer screen. The pixels of light that form the image on the screen equate to the millions of light receptors on the retina of the eye. The fewer pixels on a screen, the less distinct will be the images it will display. Fewer than 10 percent of the light receptors in the eye receive the colored, high intensity light seen in bright light or daylight conditions. These receptors are located in the center of the circular retina. The remaining 90 percent of light receptors receive gray-scale, low intensity light used for low light and night vision and are located around the periphery of the retina. RP destroys light receptors from the outside inward, from the center outward, or in sporadic patches with a corresponding reduction in the efficiency of the eye to detect light. This degeneration is progressive and has no known cure.

**Signs and symptoms**
The same view with tunnel vision from retinitis pigmentosa. The blackness surrounding the central image does not indicate darkness, but rather a lack of perceived visual information.

People may experience one or more of the following symptoms:

- Night blindness or nyctalopia;
- Tunnel vision (no peripheral vision);
- Peripheral vision (no central vision);
- Latticework vision;
- Aversion to glare;
- Slow adjustment from dark to light environments and vice versa;
- Blurring of vision;
- Poor color separation; and
- Extreme tiredness.

**Associated conditions**

Retinitis pigmentosa (RP) is seen in a variety of diseases, so the differential of this sign alone is broad.

- RP combined with deafness (congenital or progressive) is called Usher syndrome.
- RP combined with ophthalmoplegia, dysphagia, ataxia, and cardiac conduction defects is seen in the mitochondrial DNA disorder Kearns-Sayre syndrome (also known as Ragged Red Fiber Myopathy)
- RP combined with retardation, peripheral neuropathy, acanthotic (spiked) RBCs, ataxia, steatorrhea, is absence of VLDL is seen in abetalipoproteinemia.
- RP is seen clinically in association with several other rare genetic disorders (including muscular dystrophy and chronic granulomatous disease) as part of McLeod syndrome. This is an X-linked recessive phenotype characterized by a complete absence of XK cell surface proteins, and therefore markedly reduced expression of all Kell red blood cell antigens. For transfusion purposes these patients are considered completely incompatible with all normal and K0/K0 donors.

Other conditions include neurosyphilis, toxoplasmosis (Emedicine "Retinitis Pigmentosa") and Refsum's disease.
Genetics

Retinitis pigmentosa (RP) is one of the most common forms of inherited retinal degeneration. This disorder is characterized by the progressive loss of photoreceptor cells and may eventually lead to blindness.

There are multiple genes that, when mutated, can cause the retinitis pigmentosa phenotype. In 1989, a mutation of the gene for rhodopsin, a pigment that plays an essential part in the visual transduction cascade enabling vision in low-light conditions, was identified. Since then, more than 100 mutations have been found in this gene, accounting for 15% of all types of retinal degeneration. Most of those mutations are missense mutations and inherited mostly in a dominant manner.

Types include:

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<td>RPGR</td>
<td>Retinitis pigmentosa, X-linked, and sinorespiratory infections, with or without deafness</td>
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The rhodopsin gene encodes a principal protein of photoreceptor outer segments. Studies show that mutations in this gene are responsible for approximately 25% of autosomal dominant forms of RP.\(^3\)\(^6\)

Mutations in four pre-mRNA splicing factors are known to cause autosomal dominant retinitis pigmentosa. These are PRPF3 (human PRPF3 is HPRPF3; also PRP3), PRPF8, PRPF31 and PAP1. These factors are ubiquitously expressed and it is proposed that defects in a ubiquitous factor (a protein expressed everywhere) should only cause disease in the retina because the retinal photoreceptor cells have a far greater requirement for protein processing (rhodopsin) than any other cell type.\(^7\)

Up to 150 mutations have been reported to date in the opsin gene associated with the RP since the Pro23His mutation in the intradiscal domain of the protein was first reported in 1990. These mutations are found throughout the opsin gene and are distributed along the three domains of the protein (the intradiscal, transmembrane, and cytoplasmic domains). One of the main biochemical causes of RP in the case of rhodopsin mutations is protein misfolding, and molecular chaperones have also been involved in RP.\(^8\) It was found that the mutation of codon 23 in the rhodopsin gene, in which proline is changed to histidine, accounts for the largest fraction of rhodopsin mutations in the United States. Several other studies have reported other mutations which also correlate with the disease. These mutations include Thr58Arg, Pro347Leu, Pro347Ser, as well as deletion of Ile-255.\(^8\)\(^9\)\(^10\)\(^11\)\(^12\) In 2000, a rare mutation in codon 23 was reported causing autosomal dominant retinitis pigmentosa, in which proline changed to alanine. However, this study showed that the retinal dystrophy associated with this mutation was characteristically mild in presentation and course. Furthermore, there was greater preservation in electoretinography amplitudes than the more prevalent Pro23His mutation.\(^13\)

**Pathophysiology**

Animal models suggest that the retinal pigment epithelium fails to phagocytose shed rod outer segment discs, leading to accumulation of rod outer segment debris. In mice homozygous recessive for retinal degeneration mutation, rod photoreceptors stop developing and undergo degeneration before cellular maturation completes. A defect in cGMP-phosphodiesterase has also been documented; this leads to toxic levels of cGMP.
Symptoms

Retinitis pigmentosa (commonly referred to as "RP") is a disease characterized by loss of the light sensing photoreceptor cells that line the back of the eye, like the film of a camera. Usually the rod photoreceptors (responsible for night vision) are affected first, which is why loss of night vision (nyctalopia) is usually the first symptom. Daytime vision (mediated by the cone photoreceptors) is usually preserved until the late stages of the disease. Mottling of the retinal pigment epithelium with black bone-spicule pigmentation is typically indicative (or pathognomonic) of retinitis pigmentosa. Other ocular features include waxy pallor of the optic nerve head, attenuation (thinning) of the retinal vessels, cellophane maculopathy, cystic macular edema, and posterior subcapsular cataract.

Diagnosis

The diagnosis of retinitis pigmentosa relies upon documentation of progressive loss in photoreceptor cell function by electoretinography (ERG) and visual field testing. The mode of inheritance of RP is determined by family history. At least 35 different genes or loci are known to cause "nonsyndromic RP" (RP that is not the result of another disease or part of a wider syndrome).

DNA testing is available on a clinical basis for:

- **RLBP1** (autosomal recessive, Bothnia type RP)
- **RP1** (autosomal dominant, RP1)
- **RHO** (autosomal dominant, RP4)
- **RDS** (autosomal dominant, RP7)
- **PRPF8** (autosomal dominant, RP13)
- **PRPF3** (autosomal dominant, RP18)
- **CRB1** (autosomal recessive, RP12)
- **ABCA4** (autosomal recessive, RP19)
- **RPE65** (autosomal recessive, RP20)

For all other genes (e.g. DHDDS), molecular genetic testing is available on a research basis only.

RP can be inherited in an autosomal dominant, autosomal recessive, or X-linked manner. X-linked RP can be either recessive, affecting primarily only males, or dominant, affecting both males and females, although males are usually more mildly affected. Some digenic (controlled by two genes) and mitochondrial forms have also been described.

Genetic counseling depends on an accurate diagnosis, determination of the mode of inheritance in each family, and results of molecular genetic testing.
Treatment

Currently there is no cure for retinitis pigmentosa, but treatments are now available in some countries.

The progression of the disease can be reduced by the daily intake of 15000 IU (equivalent to 4.5 mg) of vitamin A palmitate in some patients. Recent studies have shown that proper vitamin A supplementation can postpone blindness by up to 10 years (by reducing the 10% loss pa to 8.3% pa) in some patients in certain stages of the disease. When it received market approval in February 2011, the Argus Retinal Prosthesis became the first approved treatment for the disease, and it is available in Germany, France, Italy, and UK. Interim results on 30 patients long term trials were published in 2012. The Argus II retinal implant has also received market approval in the USA. The device may help adults with RP who have lost the ability to perceive shapes and movement to be more mobile and to perform day-to-day activities. In June 2013 12 hospitals in the USA announced to soon accept consultation for patients with RP in preparation for the launch of Argus II later that year.

A Curry Spice May Cure Some Forms of Retinitis Pigmentosa

Turmeric is a versatile spice. This yellow orange root is used to flavor curry, dye food and clothing, and has been an ancient Asian remedy for many ailments (see Figure 1). Recently, a group of National Eye Institute-funded researchers found that curcumin, the active ingredient in turmeric, may also be effective in treating the blinding disease retinitis pigmentosa.

Figure 1: Curcumin is the active ingredient in turmeric, a yellow orange root that is dried and ground into powder used to spice curry.

Courtesy of the NEI.
Figure 2: After entering the eye, light shines through the retina to the photoreceptor layer where it is detected by rod and cone cells. RP destroys the rod and cone photoreceptor cells. Courtesy of the NEI and Washington University School of Medicine Neuroscience Tutorial, St. Louis, MO.

Figure 3: Retinas, from RP rats fed curcumin (right-hand image) were thicker than retinas of rats not fed curcumin (left-hand image). Some thickening was caused by preservation of rods and cones located in the outer nuclear layer (ONL; white arrow) of the retina. Courtesy of Dr. Ayyagari, UCSD.

Figure 4: Curcumin prevents abnormal clustering of mutant rhodopsin. Retinas from RP rats were stained with antibodies that detect rhodopsin (red) and with DAPI, a chemical that stains cell nuclei (blue). Mutant rhodopsin abnormally clustered near rod cell nuclei (left-hand image). Feeding curcumin to RP rats preserved the normal location of rhodopsin clusters to the outer segment (OS) of rods (right-hand image). Courtesy of Dr. Ayyagari, UCSD.

Affecting more than 1 in 4,000 people worldwide, retinitis pigmentosa (RP) is an untreatable disease that leads to severe vision loss and blindness. RP is a group of hereditary degenerative diseases caused by mutations in over 45 different genes. Current experimental treatments with gene transfer involve injecting healthy copies of the mutant gene into the eyes of patients. Although early clinical trials are promising, this approach is expensive and requires developing a treatment for every mutant gene that causes the disease. Now, with the publication of a study led by Radha Ayyagari, Ph.D., associate professor of ophthalmology at the University of California San Diego, doctors may look to curcumin as a simpler way to treat some common forms of RP.

"In my opinion the most advantageous way to treat RP is by identifying a molecule that can be used to treat multiple causes of the disease," Dr. Ayyagari explained.
The changes that cause RP destroy light-detecting photoreceptor cells called rods and cones (see Figure 2), which are located in the retina, the part of the eye that converts light into electrical signals sent to the brain. About 10% of RP cases are caused by mutations in the gene that encodes rhodopsin, a protein rod cells use to detect light. Many of these mutations, such as one called P23H, cause rhodopsin to abnormally cluster inside of rod cells, which impairs rod cell function and ultimately leads to cell death.

Previous studies suggested that curcumin may be an effective treatment for other neurodegenerative diseases caused by abnormal protein clustering. Specifically, a couple of studies showed that curcumin inhibited the formation of amyloid β plaques, the protein clusters thought to destroy neurons in Alzheimer’s disease.

In initial experiments performed by Dr. Ayyagari and her colleagues, curcumin prevented mutant P23H rhodopsin from abnormally clustering in cultured cells grown in petri dishes.

Having grown up in India, where turmeric is traditionally used to treat wounds and inflammations and where she studied nutritional biochemistry at the National Institute of Nutrition, Dr. Ayyagari has a strong appreciation for curcumin’s therapeutic potential.

"Coming from India I have a lot of faith in curcumin," Dr. Ayyagari explained.

To test this idea further, the researchers fed curcumin to rats genetically engineered to have the P23H mutation in rhodopsin. Previous studies showed that these rats have eye problems reminiscent of RP. Specifically, the eyes of these rats have poor electrical responses to light and over time, the retina thins with the loss of photoreceptor cells.

Feeding curcumin to the P23H rats alleviated these problems. Curcumin preserved the number of rods and cones in the retina (see Figure 3) and it increased the light-induced electrical response recorded from the rats’ eyes. These results suggest that curcumin may alleviate eye problems by preventing the loss of photoreceptors caused by the P23H mutation.

Interestingly, feeding curcumin to the rats caused rods to produce more rhodopsin and cones to produce more of S- and M-opsin, the proteins cones use to detect light. These results further support the idea that curcumin may preserve rod and cone cell function in retinas of patients who have RP caused by mutant P23H rhodopsin.

The P23H mutation causes rhodopsin to accumulate in the endoplasmic reticulum (ER), a maze of membranes located near a cell’s nucleus where new proteins are made. Rhodopsin is normally found in the outer segments of rod cells, where it captures light. The researchers looked at the location of the mutant P23H rhodopsin in rod cells by staining retinas from the engineered rats with antibodies that detect rhodopsin. As expected, rhodopsin was abnormally clustered near the nuclei of rod cells from rats that were not fed curcumin (see Figure 4). In contrast, rhodopsin was found in the outer segments of rats fed curcumin, suggesting curcumin preserved normal rhodopsin clustering.

Abnormal protein accumulation in the ER can stress cells, in general. In response, cells often turn on, or express, genes that at first may free the accumulating proteins from the ER but later may instruct the cells to die.

Experiments performed on cultured cells showed that curcumin prevented the expression of two stress genes, called immunoglobulin-binding protein (BiP/Grp78) and C/EBP-homologous protein (CHOP), which was induced when P23H rhodopsin accumulated in the ER. When the researchers looked at retinas from the engineered rats they found that feeding them curcumin prevented the expression of
the BiP/Grp78 gene in rod cells but not of the CHOP gene. Although further work is needed, these results suggest that curcumin may partially prevent stress responses in rods.

Delivering drugs to the retina is often hindered by the blood-retina barrier, a protective meshwork of cells surrounding the retina which prevents many compounds and chemicals in the bloodstream from entering the retina. The researchers detected curcumin in the retinas of rats after only two days of feeding, indicating that it passes through the blood-retina barrier. These results suggest that patients with RP could simply take curcumin pills or include turmeric in their diet rather than have a drug or gene surgically injected into their eyes.

Demonstrating its passage through the blood-retina barrier also suggests that curcumin could be used to treat any eye disease caused by abnormal protein clustering. For instance, some mutations associated with RP appear to cause abnormal clustering of other proteins found in rods, such as phosphodiesterase 6 (PDE6), a protein that helps rhodopsin convert light into electrical signals. Abnormal protein clustering may also be associated with eye diseases that affect other cells in the retina. The results reported by Dr. Ayyagari and her colleagues suggest that curcumin could treat all of these cases.

Do her results mean that RP patients should include turmeric in their diet?

"We don’t know yet. The amounts used in this study are more than one would get in a normal diet. We need to test the effective dose of curcumin in patients," Dr. Ayyagari replied as she explained the next step in her promising research.

- By Christopher G. Thomas, Ph.D.
Publication Citation:


**Study Shows Vitamin A Slows Retinitis Pigmentosa**

*June 1993 Archives of Ophthalmology*

Most adults with blinding Retinitis Pigmentosa should take a daily 15,000 IU vitamin A palmitate supplement and avoid high dose vitamin E to help prolong their vision.

This recommendation is the first from a well-designed clinical trial indicating that people can be treated for Retinitis Pigmentosa. Alan M. Laties, M.D., chairman of The Foundation Fighting Blindness’ Scientific Advisory Board, said of these findings, “This treatment can be a benefit, a very real one, for people who have Retinitis Pigmentosa. Although not a cure, it will improve quality of life, potentially adding many years of useful vision. The discovery that vitamin E is of no use, and in fact potentially harmful, is not only important on its own, but further justifies this extensive study.”

Eliot L. Berson, M.D., the study’s principal investigator and Professor of Ophthalmology at Harvard Medical School, said that adults who supplemented their diets with 15,000 IU of vitamin A palmitate daily had on average about a 20 percent slower annual decline of remaining retinal function than those not taking this dose.
Based on this finding, the investigators estimated that an average patient in the study, who started taking a daily supplement of 15,000 IU vitamin A palmitate at age 32, would retain some useful vision until age 70, whereas a patient not on this dose would lose useful vision by age 63.

The investigators also recommended that adults with Retinitis Pigmentosa should avoid taking high-dose vitamin E supplements. In the study, the disease appeared to progress faster on average in patients on a daily 400 IU vitamin E supplement than in those taking a trace amount of the vitamin. However, the study showed no evidence that normal dietary or small supplemental amounts of vitamin E have an adverse effect on the disease.

The carefully designed and monitored clinical trial involved 600 patients between the ages of 18 and 49, who were at different levels of visual function. The six-year study was conducted at a cost of $5 million at the Berman-Gund Laboratory for the Study of Retinal Degenerations at Harvard Medical School, with the support of The National Eye Institute and The Foundation Fighting Blindness.

The investigators stressed that adults considering vitamin A palmitate supplementation should first consult with their doctor. Fasting blood levels of vitamin A should be measured and liver function tests administered before starting treatment. People with certain pre-existing medical conditions may not be eligible for this treatment.

Make a point of eating a balanced diet, without selecting foods that are especially high in vitamin A. Avoid taking high-dose vitamin E supplements.

Commenting on the results of the vitamin A study, Dr. Berson stated, "One of my biggest concerns is that people will make the mistake of thinking that vitamin A supplementation in excess of the 15,000 IU recommended will provide even greater benefit. We have evidence in fact that supplementation of a regular diet with greater than 15,000 IU of vitamin A does not provide greater benefit. Moreover, a daily vitamin A intake exceeding 25,000 IU over the long-term can be toxic in adults and may cause side effects such as liver disease.

“Our interpretation of the study results is that the course of the common forms of retinitis pigmentosa is slower on average among adult patients on a regular diet who take a daily 15,000 IU vitamin A supplement in the palmitate form compared with the course of those patients not on this supplement.”

Because the study involved adults between the ages of 18 and 49, no formal recommendations can be made for patients under the age of 18. Women with Retinitis Pigmentosa should not take 15,000 IU vitamin A palmitate supplements during the time leading up to planned pregnancy and during pregnancy, because high doses of vitamin A have been linked to birth defects.

If you have questions regarding the vitamin A treatment, The Foundation publishes a free booklet entitled, Vitamin A treatment for Retinitis Pigmentosa. The Foundation also distributes a list of mail order companies that provide vitamin A palmitate in 15,000 IU capsules or tablets as the correct dose is not available in most local food stores. Beta carotene is not a suitable substitute for Vitamin A Palmitate in the context of this treatment.
Pure Focus™ - Lutein Sublingual Spray  the doctors in the USA who pioneered this alternative treatment for Macular Degeneration also found that Retinitis Pigmentosa also responded. Quite why this is if the problem is genetic ‘in the eye’ is baffling. If the problem is lack of nutritional uptake in the digestive tract (genetic) or a problem absorbing the nutrients in the eye then it may explain ‘why’. The simple fact is it does respond at any age and the average person could not care less why it works.

Lutein Formula Spray is a sublingual spray containing Lutein, Zeaxanthin, L-Lysine, Bilberry and Gingko. This is the best product for the delivery of these specific nutrients to the eye. Studies have shown these nutrients help improve and prevent vision loss due to Macular Degeneration. Lutein and Zeaxanthin filter light and serve as potent free radical scavengers for the Retina. L-Lysine and Bilberry help maintain healthy blood vessels. Gingko improves blood flow to the blood vessels in the eye and improves the utilisation of glucose and oxygen, increasing ATP production and preventing vasoconstriction to the brain and retina (and improving memory).

Pure Focus™ - Lutein Sublingual Spray has 800% more benefit than tablets or capsules. Can improve vision and prevent loss due to Macular Degeneration and other eye diseases. Recommended dosage: 6-8 sprays per day for the first two months then 3 sprays per day for the next two months and lastly 1 spray per day as a preventative.

Additional Natural Remedies for Retinitis Pigmentosa
Essential EFAs in liquid form must contain at least 480mg of DHA and 720mg of EPA per teaspoon. This helps improve circulation, integrity of blood vessels, brain function, flexibility and permeability of cell membranes. It also helps protect the retina’s photoreceptor cells. HEMP OIL is the most balanced single source of essential fatty acids, (recommended dose one dessertspoon daily) but courses of cod-liver oil (one dessertspoon daily, not the capsules) also recommended.

Zell Oxygen provides a broad range of nutrients anti-oxidants and enzymes that maintain healthy cellular activity. At least one dessert spoon daily.

Alpha Lipoic Acid is the only antioxidant that is both fat and water-soluble. Studies have shown that it is able to repair oxydative and free radical damage and its protective effect extends to nearly every cell in the body. It has also been found to enhance the action of Glutathione, vitamins E and C. Recommended dose one or two capsules daily.

L- Carnosine rejuvenates cells, this nutrient has been the subject of scientific study and has been found to help rejuvenate dying cells. We do not know if this is applicable to degenerative eye diseases, Recommended dose 3 capsules daily

MicroCurrent Stimulation with SCIO/Eductor/Indigo
Stimulates ATP and cellular regeneration and has been shown in studies to improve the overall success. Recommended use: Stimulate Appropriate SCIO, Indigo, Eductor MicroCurrent Points. Treatments per day for the first 2-4 weeks and once a day thereafter.

Use the Scalar Cybermagnetic Wand to treat these points in Numerical Sequence

Set Device to Anti Inflammation 1-2 on the 1st day 3-4 next day
Use the Cybermagentic Chair to treat the chromosomal and gene problems.

Cybermagnetic

Using the computers headphone and microphone jacks we can first analyze the patient's voice patterns for energetic disturbance and then chose sound files for relaxation, healing or energy. The music is sent into the body thru the headphones and a magnetic field generator. A magnetic field detector then receives the signals from the body establishing a cybermagnetic loop. The computer can then change the music to help the patient's body electric.

The Cybermagnetic Chair can be purchased with the zero gravity chair you see for 1200 extra, or with the simple back cybermagnetic pads to put on your own chair for 5,000 euro with the QT software included. This sytem can operate indeendently or interf ace with your QXCI, SCIO, Indigo or Eductor.

Turn Back the Hands of Time of Your DNA Clock

With Cybermagnetics
Research

Future treatments may involve retinal transplants, artificial retinal implants,[18] gene therapy, stem cells, nutritional supplements, and/or drug therapies.

2006: Stem cells: UK Researchers working with mice, transplanted mouse stem cells which were at an advanced stage of development, and already programmed to develop into photoreceptor cells, into mice that had been genetically induced to mimic the human conditions of retinitis pigmentosa and age-related macular degeneration. These photoreceptors developed and made the necessary neural connections to the animal's retinal nerve cells, a key step in the restoration of sight. Previously it was believed that the mature retina has no regenerative ability. This research may in the future lead to using transplants in humans to relieve blindness.[19]

2008: Scientists at the Osaka Bioscience Institute have identified a protein, named Pikachurin, which they believe could lead to a treatment for retinitis pigmentosa.[20][21]

2010: A possible gene therapy seems to work in mice. [1]

2010: R-Tech Ueno (Japanese Medicine manufacture enterprise) completes phase II clinical study on ophthalmic solution UF-021 (Product Name Ocuseva (TM)) for Retinitis Pigmentosa

2012: Scientists at the Columbia University Medical Center showed on an animal model that gene therapy and induced pluripotent stem cell therapy may be viable options for treating retinitis pigmentosa in the future.[22]

2012: Scientists at the University of Miami Bascom Palmer Eye Institute presented data showing protection of photoreceptors in an animal model when eyes were injected with mesencephalic astrocyte-derived neurotrophic factor (MANF).[23]

References

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Naturopathic Causes and Treatment of Retinitis Pigmentosa

Please see also Carahealth Eye.

Retinitis pigmentosa (RP) is a group of genetic eye conditions. In the progression of symptoms for RP, night blindness generally precedes tunnel vision by years or even decades. Many people with RP do not become legally blind until their 40s or 50s and retain some sight all their life. Others go completely blind from RP, in some cases as early as childhood. Progression of RP is different in each case.

RP is a type of hereditary retinal dystrophy, a group of inherited disorders in which abnormalities of the photoreceptors (rods and cones) or the retinal pigment epithelium (RPE) of the retina lead to progressive visual loss. Affected individuals first experience defective dark adaptation or nyctalopia (night blindness), followed by reduction of the peripheral visual field (known as tunnel vision) and, sometimes, loss of central vision late in the course of the disease.

Signs

Mottling of the retinal pigment epithelium with black bone-spicule pigmentation is typically indicative (or pathognomonic) of retinitis pigmentosa. Other ocular features include waxy pallor of the optic nerve head, attenuation (thinning) of the retinal vessels, cellophane maculopathy, cystic macular oedema and posterior subcapsular cataract.

Diagnosis

The diagnosis of retinitis pigmentosa relies upon documentation of progressive loss in photoreceptor function by electroretinography (ERG) and visual field testing. The mode of inheritance of RP is determined by family history. At least 35 different genes or loci are known to cause "nonsyndromic RP" (RP that is not the result of another disease or part of a wider syndrome). Genetic counselling depends on an accurate diagnosis, determination of the mode of inheritance in each family, and results of molecular genetic testing. RP combined with progressive deafness is called Usher syndrome.

RP and Autoimmune Disease

There is seems to be an autoimmune component to RP. This is good news for sufferers who have no family history of the condition. And it may also means that treatment can now be directed towards sedating an overactive immune system wither with pharmaceutical drugs or with Complementary and Alternative Medicine (CAM).

Research unveiled at The Second International Conference on Cortisols and Anti-Cortisols reported that retinitis pigmentosa (RP) is composed of two separate diseases -- one genetic and the other autoimmune. While the inherited
genetic component has no present therapy, the auto-immune condition can be treated with anti-cortisol nutrients and drugs.[1] Increased risk of autoimmune hypothyroidism in patients affected by retinitis pigmentosa.

Abstract
Patients with Retinitis Pigmentosa (RP) show hemeralopia, restricted field of vision and reduced visual acuity, owing to the degeneration and proliferation of photoreceptors and of retinal pigment epithelium. The prevalence in Italy is 1:4,000. A certain number of syndromic associations have been described, and, in particular, also that with hypothyroidism, but very few cases have been studied. We describe a family of 40 people, spanning four generations, in which we have recorded the presence of autosomic dominant RP, associated with autoimmune hypothyroidism or with circulating antithyroid autoantibodies (ATA), currently considered as the expression of active autoimmune thyroiditis or a risk factor for this complaint. We measured, in all members, TSH, FT3, FT4, antithyroglobulin and antithyroxperoxidase autoantibodies. A fundus oculi examination was performed in every subject, as well as a careful examine of the anterior region of the neck. A control population of 100 healthy people was also studied. Our data show a higher prevalence of ATA, statistically significant, in the patients with RP and in their relatives, compared with the control population and the data from the literature (13 cases over 40 = 32.5% ; p<0.01).

3 patients with RP and ATA were affected by clinically evident hypothyroidism. 10 patients with ATA were clinically euthyroid ; 8 patients affected by RP did not show circulating ATA at the time of the study.

The interest for the physician in this syndromic retinal distrophy reflects the need, emerging from our data, to test the thyroid function in the subjects with RP and in members of their families, since circulating ATA are considered a risk factor for the development of autoimmune hypothyroidism.[2]

Pigmentary retinal degeneration has been seen in a number of diseases considered to have an immunological basis e.g. Behcet's disease, polyarteritis nodosa, rheumatoid arthritis, multiple sclerosis and Harada's disease.[3] [4]

Antigenicity of retinal neuroepithelium has been well documented. It is known that the cornea, uvea, pigment epithelium of retina and the lens share some common antigens. Uveitogenic nature of retina has been demonstrated in various animal experiments by different workers.[5]Further work has demonstrated that is the photoreceptors in the retina which are antigenic[6] [7]. Various immunoglobulin abnormalities have been described in retinitis pigmentosa. These include rheumatoid factor positivity in the serum in as many as 38% cases and significantly elevated IgM level in the serum[8] [9]Some initial success has been reported in treating cases of retinitis pigmentosa with D-penicillamine[10]. The utility of D-Penicillamine in treating diseases like rheumatoid arthritis and PSS has been well established for sometime now.

These observations have raised interesting possibilities:

a) Since it is known that autologous 1gM can lyse neuraminidase treated host cell, it has been suggested that naturally occurring cytotoxic antibodies of this class could be important in the process of ageing[11]. However, selective degeneration of visual cells is not a feature of old age. therefore it is difficult to regard retinitis pigmentosa as a product of premature senility, hence the role of raised IgM levels in retinitis pigmentosa. remains unclear.

(b) Since the production of IgM is genetically controlled and a large production of cases of retinitis pigmentosa are hereditary in origin, it is possible that individuals with raised IgM are more susceptible to retinal degeneration.
(c) Raised IgM in retinitis pigmentosa may be suggestive of an occult infection especially in view of the suggestion that retinitis pigmentosa may be caused by a slow virus infection[12]. Thus, there is great deal of evidence to suggest that retinitis pigmentosa may be a disease of on autoimmune etiology. [13]

**Complementary Medicine Treatment for Retinitis Pigmentosa**

**Vitamin A**

There is currently no medical treatment that can completely cure retinitis pigmentosa, although the progression of the disease can be reduced by the daily intake of 15000 IU of vitamin A palmitate. Recent studies have shown that proper vitamin A supplementation can postpone blindness by up to 10 years. A study on 1603 patients with retinoisis pigmentosa found treatment with vitamin A palmitate 15,000 IU/day. Evidence is presented to support the idea that patients with a projected cone amplitude of 3.5 microV or greater at age 40 (about 25% of our patient population with typical retinitis pigmentosa) would be expected, on average, to retain some useful vision for their entire lives without treatment. Knowledge of the amount of remaining cone function in the ERG often reduces patient anxiety and helps patients plan for their future. [14]

**Vitamin E and Vitamin A**

Vitamins A and E in the treatment of retinitis pigmentosa This study, from the Massachusetts Eye and Ear Infirmary, Boston, was a randomised, controlled, double-masked trial of vitamin A and vitamin E supplementation in 601 patients with retinitis pigmentosa. The patient, aged 18-49 years, were randomly assigned to receive 15,000 IU of vitamin A (as retinyl palmitate), 400 IU of vitamin E, both vitamins, or a placebo (containing trace levels of the vitamins) daily for four to six years. The principal outcome measure was cone electroretinogram amplitude, an objective measure of retinal function.

Patients receiving vitamin A (with or without vitamin E) showed a slower rate of decline in retinal function, as measured by cone electroretinogram amplitude, in comparison with those not receiving vitamin A. Vitamin E showed no effect in the group as a whole. However, in a subgroup of patients with initially better retinal function, those receiving vitamin E showed a faster rate of decline in retinal function than those not receiving the vitamin.

“These results support a beneficial effect of 15,000 IU/ d of vitamin A and suggest an adverse effect of 400 IU/ d of vitamin E on the course of retinitis pigmentosa.” The authors recommend that “most adult patients with the common forms of retinitis pigmentosa” should take a vitamin A supplement under medical supervision and that these patients should avoid the use of high-dose vitamin E supplements. [15]

**Taurine, Diltiazem and Vitamin E**

Treatment with taurine, diltiazem, and vitamin E retards the progressive visual field reduction in retinitis pigmentosa: a 3-year follow-up study. The purpose of this study to assess the effect of the formula taurine/diltiazem/vitamin E on the progression of visual field loss in retinitis pigmentosa.

A double blind, placebo controlled study in 62 patients: visual field threshold values were obtained in a Humphrey Field Analyzer from center (30 degrees) and periphery (30-60 degrees), every 4 months during 3-year follow-up. Data were analysed by univariate regression, with slopes obtained from the best fit lines. Based on slope values, three groups of patients were identified as those showing negative, positive, or zero slope: > or = 1 to < or = +1. In controls (32 patients), at central area, the distribution in negative, zero, or positive slope was, respectively, 16 (50%), 11
(35%), and 5 (15%). In the treated group (30 patients) this distribution was 6 (20%) negative, 17 (53%) zero, and 7 (23%) positive slope. In periphery, 16 control patients were distributed as 11 (69%) negative, 4 (25%) zero, and 1 (6%) positive slope. In the treated group (17 patients), the distribution was opposite: 1 (6%) negative, 7 (41%) zero, and 9 (53%) positive slope. Nineteen patients receiving treatment up to 6 years showed similar distribution by slope values. Eight out of 9 patients switched from placebo (2 years) to treatment (2-3 years), showed improving changes in their slope values. A beneficial effect of the treatment decreasing the rate of visual field loss was observed, likely through a protective action from free radical reactions in affected photoreceptors.[16]

Reported effects of non-traditional treatments and complementary and alternative medicine by retinitis pigmentosa patients.

BACKGROUND:
Benefits of complementary and alternative medicine (CAM)-related interventions have been demonstrated for patients with chronic, systemic diseases in which stress, anxiety and disability are prevalent. Subjects with retinitis pigmentosa (RP) commonly indicate that they have 'good' and 'bad' vision days, stating that stress causes a decrease in vision and that vision improves when the stress is alleviated. We assessed CAM use by RP patients and its perceived effectiveness. METHODS: We enquired about nine CAM areas: meditation, mind-body therapies, yoga, movement therapies, energy therapies, acupuncture, massage therapy, spirituality/religion and herbal therapies/aromatherapy. Ninety-six RP patients with any level of vision completed an anonymous internet survey.

RESULTS:
Ninety-five per cent of respondents tried at least one of the nine CAM areas. Seventy-five per cent have used nutritional supplements, including lutein (47 per cent), bilberry (32), vitamin A palmitate (36) and docosahexaenoic acid (23 per cent). Some tried meditation (47) and yoga (31 per cent). Stress and anxiety levels were reported as improved in 93, 92 and 87 per cent of those who used yoga, meditation and mind-body therapies, respectively. Many of those who tried mind-body therapies (40) or acupuncture (50 per cent), used it with a desire to fight RP. Vision was subjectively affected in 65 per cent of acupuncture users and from 20 to 35 per cent of the users of the other CAM areas. Those who indicated that their vision was affected by at least one type of CAM (35 per cent) were statistically more likely to require magnification to read (that is, they had lost more vision and RP had progressed), than those who did not believe vision was impacted (59 versus 84 per cent).

CONCLUSIONS:
RP patients are using CAM and are experiencing some impact on vision and physical/emotional well-being. Clinicians and researchers should be aware of its use. Clinical trials with CAM interventions are necessary to attempt to validate these findings.[17]

Scientists at the Osaka Bioscience Institute have identified a protein, named Pikachurin which they believe could lead to a treatment for retinitis pigmentosa.[18]

In a study published in the journal Nature researchers working with mice at the University College London Institutes of Ophthalmology and Child Health and Moorfields Eye Hospital transplanted mouse stem cells which were at an advanced stage of development, and already programmed to develop into photoreceptors, into mice that had been genetically induced to mimic the human conditions of retinitis pigmentosa and age-related macular degeneration. These photoreceptors developed and made the necessary neural connections to the animal’s retinal nerve cells, a
key step in the restoration of sight. Previously it was believed that the mature retina has no regenerative ability. This research may in the future lead to using transplants in humans to relieve blindness.[19]

Bilberry and retinitis pigmentosa
Loss of dark adaptation is characteristic of retinitis pigmentosa. Studies have shown that dark adaptation is greatly improved by bilberries (European blue berries).

Electrical Currents and Zinc supplementation and retinitis pigmentosa
Zinc supplementation can slow but not stop vision loss. A study by Michael and Allen used nutrients and zinc. They also applied 200 microamperes of electricity ±9 volts square wave, 10 cycles/sec.) to closed eyelids. Acuity improved or was stabilised for 15 out of 25 macular degeneration patients, monitored for five years. Other studies have shown that the application of weak electrical currents to the eye has positive benefit in macular degeneration and other conditions.[20]There seem to be no known adverse effects from using microamperage electric current on the eyes. Our use of 200 micro amperes, ±9 volts at 10 cycles per second on moist dosed eye lids, produces only a sensation of flickering light.

Anti-Cortisols May Offer New Hope For Retinitis Pigmentosa
LAS VEGAS, NV -- November 17, 1997-- New research revealed at The Second International Conference on Cortisols and Anti-Cortisols reported that retinitis pigmentosa (RP) is composed of two separate diseases -- one genetic and the other autoimmune. While the inherited genetic component has no present therapy, the auto-immune condition can be treated with anti-cortisol compounds.

These new findings were disclosed in a study presented by Alfred Sapse, M.D., a leader in cortisol/anti-cortisol research and former director of ophthalmic immunology at Cedars-Sinai Medical Centre in Los Angeles. "RP was previously viewed as an untreatable, solely genetic condition," Sapse said. "By uncovering its auto-immune component, we can attack the disease with a new arsenal of anti-cortisol compounds.

"RP's auto-immune component is believed to be associated with elevated levels of the stress hormone cortisol. Known as the fight or flight stress hormone, cortisol can throw the immune system into chaos and ravage the human body. While it was previously regarded as merely a symptom or marker of serious diseases, high levels of cortisol are now believed to be a major component of such diseases and conditions as RP, AIDS, MS, Alzheimer's, the aging process and several forms of cancer. According to Sapse, RP can be treated initially with a cocktail of anti-cortisol nutritional compounds including vitamin A, zinc, ginkgo biloba and acetyl-L-carnitine, followed by treatment using anti-cortisol/steroidogenesis inhibitor drugs.

The key to treatment of RP with anti-cortisol drugs is the ability to accurately measure when cortisol levels are elevated. In this regard, Sapse has introduced a new 24-hour circadian cortisol chart to accurately measure levels of the hormone in RP patients.[21]

Herbal medicine for retinitis pigmentosa
Please see also Carahealth Eyes
This mix contains a blend of herbs known as ophthalmics that treat eye diseases. The herbs are specifically rich in Vitamin A, carotenoids and anthocyanidins. Anthocyanidins, are antioxidants that neutralise free radical damage to the collagen matrix of cells and tissues that can lead to cataracts, glaucoma, varicose veins, haemorrhoids, peptic
ulcers, heart disease and cancer. The herbs have been shown in numerous studies to improve night time visual acuity and promote quicker adjustment to darkness and faster restoration of visual acuity after exposure to glare. Ideal for pilots!

Clinical Success in China

Chinese herbal doctors have reported some successful applications of these herbal combinations;

**Concha Haliotidis Source:** The shell of *Haliotis diversicolor* Reeve, *H. Gigantea discus* Reeve and *H. ovina Chemnitz*, family Haliotidae.

**Indication:**
(a). Calm the liver, benefit yin and suppress the sthenic yang: for sthenia of liver-yang or deficiency of yin leading to hyperactivity of yang manifested as dizziness, headache, tinnitus, irritability and insomnia.

(b). Clear away liver-fire to improve visual acuity: For conjunctivitis of liver-heat type; for night blindness; for blurring of vision due to liver-deficiency; for nebula, ointment should be applied topically.

(c). Antacid and analgesic: For stomachache and regurgitation.

**Pharmacological Actions:**
(a). Improves eyesight.

(b). Hemostatic.

**Fructus Corni Source:** The pulp of *Cornus officinalis* Sieb. et Zucc., family Cornaceae.

**Indication:**
(a). Invigorate the liver and kidney, supplement the essence and improve visual acuity: For deficiency of the liver and kidney manifested as soreness of the waist and knees, flaccidity of lower limbs, impotence, frequent micturition, sterility, dizziness, tinnitus and blurring of vision.

(b). Astringe and preserve essence: For hypofunction of liver and kidney manifested as emission, enuresis, frequent micturition, metrorrhagia, menorrhagia, spontaneous perspiration, night sweat, collapse with profuse perspiration and dyspnea of asthenic type.

**Pharmacological Actions:**
(a). Relieving cyclophosphamide-induced leukopenia inmice.

(b). Diuretic and hypotensive.

(c). Inhibiting the growth of *Bacillus dysenteriae* in vitro.

**Colla Cornus Cervi Source:** The gelatin made of the antler of *Cervus nippon Temminck or Cervus elaphus Linnaeus*, family Cerridae.

**Indication:**
Warming and tonifying liver and kidney, enriching essence and blood. Indicated for impotence, spermatorrhea, aching and coldness in the loin and knees, and metrorrhadia.
Fructus Lycii Source: Fruit of Lycium barbarum L., family Solanaceae.

Indication:
Nourish yin, enrich blood, benefit essence and improve visual acuity: For deficiency of liver-yin and kidney-yin and insufficiency of essence and blood manifested as dizziness, blurring of vision, hypopsia, tinnitus, emission and soreness of the loin and extremities; also for diabetes.

Pharmacological Actions:
It contains vitamins C, B1 and B2, carotene, nicotinic acid, β-sitosterol and betaine, and can relieve the liver damage induced by CCl4 in mice.

Fructus Schisandrae Source: Fruit of Schisandra chinensis (Turcz.) Baill. and S. sphenanthera Rehd. et Wils., family Magnoliaceae.

Indication:
(a). Benefit vital energy and promote the production of body fluid: For insufficiency of vital energy and body fluid manifested as fatigue, shortness of breath, palpitation, hyperhidrosis (spontaneous perspiration or night sweat), thirst, diabetes, etc.

(b). Astringe lung-energy, relieve dyspnea and cough: For cough and dyspnea of lung-deficiency type, lung-cold type and the type of deficiency of both lung and kidney.

(c). Invigorate kidney to preserve essence: For heart-deficiency syndrome with palpitation, insomnia, and dreaminess. In addition, its powder preparation is used for chronic hepatitis with elevation of serum transaminase.

Pharmacological Actions:
(a). Its decoction exerts bacteriostatic effect on Pseudomonas aeruginosa (1:128), Staphylococcus aureus (1:64), Flexner's bacillus (1:32) and typhoid bacillus (1:8) in vitro.

(b). Schizandrin, one of its active component, can protect the liver from damage.

(c). Enhancing lymphocyte-blastogenesis rate.

(d). Cardiotonic and sedative.

Rhizoma Cimicifugae Source: Rhizome of Cimicifuga heracleifolia Kom., C. dahurica (Turcz.) Maxim. and C. foetida L., family Ranunculaceae.

Indication:
(a). Expel wind and heat, clear away toxic materials and let out skin eruption: For common cold of wind-heat type, and measles with indistinct eruptions.

(b). Lift up yang-energy: For visceroptosis.

(c). Clear away heat and expel fire: For toothache due to stomach-heat, aphthae and headache.

Pharmacological Actions:
(a). Antipyretic, analgesic and anti-inflammatory. Gastric infusion of its extract (mainly containing ferulic acid) in a dosage of 1.0g/kg can lower the body temperature of normal rats, and relieve the pain elicited by acetic acid in mice.

(b). Inhibiting myocardium, slowing heart rate and lowering blood pressure.

**Semen Cuscutae Source: Seed of Cuscuta chinensis Lam., family Convolvulaceae.**

**Indication:**

(a). Invigorate the kidney and supplement essence: For insufficiency of kidney-yang or kidney-essence manifested as impotence, emission, enuresis, frequent micturition, sterility, leucorrhagia, soreness of the loin and knees, tinnitus and deafness.

(b). Soothe the foetus: For deficiency of the liver and kidney manifested as threatened abortion and habitual abortion.

(c). Nourish the liver and improve visual acuity: For blurring of vision and hypopsia due to insufficiency of the liver and kidney.

(d). Benefit the spleen to relieve diarrhea: For loose stools or diarrhea due to deficiency of the spleen and kidney. recently, also used for aplastic anemia and chyluria.

**Pharmacological Actions:**

(a). Cardiotonic.

(b). Promoting lymphocyte-blastogenesis

**Radix Bupleuri Source: Root of Bupleurum chinense DC. and the root or herb of B. scorzonerifolium Willd., family Umbelliferae.**

**Indication:**

(a). Expel the exogenous evil from the body surface, let off heat, and clear away evil heat from shaoyang channel: For common cold with fever, alternating episodes of chilliness and fever, malaria.

(b). Disperse the stagnated liver energy: For stagnation of liver energy with hypochondriac pain, dizziness, mental depression and irregular menstruation.

(c). Life up yang-energy; For visceroptosis.

**Pharmacological Actions:**

Saikosaponin is the active component.

(a). Increasing the hypnotic effect of barbital in mice.

(b). Analgesic, anti-inflammatory, antitussive and antipyretic.

(c). Decreasing the damage of liver by CCl4 and increasing biliary secretion in rats.

(d). Hemolytic in vitro.
(e). Lowering blood pressure in rabbits and inhibiting the heart of frog and guinea-pigs in vitro.

**Fructus Ligustri Lucidi Source**: Fruit of *ligustrum lucidum* Ait., family Oleaceae.

**Indication**:
(a). Tonify the liver and the kidney, darken the hair and promote the visual acuity: For deficiency of liver-yin and kidney-yin manifested as dizziness, tinnitus, blurring of vision, weakness of the loin and knees, hectic fever, nocturnal emission, alopecia and poliosis. Recently also used for seborrheic alopecia, central retinitis, early cataract, etc.

(b). Tranquilize the mind by nourishing the heart: For insufficiency of heart-yin manifested as insomnia, palpitation and precordial pain. Recently, also used for angina pectoris, hyperlipemia and neurasthenia, especially those of yin-deficiency type. In addition, also used for leukocytopenia, viral hepatitis with yin-deficiency syndrome; its component oleanolic acid for various kinds of hepatitis.

**Pharmacological Actions**:
(a). Its component oleanolic acid can prevent and relieve cyclophosphamide-induced leukocytopenia in mice.

(b). Enhancing the anoxia tolerance of mice under atmospheric pressure.

(c). Increasing coronary flow in rabbits in vitro.

(d). Relaxing adrenaline-induced vasoconstriction in rabbits in vitro.

(e). Lowering the level of blood lipid. During observation on 22 patients receiving treatment with herbal remedy, 4 cases were judged as significant improvement (visual acuity values incremented to over 1.0, and visual angle magnified by over 50 degree), 17 cases other moderate improvement (visual acuity values incremented by over 2 rows, night vision improved, and visual angle magnified). The treatment duration varied from 1 to 4 months. Physicians judged the clinical efficacy to be good or excellent in 95.5%.


[4] Online 'Mendelian Inheritance in Man'(OMIM) RETINITIS PIGMENTOSA; RP -268000


[9] Rockland Corporation, 12320 E. Skelly Drive, Tulsa, OK 74128


[16] Pasantes-Morales H, Quiroz H, Quesada O., Treatment with taurine, diltiazem, and vitamin E retards the progressive visual field reduction in retinitis pigmentosa: a 3-year follow-up study. Metab Brain Dis.. 2002 Sep;17(3):183-97


Changes in Microcurrent Stimulation for the treatment of eye disease.
I would like to share with you the changes that have taken place with microcurrent and the microcurrent equipment since 1998. Many patients become interested in microcurrent after reading my book Microcurrent Stimulation: Miracle Eye Cure and they expect to experience the same type of equipment and treatment that was outlined in the book. The equipment and treatment has evolved since this book was published and I hope this report will give you a much better understanding. I became interested in using microcurrent in 1999 after I read the report of Sam Snead having a marked improvement of his vision.

This was reported by Tim Franklin Publisher of the Hot Springs Star on March 17, 1998. Sam was quoted as saying, “I am going to get a drivers license next year”.

The mechanism of microcurrent is felt to be the following; 1) Increases the circulation to the eye, 2) Stimulates the function of the retinal cells and 3) Reduces scar tissue. The effects of 10 to 500 microamps on the cellular level have been documented by Dr. Cheng to increase ATP production by 500%, increase protein synthesis by 70% and increase cell transport by 40%. The history of microcurrent can be divided into 3 distinct periods.

The Microstim 100 with a single channel probe, the OMCS unit with two channel glasses and the Inspirstar 2 channel biospecific digital frequency machine. Microstim 100 – single channel utilizing a probe I began using the Microstim 100 in 1998. This machine utilized a probe to treat 8 acupuncture points around the eye. 4 points were located above and 4 below each eye. Each point was treated for 12 sec. utilizing 4 different frequency settings (292 HZ, 30 Hz, 9.1 Hz and 0.3 Hz).

The data from this instrument was published in my book Microcurrent Stimulation Miracle Eye Cure 2001 and the Townscend Letter (a peer review journal) in October 2002. This machine only had one channel and only 4 frequencies. It was postulated that the 2 higher frequencies (292 HZ and 30 HZ) reduced inflammation and the two lower frequencies (9.1 HZ and 0.3HZ) improved cellular function. This machine delivered generic frequencies that were not specific to the eye but could be used anywhere in the body.
Treating the eye with the Microstim 100 and Probe

There were several disadvantages to this machine. First was the cumbersome nature of treating 8 points with a probe around the eye. Many patients with macular degeneration had difficulty locating these points and treatment was also difficult because a steady hand and good vision was needed to keep the probe fixed on these points. The machine also had a current adjust knob. Patients were instructed to turn up the current until they felt a tingling then reduce the current until nothing was felt. Many patients treated their eyes with a much too high current and because of this the results were very unpredictable. The microstim 100 was later modified into a delivery system with eyeglasses but this this system still had the limitation of the current adjust and unpredictable delivery of current.

Treating Sam Snead

I had the pleasure of treating Sam Snead the late professional golfer with the Microstim 100 in Feb 2002. In exchange for 4 days of treatment Mr Snead gave me a few golf lessons. What else did he tell me about my swing? “Dr. Kondrot I would suggest cutting back over the next year and then QUIT!!” OMCS unit with two channel glasses.
My research led to the investigation of a 2 channel machine. 2 separate channels with a frequency off set by 0.7 HZ would deliver a more broad spectrum effect and have a greater therapeutic effect. This machine had the advantage of a fixed current at 100 microamps regardless of the resistance present as the current passed into the body. This machine was developed to have the following frequencies with a 0.7 HZ off set.

Off Set Frequencies in the OMCS

292/ 292.7 HZ
30/30.7 HZ
9.1/ 9.8 HZ

0.3/ 1.0 HZ The glasses were discontinued because of manufacturing problems and problems with delivering a consistent current into the eye. This machine had a distinct advantage of the safety of the fixed current and broader action of the dual channel delivery system. Two channel biospecific digital frequency machine
Through the brilliant work of Ning Wu and his team of electrical engineers we have developed the first home programmable microcurrent machine. This machine has 5 specific programs which can be customized for each patient. Programs are designed not only to treat the eye but also to detoxify the body, reduce stress and to treat other physical problems.

Current is delivered by using sliver meshed gloves that are wrapped in a damp washcloth. This delivery system has demonstrated to deliver a consistent microcurrent energy into the eye. The gloves and washcloths also add the versatility to be used in detox and body programs. A new technique called Frequency Specific Microcurrent (FSM) has produced a dramatic improvement in treatment outcomes of macular degeneration. Instead of using basic generic frequencies which have a low level affect on the diseased eye tissue we now can use frequencies specific to the retinal tissue and the pathology. The energy is driven into the area needed for tissue repair.

The roots of Frequency Specific Microcurrent (FSM) date back to the early 1900’s from Dr. Albert Abrams, who was the first physician to use calibrated instruments capable of detecting the radiations of living tissue. Dr. Abrams concluded that all matter radiates electromagnetic energy and the characteristics of the radiation depends upon the unique molecular structure. Modern FSM utilizes hundreds of frequencies within the range of .01 to 999 Hz with varying intensities of 20 to 600 microamps. Each tissue in the body has an individualized frequencies for example the retina has a frequency of 95 Hz and macula 137 Hz. Each type of pathology also has a frequency. Hemorrhage has a frequency of 18 and edema is 14. FSM
is “frequency specific” because the frequencies of the tissue and that of the pathology are “matched” with two frequencies. For example hemorrhage in the macula the FSM treatment would use 18 Hz and 137 Hz. This coupled frequencies then matches the exact abnormalities that are present in the damaged tissue. The desired effect is to neutralize those frequencies that are in disharmony.

ScyFIX became the only company in the world to initiate FDA guidelines compliant Pilot trials using Neuromodulation for ophthalmic blindness-causing diseases in 2007. The company develops and intends to make available to patients medical devices based on the use of its proprietary MCN technology to address a range of degenerative ophthalmic conditions. MCN is a therapeutic form of neurostimulation (often referred to as neuromodulation) that involves the trans-corneal application of small, precise dosages of electrical current to the retina, via electrodes placed over the patient’s closed eyelids. ScyFIX owns the intellectual property related to the delivery of electrical current to the eye. The initial target markets are Retinitis Pigmentosa (RETINITIS PIGMENTOSA) and the dry form of Age-Related Macular Degeneration (ARMD). ScyFIX believes that MCN could be effective in treating several other degenerative conditions such as Presbyopia, Open-Angle Glaucoma, Diabetic Retinopathy and Stargardt's Disease, among others.

Retinitis Pigmentosa affects approximately 75,000 people in the U.S. and 1.75 million worldwide. Retinitis Pigmentosa is a genetic eye condition causing a loss of function in retinal photoreceptor cells outside the macula. There is currently no medical treatment that can either slow the progression of disease or cure Retinitis Pigmentosa. The primary treatment to slow progression of the disease includes daily ingestion of up to 15,000 IU of Vitamin A palmitate. Other options currently being evaluated are very costly, investigative, highly invasive and may involve retinal transplants, prosthesis, and/or gene therapies. Recognizing the genetic cause and rarity of Retinitis Pigmentosa in the US, the FDA has granted the Humanitarian Use Device designation to ScyFIX for the treatment of Retinitis Pigmentosa.

Approximately 50 million people are affected by Age Related Macular Degeneration (ARMD) worldwide, making it a leading cause of blindness. The prevalence and severity of Age Related Macular Degeneration increases with age, accounting for 55% of blindness cases in adults over age 40 in the U.S. and the prevalence of Age Related Macular Degeneration is expected to grow significantly with the aging baby-boomer population. The current treatment for this condition is the long-term use of pharmaceuticals, which are costly and often involve regular physician visits in which the patient receives an injection into the eye. The alternative is complex, invasive surgery, typically reserved for late stage conditions as a treatment of last resort.

The FDA has approved treatments for the Wet form of Age Related Macular Degeneration only, which accounts for approximately 10% of all Age Related Macular Degeneration cases. There is no approved treatment for the much larger Dry Age Related Macular Degeneration population which accounts for 90% of all Age Related Macular Degeneration cases. Consequently, most of the Age Related Macular Degeneration population is not treated as their degenerative condition progresses. The ScyFIX MCN device offers a non-invasive, patient-administered treatment option that eliminates the extended patient recovery of the alternative, invasive techniques and frequent physician visits for the limited pharmaceutical therapies available (for the Wet form of Age Related Macular Degeneration). The device consists of a portable controller, disposable patches and lead wires. The ScyFIX 700 MCN device is CE Mark approved and is the commercialized product version of the ScyFIX 650 MCN device which was used in the ScyFIX’s FDA trials.
ScyFIX recently completed two FDA guidelines compliant Pilot studies with 24 months follow up to evaluate safety and potential efficacy in patients with Dry Age Related Macular Degeneration and Retinitis Pigmentosa. No serious device related adverse events have been reported to date. Additionally, data through twenty-four months suggests that MCN therapy is clinically effective. Observations from the Pilot Trial data, suggest that MCN therapy not only slowed down the progression of disease symptoms as intended in a majority of patients in the study, but has in many cases halted, and in some cases reversed the progression of the disease. Early data from these Pilot trials was presented at three major International ophthalmic conferences: the 2008 ISCEV Symposium (International Society of Clinicians in the Electrophysiology of Vision), American Society of Retina Specialists, and the American Academy of Ophthalmology (AAO) conferences.

In the US, ScyFIX intends to make the ScyFIX 700 MCN system available under an FDA HDE approval, when received, to patients suffering from Retinitis Pigmentosa. The Company received its Humanitarian Use Device (HUD) distinction in March 2008 from the FDA, and will be submitting the 24 months data and HDE application to the FDA for approval. In addition to its ISO 13485 medical quality system certification, ScyFIX has met the Canadian regulatory requirements, and received the CE MARK for European approval for the new ScyFIX 700 device during May 2009 which allows for making the device commercially available to patients with Retinitis Pigmentosa and Dry Age Related Macular Degeneration, in international (outside the USA) markets where approval is achieved. Additional Pivotal Clinical studies are anticipated for Dry Age Related Macular Degeneration before the ScyFIX MCN therapy will be available in the US. ScyFIX is working diligently on the requirements of the regulatory pathway to facilitate FDA approval in the US.

For a short introduction to MicroCurrent Neuromodulation (MCN), read this: **Mechanisms of Action**

For information on previously published studies which indirectly support the results with ScyFIX technology, please follow the links below.

1. Treatment of Macular Degeneration Utilizing Micro-Current Stimulation

2. Treatment of Retinitis Pigmentosa Utilizing Micro-Current Stimulation

3. Nutritional Supplementation, Electrical Stimulation And Age Related Macular Degeneration

4. The Treatment of Retinal Diseases With Micro Current Stimulation And Nutritional Supplementation

5. Bioelectrical Stimulation In An Integrated Treatment for Macular Degeneration, Retinitis Pigmentosa, Glaucoma, CMV -Retinitis, & Diabetic Retinopathy

6. The Effects of Electric Currents on ATP Generation, Protein Synthesis, and Membrane Transport

7. Macular Degeneration Treatment with Nutrients and Micro Current Electricity

**Treatment for Macular Degeneration Utilizing Micro-Current Stimulation**


**Abstract**

Forty-three patients were treated, twenty two with both eyes, with direct Micro-current of 200 Micro-amps
Treatment for Retinitis Pigmentosa Utilizing Micro-Current Stimulation


Abstract

A small sample of patients diagnosed with Retinitis Pigmentosa were treated with direct current of 200 Micro-amps. Visual acuity ranged from 20/40 to 20/400. Of these patients, 66% had significant improvement in visual acuity. All of the patients demonstrated improved visual fields as measured by Humphrey Field testing. The results indicated improved RPE metabolism, ocular blood flow, and/or the release of trophic factors that may reduce photoreceptor degeneration.

Nutritional Supplementation, Electrical Stimulation And Age Related Macular Degeneration

Leland D. Michael, O.D. and Merrill J. Allen, O.D., Ph.D.

Abstract

This is a preliminary report of an ongoing study of the rate of development of age related macular degeneration in people using nutritional supplements and treated with weak electrical currents. Twenty-five subjects, ages 48 to 79 years, have been treated for periods from two to seven years. Fifteen subjects have improved their acuity. Ten have lost acuity. Two have had laser treatment, two had to stop because of age and transportation problems and two have died. The subjects have lost an average of 0.30 letters of acuity over an average of 4.0 years. By comparison, Newsome's test group lost 4.1 letters and his placebo group lost 7.1 letters of acuity in 2 years using nutritional supplements without electrical treatment. It appears that electrical stimulation of the eyelids can enhance the success rate of nutrients alone in controlling AMD.

Keywords

Age–related macular degeneration, nutrition, zinc, electrical stimulation, micro ampere

Introduction

Age-related macular degeneration (AMD) and cataract are the major causes of visual impairment and blindness in the United States in persons over 55 years of age.1 AMD damages the retinal tissues in the macular area causing fine pigmented stippling, pigment epithelium changes and the development of drusen. Drusen usually occur in a mirror pattern in both eyes. AMD is called "dry" or "wet". The dry type is characterized with either normal acuity or only a moderate acuity loss, while the wet type may progress to
rapid and severe vision loss. The average rate of acuity reduction has not been clearly defined in the literature. At the present time there is no recognized therapy for the dry type of AMD. A study by Newsome et al. showed that a slower progression of AMD occurred over a two year period, for subjects receiving vitamin and mineral supplements high in zinc compared to unsupplemented (control) subjects. The treatment for the "wet" type of AMD involves using a laser to seal off the leakage in the choriocapillaris layer up through Bruch's membrane. There is the real possibility of further vision loss due to leakage in the area around where the laser burn was applied. This stage of AMD is a depressing time for the patient. In 1983, one of us (LDM) upon recovering from retinal detachment, decided to try to help patients with early signs of macular degeneration. He began a study of the effects of nutritional supplements and microampere electrical treatments on the rate of progress of the "dry" type of AMD. Electrical treatment was incorporated because the eye has an electrical potential that may play a role in retinal health and that can be modified with an external source of electricity. (The first patient, age 74 has been in the study for seven years and has had no further acuity decline.)

Procedure

Each patient who was diagnosed as having AMD was referred to a local ophthalmologist who confirmed the diagnosis. The patient was then offered three alternatives; a) evaluation every 6 months; b) use a nutritional supplement and be evaluated every 6 months; or c) use a nutritional supplement and receive microampere electrical treatments at least once every month. Most AMD patients chose alternative c) and became part of this study. The control for this study is the data presented by Newsome because this study differs from Newsome's study only by the addition of electrical treatment to the area around the eyes.

Figure 1. The formula for the nutritional supplement that we provided to all subjects.

<table>
<thead>
<tr>
<th>Eight Tablets Contain</th>
<th>%RDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A (Fish Liver Oil)</td>
<td>500</td>
</tr>
<tr>
<td>D (Fish Liver Oil)</td>
<td>200</td>
</tr>
<tr>
<td>Vitamin E (d-alpha Tocopherol Acetate)</td>
<td>1336</td>
</tr>
<tr>
<td>Vitamin C (Ascorbic Acid)</td>
<td>333</td>
</tr>
<tr>
<td>Vitamin B1 (Thiamin HCL)</td>
<td>6567</td>
</tr>
<tr>
<td>Vitamin B2 (Riboflavin)</td>
<td>8838</td>
</tr>
<tr>
<td>Niacin</td>
<td>400</td>
</tr>
<tr>
<td>Vitamin B6 (Pyridoxine)</td>
<td>7500</td>
</tr>
<tr>
<td>Pantothenic Acid (dCalcium Pantothenate)</td>
<td>550</td>
</tr>
<tr>
<td>Inositol</td>
<td></td>
</tr>
<tr>
<td>Calcium (Phosphate)</td>
<td>50</td>
</tr>
<tr>
<td>Magnesium (Oxide)</td>
<td>62</td>
</tr>
<tr>
<td>Selenium</td>
<td></td>
</tr>
<tr>
<td>Zinc (Gluconate)</td>
<td>495</td>
</tr>
<tr>
<td>Bioflavinoids</td>
<td></td>
</tr>
<tr>
<td>Supplement</td>
<td>Dosage</td>
</tr>
<tr>
<td>--------------------</td>
<td>--------</td>
</tr>
<tr>
<td>Glycine</td>
<td>200 mg</td>
</tr>
<tr>
<td>L-Glutamine</td>
<td>200 mg</td>
</tr>
<tr>
<td>L-Arginine</td>
<td>300 mg</td>
</tr>
<tr>
<td>L-Cysteine HCl</td>
<td>400 mg</td>
</tr>
<tr>
<td>Glutathione</td>
<td>40 mg</td>
</tr>
</tbody>
</table>

* RDA has not been established. + Vision-Eze Professional Products. 925 Hale Place, Suite A4. Chula Vista, CA 91914

Suggested Use: four tablets two times daily

Figure 2: Each point on the eyelids was stimulated for about one minute with a square wave alternating electric current of 206 micro amperes at 10 cycles per second. The eyelid contact was via a wet cotton electrode and the ground was the subject’s hands.

Figure 3: Summary of the data for 25 subjects treated with nutritionsupplements and weak electrical stimulation during the period from July ’85 to July ’92. Subject 11 was dropped from the study when she received a cardiac defibrillator because electrical stimulation might have adversely affected it. Subject 14 suffered severe vision losses after lasering because she had begun to show signs of leakage. One line of acuity is 5 letters.
Supplemental vitamins and minerals (see Figure 1) were assigned to be taken twice per day with meals. During the first 10 weeks the patient was seen 20 times for fundus and acuity examination and for electrical treatments. After this initial period the subject was examined and treated once a month. The electrical treatments of 200 micro amperes were applied to pints on the closed eyelids as shown in Figure 2.

For a total of about 7 minutes for each eye using the ElectroAcuscope.

Results

Figure 3 shows the initial age of the subjects who have been in the study from two to seven years. It also shows the duration of involvement, the entering acuity, the acuity at the end and the number of letters change in acuity during the study. For those who have received laser treatment, the final acuity was taken just before lasering. Fifteen subjects improved acuity from 1 to 17 letters and 10 lost 1 to 66 letters. On average a slight reduction of vision occurred during this study amounting to 0.3 letters where 5 equals one line of test letters.

Discussion

To evaluate the results, Newsome’s data were used for comparison. The visual acuity test letter sequence that Newsom used was as follows:

<table>
<thead>
<tr>
<th>#</th>
<th>Age</th>
<th>Sex</th>
<th>Date Start</th>
<th>Date End</th>
<th>Beginning Acuity</th>
<th>End Acuity</th>
<th>Number of Letters change</th>
<th>REASON FOR LEAVING STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>74</td>
<td>F</td>
<td>7-18-85</td>
<td>7-92</td>
<td>30</td>
<td>20</td>
<td>30+2 15.2</td>
<td>+2 +3</td>
</tr>
<tr>
<td>2</td>
<td>71</td>
<td>F</td>
<td>7-21-85</td>
<td>7-92</td>
<td>30</td>
<td>25</td>
<td>30+2 20.2</td>
<td>+3 +7</td>
</tr>
<tr>
<td>3</td>
<td>74</td>
<td>M</td>
<td>9-10-85</td>
<td>11-91</td>
<td>100</td>
<td>30-2</td>
<td>100+1 50+2</td>
<td>+1 +4</td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>M</td>
<td>12-2-85</td>
<td>7-92</td>
<td>20</td>
<td>30</td>
<td>15+2 20+3</td>
<td>+5 +8</td>
</tr>
<tr>
<td>5</td>
<td>77</td>
<td>M</td>
<td>5-28-86</td>
<td>7-92</td>
<td>20</td>
<td>15</td>
<td>20.2 20.2</td>
<td>+2 +7</td>
</tr>
<tr>
<td>6</td>
<td>69</td>
<td>F</td>
<td>7-29-86</td>
<td>10-91</td>
<td>20</td>
<td>30</td>
<td>20+3 20+3</td>
<td>+3 +5</td>
</tr>
<tr>
<td>7</td>
<td>76</td>
<td>F</td>
<td>10-14-86</td>
<td>9-91</td>
<td>20</td>
<td>25+1</td>
<td>20-1 400</td>
<td>-1 -66</td>
</tr>
<tr>
<td>8</td>
<td>48</td>
<td>M</td>
<td>1-19-87</td>
<td>7-92</td>
<td>40-3</td>
<td>20.2</td>
<td>30+1 20.1</td>
<td>+6 +1</td>
</tr>
<tr>
<td>9</td>
<td>79</td>
<td>F</td>
<td>5-24-87</td>
<td>7-92</td>
<td>20-3</td>
<td>25.2</td>
<td>20 30+3</td>
<td>+1 +5</td>
</tr>
<tr>
<td>10</td>
<td>65</td>
<td>F</td>
<td>8-17-87</td>
<td>7-92</td>
<td>30-2</td>
<td>15-3</td>
<td>40-2 15.2</td>
<td>+5 +1</td>
</tr>
<tr>
<td>11</td>
<td>67</td>
<td>F</td>
<td>2-25-88</td>
<td>4-90</td>
<td>25</td>
<td>25</td>
<td>15+5 15+1</td>
<td>+10 +10</td>
</tr>
<tr>
<td>12</td>
<td>79</td>
<td>F</td>
<td>6-29-88</td>
<td>7-92</td>
<td>25</td>
<td>40+3</td>
<td>20-3 25.1</td>
<td>+2 +6</td>
</tr>
<tr>
<td>13</td>
<td>74</td>
<td>F</td>
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<td>6-89</td>
<td>40-2</td>
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<td>-7 0</td>
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<tr>
<td>14</td>
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<td>8-90</td>
<td>20-1</td>
<td>60+1</td>
<td>25.1 60+1</td>
<td>-5 0</td>
</tr>
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<td>4-92</td>
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<td>400 70</td>
<td>-2 -2</td>
</tr>
<tr>
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<td>F</td>
<td>5-30-88</td>
<td>7-92</td>
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<td>30-1 40-1</td>
<td>-8 0</td>
</tr>
<tr>
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<td>73</td>
<td>F</td>
<td>11-15-88</td>
<td>4-92</td>
<td>20</td>
<td>25</td>
<td>25 50</td>
<td>-5 +15</td>
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<tr>
<td>18</td>
<td>76</td>
<td>F</td>
<td>1-20-90</td>
<td>6-92</td>
<td>25</td>
<td>30</td>
<td>25-2 25-2</td>
<td>-2 +3</td>
</tr>
<tr>
<td>19</td>
<td>79</td>
<td>F</td>
<td>1-02-90</td>
<td>7-92</td>
<td>30</td>
<td>30</td>
<td>20-1 20-3</td>
<td>+9 +7</td>
</tr>
<tr>
<td>20</td>
<td>65</td>
<td>M</td>
<td>1-09-90</td>
<td>6-91</td>
<td>25+2</td>
<td>100+1</td>
<td>100 100</td>
<td>-28 -1</td>
</tr>
<tr>
<td>21</td>
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<td>F</td>
<td>1-29-90</td>
<td>7-92</td>
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<td>30-1</td>
<td>20-3 25</td>
<td>+8 +6</td>
</tr>
<tr>
<td>22</td>
<td>70</td>
<td>M</td>
<td>3-35-90</td>
<td>7-92</td>
<td>400</td>
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<td>400 40-3</td>
<td>0 -6</td>
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<tr>
<td>23</td>
<td>66</td>
<td>F</td>
<td>4-09-90</td>
<td>7-92</td>
<td>50-2</td>
<td>25</td>
<td>25 20</td>
<td>+17 +5</td>
</tr>
<tr>
<td>24</td>
<td>79</td>
<td>F</td>
<td>5-17-90</td>
<td>7-92</td>
<td>30-1</td>
<td>30-3</td>
<td>25-1 25-2</td>
<td>+5 +6</td>
</tr>
<tr>
<td>25</td>
<td>66</td>
<td>F</td>
<td>8-12-90</td>
<td>7-92</td>
<td>70</td>
<td>20+4</td>
<td>50+2 10+3</td>
<td>+8 -1</td>
</tr>
</tbody>
</table>

![Table](https://example.com/table.png)

SUM: +34.0 - 50.0
AVG: +1.4 - 2.0
AVG Both Eyes: +0.30
There were five letters in each line. Three lines totaling 15 letters doublesthe visual angle of the test letters.

Newsome found that for his 71 untreated control subjects, average age 68 years (42 to 88). 18.3% lost 10 to 14 letters of acuity, 8.45% lost 15 to 18 letters and 7% lost 20 or more letters over a period of 24 months using a standardized visual acuity test.4 Newsome's zine-treated experimental group contained 80 subjects of average age 67.6 (46 to 89) years, of whom 6.25% lost 10 to 14 letters, 5% lost 15 to 19 letters and 2.5% lost 20 or more letters.

Newsome has shown a positive effect of selected nutrients on AMD. The average rate of acuity reduction that Newsome found was 7.1 letters for the control population and 4.1 letters for the test population over a two year period. Our study can be compared to Newsome's because our study uses similar visual acuity criteria, the same diagnostic procedures, and includes pharmacological dosages of supplemental zinc.

The possibility of a Hawthorne effect causing an improvement in visual acuity was considered. In situations where the rewards are high as in a driver license test, the acuity test must be randomized to obtain accurate acuity measures. However, when the subject is concerned about permanent vision loss, we believe that learning the charts will have minimal effect on acuity measures.

According to Lane, "the dry type of AMD is different from the wet type and is related to inadequate amounts of zinc. If the wet type AMD subjects had been omitted from this study the results would have shown a gain rather than a loss. We did not do this because Newsome did not do it.

There have been no reported adverse effects either from the nutritional supplements or the electrical stimulation. Subjects are protected by examinations once a month. In the event of fundus leakage, these monthly examinations assure early detection and immediate referral to the retinal specialist and laser expert. These professionals and the subject then decide whether to proceed with laser therapy.

Conclusion

This is a preliminary report of an ongoing study, and so far the results are very encouraging. On average, the subjects have shown only a slight reduction in visual acuity. By comparing this study to Newsome's study, it appears that adding electrical stimulation has improved the ability of nutritional supplements to slow the progression of AMD. To permit a proper statistical evaluation more subjects are needed. Thus the experiment is continuing for another four to five years.

References

The Treatment of Retinal Diseases with Micro Current Stimulation and Nutritional Supplementation

Edward L. Paul, Jr., O.D., Ph.D., Visiting Professor of Ophthalmology, Chairman, Department of Continuing Medical Education, St. Luke’s University School of Medicine

Abstract

From May 2001 to November 2002, 94 eyes diagnosed with typically untreatable retinal diseases including age-related macular degeneration, retinitis pigmentosa and Stargardt’s were treated with an integrated treatment protocol employing micro current electrical stimulation and nutritional supplementation. Overall, 68 % showed a marked increase in vision function and visual acuity following therapy. The success rate in age-related macular degeneration was 72 % (26 out of 36 eyes), in retinitis pigmentosa 53 % (18 out of 34 eyes), and in Stargardt’s 83 % (20 out of 24 eyes). The average level of improvement was 2-3 lines as measured using the Snellen eye chart.

Keywords

Micro Current Stimulation, Macular Degeneration, Retinitis Pigmentosa, Stargardt’s, AREDS, Nutrition, Lutein, DHA, Taurine, Micro Amp, ATP, RPE

Discussion

Micro Current Stimulation (MCS) therapy is a noninvasive procedure which involves stimulating the retina and nerve fibers with very low intensity electrical current using a FDA and CE approved electrical stimulation device. The current is delivered in the micro amp range at different electrical frequencies through electrodes applied over closed eyelids. The treatment causes no discomfort or pain and is administered for 12 minutes twice each day. While a very effective form of treatment, MCS therapy is not a cure for retinal diseases and must be continued for the life of the patient. Overall, no side effects or adverse reactions related to this procedure have been observed.

It is theorized that MCS Therapy works by increasing intracellular ATP (adenosine triphosphate) concentrations, enhancing protein synthesis, and stimulating the cells ability to absorb nutrients. Through these mechanisms, MCS therapy improves RPE (retinal pigment epithelium) efficiency and thereby may restore and/or improve retinal function.

ATP is synthesized in the mitochondria process known as the Kreb’s Cycle, the sequence of reactions in the mitochondria that complete the oxidation of glucose in respiration. Kroll and Guerrieri have shown that there are age related changes in mitochondrial metabolism resulting in a decrease of the ATP synthase activity in the retina with age. Guerrieri has gone further to show functional and structural differences of the mitochondria F0F1 ATP synthase complex in aging rats. It is theorized that many retinal diseases, at least in part, are due to a decrease in mitochondria function and the subsequent decrease in intracellular ATP. This decrease in mitochondria function results from free radical damage and the mutation of mtDNA (mitochondria DNA). It is interesting to note the genetic link between ATP and retinal disease. ATP Synthase (ATPase) is an enzyme which catalyzes the synthesis of ATP. A genetic defect in the ATPase 6 Gene has now been implicated in retinitis pigmentosa.

In October 2001 the National Eye Institute, a division of the National Institutes of Health, published the Age Related Eye Disease Study which stated unequivocally that nutritional supplementation is an effective therapy against macular degeneration. This study was based on a seven year double blind study.
conducted by the NIH at five medical centers across the United States. It is clear that proper nutritional support can help protect us from diminishing eyesight and degenerative ocular complications as we grow older.

In evaluating MCS therapy in the treatment of retinal disease, clinical testing has shown that nutritional supplementation serves as a synergistic catalyst in boosting the effectiveness of MCS therapy. Subsequently, nutritional supplementation is critical part of the MCS therapy program. The formula used on the test subjects was identical to that used in the Age Related Eye Disease Study with the addition of Lutein, Taurine, and DHA (DocosaHexanoic Acid).

In respect to the legal status of MCS therapy, the Food and Drug Administration does not regulate the practice of medicine, however they do regulate the sale of medical devices. Before a medical device can be legally sold or used in the U.S., the person or company that wants to sell or use the device must seek approval from the FDA. To gain approval, they must present evidence that the device is reasonably safe and effective for a particular use, or the “indication.” The devices used in MCS therapy are approved, however they were originally developed and approved for the symptomatic relief of chronic intractable pain and as an adjunctive treatment in the management of post surgical traumatic pain problems. Once the FDA has approved a medical device, a doctor may decide to use that device for other indications if the doctor feels it is in the best interest of a patient. Subsequently, the use of an approved device for anything other than its FDA approved indication is called off-label. MCS therapy is considered an off-label use.

The author was not the first research scientist to report on the effects of MCS therapy. At least twenty other studies have been published regarding electrical current’s effectiveness in dealing with degenerative disease, tissue repair, and cell regeneration. Four other studies have been published specifically addressing MCS therapy's effect on retinal disease.

The American Academy of Ophthalmology issued a position statement regarding micro current stimulation which states “ … the overall rate of adverse effects from electrical stimulation appears to be low. In the study of AMD and micro current stimulation, there were no reported adverse side effects from the electrical stimulation … long-term studies with larger samples of patients, and adequate control groups compared to micro current stimulation are critical to establishing a base of evidence regarding effectiveness.”

References

Bioelectrical Stimulation In An Integrated Treatment for Macular Degeneration, Retinitis Pigmentosa, Glaucoma, CMV-Retinitis, & Diabetic Retinopathy

Presented: Fourth Annual Symposium on Biologically Closed Electrical Circuits, October 27, 1997, Sponsored by Mankato University, Minnesota by Grace Halloran, Ph.D. & August L. Reader, M.D., F.A.C.S. 655 Lewelling Blvd., San Leandro, CA 94579 510 357-0477

Abstract
From December 1995 to September 1997, thirty individuals diagnosed with typically untreatable eye diseases including retinitis pigmentosa, macular degeneration, CMV-retinitis, Stargaardt's and others attended an integrated treatment protocol employing bioelectrical stimulation, nutritional and herbal supplementation (including Ginkgo Biloba, Lutein, DHA) and other health care modalities. The study was monitored by a neuroophthalmologist, evaluating standard clinical visual function examinations, including objective field of vision tests obtained by the Humphrey FOV analyzer, visual acuity and color discrimination. Four controls were evaluated, with the monitors masked, and although the sample was small, the results were significant in their lack of change. Follow-up examinations of the graduates were provided, establishing efficacy of the rehabilitative progress made originally, including a review of two graduates who participated in a five-day course of treatment at the six month-post treatment period. Therapy protocol consisted mainly of bioelectrical stimulation with the Electro-Acuscope 80. Overall results showed remarkable increase in visual function in visual acuity in most, and clearly established the safety of the integrated treatment protocol. Long-term follow-up indicate maintenance and continued improvement when compliance of home program is continued. Participants of the five-day refresher demonstrated a marked increase in visual function, in visual acuity and field of vision.

Keywords
Bioelectrical, CMV-Retinitis, DHA, Electro-Acuscope, Ginkgo Biloba, Lutein, macular degeneration, nutrition, retinitis pigmentosa, Stargaardt's.

Discussion
For the past twenty-five years, both of us have dealt with significant visual impairment. Halloran as a practitioner and patient and Reader as a medical specialist Most of the diseases that we are dealing with
have been designated as chronic. Progressive, untreatable and incurable. The majority of these patients are left on their own with no resources available to try to improve their situation. The numbers are staggering and increasing as our population ages. The National Institutes of Health estimates that there are nearly eighteen million Americans suffering from serious visual impairments, with nearly half being diagnosed with macular degeneration.

Halloran was diagnosed with a genetic eye disorder, retinitis pigmentosa, and Reader as a neuroophthalmologist, have individually and collectively been searching for methods and therapies that may be of some benefit. We feel that we have been fortunate to rediscover some ancient and natural methods that definitely impact positively on visual function. Also, we have integrated the most technically advanced bioelectrical stimulation devices available to promote cellular healing. We believe that this marriage of western medical technology and eastern traditional healing practices provides the most effective treatment modality for those diagnosed with degenerative and progressive eye disorders.

From December 1995 to September 1997, thirty sight impaired individuals participated in a two-week course of an integrated treatment protocol for visual rehabilitation. The course is based on the Integrated Visual Healing program, developed by Halloran in the 1980's). This report is an extension of a pilot study conducted from 1983-1985, documenting 114 participants with a similar treatment protocol and results as encompassed in this current two-year study. The 1983–85 study was monitored by independent optometrists. This study has more objective and medically monitored documentation. Although this study lacks the electrophysiological ERG’s, the intent of this two-year study was to demonstrate safety and the need for further investigation.

Material and Methods
The 1995-97 group had an age range of 13 to 83, with the following diagnoses: twenty cases of retinitis pigmentosa (RP), seven macular degeneration (AMD - age related macular degeneration) including two cases of Stargaardt's, a juvenile form of macular degeneration, one diabetic retinopathy, one glaucoma (GL), and one CMV -retinitis (related to the AIDS virus).

Pre- and post-treatment visual testing was monitored by August L. Reader. M.D. F .A.C.S. Visual examinations consisted of field of vision (utilizing the Humphrey Field Analyzer Test, 30-2 Central, a computerized objective test of peripheral vision). standardized testing of best-corrected visual acuity (reading and fine recognition sight). Ishihara Color Plate identification. slit lamp examination and intraocular pressure.

A two-week intensive therapeutic session provided approximately thirty hours of primary treatments. An average of thirty treatments of bioelectrical stimulation of the Acu- Eye and Acuscope protocol With the Electro-Acuscope 80 were performed using 2.5 Micro Hertz and 25-50 micro amps intensity. These therapies were performed initially by the therapist and later taught to the individual patients for their self-application. The patients were encouraged to use the unit a minimum of three times per day, and up to six times per day. The patients received other supportive therapies including eight sessions of applied kinesiology and neuro-lymphatic deep stimulation, eight treatments of deep tissue acupressure in the head/neck and shoulder region, twenty sessions of color-shape identification therapy (Tyro Instrument). Nutritional and herbal support was provided for one group of seven participants (September 1996), all others were instructed to incorporate the supplemental program for on-going long term use? Nutritional regime consisted of a broad based complete multiple vitamin and mineral supplement (Life Pat, IDN, Provo Utah), emphasizing specific nutrients known to impact the visual system which included: DHA 8, Omega 3 Fatty Acid. Lutein9, Ginkgo Biloba 1a, Pycnogenol11. and a combination of antioxidants such as carotenoids.

The integrated rehabilitative program included other disciplines such as stress management4, acupressure based on acupuncture points for improving eye health. And other exercises to keep circulation optimum for on-going overall health benefits.
Results
The following tables (fables 1 .4) illustrate the improvements noted in this two-year study. These tables depict the mean deviation on visual field testing from normal compared from the pre-treatment period to the post-treatment period. Also included are the visual acuities and the color vision testing performed before and after the treatment protocols. The mean improvement in visual field function for all patients was 3.16 decibels. The improvement in the RP patients was only 2.58 decibels, while Macular Degeneration improved 4.61 db. Average visual acuity improvements were 0.98 lines. Color vision improved on average of 1.71 out of 18 color plates per eye in patients with Macular Degeneration, but only 0.35 of 18 plates in the Retinitis Pigmentosa patients (Ishihara Color Plate test for color vision anomalies is not considered the most reliable method of color vision testing).

Key Code Explanation
ID-Code = Diagnosis: First & Last Initials-Age -Right Eye MD=Macular Degeneration RP=Retinitis Pigmentosa GL=Glaucoma CMV=Retinitis MD-A=Mean Deviation on Humphrey Field of Vision Analyzer Pre-treatment MD-B=Post treatment Test Normal Mean Deviation Range: -6 to +4 VA-A=Visual Acuity (Distance) Pre-treatment VA-B=Visual Acuity Post-treatment CT-A=Ishihara Color Test (Ishihara) Pre-treatment (18 color plates total) CT-B=Ishihara Color Test at Post treatment

Table 1 depicts the most significant objective evidence demonstrated during the two-year period in the field of vision test, by the Humphrey FOV Analyzer (30-2 Central). The measurements outlined reflect the Mean Deviation, an analysis produced by the computerized testing device. Mean deviation is a comparison of the individual testing to a 'normal' population by sex and age. Normal range of mean deviation measurement for healthy population is -6 to +4. Table 1 demonstrates the difference between a control (masked to the monitor) groups of RP to RP participants. The control group was tested with the participant group on both pre- and post-examination days, receiving the identical testing procedure in a two week period.

Control data for the most part was the same. Participants in the integrated treatment protocol showed significant improvement in post field of vision analysis with the Humphrey Fay device. Recovery of field of vision is not usually associated with Retinitis Pigmentosa or any of the other disorders involved in the study.
Table 2 - Macular Degeneration Results

<table>
<thead>
<tr>
<th>ID-CODE</th>
<th>MD-A</th>
<th>MD-B</th>
<th>VA-A</th>
<th>VA-B</th>
<th>CT-A</th>
<th>CT-B</th>
</tr>
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<tbody>
<tr>
<td>MD-AH-83-RE</td>
<td>-7.19</td>
<td>-5.19</td>
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<td>CF@1’</td>
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<td>20/60</td>
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<td>20/40+1</td>
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<td>15</td>
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<td>11</td>
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<td>-3.73</td>
<td>20/50-1</td>
<td>20/50+1</td>
<td>15.5</td>
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N/A = not able to perform test due to poor visual function

Table 3 - Glaucoma & CMV-Retinitis Results

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<td>CMB-GW-41-RE</td>
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<td>18</td>
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<td>20/20+</td>
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N/A = Unable to locate pre-testing data

Table 4 - Retinitis Pigmentosa Results
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<th>MD-B</th>
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N/A = Not available due to computer failure

**Conclusion**

This two-year study clearly shows that bioelectrical stimulation to acupuncture points around the eyes and face have definite positive affects on visual functioning. These techniques, in conjunction with other complementary therapies, have clearly demonstrated that chronic progressive visual loss from several
different sources can be reversed to some degree. More importantly, the improvements in activities of daily living and the quality of life of these patients has been dramatically impacted.

This small study in conjunction with the larger study performed in the mid 80's, emphasizes the need for more research into alternative methods. The information we have thus far obtained only corroborates our previous beliefs that these methods provide patients with some hope for cure.

**Special Acknowledgements**
We would like to thank the following individuals for their technical support in conducting these studies: John Jones - Electro-Medical; Kaloni Verdi and David B. Davis, MD. - Optima Eye Center; Dale Fast, O.D.; Eugene Lopata, Ph.D.; Martha Lopata.

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**The Effects of Electric Currents on ATP Generation, Protein Synthesis, and Membrane Transport**

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*University of Louvain, Belgium

*As Published in Clinical Orthopaedics and Related Research

**Abstract:** The effects of electro stimulation on the ATP concentrations in rat skin as well as protein synthesis and membrane transport were evaluated. It was found that electro stimulation of the skin using direct current from 10 to 1000 micro amps increased ATP concentrations in the tissue by up to 500% and stimulated amino acid incorporation into the proteins by up to 123 %. The effects of electro stimulation on ATP production can be explained by proton movements on the basis of the chemiosmotic theory of Mitchell, while transport functions are controlled by modifications in the electrical gradients across the membranes. It was noted that electro stimulation exceeding 1000 micro amps has the potential of reducing ATP levels. DNA metabolism was not affected by electrical stimulation.

**Macular Degeneration Treatment with Nutrients and Micro Current Electricity**

Merrill J. Allen, O.D., Ph.D, John B. Jarding, O.D.,Ralph Zehner, O.D.

**Introduction**

This is the second report of a study of age related macular degeneration (AMD). The first report covered from July 1985 to July 1992 and was published in the fall of 1993. The positive results to date in this and in the earlier report are the slowing or reversing of the progress of AMD for most subjects.

This is exciting because the dry type of AMD is considered to be untreatable and can progress to the wet...
type which rapidly destroys vision. ABC’s television show “20/20” (Dec. 6 ’96) explained that 13 million Americans now have AMD. By the time the baby boomers reach age 65, 25% of Americans, or 30 million people, will have AMD. Happily, we are becoming aware of nutritional and electrical factors that can retard or reverse macular degeneration.


Procedure

Each subject was independently confirmed as having dry macular degeneration. The nutritional supplements used by Zehner’s subjects were similar to those used in the earlier study. Jarding’s more recent nutrients are shown in Figure 1 (p. 212). The additional nutrients used are bilberry, rutin and taurine.

In addition to nutrients taken daily, all subjects received micro ampere electricity applied to each eye’s closed lids. Zehner’s subjects were treated once per week for six weeks, then once per month. Jarding’s subjects were treated several times per week.

Results

Figure 2 (p. 212) shows Zehner’s twelve subjects. Figure 3 (p. 213) shows Jarding’s 34 subjects. For Figures 2 and 3, start date is the date the subject received the first treatment; DOB means date of birth; Acuity means the denominator of the Snellen Fraction; R means Right Eye, L means Left Eye. Change means the number of letters lost (-), or gained (+) from the initial acuity to the final acuity. There were five letters in each line of acuity. To go from 20/30-2 to 20/20 is a gain of 12 letters. (20/30-2 to 20/30 = +2 letters. 20/30 to 20/25 = +5 letters. 20/25 to 20/20 = +5 letters.) Comments provide unusual events.

Discussion

The data are presented according to the starting date. Jarding’s subjects showed improvement while Zehner’s showed a small loss. The changes in nutrition and the increase in the number of electrical treatments explains the improved success of Jarding’s procedure compared to the earlier procedure used by Zehner.

At each office visit the patient’s acuity was checked. Because many subjects reported better-vision as they left the office, Jarding began checking acuity both at the start and at the end of the office visit. Visual acuity usually improved following the electrical stimulation of the eyes. This suggests that still more frequent treatments would be beneficial.

This study is divided into two parts: Figure 2 is data from Zehner; Figure 3 is data from Jarding. The Electro-Acuscope 80, which is no longer available, was used earlier by Michael and in this study by Zehner. Jarding used the Micro-Stim 4006 which has a different output wave form compared to the Electro-Acuscope 80. The Micro-Stim 400 may be superior to the older machine, but we can’t be sure because the the Micro-Stim 400 was used more frequently. The basic electrical stimulus parameters are: 200 micro-amperes at +9 volts, alternating, square wave current.
For Zehner’s subjects there was an average loss of 3 letters of visual acuity per eye over a 2 year period. For Jarding’s subjects there was an average gain of 8.5 letters of acuity per eye.

Newsome’s research tested the value of zinc in treating macular degeneration. He used the same nutrients in the test and control groups. He added zinc only to the test group. The result was a slowing of the loss of vision of the 80 subjects in the test group receiving zinc when compared to the 71 subjects in the control group who did not receive zinc. On average his control group lost 7.1 letters of acuity and his test group lost 4.1 letters of acuity in two years. His (and our) acuity test chart had 5 letters per line.

Conclusions

The results of this study strongly indicate that nutrition and electrical stimulation are able to delay or reverse the progress of macular degeneration.

The fact that acuity usually improved within minutes of electrical stimulation shows that micro current electricity applied to the eyelids is beneficial. The fact that a change in nutrients to include taurine, rutin and bilberry extract improved the success of treatment agrees with the recent literature on the importance of nutrition to the retina.

References

6. MicroStim Inc., 7881 NW 90th Ave., Tamarack, FL 33321

Figure 1. Nutrients used in treatment for Macular Degeneration

<table>
<thead>
<tr>
<th>Nutritional Supplement</th>
<th>Two Per Day</th>
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<td>Beta Carotene</td>
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<td>Natural Vitamin E</td>
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<td>Vitamin C</td>
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<td>Zinc</td>
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Selenium 100 mcg  
Taurine 200 mg  
N-Acetyl-Cysteine 200 mg  
L-Glutathione 10 mg  
Vitamino B-2 50 mg

Figure 2. Changes in Zehners 12 subjects using nutrients and micro-current electricity

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<th>L20/</th>
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Figure 2. Changes in Zehners 12 subjects using nutrients and micro-current electricity
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Is Acupuncture a Beneficial Treatment for Retinitis Pigmentosa?

By Dr. Steve Rose on November 5, 2013

We at the Foundation Fighting Blindness have been receiving questions about acupuncture for the treatment of retinitis pigmentosa (RP), namely because of feasibility research conducted by Ava Bittner, O.D., Ph.D., at Johns Hopkins University, in collaboration with Andy Rosenfarb, N.D., L.Ac, who specializes in acupuncture and ophthalmic Chinese medicine. Their project was funded by the National Institutes of Health (NIH).

Cutting to the chase, that study did not provide us with enough information to know if acupuncture can save or restore vision in people with RP. I will, in a moment, report on what we have learned thus far, as well as on the design of Dr. Bittner’s forthcoming acupuncture study, which will hopefully tell us more.

But I have one important comment before I discuss the research: I strongly recommend that people affected by RP and other retinal diseases hold off on trying acupuncture therapy for their retinal conditions until more is
known about its risks and benefits. While Dr. Rosenfarb has used acupuncture to treat people with RP in his clinical practice, his approach has not yet been formally studied in a randomized, controlled clinical trial.

Furthermore, Dr. Bittner informed us that acupuncture needles, if not properly administered by an acupuncturist trained in specific needling techniques for treating RP, can potentially cause nerve damage, infection and other problems. So, there are real risks if the therapy is administered by someone who does not have the appropriate skill and expertise, especially given that Dr. Rosenfarb’s protocol involves administration around (not in!) the eyes. By all means, do not try this at home.

With that important disclaimer out of the way, let me tell you what we know about acupuncture. First, it is being widely used and studied by the Western medical community, especially for the treatment of chronic pain and discomfort related to a variety of diseases and conditions. Also, I recently compiled a list of about 60 acupuncture studies that are currently funded by the NIH. So, acupuncture definitely has additional potential benefits, and the breadth of those is being aggressively explored.

Second, we have evidence from a 2006 lab study that electroacupuncture — in which a low-intensity electrical current is passed through needles — might be therapeutic for retinal degenerations. An Italian research team led by Dr. Lucia Pagani showed that electroacupuncture released neurotrophic (i.e., protective) proteins in the retinas of rats with retinal degeneration. While vision in the rats was not measured, treated rats had thicker and healthier retinas than those that were untreated.

Last, we have Dr. Bittner’s recently completed 12-person feasibility study of electroacupuncture for people with RP. Results of the research were published in the journal Clinical and Experimental Optometry. In the study, participants received 10 half-hour treatments over a two-week period from Johns Hopkins acupuncturist Jeff Gould, who was trained by Dr. Rosenfarb to administer a standardized protocol designed specifically for the RP trial.

Dr. Bittner reported that eight of those participants had significant vision improvements in night vision, dark adaptation and/or visual field. She followed three of those patients for approximately a year, and their night-vision improvements were sustained. She continues to follow those three individuals to see how their night vision changes over time.

Later this year, Dr. Bittner will launch a one-year, 21-person study of electroacupuncture funded by the National Eye Institute. In this research effort, she and her colleagues will compare a control group to two therapeutic approaches: electroacupuncture and transcorneal stimulation, which involves sending a small electrical current through a wire placed on the surface of the eye.

Transcorneal stimulation has had encouraging results (increases in visual field) in small-scale German clinical trials. Dr. Bittner and her colleagues will also look at additional parameters of retinal health and vision improvement, including retinal blood flow and retinal sensitivity as measured by an electroretinogram.

Even after the small-scale, one-year study is completed, there will still be much that we don’t know about acupuncture for retinal degenerations. If there is a benefit, we still won’t know which forms of RP will benefit most and/or if acupuncture will save vision in people with other retinal diseases. Additional research will be needed, but I think Dr. Bittner and her colleagues are on the right track to getting the answers. Good research takes time.

- See more at: http://www.blindness.org/blog/index.php/is-acupuncture-a-beneficial-treatment-for-retinitis-pigmentosa/#sthash.4AFL98H5.dpuf