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Age related macular degeneration (ARMD) is one of the leading causes of severe visual impairment among older Americans. Several hypotheses have been proposed regarding the pathogenesis of ARMD. Studies concerning the relationship between cigarette smoking and ARMD are identified through the use of articles published since 1970. Due to the recently reported correlation found between Age Related Macular Degeneration (ARMD) and smoking we are interested in determining if smoking affects the macula in a young population. The purpose of the study is to determine how quickly the maculae of the young smoking population recover after the Photostress Recovery Test compared to young nonsmokers. The Photostress Recovery Test is a quick, simple and direct procedure used to assess the initial sensitivity loss and the speed of the return of visual function to the macula after a timed exposure to a bright light stimulus. If a correlation between smoking and the health of the macula is found it could lead to increased awareness and prevention of macular defects in later stages of life.

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DOES SMOKING LENGTHEN THE
PHOTOSTRESS RECOVERY TEST IN
YOUNG ADULTS

By

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A thesis submitted to the faculty of the
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Biography

Sherry Salkhordeh is a fourth year optometry student at Pacific University College of Optometry and will be graduating with an O.D. in May, 1999. Born in Tehran, Iran in 1972, she received a varied educational experience through schools in Iran, India, and U.S. She attended the University of Utah from 1990 and graduated with a B.S. in Biology in 1995.

Her interests are Geriatrics and low vision and she plans to concentrate in these fields throughout her practice as an Optometry Doctor.
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Abstract

Age related macular degeneration (ARMD) is one of the leading causes of severe visual impairment among older Americans. Several hypotheses have been proposed regarding the pathogenesis of ARMD. Studies concerning the relationship between cigarette smoking and ARMD are identified through the use of articles published since 1970. Due to the recently reported correlation found between Age Related Macular Degeneration (ARMD) and smoking we are interested in determining if smoking affects the macula in a young population. The purpose of the study is to determine how quickly the maculae of the young smoking population recover after the Photostress Recovery Test compared to young nonsmokers. The Photostress Recovery Test is a quick, simple and direct procedure used to assess the initial sensitivity loss and the speed of the return of visual function to the macula after a timed exposure to a bright light stimulus. If a correlation between smoking and the health of the macula is found it could lead to increased awareness and prevention of macular defects in later stages of life.
Does Smoking Lengthen the Photostress Recovery Test in Young Adults?

Age related maculopathy (ARM) is a degenerative disorder of the central area of the retina, the macula. The term “Age related maculopathy” is used for a variety of abnormalities that are observed in the macular area including drusen accumulation, characterized by geographic atrophy and neovascular changes. The condition was previously known as age related macular degeneration (ARMD). It is often associated with visual impairment and is more frequent after 65 years of age. Age related maculopathy is a leading cause of blindness in most western countries including the United States and Australia. Activities essential for independent living, including reading, driving and writing are most impaired by the loss of central vision due to this disease that affects the macula, the small central part of the retina. The prevalence of AMD and the associated social and economic consequences of blindness from AMD are increasing as the number of older people in our population continue to increase. The causes of ARM are unknown, but many risk factors are hypothesized.

There are two main forms of ARMD. The first, neovascular AMD, also called “wet or exudative AMD”, includes serous or hemorrhagic detachment of retinal pigment epithelium or sensory retina with the presence of sub-retinal pigment epithelial hemorrhages or sub-retinal fibrous scar tissue. This form of ARMD is usually characterized by a sudden loss of visual acuity and final visual acuities worse than 20/200. The second type, geographic atrophy or “dry AMD” is defined as a discrete area of retinal pigmentation greater than or equal to 175 μm in diameter. It is characterized by a sharp border and the presence of visible choroidal vessels.

The Framingham Eye Study found a prevalence of age-related macular degeneration of 5.7% in 2,631 people age 65 and over. The incidence was 19.7% in 396 patients age 75 and older. 5.3% of the patients with ARM were felt to have exudative form while 2.4% were felt to have a combination of the atrophic and exudative forms. Ferris, et al., attempted to determine the percentage of eyes that were legally blind due to the exudative form of ARM. They reviewed fundus photographs of the patients with ARM and visual acuities of 20/200 or worse. While this was a small sample, they
concluded that 79% of the legally blind patients with ARM had the exudative form, while 21% had the atrophic form of ARM.\textsuperscript{5}

The prevalence of age-related maculopathy was determined using fundus photographs of 6251 participants in a single-center prospective follow up study of persons 55 to 98 years of age called the Rotterdam study. The photographs were reviewed for the presence of drusen, pigmentary abnormalities or neovascular age related macular degeneration. This study showed atrophic or neovascular age related macular degeneration was present in 1.7% of the total population. Atrophic age related macular degeneration increased from 0.1% in persons 55 to 64 years of age to 3.7% in those 85 years of age or older. Neovascular age related degeneration increased from 0.1% to 7.4% in these same age groups. No gender differences were observed in this study.\textsuperscript{6} The Rotterdam Study results were similar to those of the Framingham Eye Study and concluded that in those greater than 55 years of age, prevalence of neovascular age-related macular degeneration increases strongly with age.

There are many risk factors associated with ARM. These include hypertension, history of lung infection, family history of macular diseases, smoking, sunlight exposure and refractive error. Smoking and its relationship to ARM has been an area of interest because it is a modifiable risk factor. Although reports on the association between tobacco smoking and ARM have conflicted in the past, current studies support that there is positive correlation between the two. In the United States, cigarette smoking has been implicated as one of the largest contributing factors in the development of preventable chronic and life-threatening diseases. The correlation between cigarette smoking and systemic diseases has been well proven.\textsuperscript{7,8} Toxicological pathways supporting cigarette smoking as a cause of systemic disease have been established. These include the well-accepted free-radical pathway theory.\textsuperscript{9} In both its tar and gas phase, cigarette smoke contains free radicals that are capable of reducing molecular oxygen to superoxide molecules. These molecules lead to the formation of reactive free radicals, which can initiate and propagate the process of lipid and polyunsaturated fatty acid peroxidation. They are implicated in the formation of atheromatous plaques.\textsuperscript{7} The rod and cone outer segments are at particular risk of oxidative damage because of their high concentration of polyunsaturated fatty acids.\textsuperscript{10}
One emerging hypothesis regarding the pathogenesis of AMD is that antioxidative stress or reduced levels of antioxidants play a role. Another hypothesis suggests that AMD may be associated with vascular insufficiency or vascular abnormalities. Cigarette smoking relates to both of these potential mechanisms; it increases oxidative stress, reduces plasma antioxidant levels and increases the risk of vascular disease.

With increased cigarette consumption, smokers tend to lower their intake of fruits, fiber, and vitamins. These contain micronutrients which play an important role in the oxidative stability of the cell. Many dietary components, especially vitamins A, C, D, and selenium, are involved in the antioxidant defense of the cell. Smokers have been found to have lower levels of these antioxidants.

Several biological hypotheses have been proposed to explain the association between smoking and ARM. First, smoking may promote the development and progression of subretinal new vessels. Second, smoking may promote atherosclerotic and hypoxic damage to the choroidal vasculature, leading to maculopathy. Third, smoking may lead directly to increased oxidative damage to outer retinal cells.

Currently, there is no proven treatment for atrophic ARM although in many cases low vision aids are prescribed with good results. In recent years, laser photocoagulation has been utilized to treat choroidal neovascular membranes in exudative ARM. However laser photocoagulation is useful only for a minority of patients with new blood vessel growth in and around the fovea. Furthermore, this treatment merely delays, and does not prevent, subsequent vision loss. For these reasons, the identification of avoidable causes of AMD has important public health implications. There is an increasing speculation that dietary factors, particularly antioxidants may prevent or impede the progression of AMD. The theory is biologically plausible. The outer retina rich in polyunsaturated fatty acids may be altered adversely by free-radical production and oxidation and conversely may be protected by nutrients that block this oxidative damage. Antioxidants may also help to maintain the integrity of the choroidal blood vessels that supply the macular region of the retina.

Basic and clinical research suggest that nutritional factors may be associated with AMD. Animals of several species, including primates that are deprived of nutrients with
antioxidant potential are more prone than those not deprived of such nutrients to develop retinal degeneration. This effect is enhanced by bright light. Animals that are given nutrients that have antioxidant potential are less likely to demonstrate retinal degeneration than those who are not given such nutrients.

The Multicenter Eye Disease case control study carried out in five ophthalmology centers in the United States evaluated a total of 356 case subjects who were diagnosed with an advanced stage of AMD within one year of their enrollment. These subjects were 55 to 80 years of age. 520 control subjects who were from the same geographic areas as case subjects and had other ocular diseases, were matched to cases according to age and gender. The average ages were 71 years for subjects and 68 years for controls. Fifty-six percent of case subjects and 55% of control subjects were female.²

Results of this study showed a reduced risk for exudative neovascular AMD with higher levels of carotenoid intake. This was statistically significant, apparently linear trend. When Carotenoid fractions were evaluated separately, higher intake of beta carotene and lutein/zeaxanthin were associated with a statistically significant reduction in risk for AMD. Total vitamin A, which is composed mainly of carotenoid was also associated with a reduced risk for AMD in the multivariate model. Total vitamin C intake had no consistent association with AMD. However, the results for vitamin C intake from foods, excluding supplement use, suggested a protective effect that was not statistically significant. A statistically significant trend for a lower risk for AMD was seen with a greater intake of spinach and green vegetables.

The inverse trend associated with lutein/zeaxanthin intake was seen for all categories of smoking and was somewhat stronger in current smokers. For those with the highest level of lutein/zeaxanthin intake, there was little apparent effect of cigarette smoking. For vitamin C and E, a protective association for higher intake was suggested only among those who had never smoked. However, these associations were not statistically significant. The dominant pigments in the macula are lutein and zeaxanthin, which are selectively accumulated in the retina from plasma.¹⁷ ¹⁸ These yellow pigments can filter out visible blue light, which reaches the retina and theoretically can cause photic damage.¹⁸ This study concluded that increasing the consumption of foods such as
dark green, leafy vegetables rich in certain carotenoids, may decrease the risk of developing advanced or exudative AMD.

There have been studies emphasizing the effect of smoking on the macula in people who have been smoking for a long time and in those who have recently started. Most of these studies support the importance of educating our younger population about the harmful effects of smoking on vision and the importance of not smoking. Among these studies some have found no correlation between smoking and nonexudative ARMD but have found a correlation with exudative ARM. One of these is the Beaver Dam Eye Study of Wisconsin, which evaluated looked at the macular status of 4,771 patients between the ages of 43 to 86 years old and correlated the macular appearance with smoking history. The maculae of the study group were evaluated for 2 years between 1988 and 1990 using stereo 30-degree color fundus photographs, which were graded in a masked fashion. Drusens were evaluated as to type, size, and confluence with other nonexudative changes also noted. Cigarette smoking status at the time of the examination was determined as follows: a subject was classified as a never smoker if he/she had smoked less than 100 cigarettes in his/her lifetime; as an ex-smoker if he/she had smoked more than 100 cigarettes in his/her lifetime, but had stopped smoking before the examination; or as a current smoker if he/she had not stopped smoking. Passive smokers were never smokers or ex-smokers who had lived in a household with a smoker.

The result of the study showed a significant association between exudative macular degeneration and current smoking in both genders. Based on all the data collected together, it suggests that smoking may promote the development and progression of subretinal neovascularization. In summary, cross-sectional data from this study suggest that cigarette smoking is associated with exudative macular degeneration.

The Blue Mountains Eye Study concluded that current tobacco smoking was significantly associated with neovascular AMD, geographic atrophy and early AMD. This study was a population-based survey of vision and common eye disease involving an urban, elderly population 49 years or older in the Blue Mountain region West of Sydney, Australia. Excluding the subjects that died during the two-year study and those who moved form the area, the response rate was 87-90%.
Smoking history was ascertained using an interviewer-administered questionnaire to find out whether subjects had ever smoked and if so, at what age did they start smoking. They also determined when, at what age, for how long participants had quit smoking, what form of tobacco they used, and how much they usually smoked. This study was similar to the Beaver Dam Study in considering passive smokers. Additional questions about dietary supplement intake and history of medical problems were included. Among the Blue Mountains Eye Study participants, 61% of the women and 31.9% of the men had never smoked. Among those who had never smoked, 42.9% had a smoking spouse and 90% were women.

The Blue Mountain Study concluded that there was a causal linkage between exposure to tobacco and ARM. This linkage is strengthened by the increased risk for ARM with increased exposure to tobacco. The hypothesis that is best supported by this study is that smoking may lead directly to increased oxidative damage to outer retinal cells. The findings of the Blue Mountains study agree with other published data including a magnitude of risk similar to those previously reported in the multi-center eye disease case control study.19

The Rotterdam study was also done to assess the relation between cigarette smoking and age-related macular degeneration (AMD) in a population of elderly persons. A total of 6174 persons, 55 years and older participated in this study. The average age of subjects was 80.2 years. All subjects underwent an extensive ophthalmologic examination, with current and past smoking habits assessed by interview. Age-related macular degeneration was present in 104 of the 6174 subjects. The prevalence increased from 1.1% in subjects younger than 85 years to 11% in subjects 85 years and older. In the younger group, current and former smoking was associated with neovascular AMD. Former and current smokers younger than 85 years had a 3.2 and 6.6 fold increased risk of neovascular AMD respectively, compared to those who had never smoked. This association was not present in persons 85 years and older. Unlike neovascular AMD, atrophic AMD was not associated with former smoking. A strong increased risk of neovascular AMD was present in persons who had smoked 10 or more pack-years. This study adds to the evidence that smoking is a risk factor for neovascular AMD.15
The Copenhagen City Heart Study is a prospective cardiovascular study comprising an age stratified sample of 20,000 men and women aged 20 years or more, randomly selected from a population of 90,000 subjects living within a district in Copenhagen. The relationship between age related macular degeneration, cardiovascular risk factors and certain life-style factors was also studied from 1981-83. The ophthalmological study sample comprised 1000 randomly selected persons, aged 60 to 80 years. Separate analyses were made for the atrophic and the exudative forms of age related macular degeneration. Multiple logistic regression analysis was used to evaluate the influence of potential risk factors on the frequency of AMD. Some of the risk factors were evaluated include: age, gender, corneal arcus, hypertension, intermittent claudication, ECG-abnormalities, angina pectoris, plasma cholesterol concentration, myocardial infarction, stroke, physical inactivity, smoking, alcohol intake, diuretics and heart medicines. Three variables were significantly associated with presence of AMD. Age exerted a strong influence on the risk of AMD. Among persons 75-80 years old the risk of AMD was around 30 times higher than in persons aged 60-64 years. This was the case for both the atrophic and the exudative form of AMD. Smoking was significantly associated with the risk of AMD. Compared to nonsmokers, smokers who inhaled had a 2.4 times higher risk of all types of AMD. The 2.5 times risk for atrophic form of AMD was also statistically significant. Although a 1.5x increased risk for the exudative form of AMD was found, it was statistically insignificant. The results from present study indicated that the association between smoking and ARM is only significant for atrophic AMD. Smoking has previously been found associated with arteriosclerotic disease. However, atherogenesis does not seem to give a plausible explanation of the increased risk for AMD effect, as there was no relation between the other arteriosclerotic manifestations and AMD. A variety of substances, including free radicals, from the inhaled smoke might influence the macula area, because of its high metabolism.

The Nurses' health study, a prospective study of cigarette smoking and age related macular degeneration in women was performed with 12 years of follow up (1980 to 1992). In this study, information on smoking habits was updated every 2 years. A total of 31,843 registered nurses who were aged 50-59 years in 1980 and did not report a diagnosis of cancer or AMD were enrolled at the beginning of the study. Additional
women entered the analytic cohort as they reached 50 years of age. During 556,338 person-years of follow up, 215 women were newly diagnosed as having AMD. After adjusting for other factors for AMD, women who currently smoked 25 or more cigarettes per day had a 2.4x increased risk of developing AMD, compared to never smokers. Past smokers of 25 or more cigarettes per day had a 2-fold increased risk compared to nonsmokers. Compared with current smokers, little reduction in risk was suggested even after quitting smoking for 15 or more years. Risk of AMD also increased with an increasing number of pack-years smoked. Among women who smoked for 65 or more pack-years, the risk was 2.4 times the risk of never smokers. Analyses of dry and exudative type of AMD and other alternative definitions of AMD revealed similar results. This study concluded cigarette smoking is an independent and avoidable risk factor for AMD among women.21

A similar prospective study has been done on 21,157 US male physicians who did not have a diagnosis of AMD at baseline and were followed at least 7 years while participating in the Physician’s Health Study. Based on information reported at baseline, 11% were current smokers, 39% were past smokers and 50% were never smokers. This study indicates that cigarette smoking increases the risk of AMD in a dose-dependent fashion. In multivariate analysis, current smokers of 20 or more cigarettes per day, compared with never smokers, had an increased risk of AMD. Past smokers had a modest elevation in risk of AMD. For current smokers of fewer than 20 cigarettes per day, there was a statistically nonsignificant 26% increased risk of AMD. Current smokers of one pack or more of cigarettes daily had a 2- to 3-fold increased risk of AMD with vision loss compared with never smokers.16

This dose-dependent relationship between cigarette smoking and AMD indicates that the biological effects of smoking on the retina and choroid depend on accumulated lifetime exposure. The finding among former smokers of an elevated risk of AMD that is maintained over an extended period following smoking cessation further suggests that the biological effects diminish gradually, if at all, over a number of years. The last two studies of US male physicians and US female nurses could have significant public health implication as they appear to identify an important avoidable cause of a major source of morbidity that at present time, is generally not amenable to treatment. The last two
studies also provide significantly strong evidence of the role of cigarette smoking in increasing the risk of AMD.

Clinical methods for the evaluation of macular function are limited in number. Snellen visual acuity and Amsler grid testing are the common methods that are easily employed in the eye care practitioner’s office. The central 30-degree visual fields can document the area of macular scotoma. Foveal and macular electroretinography and visual evoked response testing can be used to quantify macular function, but these techniques are not widely available in the eye care practitioners or optometrist’s offices. Snellen visual acuity and Amsler grid testing do not evaluate the pathophysiologic features of the disease process. A study by a group of ophthalmologists proposes that the clinical testing of macular function can be performed with the macular photostress test (MOT), a simple and easily accessible procedure that can be performed in an outpatient office setting. In addition, it provides another means of testing the retina and its physiologic function. In the study by Wu Gloria, et.al. eighty eyes were tested and divided into four diagnostic categories: background diabetic retinopathy, diabetic macular edema, age-related macular degeneration, and normal. Patients with age-related macular degeneration had longer recovery times than did age matched subjects without age-related macular degeneration. This reflects that the anatomic lesion in ARM is located in the retinal pigment epithelium-photoreceptor complex. In comparison, the eyes with macular edema, whose lesion is in the inner retina and not the retinal pigment epithelium, show a less-prolonged recovery time than the eyes with age-related macular degeneration.

An evaluation of Macular dazzling test, very similar to the Photo stress Recovery test, has been done by Gomez-Ulla, et.al. This test subjectively explores macular function by dazzling the retina and then measuring the length of time, which the subject takes to regain the level of visual acuity he had before the dazzling. Since what is actually being explored with this method is the function of the macula, these authors called it the macular dazzling test. Gomex-Ulía, et.al. performed the MDT on 240 healthy eyes. The ages of the subjects ranged from 10 to 69 years, the ratio of genders being 50:50. The subjects were divided into age groups as follows: from 10 to 19, 20 to 29, 30 to 39, 40 to 49, 50 to 59, and 60 to
69 years. Each age group contained 20 subjects, 10 male and 10 female. In all cases subject's visual acuity was equal to or greater than 20/25, with or without optical adjustment. Subjects were only included if they had a refraction of not more than three diopters of hypermetropia, myopia, or astigmatism. To produce the dazzle, a Minolta electronic flash light was used. A Heur chronometer was used to measure the recovery time for visual acuity following the dazzling. Long distance visual acuity was explored through the use of Snellen optotypes and short distance vision by means of Parinaud test. The exploration was systematically begun on the right eye, and later continued on the left eye. The MDT test is similar to the Photostress Recovery Test. Both are easy tests to perform, require a minimum of cooperation by the subject. The MDT value increases significantly as the age of the subject increases. Starting from age 10-69 the gender of the subject has no influence on the expected results, and there are no significant differences between normal subject's right and left eyes. Mydriasis does not affect the MDT, but miosis, reduces the recovery period.  

Due to the recently reported correlation found between age-related macular degeneration and smoking a study will be designed to see if smoking affects the macula in a young population. The essential point in our future study will be to determine how quickly the maculae of the young smoking population recover after the Photostress Recovery Test. The Photostress Recovery Test is a quick, simple and direct procedure used to assess the initial sensitivity loss and speed of visual function return to the macula after a timed exposure to a bright light stimuli. The Bailliant test, a form of Photostress Recovery Test was formulated to determine the functional macular reaction by dazzling the retina and then measuring the length of time the subject takes to regain the level of visual acuity he had prior to the dazzling. The macular photostress test involves the bleaching of the retinal visual pigments by an intense light stimulus, a transient state of visual insensitivity and production of a scotomatus afterimage, and then the return of retinal sensitivity due to resynthesis of visual pigments in the retinal pigment epithelium-photoreceptor complex. The length of time for the initial recovery from the bright light stimulus, which allows successful reading of the previously achieved snellen acuity chart, is referred to as the recovery time. The recovery time presumably is dependent on the anatomic and biochemical events that occur in the retina as it performs the photopic
process of vision. If a correlation between smoking and the function of the macula is found in this study, it could lead to increased awareness and prevention of macular defects in later stages of life by educating our young population of quitting their habits early in life and by making them understand the serious effects of smoking on their vision.

The first plan of the study would be to find out about the smoking history of our subjects using an interviewer-administered questionnaire. This questionnaire would ask whether subjects had ever smoked, and if so, at what age smoking began, if they had quit, at what age and for how long, what form their tobacco intake took (cigarettes, hand rolled, cigars, or pipe), and how much they usually smoked. Questions to determine if the subject is a passive smoker would also be asked in the questionnaire. Age, gender and eye color of subjects would be considered and matched to non-smoking controls. Subjects with medical problems such as high blood pressure, angina, stroke, acute myocardial infarction, any other major medical problems or best corrected visual acuity of less than 20/20 would be excluded from the study. The study would be designed to test 80 patients between the ages of 18 to 30 of whom 40 are smokers and 40 are control group, to evaluate how quickly each group recovers from the Photostress Recovery Test. Only subjects who have smoked at least one pack of cigarette per day for 6 months would be selected.

The Photostress Recovery Test will be done in the standard method as described in optometry literature. The transilluminator is placed 3 to 5 cm from the eye with the opposite eye occluded. The patient is asked to stare at the light coming from the transilluminator for 10 seconds. After replacing the spectacle Rx, the patient is asked to look at the distance acuity chart and the time until the acuity returns to one line larger than the original best correctable visual acuity is measured. The same procedure is repeated on the opposite eye. This is a valuable test that has been done on patients with diabetic retinopathy and age related macular degeneration.

All of the studies that are discussed have the same goal of proving the relationship and the positive correlation between smoking and macular degeneration. Our goal is not far from their goal. Our major plan is to prevent the occurrence of ARM by educating smokers of the risks involved.


10 Eye Disease Case-Control Study Group. Antioxidant status and neovascular ARMD. Arch Ophthalmol 1993;111:104-9


