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MD Research News

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Drug Treatment

Retina. 2011 Mar 19. [Epub ahead of print]

RANIBIZUMAB FOR THE TREATMENT OF EXUDATIVE AGE-RELATED MACULAR DEGENERATION ASSOCIATED WITH RETINAL PIGMENT EPITHELIAL TEAR.

Rouvas AA, Ladas ID, Georgalas I, Vergados I, Papakonstantinou D, Kotsolis AI.

From the *Second Department of Ophthalmology; and †First Department of Ophthalmology, Medical School of Athens University, Athens, Greece.

PURPOSE: To evaluate the efficacy of intravitreal ranibizumab in eyes with exudative age-related macular degeneration associated with retinal pigment epithelial tear.

METHODS: In this retrospective case series, patients with active exudative age-related macular degeneration associated with retinal pigment epithelial tear were treated by repeated injections of intravitreal ranibizumab. The outcome measures were best-corrected visual acuity and the signs of lesion activity, as evaluated by optical coherence tomography, fluorescein angiography, and indocyanine green angiography.

RESULTS: Twenty-one eyes of 20 patients were followed-up for a median of 12 months (range, 6-28 months). The median number of injections was 7 (range, 3-15). The best-corrected visual acuity improved in 6 eyes (28.57%), remained stable in 12 (57.14%), and decreased in 3 (14.28%). At the end of the follow-up time, 19 eyes (90.47%) had an inactive neovascular lesion in angiography, while 18 eyes (85.71%) had no signs of intraretinal or subretinal fluid.

CONCLUSION: Intravitreal ranibizumab was effective in improving or stabilizing vision and resulting in a quiescent lesion in the majority of patients with exudative age-related macular degeneration associated with retinal pigment epithelial tear. The functional results were apparently better in eyes without foveal involvement by the retinal pigment epithelial tear.

PMID: 21427630 [PubMed - as supplied by publisher]

J Pediatr Ophthalmol Strabismus. 2011 Mar 15;48 Online:e19-22. doi: 10.3928/01913913-20110308-02.

Intravitreal Ranibizumab for Choroidal Neovascularization in Best's Vitelliform Macular Dystrophy in a 6-Year-Old Boy.

Heidary F, Hitam WH, Ngah NF, George TM, Hashim H, Shatriah I.

Abstract

A 6-year-old boy with a family history of Best's vitelliform macular dystrophy presented with a unilateral choroidal neovascularization. He responded well to a single injection of intravitreal ranibizumab. His visual acuity improved from 6/18 to 6/9 in the affected eye. His condition remained stable for 1½ years after treatment.

PMID: 21417187 [PubMed - in process]

Curr Opin Ophthalmol. 2011 Mar 21. [Epub ahead of print]

Preferred therapies for neovascular age-related macular degeneration.

Chiang A, Regillo CD.

Retina Service, Wills Eye Institute, Jefferson Medical College, Philadelphia, USA bMid Atlantic Retina, Wyndmoor, Pennsylvania, USA.

PURPOSE OF REVIEW: This report reviews the current treatment strategies and ongoing clinical trials in the treatment of neovascular age-related macular degeneration (AMD).

RECENT FINDINGS: The functional and anatomic outcomes achieved in the pivotal ranibizumab trials with monthly injections set the standard for comparison. Since then, various modified dosing regimens with the aim of lessening the treatment burden associated with monthly injections have been investigated. Combination therapy incorporating photodynamic therapy and antivascular endothelial growth factor (anti-VEGF) therapy may represent an alternative treatment approach and randomized multicenter clinical trials are ongoing. In addition, new pharmacologic agents like VEGF Trap-Eye are being developed and investigated; preliminary 1-year results with VEGF Trap-Eye are encouraging.

SUMMARY: Ranibizumab or bevacizumab monotherapy remains the preferred therapy in the management of neovascular AMD at the present time. Ongoing clinical trials will help determine the efficacy of ranibizumab relative to bevacizumab, evaluate the long-term efficacy and safety of combination therapy modalities, and assess the role of new pharmacologic agents.

PMID: 21427571 [PubMed - as supplied by publisher]

Curr Opin Ophthalmol. 2011 Mar 21. [Epub ahead of print]

Treatment for atrophic macular degeneration.

Meleth AD, Wong WT, Chew EY.

National Eye Institute/National Institutes of Health, Bethesda, Maryland, USA.

PURPOSE OF REVIEW: To summarize ongoing and recently completed trials on geographic atrophy associated with age-related macular degeneration.

RECENT FINDINGS: A large number of investigational agents are under development for treatment of geographic atrophy in both pre and early-phase clinical testing.

SUMMARY: Therapies targeting all putative phases of pathogenesis in geographic atrophy are under development. Thus far, no therapies have been demonstrated to be effective.

PMID: 21427574 [PubMed - as supplied by publisher]

J Glaucoma. 2011 Mar 16. [Epub ahead of print]

Sustained Increased Intraocular Pressure Related to Intravitreal Antivascular Endothelial Growth Factor Therapy for Neovascular Age-related Macular Degeneration.

Tseng JJ, Vance SK, Della Torre KE, Mendonca LS, Cooney MJ, Klancnik JM, Sorenson JA, Freund KB.

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PURPOSE: To describe a series of previously normotensive eyes experiencing sustained elevated intraocular pressure (IOP) associated with long-term intravitreal antivascular endothelial growth factor (VEGF) therapy for neovascular age-related macular degeneration (AMD).

PATIENTS AND METHODS: Clinical data were reviewed for 25 eyes of 23 patients with neovascular AMD who had increased IOP while receiving interval doses of intravitreal ranibizumab and/or bevacizumab. All eyes had tolerated multiple anti-VEGF injections in the past without IOP elevations.

RESULTS: After a mean of 20.0 anti-VEGF injections (range, 8-40 injections), the mean IOP was 29.8 mm Hg (range, 22-58 mm Hg), compared with a baseline of 16.9 mm Hg (range, 14-21 mm Hg). The mean highest IOP while receiving intravitreal anti-VEGF therapy was 35.8 mm Hg (range, 23-58 mm Hg). Overall, 23 of 25 cases required IOP management. In the remaining 2 cases, anti-VEGF dosing was switched from regular interval dosing to an optical coherence tomography-guided variable regimen, with subsequent improvement in IOP without antiglaucoma treatment.

CONCLUSIONS: Serial injections of anti-VEGF agents may lead to persistent IOP elevations that require glaucoma therapy. The clinician should recognize this phenomenon, as it can occur even if the patient has tolerated multiple prior injections without IOP elevation. Further exploration of the relationship between anti-VEGF therapy and IOP is needed.

PMID: 21423038 [PubMed - as supplied by publisher]

Curr Diabetes Rev. 2011 Mar 21. [Epub ahead of print]

Diabetic Papillopathy: Current and New Treatment Options.

Giuliari GP, Sadaka A, Chang PY, Cortez RT.

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Abstract

Diabetes mellitus is a growing global epidemic. Patients with this disease present with a variety of health conditions, including a number of ocular complications that threaten vision, such as proliferative diabetic retinopathy and macular edema. Diabetic papillopathy, another potential ocular complication from diabetes, is a self-limiting, sometimes bilateral disease that may affect both type 1 and type 2 diabetics. It is characterized by optic disc swelling caused by vascular leakage and axonal edema in and around the optic nerve head. Occasionally, it may be accompanied by intraretinal hemorrhages and hard exudates. Diabetic papillopathy tends to be mild and is usually associated with good visual prognosis; however, there are some cases in which permanent visual impairment can develop. The pathogenesis remains largely unknown, but there has been evidence suggestive of its associations with a small cup/disc ratio and rapid reduction in glycemia. There is no validated therapy for diabetic papillopathy; however, current case reports have shown promising results after local injections of corticosteroids as well as bevacizumab (Avastin), a potent monoclonal antibody that has been employed for the treatment of ocular vaso-proliferative diseases such as choroidal neovascular membranes associated with age-related macular degeneration and proliferative diabetic retinopathy.

PMID: 21418004 [PubMed - as supplied by publisher]

Other treatment & diagnosis

Invest Ophthalmol Vis Sci. 2011 Mar 18. [Epub ahead of print]

Test-retest variability of reading performance metrics using MNREAD in patients with age-related macular degeneration.

Patel PJ, Chen FK, Da Cruz L, Rubin GS, Tufail A.

NIHR Biomedical Research Centre for Ophthalmology (Moorfields Eye Hospital and UCL Institute of Ophthalmology), London UK.

Purpose: To determine the test-retest variability of reading ability using the MNREAD charts in patients with stable age-related macular degeneration (AMD)

Methods: In this prospective study, reading ability was measured at 2 visits in 124 non-treated eyes of 124 patients with AMD enrolled in an ongoing clinical trial using a standardized MNREAD protocol. Only patients with stable AMD who could perform the reading test at 40 cm at both visits were included in the analysis. Different scoring rules were applied to calculate the critical print size and maximum reading speed.

Results: Data from the 59 patients that met the study criteria were analyzed with a mean (SD) age of 78 (7.6) years and mean (SD) interval of 43 (6) days between measurements. The 95% coefficient of repeatability (CR) was 0.30 LogMAR for reading acuity. The CR for critical print size and maximum reading speed varied depending on the analysis method applied.

Conclusions: We report estimates of the intersession test-retest variability of reading performance metrics in patients with stable AMD. The results are helpful in both defining end-points in clinical trials for AMD and distinguishing clinical change from measurement variability in clinical practice.

PMID: 21421873 [PubMed - as supplied by publisher]

Epidemiology & pathogenesis

Surv Ophthalmol. 2011 Mar 18. [Epub ahead of print]

A Review and Meta-analysis of the Association Between C-Reactive Protein and Age-related Macular Degeneration.

Hong T, Tan AG, Mitchell P, Wang JJ.

Centre for Vision Research, Department of Ophthalmology and Westmead Millennium Institute, University of Sydney, Sydney, New South Wales, Australia.

Abstract

Age-related macular degeneration (AMD) is the leading cause of blindness in people over 60 in western countries. Inflammatory markers have been implicated in the development and progression of AMD. C-reactive protein (CRP) is an inflammatory marker known to be associated with cardiovascular disease, and a link between AMD and CRP has been suggested. In this systematic review we summarize the currently available evidence from clinic-based and population-based studies investigating this association. A meta-analysis of evidence from eleven studies (41,690 study participants) shows that high serum levels (>3 mg/L) of CRP are associated with a two-fold likelihood of late onset AMD, compared to low levels (<1mg/L). Sub-group meta-analyses show a higher association in studies using ophthalmoscopic examination, compared to those using photographic grading (pooled odds ratio 3.83 vs 1.36), to determine AMD status, or in clinic-based samples compared to population-based studies.

PMID: 21420705 [PubMed - as supplied by publisher]

Eye (Lond). 2011 Mar 25. [Epub ahead of print]

The prevalence and analysis of risk factors for age-related macular degeneration: 18-year follow-up data from the Speedwell eye study, United Kingdom.

Ngai LY, Stocks N, Sparrow JM, Patel R, Rumley A, Lowe G, Davey Smith G, Ben-Shlomo Y.

Department of Social Medicine, University of Bristol, Bristol, UK.

Aims/Purpose: To determine the prevalence of age-related maculopathy (ARM) and age-related macular degeneration (AMD) in men aged 65-83 years living in the Speedwell region of Bristol, United Kingdom and identify modifiable risk factors.

Methods: A total of 2348 men recruited to the Speedwell prospective cohort study in 1979 were followed up in 1997 with an eye questionnaire and had retinal photographs that were assessed using the International Classification System for ARM.

Results: In all, 934 men (66.8% response rate) attended with a mean of 17.9 years (15.3-20.6 years) follow-up. Early ARM (grades 2-3) was found in 9.2% (95% confidence interval (CI) 7.4%, 11.4%) and late age-related maculopathy (grade 4, AMD) in 0.5% (95% CI 0.2%, 1.2%). The risk of ARM (grades 2-4) was increased with raised C-reactive protein and consumption of lard and solid fats, whereas triglyceride levels were associated with a lower risk. The latter were confirmed in multivariable analyses and in addition, haemodynamic measures also predicted risk (eg mean arterial pressure odds ratio (OR) per z-score 1.37, 95% CI 1.04, 1.79).

Conclusions: In a representative cohort of men aged 65-83 from Bristol, United Kingdom, many had macular changes that put them at higher risk of developing AMD. Various modifiable exposures were associated with an increased risk ARM/AMD. Opportunities for screening and undertaking secondary prevention interventions need to be explored to prevent progression of the disease and blindness.Eye advance online publication, 25 March 2011; doi:10.1038/eye.2011.56.

PMID: 21436849 [PubMed - as supplied by publisher]

Br J Ophthalmol. 2011 Mar 18. [Epub ahead of print]

Lipid metabolites in the pathogenesis and treatment of neovascular eye disease.

Stahl A, Krohne TU, Sapieha P, Chen J, Hellstrom A, Chew E, Holz FG, Smith LE.

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Abstract

Lipids and lipid metabolites have long been known to play biological roles that go beyond energy storage and membrane structure. In age-related macular degeneration and diabetes, for example, dysregulation of lipid metabolism is closely associated with disease onset and progression. At the same time, some lipids and their metabolites can exert beneficial effects in the same disorders. This review summarises our current knowledge of the contributions of lipids to both the pathogenesis and treatment of neovascular eye disease. The clinical entities covered are exudative age-related macular degeneration, diabetic retinopathy and retinopathy of prematurity, with a special emphasis on the potential therapeutic effects of ω3- (also known as n-3) polyunsaturated fatty acids.

PMID: 21421650 [PubMed - as supplied by publisher]

Klin Monbl Augenheilkd. 2011 Mar 23. [Epub ahead of print]

[The Role of Light in the Development of RPE Degeneration in AMD and Potential Cytoprotection of Minocycline.]

[Article in German]

Kernt M, Thiele S, Hirneiss C, Neubauer AS, Lackerbauer CA, Ulbig MW, Kampik A.

Augenklinik, Ludwig-Maximilians-Universität München.

BACKGROUND: Light-induced oxidative stress is an suggested reason for retinal pigment epithelium (RPE) degeneration in age-related macular degeneration (AMD). This study investigates the influence of light on intracellular reactive oxygen species (ROS) and apoptosis in the human RPE and potential cytoprotective effects of the tetracycline antibiotic minocycline.

METHODS: Primary human RPE cells were either pre- or post-incubated with minocycline and then exposed to white light or oxidative stress (600 µM, H (2)O (2)). Then viability, induction of intracellular reactive oxygen species (ROS), apoptosis and cell death was determined. Expression of apoptotic BAX and anti-apoptotic Bcl-2 protein and their mRNA were determined by RT-PCR and Western blot analysis.

RESULTS: Both light exposure and oxidative stress decreased RPE cell viability and Bcl-2 expression and increased intracellular ROS, apoptotic cell death, and BAX expression. Minocycline reduced these effects under certain conditions.

CONCLUSIONS: This study demonstrates that minocycline effectively protects human RPE cells against oxidative damage. However, in the light of minocycline's photosensitising properties its potential role in AMD treatment needs further evaluation.

PMID: 21432767 [PubMed - as supplied by publisher]

Am J Pathol. 2011 Apr;178(4):1913-21.

VAP-1-Mediated M2 Macrophage Infiltration Underlies IL-1β- but Not VEGF-A-Induced Lymph- and Angiogenesis.

Nakao S, Noda K, Zandi S, Sun D, Taher M, Schering A, Xie F, Mashima Y, Hafezi-Moghadam A.

Angiogenesis Laboratory, Massachusetts Eye & Ear Infirmary, Boston, Massachusetts; Department of Ophthalmology, Harvard Medical School, Boston, Massachusetts.

Abstract

Vascular adhesion protein-1 (VAP-1) contributes to inflammatory and angiogenic diseases, including cancer and age-related macular degeneration. It is expressed in blood vessels and contributes to inflammatory leukocyte recruitment. The cytokines IL-1β and vascular endothelial growth factor A (VEGF-A) modulate angiogenesis, lymphangiogenesis, and leukocyte infiltration. The lymphatic endothelium expresses intercellular adhesion molecule-1 and vascular adhesion molecule-1, which facilitate leukocyte transmigration into the lymphatic vessels. However, whether lymphatics express VAP-1 and whether they contribute to cytokine-dependent lymph- and angiogenesis are unknown. We investigated the role of VAP-1 in IL-1β- and VEGF-A-induced lymph- and angiogenesis using the established corneal micropocket assay. IL-1β increased VAP-1 expression in the inflamed cornea. Our in vivo molecular imaging revealed significantly higher VAP-1 expression in neovasculature than in the preexisting vessels. VAP-1 was expressed in blood but not lymphatic vessels in vivo. IL-1β-induced M2 macrophage infiltration and lymph- and angiogenesis were blocked by VAP-1 inhibition. In contrast, VEGF-A-induced lymph- and angiogenesis were unaffected by VAP-1 inhibition. Our results indicate a key role for VAP-1 in lymph- and angiogenesis-related macrophage recruitment. VAP-1 might become a new target for treatment of inflammatory lymph- and angiogenic diseases, including cancer.

PMID: 21435467 [PubMed - in process]

Genetics

Ophthalmic Genet. 2011 Mar 18. [Epub ahead of print]

The Relationship Between Angiotensin Converting Enzyme Insertion/deletion Polymorphism and Age-related Macular Degeneration.

Uçer B, Kayıkçıoğlu O, Seymenoğlu G, Var A, Cam S.

Department of Ophthalmology, Celal Bayar University, Faculty of Medicine, Manisa, Turkey.

Background: To assess the role of serum angiotensin converting enzyme (ACE) levels and ACE insertion / deletion (I/D) genetic polymorphism in Turkish age-related macular degeneration (AMD) patients and control subjects.

Methods: This prospective study consisted of 78 patients with AMD and 68 control subjects. The I/D polymorphism of the ACE was carried out by polymerase chain reaction. Serum ACE levels were determined by using the ELISA method.

Results: There was no significant difference in the mean serum values of ACE between the control and patient groups (p = 0.107). The genotypic frequencies of ACE polymorphism in the control and patient groups were not significantly different either (p = 0.218).

Conclusion: We could not show a significant role of serum ACE levels and ACE I/D genetic polymorphism in the etiopathogenesis of AMD in the Turkish population, and our findings did not support the idea that serum ACE levels and ACE DD genotype were risk factors for AMD.

PMID: 21417676 [PubMed - as supplied by publisher]

Tohoku J Exp Med. 2011;223(4):253-61.

The A Allele of the -576G>A Polymorphism of the Transferrin Gene Is Associated with the Increased Risk of Age-Related Macular Degeneration in Smokers.

Wysokinski D, Szaflik J, Sklodowska A, Kolodziejska U, Dorecka M, Romaniuk D, Wozniak K, Blasiak J, Szaflik JP.

Department of Molecular Genetics, University of Lodz.

Abstract

Age-related macular degeneration (AMD) is the leading cause of blindness among the elderly in developed countries, and its pathogenesis is underlined by genetic and environmental factors. Oxidative stress is a major environmental risk factor of AMD; namely, AMD is associated with the increased level of reactive oxygen species, which may be produced in reactions catalyzed by iron present in the retina. Therefore, variability of the genes of iron metabolism may be important in the AMD risk. In the present study, we analyzed the association between AMD and the -576G>A polymorphism of the transferrin gene or the 1892C>T polymorphism of the transferrin receptor 2 (TFR2) gene in 278 patients with AMD and 105 controls. The former polymorphism is located in the promoter region of the transferrin gene and may affect the level of its transcription, while the latter is a synonymous mutation in the exon 16, which may affect the efficiency of translation of TFR2 mRNA. Transferrin and TFR2 are important in iron homeostasis. The A allele of the -576A>G polymorphism was significantly associated with the increased risk of AMD in tobacco smokers, whereas the 1892C>T polymorphism did not influence the risk of AMD related to smoking. Moreover, each polymorphism does not influence the risk of AMD associated with age, sex or the family history of the disease. In conclusion, the A allele of the -576A>G polymorphism of the transferrin gene may increase the risk of AMD in smokers.

PMID: 21422745 [PubMed - in process]

J Lipid Res. 2011 Mar 22. [Epub ahead of print]

Epidermal expression of an Elovl4 transgene rescues neonatal lethality of homozygous Stargardt disease-3 mice.

McMahon A, Butovich IA, Kedzierski W.

UT Southwestern Medical Center, United States.

Abstract

Elongase of very long chain fatty acids-4 (ELOVL4) is the only mammalianenzyme known to synthesize C28-C36 fatty acids. In humans, ELOVL4 mutations causeStargardt disease-3 (STGD3), a juvenile dominant macular degeneration. Heterozygous Stgd3mice which carry a pathogenic mutation in the mouse Elovl4 gene demonstrate reduced levels ofretinal C28-C36 acyl phosphatidylcholines and epidermal C28-C36 acylceramides. HomozygousStgd3 mice die shortly after birth with signs of disrupted skin barrier function. In this study, wereport generation of transgenic (Tg) mice with targeted Elovl4 expression driven by anepidermal-specific involucrin promoter. In homozygous Stgd3 mice, this transgene reinstates bothepidermal Elovl4 expression and synthesis of two missing epidermal lipid groups: C28-C36acylceramides and (O-linoleoyl)-omega-hydroxy C28-C36 fatty acids. Transgene expression alsorestores skin barrier function and rescues the neonatal lethality of homozygous Stgd3 mice. Thesestudies establish the critical requirement for epidermal C28-C36 fatty acid synthesis for animalviability. In addition to the skin, Elovl4 is also expressed in other tissues, including the retina,brain and testes. Thus, these mice will facilitate future studies to define the roles of C28-C36 fattyacids in the Elovl4 expressing tissues.

PMID: 21429867 [PubMed - as supplied by publisher]

Br J Ophthalmol. 2011 Mar 24. [Epub ahead of print]

Incorrect study design and analysis: the effect of genetic variants in SERPING1 on the risk of neovascular age-related macular degeneration.

McGwin G Jr.

University of Alabama at Birmingham, Birmingham, Alabama, USA.

PMID: 21436175 [PubMed - as supplied by publisher]

Pre-clinical

Yan Ke Xue Bao. 2011 Mar;26(1):23-9.

Effect of Quercetin on Formation of Choroidal Neovascularization (CNV) in Age-related Macular Degeneration(AMD).

Zhuang P, Shen Y, Lin BQ, Zhang WY, Chiou GC.

Institute of Ocular Pharmacology,College of Medicine,Texas A&M Health Science Center,College Station,TX 77843 USA.

PURPOSE: Age-related macular degeneration (AMD) as a disease entity is "dry" at early stage and made up of two main components at late stage: atrophic AMD and exudative AMD. Quercetin acts as an anti-oxidant to protect retinal pigment epithelial cells (RPE) from damaged by oxidative stress, but its effect on formation of choroidal neovascularization (CNV) in AMD is unclear. The aim of this study is to investigate the effect of quercetin on the formation of CNV in AMD.

METHODS: The development of CNV induced by laser was detected by fluorescein angiography (FA). Colored microsphere technique was used to determine the choroidal blood flow in ocular hypertensive rabbit eyes. In in vitro studies, HUVECs were treated with NaIO3, H2O2 and NaN3 to induce oxidative cell damages. The effect of quercetin on various oxidations-induced injuries in HUVECs was measured by MTT assay. HUVECs migration was assessed using a wound healing assay.

RESULTS: Quercetin significantly inhibited the formation of laser-induced CNV. The choroidal blood flow in rabbit eyes was significantly increased after quercetin instillation. In vitro results showed quercetin enhanced various oxidations-induced injuries in HUVECs and inhibited migration of HUVECs during wound healing.

CONCLUSION: Quercetin inhibited the formation of CNV both in vivo and in vitro and increased choroidal blood flow. It could become a promising candidate for the treatment of AMD.

PMID: 21425492 [PubMed - in process]

Diet

Am J Epidemiol. 2011 Mar 21. [Epub ahead of print]

Abdominal Obesity and Age-related Macular Degeneration.

Adams MK, Simpson JA, Aung KZ, Makeyeva GA, Giles GG, English DR, Hopper J, Guymer RH, Baird PN, Robman LD.

Abstract

Evidence for an association between age-related macular degeneration (AMD) and obesity is inconsistent. The authors examined associations between adiposity and AMD prevalence using 21,287 participants from the Melbourne Collaborative Cohort Study aged 40-69 years at baseline (1990-1994). For men, each increase of 0.1 in waist/hip ratio (~1 standard deviation) was associated with a 13% increase in the odds of early AMD (odds ratio = 1.13, 95% confidence interval: 1.01, 1.26; P = 0.03) and a 75% increase in the odds of late AMD (odds ratio = 1.75, 95% confidence interval: 1.11, 2.76; P = 0.02). No other adiposity measure was associated with early AMD for men. Smoking status modified the relation between waist/hip ratio and early AMD (P = 0.05), with no association for former smokers. For women, there were inverse associations with early AMD for all adiposity measures (odds ratios = 0.89-0.93; P = 0.002-0.02), but no associations were observed for late AMD. This study confirms abdominal obesity as an AMD risk factor for men despite a survivorship effect from competing risks in morbidity and mortality. The inverse associations for women may reflect weaker true positive associations with AMD that are insufficient to overcome the survivorship effect. New data are provided on complex interactions between environmental exposures and AMD risk.

PMID: 21422060 [PubMed - as supplied by publisher]