FORM 8-K

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EYETECH PHARMACEUTICALS INC

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UNITED STATES
SECURITIES AND EXCHANGE COMMISSION

WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
PURSUANT TO SECTION 13 OR 15(d) OF THE
SECURITIES EXCHANGE ACT OF 1934

Date of Report (Date of earliest event reported): May 2, 2005

Eyetech Pharmaceuticals, Inc.

(Exact Name of Registrant as Specified in Charter)

Delaware	000-50516	13-4104684
(State or Other Jurisdiction of Incorporation)	(Commission File Numbers)	(I.R.S. Employer Identification No.)

3 Times Square, 12th Floor New York, NY	10036
(Address of Principal Executive Offices)	(Zip Code)

Registrant' s telephone number, including area code: (212) 824-3100

(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

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EX-99.1: Poster
EX-99.2: Poster
EX-99.3: Press Release
EX-99.4: Press Release

Item 7.01. Regulation FD Disclosure

On May 2, 2005, the following information related to Macugen® (pegaptanib sodium injection), the first and only FDA-approved treatment for all types of neovascular (wet) age-related macular degeneration, which is being commercialized and further developed by Eyetech Pharmaceuticals, Inc. and Pfizer Inc., was presented at the 2005 Association for Research in Vision and Ophthalmology (ARVO) annual meeting in Fort Lauderdale, Fla.:

> Safety Evaluation of Second Year Treatment of Age-Related Macular Degeneration with Pegaptanib Sodium (Macugen®): VEGF Inhibition Study in Ocular Neovascularization (VISION) presented at a poster session on Monday, May 02, 2005 at 11:15 AM − 1:00 PM, by William Mieler, M.D., Department of Ophthalmology and Visual Science, University of Chicago, Chicago, IL.
>
> $VEGF_{164}$ Governs Leukocyte Dynamics and Pathological Neovascularization presented at a poster session on Monday, May 02, 2005, 11:15 AM − 1:00 PM, by K. Nishijima, Eyetech Research Center, Lexington, MA.

The full posters are furnished as Exhibits 99.1 and 99.2 to this Current Report on Form 8-K and incorporated by reference herein.

On May 2, 2005, Eyetech Pharmaceuticals, Inc. issued the following:

> a press release announcing that the safety profile of Macugen® (pegaptanib sodium injection) in the treatment of neovascular age-related macular degeneration was maintained for two years and
>
> a press release announcing that preclinical studies demonstrate the significance of $VEGF_{164}$ in pathological neovascularization.

The full press releases are furnished as Exhibits 99.3 and 99.4 to this Current Report on Form 8-K and incorporated by reference herein.

The furnishing of the attached posters and press releases is not an admission as to the materiality of any of the information set forth therein or herein.

The information in this Item 7.01 of Form 8-K (including exhibits) shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933 or the Exchange Act, except as expressly set forth by specific reference in such a filing.

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SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: May 2, 2005

EYETECH PHARMACEUTICALS, INC.

By: Glenn P. Sblendorio
Name: Glenn P. Sblendorio
Title: Senior Vice President, Finance and Chief Financial Officer

EXHIBIT INDEX

Exhibit No.	Description
99.1	Poster of Safety Evaluation of Second Year Treatment of Age-Related Macular Degeneration with Pegaptanib Sodium (Macugen®): VEGF Inhibition Study in Ocular Neovascularization (VISION) presented at the 2005 Association for Research in Vision and Ophthalmology annual meeting on May 2, 2005
99.2	Poster of VEGF164 Governs Leukocyte Dynamics and Pathological Neovascularization presented at the 2005 Association for Research in Vision and Ophthalmology annual meeting on May 2, 2005
99.3	Press Release dated May 2, 2005 of Eyetech Pharmaceuticals, Inc. announcing that the safety profile of Macugen® (pegaptanib sodium injection) in the treatment of neovascular age-related macular degeneration was maintained for two years
99.4	Press Release dated May 2, 2005 of Eyetech Pharmaceuticals, Inc. announcing that preclinical studies demonstrate the significance of VEGF 164 in pathological neovascularization

Safety Evaluation of Second-year Treatment of Age-related Macular Degeneration With gaptanib Sodium (Macugen®): VEGF Inhibition Study in Ocular Neovascularization (VISION)

William Mieler, MD[1], for the VEGF Inhibition Study in Ocular Neovascularization (VISION) Clinical Trial Group

[1]Department of Ophthalmology and Visual Science, University of Chicago, Chicago, IL

ODUCTION

y in Ocular Neovascularization
oegaptanib sodium injection
rmaceuticals), a selective vascular
(VEGF) antagonist, in the treatment
d macular degeneration (AMD). All
3 mg, 1 mg, and 3 mg) employed
iles during the initial 54 weeks of
nts were transient, mild to moderate
l to the injection procedure itself
drug. At week 54, the per injection
retinal detachment, and traumatic
.07% and 0.08%, respectively; the
and retinal detachment were lower
ose identified in a comprehensive
00 intravitreous (IVT) injections.[2]

pre-specified time point for analysis
dpoint, the study was designed to
Safety endpoints were measured
viding an opportunity to evaluate the
ptanib therapy in patients with AMD.

RPOSE

afety of pegaptanib sodium injection in
AMD.

ETHODS

ic subtypes of AMD were enrolled in two
r, randomized, double-masked, controlled
or vision and lesion size were established.

udy, IVT pegaptanib (0.3 mg, 1 mg, or 3 mg)
nistered every 6 weeks for 54 weeks

receiving pegaptanib were rerandomized
ntinue treatment for 48 additional weeks
reatment. Those rerandomized to continue
atients who received pegaptanib for year 2.
were rerandomized (1:1:1:1:1) at week 54
n group, receive one of the three pegaptanib
ent entirely.
, masked investigators were permitted to
erapy with verteporfin at their discretion
se (i.e., for predominantly classic lesions only).
a second year of pegaptanib or 2 years of
alysis.

2004;251:2605-16. 2. Jager RD, et al. *Retina* [serial]. Genentech, Inc; South San Francisco, CA; 2004. py Study Group. *Am J Ophthalmol* 2001;131:541-60.

RESULTS

- In all, 374 patients received 2663 IVT injections during the second year (0.3 mg, N=128; 1 mg, N=126; 3 mg, N=120) and 51 patients received 388 sham injections. The mean number of treatments for all patients rerandomized to continue therapy was 7 out of a possible 8 treatments.
- An overall summary of adverse events is provided in Table 1.

Table 1. Adverse Event Summary,* N (%)

	Pegaptanib Sodium 0.3 mg (N=128)	Pegaptanib Sodium 1 mg (N=126)	Pegaptanib Sodium 3 mg (N=120)	Sham (N=51)
Patients with an adverse event	122 (95)	118 (94)	109 (91)	46 (90)
Patients with an ocular adverse event				
Study eye	92 (72)	98 (78)	92 (77)	39 (76)
Fellow eye	45 (35)	44 (35)	45 (38)	23 (45)
Patients with a serious adverse event	22 (17)	23 (18)	18 (15)	14 (27)

*Data for second year of treatment.

- Pegaptanib sodium was well tolerated at all three doses (Table 2).

Table 2. Ocular Adverse Events in ≥10% of Patients,* N (%)

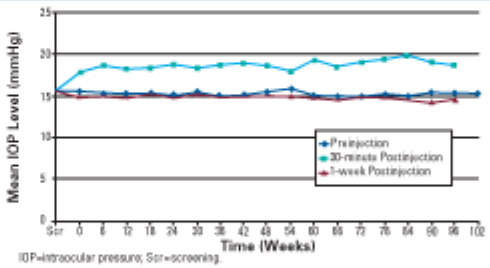
	Pegaptanib Sodium 0.3 mg (N=128)	Pegaptanib Sodium 1 mg (N=126)	Pegaptanib Sodium 3 mg (N=120)	Sham (N=51)
Eye pain				
Study eye	27 (21)	35 (28)	31 (26)	9 (18)
Fellow eye	0	1 (1)	5 (4)	0
Vitreous floaters				
Study eye	28 (22)	24 (19)	31 (26)	2 (4)
Fellow eye	3 (2)	2 (2)	2 (2)	3 (6)
Punctate keratitis				
Study eye	31 (24)	26 (21)	29 (24)	14 (27)
Fellow eye	2 (2)	1 (1)	0	1 (2)
IOP increased				
Study eye	26 (20)	22 (17)	42 (35)	4 (8)
Fellow eye	2 (2)	2 (2)	1 (1)	0
Vitreous opacities				
Study eye	13 (10)	12 (10)	21 (18)	6 (12)
Fellow eye	3 (2)	2 (2)	0	1 (2)
Corneal edema				
Study eye	12 (9)	10 (8)	13 (11)	4 (8)
Fellow eye	0	0	0	0
Lacrimation increased				
Study eye	6 (5)	15 (12)	10 (8)	6 (12)
Fellow eye	1 (1)	3 (2)	0	2 (4)
Number of phakic study eyes	78	79	87	34
Cataract in study eye, N (%)	14 (18)	18 (23)	15 (17)	8 (24)

*Data for second year of treatment. Adverse events reported in ≥10% of patients receiving pegaptanib sodium.
IOP = intraocular pressure.

Results – Continued

- Ocular adverse events occurred at a frequency similar to that observed during the first year of the study:
  - The majority of adverse events reported in study eyes were transient, mild to moderate in severity, and attributed by investigators to the injection procedure itself rather than the study drug.
  - The incidence of these ocular events was higher in the sham arm than in the fellow eye of any pegaptanib treatment arm, suggesting that the treatment preparation (use of an eyelid speculum, anesthetic drops, mydriatic drops, antibiotic drops, povidone-iodine drops or flush, and subconjunctival injection of anesthetic) rather than the IVT injection itself may be involved in causing these events.
  - In the 374 patients receiving a second year of pegaptanib sodium, anterior chamber inflammation was reported in 21 (6%) patients; the event was mild in all but 1 patient (0.3%) who had moderate inflammation, and no patient had severe anterior chamber inflammation.
- As in year 1, patients experienced an increase in intraocular pressure (IOP) at the 30-minute postinjection assessment compared with preinjection at each visit. IOP returned to preinjection levels at the 1-week postinjection visit (Figure).

Figure. Mean IOP Levels, 0.3 mg Pegaptanib Treatment Group



IOP=intraocular pressure; Scr=screening.

- Pegaptanib sodium continued to have a favorable injection safety profile during the second year (Table 3). There were no reports of endophthalmitis or traumatic cataract occurring within the second year of treatment with pegaptanib sodium. Four cases of rhegmatogenous retinal detachment were reported (0.15% per injection).

Table 3. Injection-related Serious Adverse Events in Patients Receiving Pegaptanib Sodium*

Condition	Rate: Patients	Rate: Percent per Injection	Severe Vision Loss†: Patients	Severe Vision Loss†: Percent per Injection
Endophthalmitis	0	0	-	-
Traumatic cataract	0	0	-	-
Rhegmatogenous retinal detachment	4	0.15	1‡	0.04

*Data for second year of treatment. Pegaptanib patients rerandomized to pegaptanib, N=374; total injections, N=2663. †Severe vision loss is defined as a loss of ≥30 letters. ‡Follow-up not available for 1 patient.

Results – Continued

- All doses of pegaptanib were well tolerated systemically (Table 4). No serious adverse events definitely attributed to the study drug were identified.

Table 4. All Causality Nonocular Adverse Events in >5% of Patients,* N (%)

Adverse Event (Preferred Term)	Pegaptanib Sodium 0.3 mg (N=128)	Pegaptanib Sodium 1 mg (N=126)	Pegaptanib Sodium 3 mg (N=120)	Sham (N=51)
Blood and lymphatic system disorders				
Anemia	5 (4)	7 (6)	4 (3)	2 (4)
Infections				
Upper respiratory tract infection	4 (3)	9 (7)	2 (2)	2 (4)
Urinary tract infection	8 (6)	2 (2)	3 (3)	3 (6)
Musculoskeletal and connective tissue disorders				
Back pain	3 (2)	4 (3)	6 (5)	3 (6)
Nervous system disorders				
Headache	4 (3)	7 (6)	8 (7)	1 (2)
Respiratory, thoracic, and mediastinal disorders				
Nasopharyngitis	12 (9)	8 (6)	10 (8)	3 (6)
Vascular disorders				
Hypertension	8 (6)	5 (4)	8 (7)	3 (6)

*Data for second year of treatment. Adverse events reported in >5% of patients receiving pegaptanib sodium.

- There was no evidence that pegaptanib sodium was associated with the potential VEGF inhibition related adverse events seen with the systemically administered nonselective VEGF inhibitor bevacizumab (Avastin®)† (Table 5).

Table 5. Potential VEGF Inhibition Related Adverse Events,* N (%)

	Pegaptanib Sodium (N=374)	Sham (N=51)
All thromboembolic adverse events	17 (5)	4 (8)
Arterial events	12 (3)	3 (6)
Venous events	5 (1)	1 (2)
All serious thromboembolic events	12 (3)	4 (8)
Serious arterial events	9 (2)	3 (6)
Serious venous events	3 (1)	1 (2)
Vascular hypertensive disorders	25 (7)	3 (6)
Heart failure	6 (2)	4 (8)
Serious hemorrhagic adverse events	2 (1)	1 (2)

*Data for second year of treatment.

- In the 2-year pegaptanib cohort, 7 of 374 subjects reported visual loss of any degree within 7 days of injection (7/374, 1.9%), with only 1 documented case of severe (≥20 letters) visual loss (1/374, 0.27%).
- No clinically meaningful pattern of change or findings to suggest a relationship to treatment was identified for any of the hematology or chemistry analytes evaluated.

CONCLUSIONS

In patients with neovascular AMD, pegaptanib sodium was well tolerated during the second year of treatment, and no new safety concerns were identified. In the 2-year report of verteporfin therapy in AMD, 10 patients (total N=225) reported severe visual acuity decrease within 7 days after treatment and were documented to have lost at least 20 letters of visual acuity compared to pretreatment acuity (10/225, 4.4%). Given the relative risk for acute visual loss pursuant to pegaptanib therapy, even given the small albeit finite risk of endophthalmitis, pegaptanib's safety profile, when compared to other available agents, seems superior.

GF164 governs leukocyte dynamics and pathological neovascularization

K. Nishijima[1], N. Jo[1], N. Dean[1], J. Bradley[1], Y.S. Ng[1], G.S. Robinson[1], P.A. D'Amore[2], D.T. Shima[1], A.P. Adamis[1]

[1]Eyetech Pharmaceuticals, Inc., Lexington, MA., [2] Schepens Eye Research Institute, Harvard Medical School, Boston, MA.

OUND & PURPOSE

cal vascular development and pathological angiogenesis in the retina have been shown to be controlled by vascular endothelial EGF). Previous studies have demonstrated that ischemia-induced retinal neovascularization is partly the result of local sponses. This study aims to characterize the roles of the different VEGF isoforms in inflammation and pathological on by examining the pro-inflammatory and angiogenic nature of the VEGF164 isoform through the use of VEGF164-deficient ce.

S

$VEGF^{120/188}$ mice:

eficient mice were generated by mating $VEGF^{+/120}$ male mice with $VEGF^{+/188}$ female mice, both of which were produced via genesis with Cre/*lox P*-mediated site-specific recombination in embryonic stem cells. Genotyping was performed by PCR.

ysiological and pathological neovascularization in retinopathy of prematurity (ROP):

quantitative assessments, 10μm serial sections were cut sagitally through the optic nerve, and sections roughly 50μm apart were CD31 staining 14 sections per eye for analysis.

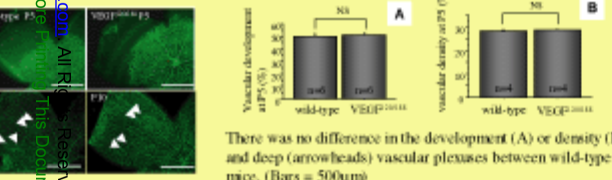
horoidal neovascularization (CNV) model:

after laser irradiation, wholemount choroids were stained with an anti-CD31 antibody and CNV areas were measured.

persensitivity (DTH) model in skin:

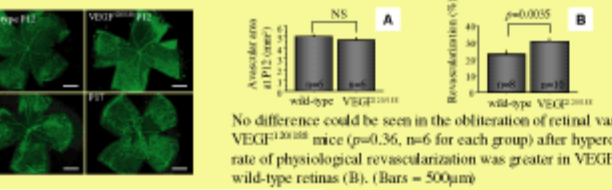
nsitized with a 50μl application of a 2% oxazolone (4-ethoxymethylene-2 phenyl-2-oxazolone-5-one) solution in acetone/olive oil applied to the shaved abdomen on day 1 and 2. On day 5, the right ears were challenged by applying 20μl of 1% oxazolone solution, left ears were treated with vehicle alone. The thickness of each ear lobe was measured at timed intervals after challenge. To estimate infiltration into the tissue, mice were sacrificed 24 hours after challenge, and 10μm serial sections started at 5mm from the apex of e collected. After fixing in PFA, immunocytochemistry was performed on every 10th slide using an anti-CD45 antibody, FITC-olectin-B4, and DAPI nuclear stain.

gical Development of Retinal Vessels



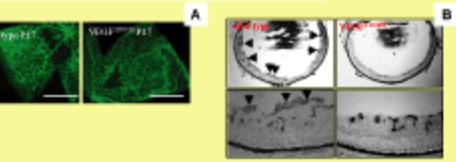
There was no difference in the development (A) or density (B) of superficial and deep (arrowheads) vascular plexuses between wild-type and $VEGF^{120/188}$ mice. (Bars = 500μm)

ation of Retinal Vessels and revascularization in ROP



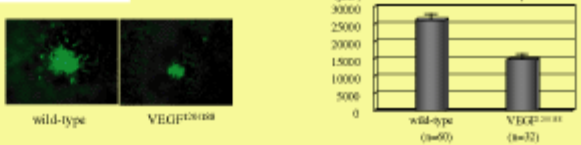
No difference could be seen in the obliteration of retinal vasculature between wild-type and $VEGF^{120/188}$ mice (p=0.36, n=6 for each group) after hyperoxia (80% O_2) (A). However, the rate of physiological revascularization was greater in $VEGF^{120/188}$ mouse retinas than in wild-type retinas (B). (Bars = 500μm)

gical Neovascularization in ROP



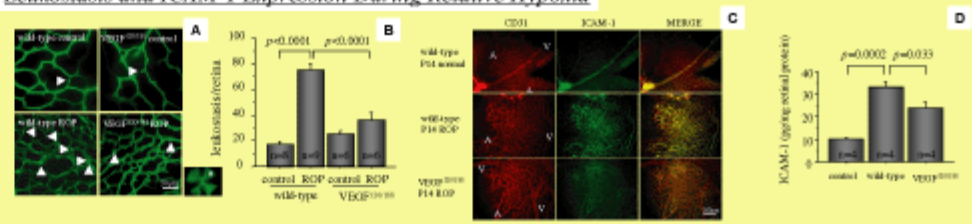
uantify neovascularization (A), we measured the area of the tufts above the inner limiting membrane Tuft formation in VEGF164-deficient mice was reduced by over 90% as compared with wild-type (C). (Bars = 500μm)

Laser-induced CNV



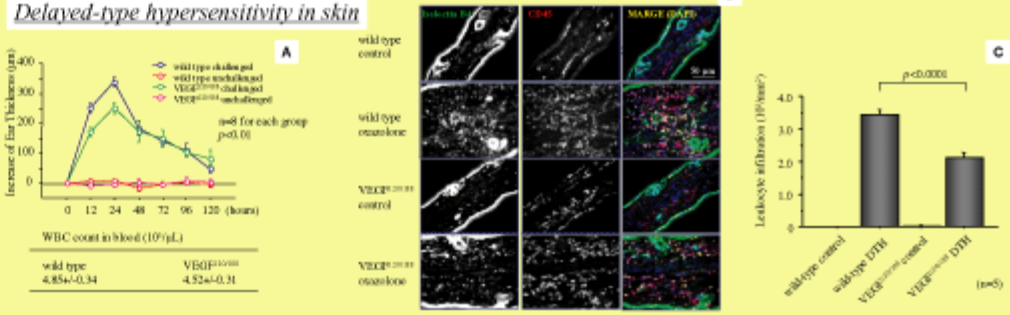
The size of CNV lesion in the VEGF164-deficient choroid was significantly smaller than that of the wild-type (p<0.001).

Leukostasis and ICAM-1 Expression During Relative Hypoxia



Quantitative analyses of lectin-perfused retinas (A) revealed a 4.3-fold increase in leukocyte adhesion in wild-type ROP mice, as compared with normal controls (B). In VEGF164-deficient ROP mice, the number of leukocytes adherent to the retinal vasculature was significantly decreased by 51.6% when compared with wild-type ROP mice. In immunocytochemistry experiments, ICAM-1 was detectable in the major veins, but not in the capillaries or arteries of non-ROP retinas at P14 (C). In wild-type ROP retinas at P14, ICAM-1 immunoreactivity was markedly up-regulated in capillaries, especially around the avascular area. In VEGF164-deficient ROP retinas, however, the expression of ICAM-1 was not significantly up-regulated in capillaries (C). ELISA results demonstrated that ICAM-1 protein levels were approximately 1.4-fold higher in wild-type ROP retinas than in VEGF164-deficient ROP retinas (D).

Delayed-type hypersensitivity in skin



Twelve hours after challenge, the thickness of the ears treated with oxazolone significantly increased in wild-type mice as compared with VEGF164-deficient mice (A). Although the ear swelling reached a peak at 24 hours post challenge in both groups of mice, this swelling was significantly less in VEGF164-deficient mice. In order to determine the degree of leukocyte infiltration, the numbers of isolectin B4+/CD45+/DAPI+ leukocytes outside the vessels were counted in a series of sections at 24 hours (B). There was a significant suppression in leukocyte infiltration into the ear tissues of VEGF164-deficient mice (C). There was no difference in the number of circulating leukocytes in the blood from challenged wild-type or $VEGF^{120/188}$ mice (A, bottom).

CONCLUSIONS

Our results demonstrate that the combination of the VEGF120 and VEGF188 isoforms is sufficient to drive normal physiological retinal vessel growth, and that VEGF164 is not required for the normal development of the retinal vasculature. Furthermore, VEGF164 is a VEGF isoform that promotes pathological neovascularization in ROP and CNV. In addition, VEGF164 shows proinflammatory characteristics in both the ROP and skin inflammation models. These findings confirm the critical role of the VEGF164 isoform in pathological angiogenesis and inflammation, and may have implications for the therapeutic roles of VEGF antagonists in the eye.

Exhibit 99.3

FOR IMMEDIATE RELEASE

Media Contact: **Chris Smith**
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Office: (212) 824-3121
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SAFETY PROFILE OF MACUGEN® (pegaptanib sodium injection) IN THE TREATMENT OF NEOVASCULAR AGE-RELATED MACULAR DEGENERATION (AMD) MAINTAINED FOR TWO YEARS

– Studies identified no new safety concerns in a continuous second year of treatment –

NEW YORK, May 2, 2005 – Eyetech Pharmaceuticals, Inc. (NASDAQ: EYET) announced today that the proven safety profile of Macugen® (pegaptanib sodium injection), the first and only FDA-approved treatment for all types of neovascular (wet) age-related macular degeneration (neovascular AMD), continues during the second year of treatment. No new safety concerns were identified in Year 2, and the safety profile was similar to that described for the first year of the study. A pharmacokinetics safety study also showed that Macugen was well-tolerated, with no evidence of systemic vascular endothelial growth factor (VEGF) inhibition or clinically significant ocular inflammation. These data were presented at the 2005 Association for Research in Vision and Ophthalmology annual meeting from May 1-5 in Fort Lauderdale, Fla. "The favorable safety profile demonstrated by Macugen in multiple clinical studies reflects the benefits that Macugen can provide to patients with all subtypes of neovascular AMD who have been waiting for a safe and effective treatment option to help preserve their vision by slowing vision loss," said William Mieler, M.D., Professor and Chairman, Department of Ophthalmology and Visual Science, University of Chicago.

“The data also underscore the tremendous advance that Macugen provides by targeting an underlying cause of the disease.”

Two-Year Safety Data

Findings from an evaluation of second-year safety data from patients who received more than one year of Macugen therapy or sham treatment in the Phase 2/3 VISION studies identified no new safety concerns with continuous Macugen treatment for more than one year. In these studies, 374 patients received 2,663 injections of Macugen during the second year (0.3 mg, N=128; 1 mg, N=126; 3 mg, N=120) and 51 patients received 388 sham injections. The most common ocular adverse events reported in the second year of the study were transient, mild-to-moderate in severity, and were attributed to the injection procedure rather than the study drug. Also in Year 2, no new systemic safety concerns or ocular safety issues emerged. Of note:

Among the 374 patients who received Macugen for more than one year, there were no cases of endophthalmitis or traumatic cataract in the second year. Four cases of retinal detachment were reported out of the 2,663 Macugen injections administered (0.15% per injection) during the second year. Two patients receiving active treatment discontinued due to this adverse event.

Among the six percent of patients who experienced anterior chamber inflammation in the second year, no severe cases of inflammation and one case (0.27%) of moderate inflammation occurred.

Ocular adverse events reported in more than 10% of study patients receiving a 0.3 mg dose of Macugen during the second year of treatment include eye pain (21%), vitreous floaters (22%), punctuate keratitis (24%), increased intraocular pressure (20%) and vitreous opacities (10%).

Similar to Year 1, patients experienced an increase in intraocular pressure during the 30-minute post-injection assessment during the second year of therapy. Levels of intraocular pressure generally returned to pre-injection levels by the post-injection visit one week later for most patients.

Overall, the mean number of treatments for all patients re-randomized to continue Macugen was seven out of eight possible treatments, indicating a high compliance rate (87.5%) throughout the second year regardless of the reported adverse events. Overall, the mean number of treatments for the two years for all patients re-randomized to continue Macugen therapy was 16 out of 17 possible treatments, indicating a compliance rate of 94 percent throughout this two-year period regardless of the reported adverse events.

Additional Safety and Pharmacokinetics Data
In a separate analysis of an open-label cohort of 37 patients treated with 3.0 mg of Macugen, results showed that Macugen was well-tolerated by all patients who received Macugen every 6 weeks for 30 weeks. Following Macugen treatment, there was no evidence of:

accumulation of Macugen

development of anti-Macugen antibodies

systemic VEGF inhibition (i.e., no systemic hypertension or proteinuria)

clinically significant ocular inflammation

"We are pleased that these data further support our vision to make Macugen a significant new medicine that will change the treatment paradigm for neovascular AMD by providing all neovascular AMD patients with a safe and effective treatment option," said David R. Guyer, M.D., Chief Executive Officer of Eyetech.

Additional Macugen Data to be Presented at ARVO
In addition to the proven safety with two years of Macugen therapy, second-year efficacy data also confirm sustained efficacy through two years, in that patients who discontinued treatment were more likely to experience vision loss. Additional data presented at ARVO also support Macugen' s broad indication, showing it may have the ability to help preserve more vision if patients are treated early in the course of neovascular AMD.

Other presentations at ARVO will highlight key data from ongoing research on Macugen/VEGF for retinal diseases. Presentations include:

$VEGF_{164}$ Governs Leukocyte Dynamics and Pathological Neovascularization to be presented at a poster session on Monday, May 02, 2005, 11:15 AM - 1:00 PM, by K. Nishijima, Eyetech Research Center, Lexington, MA.

VEGF Inhibition Study in Ocular Neovascularization (VISION): Second Year Efficacy Data to be presented during an oral presentation onTuesday, May 03, 2005 at 8:45 AM − 9:00 AM, by Donald D' Amico M.D., Harvard Medical School, Massachusetts Eye and Ear Infirmary, Boston, MA.

Intravitreous Pegaptanib Sodium (Macugen®) in Patients With Age-Related Macular Degeneration (AMD): Safety and Pharmacokinetics to be presented during an oral presentation on Tuesday, May 03, 2005 at 11:15 AM − 11:30 AM, by Antonio Capone, M.D., Associated Retinal Consultants, Royal Oak, MI.

Eyetech will file the above posters and presentations with the U.S. Securities and Exchange Commission under Form 8-K.

About Macugen
Macugen is indicated in the United States for the treatment of neovascular age-related macular degeneration and is administered in a 0.3 mg dose once every six weeks by intravitreal injection. Macugen is a pegylated anti-VEGF aptamer, which binds to vascular endothelial growth factor (VEGF). VEGF is a protein that plays a critical role in angiogenesis (the formation of new blood vessels) and increased permeability (leakage from blood vessels), two of the pathological processes that contribute to the vision loss associated with neovascular AMD.

For full prescribing information about Macugen, please visit www.macugen.com.

Important Safety Information
Macugen is contraindicated in patients with ocular or periocular infections.

Intravitreal injections including those with Macugen have been associated with endophthalmitis. Proper aseptic injection technique – which includes use of sterile gloves, a sterile drape, and a sterile eyelid speculum (or equivalent) – should always be utilized when administering Macugen. In addition, patients should be monitored during the week following the injection to permit early treatment, should an infection occur.

Increases in intraocular pressure (IOP) have been seen within 30 minutes of injection with Macugen. Therefore, IOP as well as the perfusion of the optic nerve head should be monitored and managed appropriately.

Serious adverse events related to the injection procedure occurring in <1% of intravitreal injections included endophthalmitis, retinal detachment, and iatrogenic traumatic cataract.

Most frequently reported adverse events in patients treated for up to 2 years were anterior chamber inflammation, blurred vision, cataract, conjunctival hemorrhage, corneal edema, eye discharge, eye irritation, eye pain, hypertension, increased IOP, ocular discomfort, punctate keratitis, reduced visual acuity, visual disturbance, vitreous floaters, and vitreous opacities. These events occurred in approximately 10% to 40% of patients.

About Eyetech Pharmaceuticals, Inc.
Eyetech Pharmaceuticals, Inc. is a biopharmaceutical company that specializes in the development and commercialization of novel therapeutics to treat diseases of the eye. Eyetech' s initial focus is on diseases affecting the back of the eye. Eyetech is commercializing and further developing Macugen® (pegaptanib sodium injection) with Pfizer Inc for the treatment of neovascular AMD. Macugen is also being studied for other indications including diabetic macular edema and retinal vein occlusion.

Eyetech Safe Harbor Statement
This press release contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, included in this press release regarding our safety profile for Macugen, strategy, future operations, future clinical trials, prospects, plans and objectives of management are forward-looking statements. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make. Various important factors could cause actual results or events to differ materially from the forward-looking statements that we make, including risks related to new safety and other information arising out of clinical trial results or use by patients; continued acceptance of Macugen by the medical community, by patients receiving therapy and by third party payors; supplying sufficient quantities of Macugen to meet anticipated market demand; our dependence on third parties to manufacture Macugen; the impact of competitive products and potentially competitive product candidates; our dependence on our strategic collaboration with Pfizer; obtaining, maintaining and protecting the intellectual property incorporated into our product candidates; new information arising out of clinical trial results; successful recruitment of patients for the clinical development of Macugen in other indications; successful outcomes in the further clinical development of Macugen; regulatory approval of Macugen for other indications; and the success of Macugen' s recent launch generally. These and other risks are described in greater detail in the "Risk Factors" section of our most recent annual report on Form 10-K filed with the United States Securities and Exchange Commission. Our forward-looking statements do not reflect the potential impact of any future acquisitions, mergers, dispositions, joint ventures or investments we may make. We do not assume any obligation to update any forward-looking statements.

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Exhibit 99.4

FOR IMMEDIATE RELEASE

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PRECLINICAL STUDY DEMONSTRATES SIGNIFICANCE OF VEGF$_{164}$ IN PATHOLOGICAL NEOVASCULARIZATION

– Provides further evidence of the therapeutic benefits of a VEGF inhibitor that specifically targets an underlying cause of eye diseases –

NEW YORK, May 2, 2005 – Eyetech Pharmaceuticals, Inc. (NASDAQ: EYET) announced today that research in vascular endothelial growth factor (VEGF) suggest that specific isoforms may play important roles in neovascular disease in the human eye. Results from a study in mice provide evidence that the presence of a specific isoform called VEGF$_{164}$ is not required to drive normal vascular development in the retina. However, by specifically targeting this isoform, there is a reduction in abnormal blood vessel growth (pathological angiogenesis) which results in diseases of the retina. These data were presented today at the 2005 Association for Research in Vision and Ophthalmology annual meeting in Fort Lauderdale, Fla.

VEGF and its role in the cause and progression of certain eye diseases such as neovascular age-related macular degeneration (neovascular AMD) have become increasingly important. VEGF is a protein that is responsible for stimulating abnormal blood vessel growth and blood vessel leakage in diseases such as neovascular AMD, diabetic retinopathy and retinal vein occlusion.

"This study provides further evidence that by specifically targeting the pathologic or 'bad' isoform, we can block undesirable blood vessel growth while still permitting the 'good' blood vessel growth and maintenance

required for healthy and normal function of the eye," said Anthony P. Adamis, M.D., Chief Scientific Officer of Eyetech. "Therefore, selective inhibition of pathological forms of VEGF may provide a more optimal balance of safety and efficacy in treating an underlying cause of neovascular AMD."

The study, a collaboration among researchers at Eyetech Pharmaceuticals and Schepens Eye Institute at Harvard Medical School, was designed to characterize the role of different VEGF isoforms in inflammation and pathological neovascularization. The study utilized three models – retinopathy of prematurity (ROP), laser-induced choroidal neovascularization (CNV) and delayed-type hypersensitivity (DTH) – to examine the pro-inflammatory and angiogenic outcomes in a population of study mice who were $VEGF_{164}$-deficient, but still harbored two other VEGF isoforms $VEGF_{120}$ and $VEGF_{188}$. $VEGF_{164}$ is the mouse equivalent of isoform $VEGF_{165}$ in humans. The data presented are based on observations reported from animal studies. The clinical significance in humans is unknown.

Results of the ROP model, which was used to quantify the physiological and pathological neovascularization in the eye, suggest that there were no developmental differences between wild-type mice and those deficient in the $VEGF_{164}$ isoform. However, the ROP model showed that pathological neovascularization in the study mice was reduced by 90 percent compared to wild-type mice, an outcome which the authors attribute to the significant decrease in retinal inflammatory cells, which are known to drive the abnormal angiogenesis in ROP. Results in the CNV model showed that laser-induced CNV lesions in the eyes of the study mice were 44 percent smaller than in the wild-type mice. Data from these two models indicate that $VEGF_{164}$ is not required for normal development of the retina, but instead specifically induces inflammation and pathological neovascularization, and is more potent in doing so than the other VEGF isoforms. Data from the DTH model, which was used to measure inflammation and characterize the role of $VEGF_{164}$ outside of the eye, confirmed that the absence of $VEGF_{164}$ resulted in a significant decrease in inflammatory response, characterized by leukocyte accumulation and tissue edema in the ear skin of mice. This response showed that the pro-inflammatory nature of $VEGF_{164}$ is not isolated to ocular tissues.

Anti-VEGF therapy appears to target an underlying cause of all neovascular AMD and may have potential benefits in a wide range of therapeutic areas. Macugen® (pegaptanib sodium injection) is a pegylated anti-VEGF aptamer, which binds to the specific isoform $VEGF_{165}$, which is believed to be an underlying cause for the pathological blood vessel growth and leakage in ocular neovascularization in humans. Macugen interrupts the VEGF cascade and the associated neovascularization by selectively blocking VEGF and the

science strongly suggests that this selectivity is the reason why no physical destruction of vasculature has been observed in connection with use of Macugen.

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